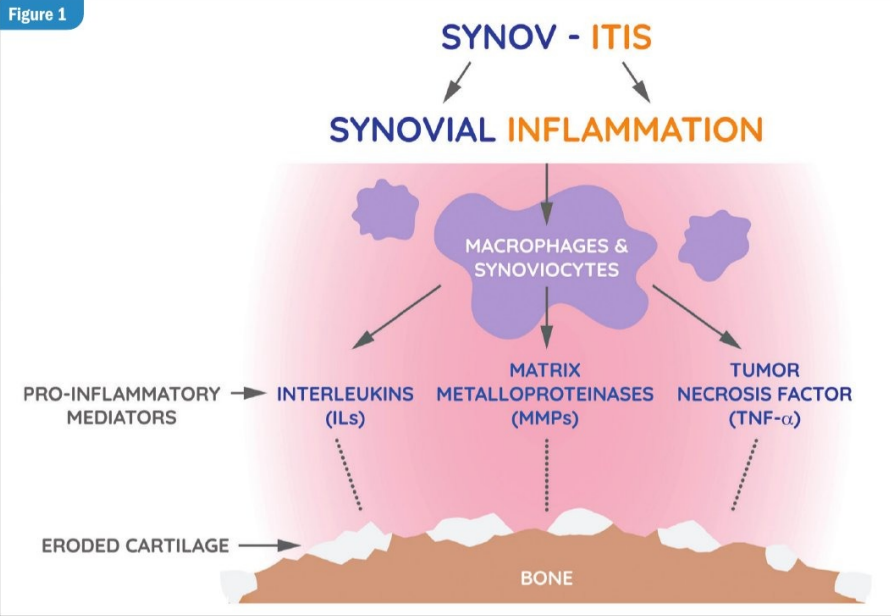
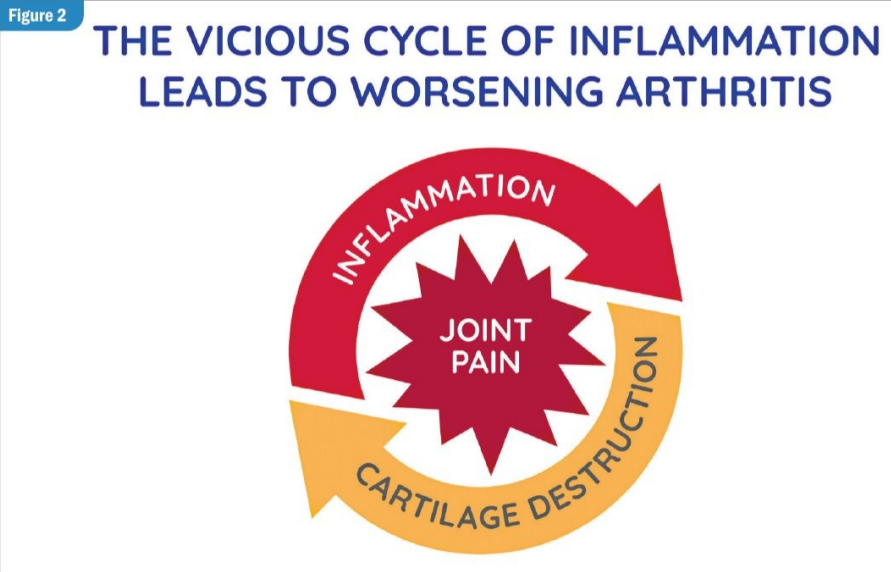


# What they don't teach you about canine osteoarthritis in veterinary school

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Osteoarthritis is driven by synovitis (inflammation of the synovium), leading to an overproduction of macrophages and synoviocytes.



The release of these pro-inflammatory mediators creates a vicious cycle of inflammation, pain, and cartilage destruction.

By Matthew W. Brunke, DVM, DACVSMR (Canine), CCAT

Veterinarians spend intense years studying anatomy, physiology, pathology, pharmacology, and surgery to learn to diagnose and treat hundreds of diseases. Yet, despite the fact canine osteoarthritis (OA) is one of the most prevalent and clinically significant diseases they will

face, many students receive only a single credit hour on OA. In fact, some veterinary schools focus significantly more hours on procedures only a small percentage will ever perform, like gastric bypass surgeries, than on studying OA, a disease seen regularly in day-to-day practice.

### OA is nothing short of an epidemic

Although OA commonly affects popular larger breeds, it is

widespread across dogs of all sizes and ages. Estimates of prevalence are truly sobering:

- At least 20 percent of dogs over one year in age develop OA during their lifetime. Some studies report radiographic evidence that shows around 40 percent of all dogs are affected in at least one joint.<sup>1</sup>
- In a general practitioner's day, three out of 10 appointments involve some sort of mobility issue.

- 61 percent of the elbow OA cases are bilateral.<sup>2</sup>

Sadly, one study researching elbow OA in dogs found this disease drove the decision for euthanasia in 41 percent of cases analyzed.<sup>2</sup> This is a serious call to action for the veterinary community to better understand the disease, diagnose it earlier, and treat it more proactively. Too often, otherwise young, healthy dogs are not diagnosed or treated until cartilage deterioration and joint damage have significantly affected their quality of life.

In another study, investigators found 39.8 percent of dogs aged eight months to four years had arthritis in one or more joints.<sup>3</sup> Clearly, we must recognize OA as a younger dog disease, as elbow and hip dysplasia are developmental orthopedic conditions caused by genes that don't allow those joints to form well as a dog goes through skeletal maturity.

### Addressing the root cause: synovial inflammation

In vet school, OA is often presented within the musculoskeletal disease section as a cartilage disease, basically a "wear and tear" problem where cartilage erodes over time, leading to pain and dysfunction.

The problem with this focus is that cartilage does not have nerve endings, which begs the question: why is OA so painful? This is because cartilage deterioration is the end result of what's happening within the synovium inside the joint.

OA is, in fact, a disease of the synovium, where nerve endings exist. In a lax joint, normal movement stretches the synovium, causing it to become inflamed. The resulting synovitis then signals macrophages and synoviocytes, which recruit other inflammatory cells. This begins a vicious cycle of inflammation that destroys cartilage and ultimately damages the joint, leading to the chronic pain and disability that are hallmarks of OA (Figures 1 and 2).

### Properly diagnosing OA

Our initial training in vet school equipped us to identify overt

signs of lameness and joint pain but has left us less prepared to recognize early or subtle signs of OA. Further, education has primarily focused on pain management rather than a more comprehensive approach tailored to the whole-joint nature of the disease.

The best practice for effectively diagnosing and treating OA is to examine every patient, even if lameness is not yet apparent.

- Make it standard to get good histories at every visit. A dog does not show reluctance to go up and down stairs because of age; it does so because of pain.
  - Watch them walk in your clinic or outside or review a video on the owner's phone of the dog standing and walking.
  - Flex and extend every patient's joints and even record goniometry (OK, that may be asking a lot).<sup>4</sup>
  - Feel the elbows, stifles, and hocks for asymmetry, both acute (swelling/effusion) and chronic (fibrosis, periarticular thickening, medial buttress).
  - Note thinner muscles, which means atrophy, when the patient is standing square.<sup>5</sup>
- Consider getting additional training like a rehab certification or taking CE courses on mobility issues.

It is essential to become proactive in diagnosis to help dogs live long, active lives. Keep in mind by the time we see osteophytes on radiographs, the patient is already in the painful, irreversible stage of OA. However, even then, all hope is not lost. We can still manage and improve the quality of life of many of these patients.<sup>6</sup>

### From pain management to disease inflammation management

First, it is vital to note having a low body mass and engaging in low-impact exercise are both essential. Studies have shown a lean, active dog lives two years longer than its fat, sedentary counterpart.<sup>7,8</sup> In fact, weight loss alone has been shown to be beneficial for managing OA in dogs.<sup>9</sup>

Beyond that, we have many tools in our toolbox. Oral NSAIDs

and amantadine remain staples in general pain management, with multiple studies supporting their use.<sup>10,11</sup> However, several oral medications (e.g., pregab, opioids, and gabapentin) do not have the evidence to support their usefulness in OA. Until we have more data, they just are not optimal options.

There are a number of other interventions we can employ, including acupuncture, photoceuticals, heat/cold therapy, extracorporeal shock wave therapy, pulsed electromagnetic field therapy, rehabilitation, and assistive devices. These various approaches can help, but results are not consistent across all cases.<sup>1</sup>

Intra-articular (IA) therapy is the area where there have been significant advances, but they have not been widely adopted into small animal medicine. In contrast, the majority of equine practitioners surveyed reported regular use of various joint injections.<sup>12</sup> We can follow suit to do better in small animals.

Here are some highlights of various IA options:

- **IA corticosteroids** (e.g. triamcinolone acetate, methylprednisolone acetate) have been widely used, but their benefits are typically short-term (weeks to months), and there is the risk of chondrotoxicity with repeated use and acceleration of cartilage degeneration if overused.<sup>13</sup>
- **Hyaluronic acid (HA)** injections aim to improve lubrication and potentially protect cartilage. HA is generally well tolerated with a low incidence of adverse effects. However, opinion remains divided on their efficacy, and considerable divergence is reported in the literature.<sup>14</sup>
- **Collagen-elastin hydrogel microparticles (CEHM)** are thought to create a joint scaffold to provide structural support for cell growth and tissue regeneration. However, a recent study comparing triamcinolone (TA + CEHM) or TA alone showed no difference between study groups, and the proportion of dogs achieving treatment success was low, ranging from 0 to 29 percent.<sup>15</sup>
- **2.5 percent intra articular polyacrylamide hydrogel (IPAAG)** is designed to provide structural support to the synovium. It is a long-acting option for treating OA in humans, horses, and dogs. An observational study evaluating owner-reported



Figure 3 A radiograph of the canine elbow showing normal joint structures.

outcomes showed it reduced canine OA signs and adjunctive therapy use.<sup>16,17</sup>

- **Platelet-rich plasma (PRP)** has been associated with significant improvements in pain reduction and increased limb function. However, the evidence supporting its efficacy is mixed and often considered low quality.<sup>18,19</sup>
- **Mesenchymal stem cells** may support tissue repair through paracrine signaling rather than direct cartilage regeneration, but clinical outcomes have been variable, and long-term efficacy is undetermined.<sup>20</sup>
- **Tin 117m (117mSn)** via radiosynoviorthesis (the same procedure that has been used in human arthritis) delivers highly targeted microparticles directly into the joint space with no reported systemic distribution or side effects. It is specifically designed to reduce synovial inflammation and pain directly at the source, along with the pro-inflammatory macrophages that drive progression.<sup>21,25</sup> Studies of tin 117m in rodents demonstrate a disease-modifying mechanism and the potential to slow progression.<sup>25</sup> Tin 117m is ideally used early to help reduce synovitis, reset the joint environment, and help mitigate significant damage to joint structures, but has shown benefits in all stages of OA.<sup>21,25</sup> Multiple peer-reviewed, published data have demonstrated up to 92 percent effectiveness and safety in delivering one year of relief with just one treatment.<sup>21, 23,25,26</sup>

I am now using tin 117m extensively in my own practice. After treating hundreds of dogs (and some cats, and a Komodo dragon), I find it to be the most powerful anti-inflammatory I can use and frequently find a single injection provides benefits for more than 12 months in many patients. Tin 117m has also been shown to not cause damage to the joint, which is key in my opinion.

OA is a chronic, whole-joint disease driven by pervasive



Figure 4 A radiograph of irreversible OA with osteophytes clearly visible.

inflammation. By reframing it first and foremost as an inflammatory disease, the profession can deliver better outcomes, more effective interventions, and more hopeful futures for animals suffering from chronic joint pain and diminished mobility. 🐾

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