**A Practical Approach to Managing Osteoarthritis**

Denis Marcellin-Little, DEDV, DACVS, DACVSMR

University of California, Davis

Osteoarthritis (OA) affects many dogs, large and small. Most often, OA is the consequence of a developmental orthopedic disease that often affects a single joint or a pair of joints, and, less often, affects multiple joints. Many developmental orthopedic diseases affect left and right joints. For example, hip dysplasia is reportedly bilateral in >80% of affected dogs,1 and elbow dysplasia is bilateral in approximately 50% of affected dogs.2 All joints can be affected: the hip (most common causes of OA in the hip: hip dysplasia, Perthes disease), stifle (patellar luxation, cranial cruciate ligament disease, osteochondritis dissecans [OCD]), elbow (elbow dysplasia, elbow OCD, fragmentation of the medial coronoid process, incomplete ossification of the humeral condyle), shoulder (shoulder OCD, developmental shoulder subluxation), tarsus (OCD of the talus), and carpus (carpal laxity, carpal subluxation secondary to chondrodystrophy). Initially, OA causes acute pain. Pain is the primary consequence of early-stage OA. Affected dogs retain a relatively normal musculoskeletal system in the near term. Often, early-stage OA remains undetected because owners (and potentially caregivers) may consider the clinical signs of early-stage OA to be unrelated to OA. These signs may be attributed to “growing pains” or to the dog’s personality rather than to early-stage OA.

Over time, OA causes chronic pain. Late-stage OA negatively impacts the musculoskeletal system: Joints become thicker and less flexible, and muscle mass is lost. Dogs with chronic pain become less fit, less playful, and less mobile as the disease progresses. Seemingly, the progressions of OA over a lifetime in large and small dogs are similar, but the consequences of OA are particularly easy to notice in large-breed dogs because their loss of mobility profoundly impacts the dogs and their owners. Small dogs, on the other hand, are seemingly easier to handle when their mobility is compromised. There is no cure for OA and, clearly, there is room for improvement in how our veterinary profession deals with the disease. Many modalities or supplements used to manage it have no proven benefits or very scant evidence of efficacy. At best, they offer minor benefits to patients; at worst, they offer no benefits at all. There are, however, several key measures to implement along the way that will lessen the impact of OA on patients.

The purpose of this presentation is to discuss these key management steps and their implementation at various stages of OA. We will concentrate on several aspects of OA management that have the clearest benefits: early identification of OA, weight optimization, regular exercise, and nonsteroidal anti-inflammatory drug (NSAID) therapy.

**WHAT IS THE IMPACT OF OA IN DOGS? HOW DOES IT CHANGE OVER TIME?**

We tend to think of OA as a disease with a uniform impact, regardless of its stage, patient age, and the joint or joints affected. As a matter of fact, the impact of OA on dogs changes dramatically over time. Its impact and consequences evolve from brief episodes of acute pain in young dogs, to loss of willingness to play in middle-aged dogs, to a loss of ability to walk in older dogs (Figure 1).



Figure 1. The range of signs of canine osteoarthritis in dogs vary greatly over a lifetime.

STAGE 1. GROWING DOGS OR YOUNG ADULTS SHOW INTERMITTENT SIGNS LASTING A FEW SECONDS OR A FEW MINUTES

Growing dogs with developmental orthopedic diseases may have hind limb lamenesses (with hip laxity, patellar luxation) or forelimb lamenesses (with elbow subluxation, elbow dysplasia, OCD of the shoulder joint). The lameness is often mild. It may be intermittent or constant. The clinical signs of early joint disease, however, are often ill defined: being generally slower, clumsier, less playful, and having a “funny” gait. Unfortunately, joint disease is often overlooked or missed at that stage in life. It is missed because owners and clinicians often think of growing dogs as being very healthy. Also, they may falsely attribute the lameness to “growing pains” or “pulling a muscle.” As OA enters its chronic phase, the clinical signs of joint disease become more discrete and more intermittent. They may become bilateral, too, and bilateral lameness is seemingly more difficult to detect than unilateral lameness. When signs subside, owners and caregivers often forget the period of lameness and no longer worry about the potential presence of a chronic orthopedic disease.

STAGE 2. YOUNG ADULT DOGS HAVE INTERMITTENT SIGNS LASTING A FEW HOURS

Young adult dogs with joint disease often have intermittent signs that subside after a night of rest. These signs are often associated with more intense periods of activity—for example, with a sudden burst of activity (the “weekend warrior syndrome”). OA may or may not be diagnosed during this second stage. The likelihood of diagnosing OA appears to vary between specific joints. Some diseases are much easier to visualize than others. For example, it is easier to detect hip subluxation on ventro-dorsal radiographs of the pelvis than to detect humero-radial subluxation on radiographs of the elbow joint.3,4 Also, dog owners and clinicians tend to be much more aware of hip dysplasia than elbow dysplasia. Surprisingly, there is not a lot of difference between the number of dogs with hip dysplasia and elbow dysplasia. Even though global statistics on orthopedic diseases in dogs are lacking, according to statistics provided by the Orthopedic Foundation for Animals for the 50 most affected breeds, hip dysplasia has been present in 21% of the 430,000 dogs that have been tested, and elbow dysplasia has been present in 16% of the 180,000 dogs that were tested.5,6

STAGE 3. ADULT DOGS BECOME EXERCISE INTOLERANT AND SHOW DIFFICULTIES PERFORMING ACTIVITIES OF DAILY LIVING

Middle-aged dogs with OA progressively become exercise intolerant. They may sit when taking a leash walk. They hesitate or refuse to climb into a motor vehicle or to climb stairs. The changes present at that stage of OA are more profound: Cartilage damage is major, the joint capsule is thickened and may restrict the motion of the arthritic joint (i.e., hip extension is limited, elbow flexion is limited, stifle joint extension is limited), and dogs often have lost muscle mass in affected limbs. A pain response to joint motion (hyperflexion, hyperextension) or to joint loading (galloping, jumping) is much more likely to be present at that stage. OA flares are easier to trigger, are more severe, last longer, and are harder to control with therapy. OA is often diagnosed at that third stage because the clinical and radiographic signs are more obvious, severe, and persistent.

STAGE 4. OLDER DOGS LOSE THE ABILITY TO WALK

The fourth stage is the most severe OA stage. It most often involves geriatric dogs that are losing the ability to walk and perform activities of daily living. The changes present in limbs with OA are similar to changes present in the third stage: loss of articular cartilage, thickening of the joint capsule, and pain present when joints are loaded or when joint capsules are stretched. The loss of muscle mass is more severe. Dogs, particularly overweight dogs, progressively lose the ability to walk. Because most households are not prepared or able to care for a dog with limited mobility, the loss of ability to walk because of OA is one of the key causes of euthanasia in large dogs, if not the main cause of euthanasia. In a lifelong study of seven litters of Labrador retrievers, the loss of ability to walk because of OA was the leading cause of end of life.7 For some dogs, the loss of ability to walk occurs much earlier in life.

**KEY GOALS TO EFFECTIVELY MANAGE OA**

Several key steps can have a profound positive impact on dogs with OA. They include identifying OA early, educating the dog and the owner, cautiously selecting a management plan, and leveraging the power of NSAIDs at all stages of OA.

1. IDENTIFYING OA EARLY

Unfortunately, OA often remains undiagnosed until dogs reach the third stage of the disease, the stage of exercise intolerance. Early interventions, however, are far more effective (and cost-effective) than late interventions. To diagnose orthopedic diseases and OA at the first or second stage, all involved in the management of OA should act proactively. The early diagnosis of orthopedic diseases and associated OA should be a high priority for all individuals involved: Owners, clinicians, and technicians should not wait for problems to be discovered by elbow, respectively), can be detected and can be effectively treated at 4 months of age. Later in life, the treatment of these orthopedic problems becomes more complex, costly, and palliative.

2. EDUCATING DOG OWNERS ABOUT OA

It is critically important to educate dog owners about the features of OA. Too many owners have misconceptions about the disease: Many think that OA is a geriatric disease, that OA develops because of wear and tear in joints, that dogs are not painful as long as they don’t cry, that being an overweight dog has no impact on OA, and that exercise is detrimental to dogs with OA. It is unrealistic to expect owners to make pragmatic management decisions regarding OA when their knowledge of the disease is so superficial and potentially flawed. Offering objective information about OA should be a key priority for clinicians so that myths and misconceptions in the dog-owning population disappear progressively. Owner education is a long-term task that should involve all members of the treatment team—particularly clinicians and veterinary technicians. Written or online material should be shared with owners so they educate themselves over time, before a crisis occurs and before big decisions such as surgical procedures or other costly treatments are warranted. Too often, OA is diagnosed in middle-aged dogs that have been suboptimally socialized, that distrust individuals they have not met, and that have minimal experience being handled with a leash or a harness. This lack of socialization and training complicates the implementation of home-based and clinic-based exercise programs. It may limit the options available to manage OA. It is therefore important to raise, socialize, and train dogs keeping in mind that close contact and repeated interactions may become necessary as part of the management of their chronic diseases, including OA. Rather than making vague recommendations about it, members of the medical team should discuss, explain, facilitate, and teach, and oversee pup socialization and leash and harness training. Owners who interact more with their dogs become better managers of OA over the long term. They learn to assess locomotion more objectively (i.e., they do not overestimate or underestimate the severity of clinical signs). They become more pragmatic and objective about flares and response to therapy.

3. MANAGING OA EFFECTIVELY: THE SCIENTIFIC EVIDENCE

Interventions should be proportionate to the severity of the disease. In other words, it seems ill advised to perform an irreversible surgery in dogs that have mild clinical signs or whose clinical signs developed a few days before their examination. For example, one should not consider doing a femoral head ostectomy in a dog with signs of hip dysplasia that are mild, recent, and easily controlled with rest and NSAIDs. The same rationale applies to the medical management of all forms of OA in dogs: Simple therapeutic measures should be implemented at first, and more complex or costly measures should be considered for the later stages of the disease. This will ensure that the therapy is adapted to the stage of the disease and that it can be sustained over a long period. Therapeutic interventions must focus on modalities and processes that have proven benefits in dogs or in humans rather than modalities and processes without proven benefits.8–10 The anticipated cost and owner involvement for long-term OA management should be discussed. Ineffective management options decrease owners’ trust in the medical team and decrease owners’ ability to pay for effective treatment strategies. Similar to OA management in humans, three management options for OA in dogs stand out because they offer clear and unequivocal benefits: NSAIDs, weight optimization, and regular exercise. The evidence for efficacy of these three forms of treatment is far greater than the potential efficacy of all other forms of treatment. These options have a solid safety record, with a low rate of side effects and complications. Their cost, compared to other forms of management, is also very reasonable and sustainable by many owners. NSAID therapy is most convenient among these three options because, unlike exercise and weight optimization, NSAID administration does not require significant lifestyle changes (e.g., walking daily with a dog or dramatically changing the dog’s food intake). NSAID therapy is described below.

Weight Optimization

Weight optimization is extremely effective when managing OA in dogs. Weight optimization includes avoiding excessively rapid growth during the first year of life because rapid growth increases the likelihood that dogs that have the genes responsible for developmental orthopedic diseases will express these genes. In other words, dogs with faulty genes responsible for a specific orthopedic disease (i.e., OCD, elbow dysplasia, hip dysplasia) are more likely to see these faulty genes expressed if they grow rapidly rather than slowly. The process of eating freely with an unlimited amount of food available is called ad libitum feeding. A limited food intake generally represents approximately 75% of the food consumed during ad libitum feeding. Dogs with such limited food intake grow much less rapidly than dogs eating ad libitum,11 but their adult size does not differ from dogs that grew more rapidly. Weight optimization is also critical for adult dogs. Many dogs are overweight or obese. OA progresses much more rapidly in overweight dogs than in dogs that are not overweight. Also, the clinical signs of OA decrease when overweight dogs lose weight.

Exercise

Exercise is also a key strategy used to manage OA. While exercise intuitively means that arthritic joints are going to have to do more work, exercise under controlled conditions is beneficial to arthritic joints. Controlled exercise maintains muscular and cardiovascular fitness over an extended period. Exercise only has limited value for weight management, and it should not be the sole strategy used to promote weight loss because exercise cannot overcome excesses in caloric intake. Weight loss should be achieved and sustained with dietary adjustments (that may be supported by the increase in basal metabolism induced by exercise). Exercise is a long-term strategy that can maintain fitness at the first and second stages of OA and regain fitness at the third and fourth stages of OA in dogs. In a study involving Labrador retrievers with hip dysplasia conducted by our research group, lameness scores were lower in dogs that were exercising more: Dogs that exercised >1 hour/day had a lameness score that was 30% lower than the dogs that exercised <15 minutes/day, showing a clear association between exercise and lameness.12

During the first and second stages of OA, dogs are naturally fit, and multiple exercise options are available. Their goals are to maintain muscular and cardiovascular fitness and joint motion (through regular exercise) and to avoid OA flares (through controlled exercise). Members of the clinical team (veterinarians and veterinary technicians) play a key role in identifying these exercises, in training the dog to perform them, and in training their owners to continue the exercise plan with their dogs. For these dogs, most of the exercises are home based. For dogs in the third stage of OA, exercise is aimed at recovering fitness and joint motion to boost exercise tolerances. Members of the clinical team should take a more active role in the selection of appropriate exercises and the implementation of the program. Similar to programs for dogs in the first and second stages of OA, the programs are initiated in the clinic and then transition toward home-based programs to control their cost and sustain the efficacy over a long period. For dogs in the fourth stage of OA, exercise programs should be more cautiously implemented and should have more modest goals. Aquatic exercises may be more effective than land-based exercises. Ambulation assistance and weight optimization may be necessary before exercises can be performed. Exercises for dogs with limited mobility are more technically challenging than exercises in dogs with less severe OA, and the transition toward home-based exercise is more progressive and challenging. Dogs with OA that are effectively managed may never reach the fourth stage of the disease.

4. OPTIMIZING NSAID USE AT ALL STAGES OF OA

NSAID administration is also a key strategy for effective OA management. NSAIDs are the most convenient of the three leading strategies: They are not as time consuming and complex as exercise programs, and they are not as emotionally challenging as weight-loss programs. This makes them the most convenient short-term and long-term OA management option. At all stages of OA, however, NSAIDs should be considered to be a means to reaching a goal rather than an end. Specifically, NSAIDs should be used to:

* Alleviate pain (at all OA stages)
* Avoid loss of joint motion (stage 2)
* Avoid loss of muscular and cardiovascular fitness (stage 2)
* Avoid the onset of demeanor changes associated with chronic pain (stage 2)
* Avoid the onset of exercise intolerance (stage 2)
* Avoid loss of mobility (stages 3 and 4)
* Recover mobility (stage 4)

NSAIDs play a critical role in slowing the progression of OA from one stage to the next so dogs never reach the third and fourth stages of the disease.

In the First Stage of OA

NSAIDs are prescribed to alleviate pain. Because the pain present at this early stage of the disease tends to be acute rather than chronic, it is logical to use NSAID therapy for a relatively brief period or on an as-needed basis. At that stage, NSAIDs most often are the only pain medication needed to successfully manage pain, in my experience. NSAID therapy is successful when the lameness subsides and when dogs remain active and happy.

In the Second Stage of OA

NSAIDs are administered to control flares. In humans with OA, flares become more common, more severe, longer in duration, and more difficult to control over time. Even though little is known about the frequency, severity, and duration of flares in canine OA, one can assume that flares are similar to the flares experienced by humans. The duration of NSAID administration should be based on how rapidly and effectively flares are controlled. At that stage, adjunctive pain management (beyond NSAIDs) is often unnecessary, in my experience, with the exception of cold-pack therapy. Cold packs are a valid consideration to assist in the control of OA flares in the elbow, carpus, stifle, or tarsal joints. Cold packs are probably ineffective for flares involving the hip or shoulder joint, because of the depths of these joints. NSAID therapy is successful when flares are controlled and do not return when NSAIDs are discontinued. One should not hesitate, however, to prescribe NSAIDs for longer periods of time when flares are harder to control. The implementation of an exercise program is another key priority at that stage so that dogs stay fit and delay the onset of exercise intolerance. Clinicians should evaluate dogs at the second stage of OA yearly; that assessment should include gait, fitness level, muscle mass, and other changes resulting from OA. Also, members of the medical team (i.e., a veterinary technician focused on OA management) should stay in contact with owners to make sure that all aspects of the long-term management program (NSAID therapy, weight management, and exercise) are successfully implemented and sustained.

In the Third Stage of OA

NSAID administration becomes more critical because dogs may be unable to exercise or function at home without sustained pain management. One should anticipate that NSAID treatment duration will be longer compared to the first and second stages of OA. Successful NSAID treatment often allows dogs to function well at home and to exercise regularly and comfortably. If clinical signs persist despite the administration of NSAIDs, adjunctive medications may be considered. In a study conducted by our research group on dogs with lamenesses that were refractory to NSAIDs, small functional benefits were observed in dogs that received amantadine in addition to their NSAID for 6 weeks, compared to the dogs that received an NSAID only.13 Weight optimization is a higher priority at that stage than at previous stages because weight loss positively impacts locomotion.14 Similarly to the previous phases of OA, clinicians should make sure that all modalities and processes are implemented and sustained because the consequences of inaction are more serious at that stage of the disease. Reevaluations should take place more regularly (e.g., every 3 to 6 months). Owners should be rewarded for successfully implementing and sustaining their management program.

In the Fourth Stage of OA

The disease becomes life-threatening. Fortunately, the onset of loss of the ability to ambulate can be delayed by many months by keeping dogs slender.15 Pain management is the most critical aspect of OA management at that stage. NSAIDs should be used (unless contraindicated due to other medical conditions) because they have an unequivocal positive impact on OA patients.16 Adjunctive pain medications and pain management strategies (e.g., cold therapy, massage) may be considered, too. Falls and situations that are likely to lead to OA flares or orthopedic injuries must be avoided. If deemed necessary, ambulation assistance should be discussed with owners. Clinicians should not hesitate to hospitalize dogs to optimize rest, to provide short-term ambulation assistance, to provide thorough and sustained pain management, and to maximize the chances of controlling the situation.

5. Adjunctive medications

6. Complementary approaches

Several complementary approaches are used to

CONCLUSION

Following the key steps described above will help dogs and their owners successfully cope with OA over the dogs’ lifetime.

REFERENCES

1. Citi S, Vignoli M, Modenato M, et al. A radiological study of the incidence of unilateral canine hip dysplasia. Schweiz Arch Tierheilkd 2005;147:173-178.

2. Haudiquet PR, Marcellin-Little DJ, Stebbins ME. Use of the distomedial-proximolateral oblique radiographic view of the elbow joint for examination of the medial coronoid process in dogs. Am J Vet Res 2002;63:1000-1005.

3. Lust G, Todhunter RJ, Erb HN, et al. Comparison of three radiographic methods for diagnosis of hip dysplasia in eight-month-old dogs. JAVMA 2001;219:1242-1246.

4. Wosar MA, Lewis DD, Neuwirth L, et al. Radiographic evaluation of elbow joints before and after surgery in dogs with possible fragmented medial coronoid process. JAVMA 1999;214:52-58.

5. Hip dysplasia statistics. Breeds having at least 100 evaluations January 1974 through December 2010. offa.org/stats\_hip.html. Accessed January 3, 2014.

6. Elbow dysplasia statistics. Breeds having at least 100 evaluations January 1974 through December 2010. offa.org/stats\_ed.html. Accessed January 3, 2014.

7. Lawler DF, Evans RH, Larson BT, et al. Influence of lifetime food restriction on causes, time, and predictors of death in dogs. JAVMA 2005;226:225-231.

8. Zhang W, Moskowitz RW, Nuki G, et al. OARSI recommendations for the management of hip and knee osteoarthritis, part I: Critical appraisal of existing treatment guidelines and systematic review of current research evidence. Osteoarthritis Cartilage 2007;15:981-1000.

9. Zhang W, Moskowitz RW, Nuki G, et al. OARSI recommendations for the management of hip and knee osteoarthritis, part II: OARSI evidence-based, expert consensus guidelines. Osteoarthritis Cartilage 2008;16:137-162.

10. Marcellin-Little DJ. Medical treatment of coxofemoral joint disease. In: Bonagura JD, Twedt DC, eds. Kirk’s Current Veterinary Therapy XIV. Philadelphia, PA: Elsevier; 2008:1120-1125.

11. Dämmrich K. Relationship between nutrition and bone growth in large and giant dogs. J Nutr 1991;121:S114-S121.

12. Greene LM, Marcellin-Little DJ, Lascelles BDX. Associations among exercise duration, lameness severity, and hip joint range of motion in Labrador retrievers with hip dysplasia. JAVMA 2013;242:1528-1533.

13. Lascelles BDX, Gaynor J, Smith ES, Roe SC, Marcellin-Little DJ, et al. Amantadine in a multimodal analgesic regimen for alleviation of refractory osteoarthritis pain in dogs. J Vet Intern Med 2008;22:53-59.

14. Impellizeri JA, Tetrick MA, Muir P. Effect of weight reduction on clinical signs of lameness in dogs with hip osteoarthritis. JAVMA 2000;216:1089-1091.

15. Smith GK, Paster ER, Powers MY, et al. Lifelong diet restriction and radiographic evidence of osteoarthritis of the hip joint in dogs. JAVMA 2006; 229:690-693.

16. Aragon CL, Hofmeister EH, Budsberg SC. Systematic review of clinical trials of treatments for osteoarthritis in dogs. JAVMA 2007;230:514-521.