

ORTHOPEDIC DISORDERS OF THE GROWING PUPPY

by

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Introduction

Sorting the multitude of bone growth diseases in puppies and young dogs is a formidable task even for a veterinarian, since one is confronted with a long list of look-alike acronyms like OCD and HOD, HD, CMO or, alternatively, long, complicated names like Osteochondrosis, Osteochondritis Dissecans, Hypertrophic Osteodystrophy, Hip Dysplasia, or Cranial Mandibular Osteoarthropathy. Not to mention Panosteitis and Avascular Necrosis of the Femoral Head (also called Legg–Perthes Disease). This article will begin to elucidate these and several other disorders of the growing dog.

First, a basic lesson in rudimentary ‘medical–ese’ will help tremendously, and you would be accurate to respond and say that it is ‘all Greek to me,’ since all these roots are Greek in origin with only a few Latin exceptions. If we examine elements in the list I’ve already presented:

Osteo = (G) bone
Chondros = (G) cartilage
–osis = (G) condition of
–itis = (G) inflammation of
Dissecans = (L) dissecting
Hyper = (G) excessive
Trophic = (G) growth as in nourishment or simple expansion
Dys = (G) abnormal
Plasia = (G) growth in the sense of molding or formation
Cranial = (G) skull
Mandible = (L) jaw
Arthro = (G) joint
Pathy = (G) pathos or suffering
Pan = (G) all
A = (G) without
Vas = (L) vessel or duct
Necrosis = (G) death

Not only are these many of the key roots for the numerous orthopedic diseases affecting our canine companions, but recognizing them will illuminate many other obscure medical terms.

Next, it is important to note that the causes of most of the diseases that will be presented are not fully understood. In fact, while hereditary factors are suspected in several disorders (OCD, CMO, Avascular Necrosis of the Femoral Head, and the various elbow dysplasias), only CMO and Hip Dysplasia are generally accepted as proven to be hereditary. Nevertheless, all these disorders, including Hip Dysplasia, are influenced by many nutritional, environmental, and even hormonal factors. All of these diseases are best considered complex syndromes rather than simply diseases.

As each syndrome is presented, we will find that certain conditions affect males or females at different rates thus suggesting a hormonal influence, others are generally reported in certain breeds over other breeds thus attesting to their inheritability, while others are clearly associated with over-nutrition and/or excessive vitamin and mineral supplementation, affecting mixed-breed dogs as well as purebreds. Some disorders will cause the dog to be systemically ill with fever and lethargy or to eventually develop persistent arthritis that may require surgery. Other conditions will be little more than a nagging or intermittent lameness that the dog eventually out-grows with minimal treatment.

Finally, many of these conditions will affect more than one joint or bone, even if lameness, pain, or discomfort seems consistently related to one anatomic site. Your veterinarian is very likely to urge you to radiograph several areas, particularly the identical joint or bone of the opposite leg. You should agree to this; not only are many of these diseases bilateral, but the anatomy of many joints, particularly of the elbow and hock, are extremely complex. These joints are further confused by the numerous growth plates of each bone. Since each growth plate closes at a different time and since there can be many breed-specific peculiarities, your dog's normal leg will provide the best radiographic atlas that your veterinarian can use as a reference. There is no text book available that presents all the intricate changes that occur normally each month in all the growing puppies of all the various pure-bred and mixed-breed dogs that come to a veterinary clinic for treatment of lameness every year.

Osteochondrosis Complex

This is a failure of articular cartilage to be sequentially replaced with bone as the dog grows. In this way, a little section of cartilage remains where bone should be, and this section of the joint is weak and easily irritated. The irritation may increase to the point of pain, excessive joint fluid production, and swelling of the joint, thus progressing to Osteochondritis. Finally, if the weakened area begins to separate (dissect) from the rest of the articular cartilage, the condition is termed Osteochondritis dissecans or OCD. While the dog may be severely lame, even to the point of muscle wasting in the involved leg, the dog is not feverish or ill, generally still eating well and interested in playing.

The articular surfaces of the joints of the large bones of young, male, rapidly-growing large and giant breed dogs are most generally affected, but multiple factors have been implicated in this process. These include hereditary, nutritional, hormonal, and traumatic

factors, and there is evidence to suggest that OCD of the different joints may each have a differing interplay of these various factors.

In osteochondrosis of the hock (ankle), we find that Rottweilers, Labrador Retrievers, and Australian Cattle Dogs are at particular risk, often showing lameness at four or five months of age. Affected dogs carry the leg particularly straight, running with a short-strided gait. In extreme cases, the dog may be three-legged lame with wasting of the muscles of the affected limb. Fluid may be felt in the hock joint, and the dog will be especially painful when the joint is forced into extreme positions of movement by bending and straightening the leg.

Osteochondrosis of the stifle (knee) is very serious and will affect male dogs 76% of the time. Moreover, of those dogs affected, nearly 70% of them will have the disease in both stifles, although few will actually show lameness in both legs. In addition to the large breeds already listed, German Shepherds and Great Danes may also be particularly prone to the condition in this joint. Generally, the onset of lameness is very gradual, but may be evident as early as three months of age.

Osteochondrosis of the shoulder rarely produces lameness before six months of age, and while the condition affects both shoulders in 43% to 65% of these dogs, only 3% to 5% of them will show lameness in both legs. Osteochondrosis of the elbow is less well studied, but lameness may be seen as early as four months of age.

Treatment will depend on the degree and persistence of lameness. Frequently, a veterinarian will simply recommend changing the puppy's diet to one of lower protein, calories, and calcium to decrease the rapid rate of growth in the pup. Aspirin and rest may also be prescribed over a two to four week period. If the lameness becomes severe or simply refuses to go away, the pup is re-examined. Your veterinarian may recommend blood tests for Lyme disease, if this is found in your area, as well as a radiograph of the abnormal joint and the corresponding joint of the opposite leg. This often requires sedation or even anesthesia to allow the proper positioning. Since the density of cartilage is the same as joint fluid, your veterinarian may see only a flattened area or nothing at all. Repeated radiographs one month later or injecting a radiographic dye into the joint (contrast arthrography) may be needed to positively diagnose osteochondrosis/osteochondritis

Once the condition is confirmed, treatment may or may not require surgery. Even in those cases that have progressed to osteochondritis dissecans where the flap of cartilage has separated from the bone, there is still a 50-50 chance that the dog will self-heal. Surgery involves entering the joint and removing the flap, yet frequently the dog will accomplish the same objective through a few months of vigorous exercise and aspirin. However, if the dog is completely lame and muscle wasting has started, surgery should be done promptly to avoid the complications related to the muscle atrophy. Most dogs do very well after surgery as it is not an especially invasive procedure. Your veterinarian is best equipped to guide you in the right decision for your dog

Hypertrophic Osteodystrophy

HOD has many other names, including metaphyseal osteopathy, Moeller-Barlow Disease, canine scurvy, osteodystrophy I, and osteodystrophy II. This no doubt reflects the degree of confusion surrounding the causes and cures of this condition. Entire litters may be affected, but

a genetic basis has not been established. Proposed causes include low blood values of Vitamin C, over-nutrition and excessive vitamin and calcium supplementation, imbalances of the body's calcium-controlling hormones like parathormone and calcitonin, and immune-mediated reactions in the bone brought about secondarily from vaccinations with modified-live vaccines.

Bones do not grow in the middle but from either end, at the site termed the metaphysis. No actual growth occurs in the middle of the bone. In HOD, any metaphysis of any long bone may become inflamed and painful, but the usual site is the lower metaphysis of the main bone of the front leg, the radius, just above the carpus (wrist) between two and four months of age. These pups are often quite sick with a fever, depression, loss of appetite, and swollen, hot metaphyses of both legs. They may refuse to stand at all. The swollen area is clearly painful when palpated, and a radiograph will demonstrate a very obvious abnormality. The metaphysis will appear to have two growth plates, instead of only one. In actuality, this abnormal area is inflammation and bleeding into that area of the bone. Advanced cases will show excess bone laid down around the entire area (hypertrophy).

These pups often require intensive treatment to relieve pain and swelling, including antibiotics, cortisones, aspirin, and vitamin C supplements. The overall diet may or may not be altered. Most pups respond well to therapy

Cranial Mandibular Osteoarthropathy

CMO, sometimes called mandibular periostitis or lion's jaw, is another condition of excessive bone deposition, but it is usually only seen in young Scottish and West Highland White Terriers, pointing to an inheritable basis. Case reports have documented the condition in the Boston Terrier, Cairn Terrier, Labrador Retriever, Great Dane, Doberman Pinscher, and Boxer, but this is generally considered a Scottie or a Westie malady. Seen most frequently between the third and the sixth month of age, this can be a very painful condition of the jaw where so much excess bone and inflammation may be laid down along the mandible and around the cheek joint that the pup cannot open its mouth to eat. In severe cases, intermittent fevers will also occur during the course of the disease. Thankfully, this condition is rare and generally self-limiting. It will resolve once most or all bone growth has stopped (9 to 18 months), and most dogs can be helped with pain killers and a liquid diet.

Panosteitis

With only rare exceptions, this is another self-limiting disease. By twenty-four months of age, when all bone growth has ended, the dog's discomfort will be over. Before that time, however, numerous bouts of lameness, frequently accompanied by fever, may have occurred. Like HOD and CMO, this is also an inflammatory disease, but, in this case, the middle areas of the long bones are generally affected. Occasionally, the condition will also involve the metaphysis, but

radiographs of this disease will show a patchy invasion of excess bone down the shaft of the long bones where normally only marrow should be present.

The causes of panosteitis are not known, but young male dogs of the large and giant breeds are most often affected. Once confirmed, an owner is generally told to stop feeding any supplements or growth formulae and to administer aspirin whenever discomfort occurs. Since the lameness can frequently shift from leg to leg, and since the dog may actually have a fever and be ill during severe bouts of panosteitis, it is necessary to rule out Lyme Disease in those areas where it occurs. Blood tests, antibiotic trials, and repeated radiographs may be necessary for those cases

Avascular Necrosis of the Femoral Head

Originally termed Legg–Calve–Perthes disease after the human physicians who described this condition in man, avascular necrosis of the femoral head is seen almost exclusively in Toy and miniature breeds. Unlike many of the other conditions described above, this disease of the hip generally affects only one joint and affects males and females equally. While a recessive mode of inheritance seems likely, it has not been proven. The condition, if hereditary, is most likely carried on a non–sex chromosome and is incompletely penetrant. In other words, like all of the conditions presented, genetic control of expression is not enough. The disease is likely to occur as a result of an interplay between genetic predisposition and environmental factors which are still unclear at this time

Essentially, the tiny blood vessels which nourish the neck of the femur at the hip joint become damaged. When the blood supply is cut off, normal closure of the femoral neck growth plate does not occur, leading, eventually, to complete or partial separation of the shaft of the femur from its head and neck located in the hip joint. This joint may collapse, leading to arthritis frequently requiring surgical correction to relieve the pain. This process begins near the time of closure of the growth plate which, in Toy breeds, may be as early as three to four months of age.

With early detection, rest and sling supports of the affected hip may prevent arthritis and the need for surgery. If arthritis progresses and becomes crippling, the required surgical procedure is a femoral head and neck excision. This same surgery may also be performed in dogs with severe hip dysplasia or dogs whose hips have been severely dislocated by trauma, such as an auto accident. The femoral neck is cut just where it joins the shaft of the femur. Essentially, there is then no true joint at all. The femur simply floats in a sling of muscle which thickens and forms a false joint of support around it. This surgery cannot be done in the giant breeds, but will work well in dogs of medium weight and exceptionally well in small and Toy breeds

Hip Dysplasia

This is the most common developmental orthopedic disease complex in the dog, but before a detailed discussion can begin, it is important to re–examine the Greek translation of the name.

Abnormal growth and development of the hip joint may lead to arthritis, but hip dysplasia must never be equated with arthritis of the hip. In fact, the dysplasia is not believed to originate in the bones themselves; they are normal at birth. Rather, the soft tissue supporting structures that help support the femoral head inside the hip socket are believed to be the main determinant of this syndrome. The dysplasia involves far more than the bony changes that are often not visible on a radiograph before twelve, or even twenty–four, months of age.

In affected puppies, frequently those of the large and giant breeds, a looseness of the femoral head within the hip socket may develop as early as eight weeks of age. Thus, the likelihood of hip dysplasia can be diagnosed in very young pups by careful palpation. Unfortunately, this absolutely must be done with the pup in a deep plane of anesthesia. Moreover, it is best performed by a veterinary clinician specializing in orthopedic diseases. Reliably evaluating laxity in a tiny puppy requires experience as well as expertise. In pups seven to eleven weeks of age, a test developed by Bardens and Hardwick is used to lift the femoral head directly out of the socket, thus evaluating the degree of fit. In pups eleven to eighteen weeks of age, methods developed by Ortolani and Barlow are used. In this test, rotations of the femur are used to determine the risk of dislocation of the femur out of the joint.

Radiographic techniques approved by the Orthopedic Foundation for Animals (OFA) are not accurate in dogs under twenty–four months of age. However, 80% of dysplastic dogs will have an abnormal radiograph at twelve months, and a certain lower percentage will show these changes earlier. In the last two years, new radiographic techniques have been developed specifically for the young pup and published in the veterinary literature. As with all methods for accurate diagnosis of hip abnormalities, general anesthesia is necessary.

The method of G. K. Smith, D. N. Biery, and T. P. Gregor places the puppy on its back, just as the OFA method for the mature dog, but the legs are not rotated in any way. Instead, tension is placed between the thighs and directed outwards, forcing the femoral head out of the joint if it is loose. The method of B. Slocum and T. M. Devine places the puppy on its stomach and directs the X–ray beam across the rim of the hip joint, effectively studying the joint in cross section. These two new techniques are not yet widely adopted, but may eventually allow veterinary clinicians in general practice to produce an objective test for the determination of hip dysplasia in young puppies, just as they now can provide OFA–certification radiographs for the mature dog. Such new techniques will also free dog owners and breeders from a dependence on board–certified orthopedic surgeons for the evaluation of puppies.

Treatment for dysplastic dogs, like that for dogs affected with OCD, varies widely according the extent of lameness and the proposed use for the dog. Many dogs whose HD has progressed to extremely pronounced arthritic changes in the joint may show no signs of pain or discomfort at all. Another dog with radiographically mild dysplasia may experience profound pain and lameness. It is important that the *dog* be treated, not the radiograph. Frequently, this will require multiple radiographic studies over time or periods of rest and anti–inflammatory drugs such as aspirin, or even cortisone, before surgery is considered necessary. Many, many dogs live to an old age with their dysplasia before arthritis is a significant health problem for them.

Surgical procedures include (1) cutting the pectineus muscle and tendon on the inside of the thigh to help reduce painful tension against the hip, (2) femoral head and neck excision as described above under “Avascular Necrosis of the Femoral Head and Neck,” and (3) complete artificial hip replacement. Additionally, a new procedure has been developed for use in young, growing dogs prior to the development of any arthritic changes. Done in two separate operations of the right and left sides, the alignment of the entire pelvic girdle is shifted by

multiple transactions of the pelvis which is then secured in its new position by bone plates. By this procedure, the femoral head is seated deeply into the hip socket before bony changes like arthritic lipping of the rim or flattening of the femoral head can occur. Extensive surgeries are more likely to be done for guard, tracking, and other working dogs, and consultation with a veterinary orthopedic surgeon may be advisable. *In any event, hip dysplastic dogs need to be identified by radiographs and neutered before they can breed. Surgical repair or treatment of dysplasia does not produce a dog that should be bred as this condition is considered hereditary.*

While strides in basic and applied clinical research and treatment in hip dysplasia are made every year, we still do not know the cause of the disease; an interplay of genetic, hormonal, nutritional, and environmental factors is assumed. Interestingly, in one study, 30% of dogs with pathologic changes in their hips also had abnormalities in one or more other joints. In another study of a colony of Labrador Retriever pups, the rate of closure of the growth plates of the hip socket itself could be shifted forward or delayed by thirty days simply by controlling the general growth rate through nutrition. The normal closure date is six months while that of the femoral head is 10 months. In rapidly growing pups, closure of the hip took five months. Since numerous studies have demonstrated a decrease in hip dysplasia when growth rate is restricted, the researchers studying the Labrador kennel theorize that premature closure of the hip growth plates could accentuate the development of hip dysplasia in those pups predisposed.

Such information has created a significant controversy over the proper diet for the growing large breed puppy. Using the same studies, some clinical researchers believe that hyper-supplementation is advisable so that the tendency toward hip dysplasia will most likely be expressed. In that way, elimination of carriers from the breeding pool can be accomplished most quickly. Other researchers recommend restricted nutrition so that growth plate closure will be delayed, and the development of hip dysplasia will be less likely.

Until the precise cause of the disease is known, precise feeding recommendations cannot be made. The best strategy at this time is simply to feed growing large and giant-bred puppies a quality, name-brand growth diet without any additional supplements, particularly avoiding calcium-rich supplements such as bone meal or dairy products. A stool specimen should be analyzed monthly for parasitic infections, and each puppy's weight checked at each monthly veterinary visit. In this way, overall condition and growth rate can be evaluated. Supplements are rarely needed if a puppy is free of internal parasites and is fed a quality growth formula.

Fortunately, thanks to the advances made over the past two decades in both surgery and anti-inflammatory drugs, few dogs today must be euthanized due to the unrelenting pain of arthritis, regardless of its cause.