HYPEROSTROPHIC OSTEODYSTROPHOSY (HOD)
by Fred Lanting

The pup the Smiths bought was a very promising individual with a great pedigree. Here, they hoped, was the foundation of their successful showing and breeding future. But in a matter of a couple of weeks, a previously unnoticed cowhocked condition developed and worsened. They shrugged this off, having heard that dogs with “extreme” rear angulation sometimes develop loose hock action between 2 and 6 months of age. The pup will outgrow it, they thought.

But in another month the pup was acting sickly, evidencing an uncharacteristic listlessness. Rectal temperature showed a fever, and because the pup had intermittent diarrhea, it was started on antibiotics. It had already been routinely treated for worms even though there were no eggs or spores in recent stool checks. After another week the pup “went down” with partial paralysis in the rear, and had very weak (slanted) pasterns with splayed feet and swollen “wrists” or carpal-foreleg joints.

There followed a succession of treatments: Mediprin™, Bufferin™, Prednisolone™, antibiotics, Vitamin D, calcium gluconate, Vitamin C, and more. Sometimes it seemed one course of action was working when suddenly the condition would worsen. Months of worry, pity, temporary relief of pain, nursing care, and assistance passed before the pup pulled out of this perplexing condition. Never did it attain the stature and weight of most others of its breed and line, nor did it lose its cowhocked stance and gait, although it finally gained normal health.

Many variations on the above theme have been played by a frustrating and painful disease known as HOD, which stands for hypertrophic osteodystrophy. Hyper- means excessive, and -trophic or -trophic refers to growth, so the name describes an abnormal and excessive growth of bone (os-) in certain locations. Since this excessive growth does not usually occur as much around the shaft, but certainly at the area of the metaphysis, the term “metaphyseal osteopathy” had at one time been suggested. It has also been called Osteodystrophy I and/or II, Barlow’s or Moeller-Barlow’s disease, skeletal scurvy, and just Vitamin C deficiency at one time or another, although some of these terms are misleading or inaccurate.

BREED, SEX, AND AGE CORRELATION

Once thought to be strictly a problem in giant breeds, HOD is also seen in large and medium size breeds, including Setters, Labrador Retrievers, Doberman Pinschers, Weimaraners, Pointers, German Shepherd Dogs, Collies, Boxers, Basset Hounds, Great Danes, and Borzoi. Probably the greatest occurrence has been reported in Irish Setters. It appears that early rapid growth rate is a factor as it is in the case of hip dysplasia and panosteitis, but size of the individual does not appear to play any role. Several years ago the Irish Setter and the German Shepherd Dog were two of the breeds most seen with HOD but while the incidence truly is higher in the Irish Setter, the inclusion of the GSD in 1979 may be mostly because of its greater numbers rather than a high percentage or incidence in this breed.

One Greyhound, 20 months old, was diagnosed in Sydney, Australia as suffering from HOD, but because of its untypical age and breed, it may possibly have been something else. One group of researchers in 1975 gave the age range as 3 to 8 months, and a 1990 textbook gave 2 to 8 months, and may have included rare early incidents such as the 1973 Australian study that reported an 8-week old pup having had indications for 2 weeks before being presented at the clinic. A 4-year Norwegian compilation of 26 affected dogs indicated that slightly less than half of the cases are diagnosed between 13 and 15 weeks of age, and an equal percentage spread out between 15 and 24 weeks of age. The first half is at approximately the age at which distemper and hepatitis (and many other, in some cases) vaccines are given to most dogs, which may give rise to speculation about challenges to the immune system. In that same study, twice as many males were afflicted as females, which ratio has been noted elsewhere by breeders. It has been shown that females are generally more able to handle stresses.

CLINICAL SIGNS

A description of symptoms should be prefaced by the warning that any one or several can be evident in other diseases as well, and either some or all can be present in the disorder discussed here. It may reappear three or four times, and in this respect it is a little like panosteitis. In a few cases, serious multiple relapses have been noted, enough to warrant euthanasia for relief of the pain. Recovered dogs may have radiographic evidence of residual bone change, or may have frank limb deformities.

In addition to the foregoing example of the Smith's dog, HOD's clinical signs (symptoms) can include a clear discharge at the eyes, bowing of the foreleg below the elbow, and turned-out (“east-west”) front feet. There may be depression, pain (even in the jaws), lameness, reluctance to stand, and anorexia (loss of appetite and weight), with painful joint swelling at the distal metaphyseal regions of the long bones. Although most physical signs are in the distal radius/ulna (pastern area), they are also seen in the distal tibia (hock area). Fever might not be manifest in the early stages of the disease. Diarrhea often, but not always, precedes the joint episodes by a couple of days to a couple of weeks. Usually the severe pain in the lower area of the leg, where either the pastern or the hock begins, typically gives the dog anything from a stiff gait to slight or severe. Extremely adducted pasterns, often described as “soft” or “down”, are very characteristic of HOD. Carpal subluxation, in which the dog stands and walks on its pasterns, is an extreme example of being down at the pasterns, and the similarity is known by breeders who have fed very high-energy, high-protein foods.

HOD, inherited carpal subluxation, and panosteitis share the similarity in that they are all much worse when such diets are consumed. Death is not unknown but fairly rare, and preventable.
RADIOGRAPHIC SIGNS

Correct diagnosis is best made by X-ray film examination along with observation of the clinical signs (symptoms). As young leg bones grow, the end sections are continually changing in composition between cartilage and bone. A short distance from the ends, in the metaphyseal region, is a transverse line of cells known as a growth plate. In order to make the bone increase in length, you’ll remember, cartilage near the end of the shaft is replaced by bone cells while bone in the epiphysis is transformed to cartilage at the growth plate. Meanwhile, cartilage on the far end of the epiphysis ossifies and is itself added to by simple cell-division growth. The greatest change occurs in the distal end of the lower leg, where growth is apparently most rapid in the breeds with medium or long legs.

Radiographically, early signs include a small and irregular radiolucent (clearer) line in the metaphysis just above and parallel to the growth plate and separated from it by a relatively dense band. It’s most easily seen in the lower end of the forearm. In some advanced cases much proliferation of new bone (“calcium deposits”) appears around the metaphysis. This mineralization does not appear to be firmly attached to the cortical bone, but lies on the surface of the periosteum. The longer the condition persists, the further up the limb this ossification of the tissue of the periosteal cuff proceeds. In the worst stages, this new bone will be incorporated with the cortical bone, the hard, dull, dense bone beneath the periosteum. If it goes this far, some remodeling and permanent change will occur. Radiographic evidence may also be seen elsewhere on the bone, at some distance from the joint most affected, and soft-tissue swelling may also be evident.

When HOD strikes, the metaphysis becomes generally more dense and thus more opaque to X-rays, and usually becomes more enlarged; in the growing pup it already is normally wider in proportion to the shaft than it is in the adult. The epiphysis and growth plate largely retain normal appearance, but the radiolucent line is quite noticeable in the increased-radiodense end of the metaphysis closest to the growth plate. Because the opaque appearance is sometimes irregular, granular, and discontinuous, some investigators in the 1960s felt these signs to indicate either a separate disease or a variant of HOD which they called “hypertrophic osteodystrophy type II”. Almost all of us now realize that the use of the term osteodystrophy-II is superfluous as a description of a supposedly different disorder, because early cases of HOD eventually develop new bone growth in and around the surface of the bones.

As the disorder advances, or in dogs suffering from a more severe form or phase, the enlargement of the end of the ulna above the epiphysis, and the bony calcium deposits that form on the outside of the periosteum, are preceded or accompanied by hemorrhage beneath as well as outside the periosteum, and blood cell infiltration into the bone itself.

The periosteum is the tough, smooth, elastic white covering of bones, and it serves as a point of attachment for other connective tissues such as ligament, cartilage, and the fascia of muscles. The ossification shows up on the radiograph as a billowy or beaded opaque deposit separated from the metaphysis by a translucent line at the periosteum. The swelling resulting from such hemorrhage and bony growths is often very warm and always painful. Such deposits are not usually found in areas of slower growth, such as the proximal metaphysis of the radius and ulna, but can be quite massive on the distal end in just a couple of weeks after symptoms commence. As the disease runs its course and the patient recovers, the mineralization outside the periosteum is gradually resorbed and the radiographic appearance of the metaphysis resumes normal shape and density. At the same time, body temperature has returned to normal, lameness begins to subside, and appetite returns. While repair and remodeling are usually complete, there are some cases in which distortion of the diaphysis and residual osteophytes can remain.

HEMATOLOGIC AND HISTOLOGICAL INDICATIONS

A higher than normal level of white blood cells (primary infection fighters) is often an indication of the presence of a viral or bacterial agent, and in HOD there is sometimes a high leukocyte count in bones as well as in the blood. However, biochemical analysis and hematologic tests are not very fruitful in this disease, even though neutrophilia, lymphocytopenia, and monocytosis may be findings during the late or active stages; these probably reflect stress and inflammation, and are effects rather than causes. Blood tests can show mytosis, an increase in a certain type of leukocyte, and anemia to a slight degree. Chemical analysis discovers low serum ascorbic acid (vitamin C), but to implicate a deficiency of this vitamin would be a mistake. The reduced serum level often found is now considered a secondary change rather than a causal factor. Besides, microscopic changes in bone in these patients are different than those in hypovitaminosis C (deficiency). See discussion of vitamin C below.

The most noticeable histologic changes are seen in the metaphysis, specifically in the nature of its spongy bone. Here the pathologist can find microfractures in the trabecular bone, necrosis, osteomyelitis, and other bone defects. That dense band I mentioned earlier is a result of excess calcification spreading from the cartilage lattice of the primary spongiosa, and from abnormal trabecula cell growth. Upon necropsy, he can also confirm what the radiologist has seen, the ossification of the periosteum and nearby soft tissues.

NUTRITION

By 1957 it was obvious that dietary vitamin D increase was not effective, and in fact by the mid-1960s breeders were warned against mineral overloading (calcium supplements in the diet). Yet even in the 1980s and to a lesser extent the 1990s, calcium supplementation occasionally had still been prescribed for HOD! It is part of human nature to rest on what we’ve been told...
rather than practice continuing education, since “the world is too much with us”, so don't be surprised if your vet hasn't kept up on everything and especially on things not as necessary to his daily practice. HOD doesn't show up frequently enough to warrant that much attention, but the orthopedic specialist who gets referrals from many vets may more easily identify its symptoms.

To be fair, one must also acknowledge the report of one study that involved supplementing the diets of young large-breed dogs with minerals and vitamins A and D at three times the NRC recommendations. These dogs had no differences in growth rates, radiographs, or blood chemistry. But I feel the preponderance of evidence leans toward the previous view, that there is considerable risk to many dogs in supplementing in that way, especially those breeds or families that may have a genetic predisposition to HOD.

Most who have studied HOD now conclude that it is probably some sort of a metabolic disorder, and many if not most believe also that the tendency to fall prey to owners' practices of over-supplementation and overnutrition is genetic. An imbalance of minerals, protein, and vitamins interferes with normal deposition of calcium phosphate (bone) and turnover of cartilage which lead to the physical, visible changes. If a high-protein, high calorie/energy, and highly palatable diet producing overnutrition is indeed a candidate for cause, the process possibly traces its route through excess calcium, hypercalcemia, hypercalcitoniom, hypoparathyroidism, and retarded bone resorption. The body reacts to excess calcium by lowering the level in the blood (excretion, deposition) but it goes too far into hypocalcemia because of the persistent hypercalcitoniom. As you learned in the chapter on nutrition, this condition arises because excess dietary calcium stimulates the gut to secrete more gastrin.

On the other hand, some investigators remain skeptical because hyperthermia (fever), which is a reliable sign in most diagnosed cases, was not recorded in the experimentally over-nourished dogs in the main nutrition study, and certain histologic and radiographic signs found in HOD cases “in the field” were not seen in the experiment dogs. Further, HOD does occur in some dogs on dietary intake that would not be considered overnutrition under current guidelines.

Vitamin C

Even researchers who once felt that HOD in the late form may be associated with deficient vitamin C will admit that the vitamin theory was controversial and that the response to vitamin C therapy was variable. Clinical signs similar to those of scurvy (frank vitamin C deficiency) have been reported in young dogs but no direct link was firmly established by that work. Later, a Finnish team at the vet school in Helsinki published a poorly-founded one-page anecdotal report of two cases they diagnosed as being HOD. The undated and unidentified copy of “Scurvy as a cause of HOD in two young dogs” that I received from a vitamin supplement salesman in Finland had poorly-documented references but quoted, among other sources, work by Kivirikko as stating that insufficient vitamin C resulted in the failure to biosynthesize collagen. The deduced chain of events was given as disappearance of collagen bundles, and cartilage becoming thin and watery in appearance. They concluded that the livers of young pups become stressed by the vaccines against distemper, hepatitis, and other viral disorders, and since the liver is where most of the dog's vitamin C comes from, the shots temporarily disrupt the natural in-situ synthesis of this vitamin. They also implied that larger, fast-growing breeds required more ascorbate/vitamin C than their livers could make to keep up with the enormous amount of collagen production needed. Respected nutritionists have debunked this work, and one I won't name here (but I have in personal correspondence) gives it the name of a famous rodent created by Walt Disney.

Many people with some degree of hypochondria imagine themselves as having a particular disease or group of ailments, based on the descriptions of the symptoms common to them. Similarly, some people see what they think are indications of disorders in their dogs, jump to conclusions, and make a diagnosis, often erroneously. It is a normal thing for many breeds, especially the giant and some of the other large breeds, to have somewhat "knobby” carpal joints, so this should not lead one to a supposition of HOD. On the other hand, it would be exercising wisdom not to over-feed or supplement with vitamins A or D and calcium.

The great vitamin C controversy is far from over, as can be seen by the frequent resurrection of “scientific studies” which have little or no basis, but represent a “magic pill” answer for the hopeful producers and readers of club newsletters and magazines who are careless about what they promote or believe. These articles are resurrected in greater frequency than the independent return of HOD.

Man has apparently benefited from very large doses of this vitamin during times when the body is under stress as a result of viral and other infections, but most animals make their own ascorbate (vitamin C). Newborn puppies synthesize their own even when the colostrum and bitch milk have elevated vitamin C levels, and relatively large doses of the vitamin sometimes have little effect on either the production rate of self-synthesized serum ascorbate or on the course of certain diseases such as HOD, hepatitis, kennel cough, etc. Vitamin C salesmen usually claim the doses were not large enough.

HOD is associated with retardation of bone resorption and by excessive bone formation, and is linked to excessive dietary carbohydrates and protein. That is its only relationship or similarity to HD, as much as we can tell. Vitamin C deficiency as a factor in causing HOD is highly unlikely, and vitamin C supplementation can only make HOD worse, according to University of Liverpool researcher Dr. David Bennett.
The measurement of ascorbate or ascorbic acid is difficult and often inaccurate, as it is very unstable. I remember the frustration I encountered when finding this to be true in graduate work, in my organic chemistry lab. Therefore, I don’t get excited when I read reports of ascorbic acid measurements in blood and urine in connection with HOD.

Extra vitamins C and A can enhance calcium absorption from the intestine, and more calcium certainly is not what you want when a dog is suffering from “calcium deposits”. Because of the report of low serum ascorbate (vitamin C in the bloodstream) some work was undertaken at Cornell which contradicted the suggestion to supplement, and found that large doses failed to give consistent results. This was in agreement with Bennett’s work. Those researchers at Cornell claimed the earlier studies supporting the use of vitamin C were uncontrolled and the results equivocal. Some dogs in the Cornell investigation had a temporary remission, others were totally unaffected.

More about diet

The effect of diet as a causative factor may be equivocal, but there is no doubt that excessive calcium supplementation can greatly exacerbate the pain and radiographic signs. As a general rule, stay away from calcium/vitamin D additions to the food, since it not only makes the HOD worse, it contributes to the severity of other orthopedic and systemic disorders as well. Even ad libitum feeding of high-nutrient density, balanced dog food without extra calcium has resulted in experimentally-induced HOD. If a growing dog eats all it wants of a “good” dog food, it can absorb more calcium than is beneficial compared to a pup on a restricted diet. Keep your puppies on the thin side, and you can avoid some health problems.

Use a high-quality, but not high-energy (calorie) dog food, don’t feed more than the dog needs or wants in a short mealtime, and don’t supplement with Vitamins C or D or calcium if HOD is known in your breed, unless your nutrition-expert veterinarian prescribes these for some specific reason. Certainly don’t supplement if the symptoms of HOD appear. It would probably be wise to switch the HOD patient’s diet to a lower-calorie, lower-protein food as quickly as you can without causing diarrhea. Make sure it has a low enough calcium level to satisfy your veterinarian (whom you’ve already asked to confer with perhaps a specialist in the veterinary teaching hospital at the university.)

CAUSE

The cause of HOD is unknown. This is the message that comes out of all the work done so far, and the picture is unlikely to get any better until there is sufficient information and controlled studies to yield some scientific conclusions. One veterinarian/breeder published in the newsletter of the Irish Setter Club of America a questionnaire in which he sought answers to some 32 questions designed to uncover a connection with another disease, diet, or genetics. Almost no one responded, although Irish Setter publications had carried a number of tear-jerker case histories and warnings about HOD. Apathy will certainly hinder the fight. Perhaps some group will find the interest and the contributions, and fund sufficient research to solve the HOD enigma. But then, I had expressed the same hope in 1980.

I once suspected a viral agent might have been directly connected with HOD, but no evidence has come to light to support that, although further work is needed before we can exclude microbial infections. It now appears HOD is quite possibly a result of nutritional and immunological forces acting in dogs, most of whom may be genetically at risk. Familial relationships have been claimed in anecdotal reports gleaned from discussions I’ve had with many breeders. Most cases are diagnosed at approximately the age at which distemper vaccine (at least, the “adult shots”) is administered, and many have shown initial symptoms one to six weeks after vaccination. The fact that fever accompanies other signs, and the additional history of diarrhea frequently preceding the onset of pain are indications that if a virus or bacteria is not a direct causative agent in genetically-susceptible dogs, it is possible that an inactivated, killed, or modified live virus somehow upsets the dog’s immune system. Immune response is related to the function of many endocrine glands that produce hormones, and hormones have been identified as playing a part in mineral absorption and joint development. Too tenuous a thread for you to follow with credulity? Understandable. That’s why I present it only as an idea to run up the flagpole. If nobody ever salutes, there’s no harm done. In humans, measles vaccine has caused bone disease surprisingly similar to canine HOD. Nearly every dog fancier knows of the similarity between measles and distemper viruses, the former being commonly used in a modified version to immunize very young puppies against distemper before the pups are completely weaned. Incidentally, there is some suspicion that human multiple sclerosis is related to canine distemper virus, though it is an unproven theory as yet.

An attempt to induce HOD in healthy dogs by transferring the disease from affected dogs was planned in Norway and partially completed, but with somewhat disappointing results. Blood was transfused from a dog in the acute stage of HOD to healthy dogs of different breeds, and some developed distemper and died! Their blood donor had been vaccinated against distemper and hepatitis one week before it had shown signs of HOD. Other dogs died after receiving blood from another HOD-afflicted donor, but none of that dog’s recipients developed signs of HOD. Interestingly, there was an epidemic of distemper in the area at the time, but less than 3 years after that, HOD had almost disappeared, with only two dogs being diagnosed for it since the 26 cases at the clinic in 1976. Could whatever causes HOD be transmitted in the blood and also cause distemper? Could some individuals challenged with either virulent distemper virus or modified viral vaccine come down with HOD, and others get distemper?

Why does HOD seem to come and go, like consecutive bad years for dog ticks or cicadas, followed by a respite for a few or several years? That is hard to answer, and any attempt is speculative. HOD could be a single disease with one or more causes, or it could be a syndrome. You remember that means a collection of symptoms, and that there could be up to several disease “events” going on at one time. Your dog with HOD might have a copper deficiency, a diet too high in protein and calories, a microbial infection, a challenged immune system, or any combination of these and other processes going on at once. That HOD seems to come in “waves” lends credence to the multiple-cause hypothesis.
TREATMENT OF HOD

Penicillin, streptomycin, sulfa, and other antibiotics, and a host of analgesics ("pain killers") such as aspirin preparations, Mediprin™, and others have been administered with no reliable beneficial or conclusive results. Steroids and other medications were given to no avail as far as the primary lesion was concerned. Because of spontaneous remissions and unforeseen worsening or relapses, the success or value of any treatment will continue to be elusive. Remission and drug use are probably coincidental in almost all cases. As in the case of panosteitis, it appears that in most cases the dog will get better whether or not it is treated at all, and regardless of diet except for the harmful addition of calcium, vitamin D, and possibly vitamin C. Some owners reported apparent improvement with one choice one time, and then did not repeat their success the next time. It may be wisest to treat symptoms conservatively and assume we have another self-limiting disorder in HOD-afflicted dogs, with TLC (tender loving care) and patience the best tools at your disposal.

The difference in this conservative management approach to HOD compared to panosteitis is that the complications in HOD may be very serious. The dog may not die from the HOD itself, whatever the cause may prove to be. This is similar to the situation in human medicine wherein the patient does not die of the AIDS virus directly but of the complications it brings on, such as cancer or disorders in the lungs, heart, and other organs and systems. Therefore, medical management of HOD should be directed toward halting the diarrhea, lowering the fever, getting rid of parasites, and relieving whatever pain you can. Symptomatic treatment might make the difference between losing your dog and saving him, but death is such a rare consequence that the owner is cautioned not to go overboard on treatment. Don’t try to eliminate all symptoms, in other words, or the remedy might be worse than the disease. At present, the only generally recognized treatment prescribed is purely symptomatic: relief of pain through buffered aspirin or sometimes corticosteroids. Some few of the many owners I have corresponded with have been positive that if they had not treated the symptoms such as appetite loss, diarrhea, etc., they would have lost their pups. Most people who have studied this disorder agree that the best you can do is give the dog rest, aspirin if the dog is obviously in pain, and a diet not excessive in protein or energy. In the worst cases, the dog might have to be force-fed or given fluid therapy to prevent dehydration, and other symptom-oriented treatments.

DISORDERS WITH SIMILAR SIGNS

Besides reading the following short comments about disorders that could conceivably be mistaken for HOD, it would be a good idea to read more about them in works dealing with other miscellaneous disorders. Your vet will be especially careful to avoid a poorly exposed or focused radiograph, as the wrong diagnosis otherwise could easily be made.

Panosteitis usually occurs in older pups than does HOD, and is less severe with a zero fatality risk in itself. Moeller-Barlow’s Disease was once thought to be a separate problem with less fever associated with it than HOD, according to some reports but most now believe it to be the same lesion. The signs of HOD resemble those of scurvy in humans, and radiology shows features of both clinical rickets and scurvy. Osteodystrophy-II, mentioned earlier, is probably a stage in the progression of HOD, beyond which some individuals never go before recovering. Hypertrophic pulmonary osteoarthropathy also has periosteal new bone formation at the distal ends of the extremities, but it is almost always accompanied by lung disease, and the osteophytes are more in the wrist and hock than in the long bones. Legg-Calvé-Perthe’s disease and hip dysplasia involve the proximal end of the femur and is usually a problem just in toy breeds. OCD of the shoulder and knee (stifle) and some elbow disorders can give somewhat similar clinical signs, but are readily identifiable radiographically.

Another, very similar disorder is hypertrophic osteopathy or hypertrophic osteoarthropathy, again characterized by osteophytes on the outside of the ulna, radius, and other long bones, usually worse at the distal ends near the pastern or hock. How this differs from HOD is not so much in radiographic and direct visual appearance, but in age of onset, recommended treatment, and probably the cause. While HOD strikes puppies in a certain age range, this disorder affects dogs of all ages, most often adults.

Fungal infection

Sometimes bony involvement of long bones caused by Blastomyces will cause radiographic shadows with similarities to HOD, but this fungal disease may also show up as skin lesions around the distal end of the limb section where the apparent increase in bone density is found on film. It shouldn’t be difficult to differentiate between them.

Multiple Cartilaginous Exostoses

Yet another condition with radiographic similarities to HOD is found in humans, cats, horses, and dogs. This is also known as osteochondromatosis, hereditary multiple exostosis and, in Britain, diaphyseal aclasis. It is seen mostly on ribs, vertebrae, and the long bones, although it has been seen on every bone except the skull. Because the growth, a gross exostosis, seems to involve mostly the metaphyseal area (near the growth plate at the ends of the long bones), it can be mistaken for HOD.

Polyarthritis

The name suggests the problem: arthritic inflammation in several locations. This disorder can be classified as erosive or non-erosive, but both types can mimic HOD upon cursory observation, especially in that they can produce swelling at the carpus and tarsus (pastern and hock), but also in other symptoms. Where HOD usually occurs between 3 and 5 months, this disorder’s onset is more typically between 9 and 10 months of age.
HOD is an orthopedic disease seen in medium, large, and giant breeds, more common in some than others. There may be several causative factors including heredity, infection, and possibly vaccines, with contributing factors being both genetic susceptibility (“weakness”) and calcium supplementation or unlimited/excessive feeding of pups resulting in mineral overloading as an intensifier of pain and abnormal bone growth.

As in the case of panosteitis, the disease appears to be both self-limited and transient, independent of treatment. Although there are rare deaths, probably due to “complications”, most pups outgrow HOD within one to a few months. The fatality rate is too erratic to reliably measure. In some reports it has been 25-35% (almost certainly inflated via poor statistics and diagnoses) and in others it was less than 4%. In every case, it is traumatic because of the pup's pain and the owner's helplessness and frustration.

Multiple relapses are not uncommon, and the same bones can be affected more than once. Extraperiosteal calcification is slowly resorbed and radiodensity of the affected limbs returns to normal or nearly so. Some individuals are left with permanently bowed forelegs because the ulna has grown at a different rate than the radius (as is the case in some elbow dysplasias), and some are cowhocked for life. Most, however, endure and survive the effects of HOD without permanent damage.