



surgery

internal medicine

emergency & critical care

dermatology

radiology, ultrasound & CT scan

? **INTRODUCTION:** What is osteoarthritis? Osteoarthritis (OA) is the most common form of arthritis affecting companion animals. In the United States with an approximate dog population of 44 million, published data indicate that as many as one in five, or 20%, of the adult dogs suffer from some degree of OA. Cats frequently suffer from OA as well (see box 1 for comparison to humans). Osteoarthritis is a degenerative process, sometimes referred to as “degenerative joint disease” (DJD), and is exhibited by progressive destruction or loss of the cartilage surface in the affected joint(s).

Comparison: Americans with arthritis*

BOX 1

- 2002—70 million (1 in 3 adults)
- All age groups affected including nearly 300,000 children
- Arthritis is the nation’s leading cause of disability among Americans over age 15
- Economic impact = \$86.2 billion annually
- Arthritis (in humans) refers to >100 different diseases that affect areas in or around joints.

Causes for OA in Small Animals

BOX 2

- **Developmental Disorders:** e.g., elbow dysplasia, hip dysplasia, osteochondrosis, patella luxations, other bone or joint conformation abnormalities
- **Periarticular trauma:** e.g., articular (joint) fractures or luxations
- **Acquired Orthopedic Conditions:** e.g., cruciate ligament injuries, shoulder instability, neck or back instability, chronic overuse

Osteoarthritis typically develops as a consequence of: developmental disorders, articular trauma, or acquired orthopedic conditions (see box 2). Other causes for arthritis include immune-mediated and infectious processes. Osteoarthritis develops slowly

and progressively. Once the process has been set in motion, it is difficult to arrest and impossible to reverse. As the environment of the joint changes with the development of OA, alterations occur in the basic properties of the articular cartilage. As these changes occur and progress, the cartilage is no longer able to provide pain-free movement in the affected joint(s). Patients with OA thus typically develop stiff and painful joints.

? **DIAGNOSIS:** Diagnosis of OA begins with a thorough patient history and physical exam. Owners often report a specific lameness or distinct behavioral change. Earlier symptoms may be more subtle. Physical exam findings typically include: muscle atrophy (wasting) in the area of the affected joint, joint stiffness (loss in range of motion) and tenderness with manipulation, increased joint fluid (effusion), and increased thickness or swelling (periarticular fibrosis) around the affected joint. Currently, radiographs (x-rays) constitute the primary method of definitive diagnosis. Radiographic changes, however, occur later in the disease process and do not necessarily correlate with symptoms. As we say, “Dogs do not walk on their x-rays.” This implies that patients with significant OA, as noted on x-rays, may have minimal joint pain. Whereas patients with minimal OA, as documented on x-rays, may have significant joint pain. Furthermore, x-rays may document that a patient has OA in more than one joint of a lame leg (e.g., hip and knee) again stressing the importance of a thorough exam to determine which joint(s) is/are painful and responsible for the lameness.

? **TREATMENT:** Treatment of OA depends to a large degree on what stage the disease process is in at the time of initial diagnosis. In every case however, a multi-modality and/or multi-agent therapy is advised. Ideally, medications for OA would relieve pain and inflammation, retard on-going joint degradation, and promote joint tissue healing. No single ideal medication exists for OA treatment. A typical multi-modality approach to treatment of OA involves the following: weight reduction, an exercise program, anti-inflammatory agents, chondroprotective agents (nutraceuticals and DMOADs), narcotic analgesics, ancillary modalities, and surgical intervention (if indicated).

* Arthritis Foundation (www.arthritisfoundation.org)

Osteoarthritis

? **WEIGHT REDUCTION:** Excess body weight contributes significantly to the disability of OA. Thin is good. Thin dogs live longer, and develop less arthritis, than obese dogs. A recent study evaluated leanness in relation to OA and the crippling disease of canine hip dysplasia (CHD). Keeping dogs lean does not change their genetic composition or predisposition to develop hip dysplasia; however, leanness has been shown to **delay or prevent** the expression of radiographic (x-ray) signs of OA in patients with CHD. Weight reduction is the first step in managing OA and often is more important than any medication. Furthermore, thin patients typically respond better to medications for OA than do obese patients.

? **EXERCISE:** Muscle atrophy (wasting) occurs as patients shift weight off a painful, arthritic joint, and further exacerbates OA changes by diminishing muscle support of the affected joint. Controlled exercise maintains the health of muscles, ligaments, and bone while decreasing joint inflammation. Exercise also helps to reduce the need for other medications and combat the depression that occurs in many arthritic and obese animals.

? **NSAIDS: NonSteroidal AntiInflammatory Drugs** represent the primary and initial medical treatment modality for OA. NSAIDs have antiinflammatory and analgesic properties, both at the local level of the inflamed joint, and more globally through their effects at limiting nerve stimulation in the spinal cord. The goal with NSAID treatment is to get the pain and inflammation of OA under control with daily NSAID use, then reduce the frequency of administration and allow other agents (below) to help maintain long term comfort. There are a number of NSAIDs available for animals, and as with people, one may work well for some patients and not for others.

? **NUTRACEUTICALS:** These compounds are also referred to as “chondroprotective agents” and consist of glucosamine, chondroitin sulfate, MSM (manganese ascorbate), GLM (green lipped muscle - Perna Canaliculus), and Omega 3 fatty acids. Glucosamine and chondroitin sulfate work by providing supraphysiologic levels of the precursors required to rebuild damaged cartilage. Glucosamine may also have anti-inflammatory properties separate from NSAIDs. Perna Canaliculus (GLM) is rich in glycosaminoglycans, as is chondroitin sulfate, and works in a similar manner. MSM and Omega 3 fatty acids are considered natural anti-inflammatory agents. Unfortunately, the U.S. FDA does not regulate the nutraceutical market and consequently there is no guarantee that the product actually has what it claims to have regarding quality, purity, strength, or shelf life of key ingredients.

? **DMOADS: Disease-Modifying Osteo Arthritis Drugs (DMOADs)** are also chondroprotective agents. Polysulfated glycoaminoglycan (PSGAG), know as “Adequan,” is approved by the U.S. FDA for dogs and classified as a DMOAD. Adequan is derived from bovine tracheal cartilage and is a mixture of highly sulfated glycoaminoglycans, predominately chondroitin sulfate. Adequan has both anti-inflammatory and cartilage building properties and has been proven in several scientific studies to be helpful in the treatment of OA in dogs. These products act as joint lubricants to decrease friction in the joint.

? **JOINT DIETS:** Several of the major dog food companies (Iams, Hills, Purina) have developed “joint” specific diets that are formulated with various antioxidants and specific levels of Omega 6 and 3 fatty acids designed to help control inflammation associated with OA. These diets have been scientifically proven to reduce the need for NSAIDS.

? **NARCOTICS:** Opioid agonist drugs such as morphine, or similar drugs, are very effective in treating chronic pain associated with advanced OA. These medications can be effective when used in combination with other therapies to treat severe OA pain. Narcotics provide central pain relief (analgesia), they do not have anti-inflammatory properties.

? **SURGERY:** Surgical intervention in the treatment of OA is indicated: to stabilize an unstable joint thus helping to limit progressive OA development; to “clean out” and/or “wash out” an arthritic joint thus removing many of the mediators of pain and inflammation; to totally replace, or remove, a severely arthritic joint. Your surgeon can discuss these options with you.

? **ANCILLARY MEASURES:** Acupuncture, massage, hydrotherapy, ultrasound, cold laser, and iontophoresis are additional therapeutic modalities that have proven efficacy in improving joint function and decreasing joint pain in patients with OA. These modalities are highly specialized and should only be carried out by a trained physical therapist or other individuals trained the respective technique.

WHEAT RIDGE OFFICE

3695 Kipling St., Wheat Ridge, CO 80033
Tel 303-940-1239 • Fax 303-420-8360

WESTMINSTER OFFICE

945 W 124th Av, Westminster, CO 80234
Tel 303-350-4733 • Fax 303-350-4734

BOULDER OFFICE

1658 30th ST, Boulder, CO 80301
Tel 720-974-5802 • Fax 303-440-0649