The Show Dog Super Site

Breeder Vet

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UNILATERAL HIP DYSPLASIA

For 22 years, I have been working with large breeds of dogs in my veterinary practice. The numbers of giant and large breeds we see far exceed the numbers of medium and small individuals. One reason for this is my long history with this type of dog; another is that we do OFA hip radiographs without anesthesia, and for whatever reason, the owners and breeders of the large and giant breeds are, as a whole, far more sensitive to anaesthetic issues than those of smaller breeds.

Over the course of these years, the kennels of many clients who raise large numbers of giants have been our laboratory. Following these breeds, these breeding lines, and individuals within these lines over their entire lifetimes, certain facts have become incontrovertible to us.

We have never taken the time to go into our records and pull out the data to quantify these observations for professional journals, so the observations here must come under the heading of clinical findings. However, a few years back we looked into the records on Newfoundland dogs, and found our database held over 400 adult individuals. Several other large and giant breeds are represented in lesser, but still very substantial, numbers.

Internet sources on hip dysplasia include a selection of abstracts from Medline for the years 1966 to 1996.(Medline Literature Search: Canine Hip Dysplasia, 1966 - 1996. http://www.working-retriever.com/library/chd6696.shtml) At the end of this article are some of these abstracts found on that page which support observations made here. Many other references are present in the literature, but these suffice to illustrate the research available making mention of the items we are interested in:

- Dietary considerations
- Influence of injury and pain

THE PREMISE

One frequently encounters discussions which assert that hip dysplasia is 50% genetics and 50% environment. We prefer to think of it as 100% genetics, then 100% environment. Genetic considerations are the entirety of what we must as breeders consider. Once that puppy is born, environment is 100% of how well that puppy will do within the possibility of his genetics. Dogs with very good hips, congenitally, may have an injury and end up diagnosed as having 'unilateral hip dysplasia'. Individuals with very shallow sockets may be mobile and free of pain to an advanced age, if they have unlimited exercise. This is the entirety of what the owner of that puppy needs to concern himself with to provide the best possible care for his dog.

Over the years, our observations of the kennel populations of giant breeds and their siblings living in private homes have led to the conclusion that there is no such thing as *congenital* unilateral hip dysplasia, but only *acquired* unilateral hip dysplasia. The kennel dogs, whose exercise opportunities are maximized, with several dogs of a similar age free to run and play all day and night in large paddocks, show us some interesting things.

- They grow much more slowly, because much of their food intake goes into play and running
- They rarely (almost never) suffer an injury.
- They always have symmetrical hip sockets, even if they are very shallow.

Their litter mates in private homes, where exercise is confined to an hour or two of intense play or jogging when their owners return from work, provide us with a different set of observations.

- They grow very large, very fast.
- They often suffer injury.
- They frequently have hip sockets of different depths.

The implications of these observations are enormous, but very simple to understand. These large breeds grow much too quickly for their biology to keep up. They frequently show some degree of clinical rickets with some bowing of the forelegs, and have large soft joints due to the inability of the body to deposit calcium in the bone at a rate equal to the rate of growth. They often show uneven growth, with the rear end growing over a few weeks, then the front end trying to catch up. As the rear leg assembly grows disproportionately, and the puppy is 'high in the rear', the mechanical leverage that the muscles are able to exert across these straighter angles is much reduced. The result of this is a decreased ability of muscle to protect joints from injury.

The owners of giant breed pets tend to overfeed their puppies, having with the best of intention, the inner desire to see a 'big dog' and to do nothing which might risk the dog not attaining the greatest possible size. Try as they may, some owners are just unable to restrict their puppy's diet. Owners are cautioned that their puppy needs a good deal of exercise, but their work schedules often conflict with their desire to do this. The result is a period of intense exercise. A 3 mile run, or a half hour of Frisbee. When a single puppy who lies around all day welcomes his owners in the evening, he is ill prepared for either intense exercise, or the uncertainty of footing on slippery floors and his always changing joint angulation. His muscle tone is a small fraction of that of a puppy which plays with other active dogs all day long. The result is an injury.

When any dog has a hip dislocated, if it is not repaired within 48 hours, the socket begins to lose depth. When an injury occurs to a rear leg, whether it be a toe injury, a stifle injury, a soft tissue injury, or a hip injury, the result is often a decreased amount of weight bearing on that leg over a period of days to weeks or months. When this happens, the mechanical forces applied to the living bone tissue change, and the hips become asymmetrical, the injured hip becoming shallower in a similar fashion.

Many will find that a very controversial statement. Dr. Corley from the OFA would reply to observations such as this, that he could prove that unilateral hip dysplasia was genetic, since it was almost always the left hip which was shallower. My response to his statement was that this proves to me that most dogs are right handed. The left diagonal being the master limb, the one with which the dog pushes off most strongly, and the one which is most liable to injury, especially stifle injury.

The conclusion from these observations is that the single most important environmental factor in a puppy's life is exercise, continuous and strenuous. Since this is often impossible for owners to arrange, the next considerations are to drastically restrict the diet of the growing puppy and to avoid strenuous exercise which will exhaust his muscles and leave him unable to protect his joints from injury. The puppy should be given frequent moderate exercise instead. This requires a different kind of time commitment from the owner.

NUTRITIONAL CONSIDERATIONS

In the past few years, more and more articles on nutrition in large breed puppies have pointed to a very early metabolic problem with over feeding. It has been shown that overeating even within the first 2 weeks of life can significantly affect the outcome of a puppy's hip conformation. Many of our giant breed bitches as well as other breeds, give way too much milk. Food is too readily available, and she doesn't have to go out and exercise in order to find it. We won't ask you to starve your bitches to reduce their milk output, but it might be wise to follow the rate of gain of your puppies and avoid the overeating problems, diarrhea and colic, as well as the eventual hip problems, by keeping these bitches out of the box for a period of time daily. These articles imply that *it is more than just weight bearing* that is involved in the eventual status of hips in puppies which are over fed.

BREEDING DECISIONS

Another issue these observations force us to confront is what to do about the breeding future of an individual which has *one very good hip* and one with damage. We must consider that the 'good' hip is the one which most closely reflects the dog's genetic makeup. Hips don't get better with age, only worse, if they change at all. This brings us into the twilight zone, the gray area. It is not the job of the OFA to speculate on what matters might affect a given animal's radiographic hip configuration. However, as breeders, especially in some breeds with many genetic defects to weigh and consider, one might wish to breed this animal. Since our clubs rely so heavily on radiographic hip interpretation in their codes of ethics, this is a matter of great soul searching for many breeders. Whether to breed an animal which no doubt has genetically good hips, but which won't pass OFA, or not. The only advice here is that it *is* necessary to have a clearing house for hip x-rays, and OFA does a good job, in general. But in some specific instances, that rating might not reflect a dog's genotype accurately, or more properly, as well as ratings with both hips symmetrical. Actually, phenotype (the actual hip conformation) is never the same thing as genotype. This is a situation where it is even more important than usual to gather hip information on parents and the dogs behind them, the parents' siblings, and any siblings of the affected dog.

PAIN RELIEF AS TREATMENT

There is a persistent conviction among some dog breeders that vitamin C can help prevent hip dysplasia. Why did the dog fancy start giving vitamin C for 'prevention' of hip dysplasia? Back in the days when Linus Pauling began touting its effects in prevention of colds, it was discovered that it has a minor role in pain relief. Vitamin C does not prevent colds; it reduces severity of symptoms. Any anti-oxidant and free radical scavenger will do this. The vitamin C pain relief factor is a pretty small one. However, the same puppies we're worried about developing bad hips always have some degree of rickets as well. Vitamin C is the treatment for the bad effects of rickets. The combination of these two beneficial effects no doubt keeps the puppy more mobile, thereby allowing him to use himself better and more evenly, and to exercise more. So then, the situation above comes into play. The puppy moves more, has fewer joint deformities during growth, and can keep himself fitter, if the effects of rickets are reduced and a bit of pain relief is realized.

The abstracts below contain several references to the use of 'PSGAG' (polysulfated glycosaminoglycan), a.k.a. 'Adequan', in puppies. And now we've hit the real answer to the challenge of keeping injured dogs mobile and reducing time of differential weight bearing. Adequan is a marvelous drug. It is given by injection in the muscle, and it works just as the body would, if there were no chronic inflammation in the joint; it begins to fill in and repair the painful cartilage injuries, reducing arthritis and keeping the puppy mobile, and *keeping weight bearing equally distributed!* It's pretty clear that some 'unilateral hip dysplasia' can be avoided through the use of anti-inflammatory drugs, and in more cases, the degree of difference between the hips can be reduced. Another aniti-oxidant/free radical scavenger used by this practice, which affords really significant pain relief, is Super Oxide Dismutase/Catalase, which is marketed by Biovet. This product and the Adequan injections fall into the class of treatments which are 'all good'; there are no down sides to their use. Aspirin remains the century's super drug, in the mind of many; aspirin too can be used to keep a puppy mobile after an injury, in the appropriate dose.

A Note About Lyme Disease. The use of Adequan in the treatment of Lyme arthritis is an absolute must. The pain of Lyme arthritis is excruciating, and aside from treatment, the use of Adequan becomes a simple humane consideration. Its ability to fill in cartilage erosions and reduce pain is nothing short of miraculous. The only caution here, is that it must be used until the spirochaetes causing the cartilage damage are gone, not the usual 5 week treatment.

ABSTRACTS

J. W. Alexander. The pathogenesis of canine hip dysplasia. Vet Clin North Am Small Anim Pract, 1992;503-11. Dogs with CHD are born with normal hips that subsequently undergo varying degrees of subluxation of the coxofemoral joint. Although the etiology of CHD is multifactorial, the pathogenesis or stages of change within the affected coxofemoral joint(s) are similar regardless of cause. With the onset of the disease, there are progressive structural changes including joint laxity and femoral head subluxation; swelling, stretching, fraying, and eventual rupture of the teres ligament; a shallow, flattened acetabulum, deformity of the head; erosion of articular cartilage, eburnation of subchondral bone, DJD; and periarticular osteophyte formation.

C. L. Fries and A. M. Remedios. The pathogenesis and diagnosis of canine hip dysplasia: a review. Can Vet J, 1995;494-502. Hip dysplasia is a common developmental problem affecting the canine population. Despite extensive research into the condition, many questions remain unanswered and numerous misconceptions are present among the general public. The purpose of this paper is to review the current knowledge on the development of hip dysplasia, factors modifying its development, and current diagnostic techniques. A computerized literature search was conducted for the period of January 1983 to April 1985 using the MEDLINE and CAB databases, and the keywords hip dysplasia, hip, dog, and canine. Other articles, wherever possible original research articles, published before 1983 were also reviewed. Animals affected by hip dysplasia are born with normal hips, but quickly develop subluxation of the femoral head. Degenerative joint disease follows. Hip dysplasia is a complex, inherited, polygenic trait. Selective breeding of only normal dogs with normal littermates, parents, and grandparents is the recommended method of reducing the incidence in the general population. Gene expression in affected individuals may be modified by a number of environmental factors. These factors do not cause hip dysplasia, but they alter manifestations of the trait and its severity. Nutrition is a major environmental factor. Excess energy consumption increases the frequency and severity of hip dysplasia in genetically predisposed dogs. Food intake should be regulated to maintain a slender figure with the ribs and dorsal vertebral spines easily palpable, but not visible. Excess dietary calcium and vitamin D contribute to hip dysplasia in genetically predisposed individuals and should be avoided. High dose vitamin C supplementation in growing puppies does not prevent hip dysplasia, and this practice should be discontinued.(ABSTRACT TRUNCATED AT 250 WORDS)

R. D. Kealy, S. E. Olsson, K. L. Monti, et al. Effects of limited food consumption on the incidence of hip dysplasia in growing dogs. J Am Vet Med Assoc, 1992;857-63. Forty-eight 8-week-old Labrador Retrievers were allotted to 2 groups of 24 dogs each; 1 group was fed ad libitum and the other group was given 25% less of the same feed until the dogs were 2 years old. Radiography of the hip joints was done when the dogs were 30, 42, 54, 78, and 104 weeks old. Subluxation was measured by the Norberg angle on radiographs made with the dog in the standard (extended limb) position. Independent of age at which the radiography was done, there was less subluxation of the femoral heads in the limit-fed dogs. Using the Swedish method of hip joint evaluation on the same radiographs, it was found that fewer dogs on limited food intake had signs of hip dysplasia. Radiographs done when dogs were 2 years old, for all the methods used (Norberg angle in standard and frog-limb position, the Orthopedic Foundation for Animals [OFA] score, and the Swedish score), revealed less hip dysplasia (less joint subluxation and less degenerative joint disease) in the limit-fed dogs. Using the OFA method, 7 of the 24 limit-fed dogs and 16 of the 24 ad libitum-fed dogs were diagnosed as having hip dysplasia. Similarly, using the Swedish method, 5 of the 24 limit-fed dogs and 18 of the 24 ad libitum-fed dogs were diagnosed as having hip dysplasia. The food-intake-related differences were significant both for the OFA score and for the Swedish score.(ABSTRACT TRUNCATED AT 250 WORDS)

J. J. de Haan, R. L. Goring and B. S. Beale. Evaluation of polysulfated glycosaminoglycan for the treatment of hip dysplasia in dogs. Vet Surg, 1994;177-81. A double-blinded, controlled clinical study was performed to compare the response of adult dogs affected with hip dysplasia to a placebo and three different dosages of polysulfated glycosaminoglycan (PSGAG): 2.2 mg/kg, 4.4 mg/kg, and 8.8 mg/kg. Dogs were randomly assigned to treatment groups. The drug was administered intramuscularly every 3 to 5 days for a total of eight injections. Response to treatment was analyzed based on changes in lameness, range of motion (ROM), and pain on manipulation of the hip joints. Evaluation for adverse reactions included complete blood cell (CBC) count, blood urea nitrogen (BUN), creatinine, and physical examination. Data were collected on a total of 111 dogs. Eighty-four met all criteria for inclusion in the study. Dogs that were given 4.4 mg/kg of PSGAG showed the greatest improvement in orthopedic scores, whereas dogs in the placebo group showed the smallest improvement; however, the differences in clinical improvement between the four treatment groups were not statistically significant. No local or systemic adverse reactions related to the drug were observed.

G. Lust, A. J. Williams, N. Burton-Wurster, et al. Effects of intramuscular administration of glycosaminoglycan polysulfates on signs of incipient hip dysplasia in growing pups. Am J Vet Res, 1992;1836-43. We tested the hypothesis that treatment of growing, susceptible (to hip dysplasia) pups by IM administration of glycosaminoglycan polysulfates would mitigate the signs of incipient hip dysplasia. In 1 experiment, 7 pups, selected at random from 2 litters, were administered glycosaminoglycan polysulfates (2.5 mg/kg of body weight, IM) twice weekly, and 7 control pups from the same litters were given sterile buffered 0.9% saline solution from the age of 6 weeks to 8 months. Hip joints were examined by radiography, with pups in the standard, limbs-extended position. At 8 months of age, all pups in this experiment did not manifest femoral head subluxation radiographically. The Norberg angle, a measure of coxofemoral congruity, improved from a mean +/- SEM value of 102 degrees +/- 1 degrees in controls to 106 degrees +/- 1 degrees in treated pups (P = 0.008). Pups were not subjected to necropsy. In the second experiment, 8 pups were selected at random from 2 litters and were administered 5 mg of glycosaminoglycan polysulfates/kg, IM, twice weekly from 6 weeks to 8 months of age. Similarly, 8 control pups were administered saline solution. At 8 months of age, hip joints were examined by radiography with pups in the standard position; at necropsy, intra-articular tissues were evaluated macroscopically and biochemically. Of 8 treated pups, none had subluxation radiographically, whereas 4 of 8 control dogs had femoral head subluxation. Mean Norberg angle on the radiographs was 109.7 degrees +/- 1.6 degrees for the treated group and was 101.5 degrees +/- 1.6 degrees for controls, representing a mean improvement in coxofemoral congruity of 8.2 degrees in the treated pups.(ABSTRACT TRUNCATED AT 250 WORDS)

D. C. Richardson. The role of nutrition in canine hip dysplasia. Vet Clin North Am Small Anim Pract, 1992;529-40. The role of nutrition in canine hip dysplasia is as multifactorial as the disease itself. Large and giant breeds primarily are at risk for the disease. Rate of growth, feeding methods, feed consumption, specific nutrients, and electrolyte balances within the diet have all been shown to influence hip dysplasia. Known nutritional risk factors are rapid weight gain and excessive calcium supplementation. Nutritional factors with less secure roles in their influence on the disease process are vitamin C, protein, and carbohydrates. There exists a need to identify further and control the various nutritional factors in the diet that influence canine hip dysplasia.

I. Hanssen. Hip dysplasia in dogs in relation to their month of birth. Vet Rec, 1991;425-6. German wirehaired pointers, English, Irish and Gordon setters, and labrador retrievers that were puppies during the spring and summer had a significantly lower incidence of hip dysplasia than those growing up during the autumn and winter. However, golden retrievers and German shepherd dogs did not show the same seasonal pattern of incidence of hip dysplasia.

C. S. Farrow and R. T. Back. Radiographic evaluation of nonanesthetized and nonsedated dogs for hip dysplasia. J Am Vet Med Assoc, 1989;524-6. The use of chemical or gas restraint was unnecessary in most large breed dogs being evaluated radiographically for hip dysplasia. Of 100 large-breed dogs, 97 were successfully radiographed for hip dysplasia evaluation without the use of sedation or anesthesia.

H. Kasstrom. Nutrition, weight gain and development of hip dysplasia. An experimental investigation in growing dogs with special reference to the effect of feeding intensity. Acta Radiol Suppl (Stockh), 1975;135-79. Thirty-one dogs from 5 litters with a high parental frequency of hip dysplasia were used in the investigation. Each litter was split in two groups, of which one was put on a high caloric intake, the other one on a low caloric intake. Each member of a group had a paired litter mate in the other group. The litter mates were paired on the basis of the result of a palpatory examination of the hip joints before 12 weeks of age. If possible, paired mates were of the same bodyweight at the time of palpation, and of the same sex. In the groups made up of pups from 3 of the litters, the protein intake was kept at an optimal level, regardless of the amount of calories given. It was found that hip dysplasia was more frequent, occurred earlier, and became more severe in the dogs with a rapid weight gain caused by increased caloric intake than in the dogs which had a low weight gain because of restricted feeding. The final diagnosis was closer correlated with feeding and weight gain than with tightness or laxity of the hip joints before 12 weeks of age.