Canine Hip Dysplasia Part VI

Treatment of this disease must be tailored specifically to the needs of your pet, whether using conventional or alternative medicine

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This article is the sixth in an eight-part series on canine hip dysplasia (CHD). What follows is written from the perspective that the readers are serious and conscientious breeders who are the guardians of the genetic pools that constitute their breeds. While this series of articles will not replace a stack of veterinary and medical texts, it is a relatively in-depth look at the whole problem of canine hip dysplasia. Furthermore, the series is designed to be retained as a reference. When you finish reading this series, you will have a sufficient background to make rational breeding choices and will be able to discuss the subject from an informed basis with your veterinarian. You may not like what you read, but you will be more competent to deal with the problem.

Conclusions from part I: Genetics is the foremost causative factor of canine hip dysplasia. Without the genes necessary to transmit this degenerative disease, there is no disease. Hip dysplasia is not something a dog gets; it is either genetically dysplastic or it is not. An affected animal can exhibit a wide range of phenotypes, all the way from normal to severely dysplastic and functionally crippled. Hip dysplasia is genetically inherited.

Conclusions from part II: While environmental effects, to include nutrition and exercise, may play a part in mitigating or delaying the onset of clinical signs and clinical symptoms, hip dysplasia remains a genetically transmitted disease. Only by rigorous genetic selection will the incidence rate be reduced. In the meantime, it makes sense to have lean puppies and to avoid breeding animals from litters that showed signs of hip dysplasia. It is probable that even normal exercise levels may increase the phenotypic expression of CHD of a genetically predisposed dog. Stay away from calcium supplementation of any kind; all it can do is hurt. There is no conclusive evidence that vitamin C can prevent hip dysplasia, but there is some evidence that vitamin C may be useful in reducing pain and inflammation in the dysplastic dog. Fortunately, large doses of vitamin C are readily excreted, but it is still possible to cause untoward side effects with megadoses.

Conclusions from part III: Canine hip dysplasia can be difficult to diagnose, as a number of other orthopedic neurological, autoimmune and metabolic problems may mimic it. Controversy surrounds the question of positioning for hip X-rays and what part joint laxity plays in hip dysplasia. Hip dysplasia may be more common in large and giant breeds and is one of the most over-diagnosed and misdiagnosed conditions.

Conclusions from part IV: Sadly, no breed registry in the United States requires genetic screening of parents as a prerequisite for litter registration or even offers a "fitness for

breeding" certification. The current registries for hip dysplasia (and other genetically transmitted problems) cover so little of the American Kennel Club-registered dog population that their impact so far has been minimal. The tools we need are there. Joint responsibility for failing to use the tools at hand lies with the AKC, United Kennel Club, parent clubs and individual breeders.

Conclusions from part V: The two major methods of diagnosing canine hip dysplasia available to the fancy in the United States are those followed by OFA and those followed by PennHIP. Both are diagnostic; however, the hip-extended protocol followed by OFA may produce false-negative results. The protocol followed by PennHIP has a prognostic or predictive capacity through the use of statistics and a carefully guarded data base that allows a prediction to be made with respect to the probability of phenotypic expression of canine hip dysplasia. No one has a clear quantification of the gray area between obviously clear and obviously dysplastic hips.

This article will address the long-term medical management of canine hip dysplasia, the goals of which are to relieve pain, restore function and hopefully mitigate or delay the progression of the disease. There are philosophical choices to be made based on the psyche and the general approach to life of the individual animal. Some breeds are noted stoics, able to tolerate what would appear to be a great deal of pain. For such animals, restoration of function is the greatest gift. For other animals more susceptible to pain, relief of that pain may be the greatest gift. Much of the philosophy of medical management of canine hip dysplasia must come from the animals themselves. The authors both are more experienced with Northern breeds (Akitas and Samoyeds), which tolerate pain well; however, our experience covers other breeds as well. The philosophy of treatment must come from multiple sources: from traditional medicine, holistic medicine, acupuncture and even chiropractic.

Caveat: Before starting medical treatment, surgical procedures may be necessary to correct anatomical malformations. Such surgical procedures will be addressed in the seventh and eighth articles in this series.

Chiropractic

William Inman, D.V.M., a clinician working in the Seattle area, is the reason we have included chiropractic. Inmanâs research, presented in the third article in this series suggests that spinal conditions, especially subluxation of between the eighth and tenth thoracic vertebrae, can cause a neurological condition that mimics the symptoms and signs of canine hip dysplasia. Inmanâs treatment method includes the traditional chiropractic "spinal adjustment," but with a twist. He has had numerous successes with a device called the "Activator." This instrument applies a small force very quickly on the affected spinal segment. Inman calls this technique "Veterinary Orthopedic Manipulation" and maintains that with this device he can "reactivate" the neurological pathway that has been compromised. The problem with discounting this whole process as being just a little too "New Age" lies in the number of apparent successes he has had.

Ask the owner of a paralyzed Dachshund and the 30 or more other people (including

respected dog breeders, veterinarians and chiropractors) who saw the Dachshund start walking after only one such treatment. This little dog had been through the veterinary process and its owner was preparing to put it down after traditional veterinary medicine had failed to relieve the pain or restore the function. In a last-ditch effort to help her dog the owner brought her to a seminar hosted by the Wenatchee (Wash.) Kennel Club on April 22. The results were those described above. The case is definitely anecdotal; however, Inman has too many such cases to be dismissed out of hand, even by those in the mainstream of veterinary medicine.

With a background in genetics, neuroanatomy and neuropathology, Inman still questions the mechanism of how his technique works. What is it that may be happening at the cellular level that promotes healing? Why is it that an animal has to be "readjusted" periodically on a specific schedule for the results to stick? "Once the body has been returned to normal neurologic function via adjustment, it stays in adjustment for about three days. Months to years of functioning out of adjustment impinge on this newly rehabilitated neurologic ability, and the spine slips back to its previous out-of-adjustment condition. This is why further adjustments are necessary . At three, seven, 14, 21, 42 and 70, the body falls out of adjustment·."[1] From the authorsâ perspective further research is clearly indicated, but meanwhile, this option of chiropractic is available to that segment of the dog population not suffering with the genetic disease of hip dysplasia, but from subluxation of the spine between the eighth and tenth vertebrae.

Acupuncture

While relatively few Western veterinarians are using acupuncture, Western medicine is beginning to respect its potential and to practice it. As with many things in life a full understanding of the process is unnecessary for employing it. In physics, for example, light and electricity are poorly understood, yet the modern way of life is predicated on the use of both light and electricity. Acupuncture survives from a time before modern science and physical mechanisms were described in philosophical terms that do not hold up to strict scientific examination, yet the phenomena exists. This appears to be the state of acupuncture in medicine and in veterinary medicine. A great body of anecdotal evidence exists to suggest that acupuncture has potential for at least temporarily reducing pain and promoting natural healing. Acupuncture has a following among not only dog people but horse people, and many are the accounts of lame animals being restored to full function. As with Inmanâs chiropractic example, acupuncture has too many apparent successes to be discounted without further study.

Drug Therapy

Because Hip Dysplasia results in abnormal forces being applied to the coxofemoral joint one of the most effective treatments is control of the dogâs weight. If indicated, even small amounts of weight loss are productive. Restricted activity also should be considered not only to avoid excessive wear on the affected joint, but to control transient inflammation. Even so, most of the pharmacological treatment alternatives function by reducing the inflammatory response. These drugs can be divided into corticosteroids, which can include but are not limited to methylprednisolone, dexamethasone and prednisone and a variety of NSAIDs

(non-steroidal anti-inflammatory drugs). Although useful in the acute stage, the corticosteroids are inappropriate for long-term treatment modalities due to their multiple undesirable side effects. Besides suppression of the immune system and loss of adrenal function, the use of corticosteroids can cause increased appetite increased thirst and gastrointestinal ulceration. Other research also indicates that corticosteroids can disrupt the articular cartilage matrix by inhibiting proteoglycan synthesis. [2,3] Proteoglycan is necessary for stiffness and compressibility of the matrix. Fortunately, this effect is reversible within two or three weeks. Experimenting to determine the right interval between injections may be necessary.

NSAIDs are not without their drawbacks, either. Common aspirin (acetylsalicyclic acid) can cause vomiting and bloody stools; bleeding times may be extended due to irreversible inhibition of platelet function, and severe overdose can lead to an abnormally high fever, electrolyte imbalance, renal hemorrhage, convulsions and coma. Clotting time returns to normal within several days, however, as a result of normal platelet turnover. There is some indication that though aspirin is often the drug of choice, it may possibly accelerate the degeneration of articular cartilage.

Another drug used to relieve the symptoms associated with hip dysplasia is phenylbutazone. This drug[4] has a potentially serious side effect in that it depresses bone marrow formation. Bone marrow is the site of red blood cell maturation. Not yet approved for dogs by the Food and Drug Administration is the promising anti-inflammatory drug carprofen. Clinical trials have shown it is a more effective anti-inflammatory than both aspirin and phenylbutazone, and when compared to placebo it is 24.8 times more efficacious. In a double-blind study of 209 dogs, it was therapeutic in relieving pain, lameness and contralateral (opposite-side) weight-bearing.[5] The drug also increased range of motion and reduced crepitus (the dry crackly sound when two dry articular surfaces rub together). An added benefit is that carprofen also seems to have a reduced potential for inducing gastrointestinal problems.

Caution: Of these drugs, only aspirin and phenylbutazone are FDA-approved for use in dogs.sup>6/sup>

"However, few NSAIDs are approved by the U.S. Food and Drug Administration for use in dogs, which has resulted in the empirical use of those approved in humans with sometimes disastrous results." In 1987 NSAID exposures comprised 8 percent of all human and veterinary medication calls to the Illinois Animal Poison Information Center.

"Many of these newer NSAIDs have a small margin of safety, due to long half lives and low rates of elimination."[7]

A few years ago dimethyl sulfoxide(DMSO), an industrial solvent, became a popular, though unapproved and unproven, treatment for arthritic joints. DMSO is a free-radical scavenger and is reported in the popular press to produce favorable results. Caution is advised.

Nutritional Therapy

The common mechanism for most of the anti-inflammatory drugs is inhibition of prostaglandin E_2 synthesis. Also referred to as the arachadonic "cascade," these drugs function by blocking the activity of the enzyme cyclooxygenase. What most people do not realize is that the antioxidant vitamins, d-alpha-tocopherals (the most biologically available form of vitamin E) and calcium ascorbate (a more effective form of vitamin C), also modulate PGE_2 synthesis by inhibiting cyclooxygenase and stabilizing the cell membrane. Even though dogs manufacture their own vitamin C, to be therapeutically effective the blood plasma concentrations of these two vitamins must be maintained at a higher than normal value. Therefor, the form of the vitamin is important, and the amount ingested is higher than that suggested by the Association of American Feed Control Officials. These nutritional supplements are not useful for acute symptoms, but if taken daily and consistently, they can be reduce inflammation without any detrimental side effects.

An added benefit of these two vitamins is that they scavenge free-radicals (highly reactive and unstable compounds generated in mammalian cells as a result of cellular metabolism and cell damage), and when taken together vitamin C can regenerate the "reduced" form of vitamin E so that it can be recycled by the cell. Free-radicals are formed also in the inflammation process and when the animal is exposed to various environmental pollutants, including ultraviolet light. Besides being implicated in arthritic disease process, free-radicals are associated with the onset of cancer, aging, cataracts, neurologic disorders and a reduced immune function. Edward A. Moser, M.S., V.M.D., suggests in his article in the November/December 1994 issue of *Veterinary Technician*, "For a thirty pound dog, giving approximately 80 I.U. of vitamin E [and] 50 mg of Vitamin C·can safely be recommended. Smaller dogs need proportionately less, larger dogs proportionately more."[8] Other sources would consider this a very conservative dosage. In a Norwegian study, 30 mgs/kg of body weight of polyascorbate was given three times a day for six months.[9] (A kilogram is 2.2 pounds)

Approximately 77 percent of the dogs treated showed marked improvement after six months, and 32 dogs out of the 45 diagnosed with hip dysplasia were symptom-free after only one week. Polyascorbate is a mineralized form of vitamin C that aids in the absorption and retention in the bodyâs tissues, and because it has a neutral pH it does not cause gastric upset. Ascorbic acid, the vitamin C we are most familiar with, is too rapidly excreted to be effective, can irritate the lining of the digestive tract, and at the higher dosage recommended will cause the formation of crystals in the urinary tract.

GAGS

The drugs and nutritional supplements mentioned so far either retard the breakdown of joint components or reduce pain and inflammation, thus improving the quality of life for the dog. None of them, except calcium ascorbate, are able to repair cartilage that has been compromised. While vitamin C is necessary for maintenance of collagen, it is also a carrier of activated sulfates needed for the synthesis of glycosaminoglycans (GAGS). An injectable

form of polysulfated glycosaminoglycan called Adequan is in the process of being approved for dogs by the FDA. Considered a chondro-protective drug, it is already available in Canada for dogs and licensed for horses here in the United States. Previous laboratory trials (in-vitro cell-line experiments) demonstrated its effectiveness in promoting the synthesis of cartilage matrix components. It also slows down the destruction of these cartilage components, decreases joint inflammation, restores the normal hyaluronic acid content in the synovial fluid (increases viscosity) and relieves pain.[10,11] Another study conducted at Cornell University has shown that PSGAG (polysulfated glycosaminoglycans), given prophylactically, are able to improve coxofemoral joint congruity in puppies prone to hip dysplasia.[12]

To understand how this product works, let us review a few pertinent facts about joint structure and the articular cartilage. Stress due to the abnormal biochemical forces in the dysplastic joint causes injury to the chondrocytes and the release of various destructive enzymes. Chondrocytes are responsible for the synthesis of collagen and proteoglycans, which constitute the ground substance (matrix) of articular cartilage. Acting somewhat like "glue," the matrix proteoglycans play an important role in the structural integrity of cartilage. A number of destructive enzymes have been isolated that break down joint matrix components; among these are the metalloproteinases. These enzymes break down proteins and depend upon the metal ions CA++ (calcium) and Zn++ (zinc) for their activity. Adequan is thought to function by inhibiting the activity of these metalloproteinases and other degenerative mechanisms, but a dual role has been suggested in that it may also act by stimulating the synthesis of proteoglycans and collagen by the chondrocytes.[13]

Both the femoral head and the acetabulum are covered with articular cartilage. The entire surface area is lubricated by synovial fluid, which is composed of and ultrafiltrate of plasma and glycosaminoglycan hyaluronic acid. Viscosity is the result of hyaluronic acid concentration, so anything that affects the concentration of HA also affects the lubricating potential of the synovial fluid. Synovial fluid, the source of nutrition for the articular cartilage, functions by eliminating metabolic waste products, and is contained by a fibrous structure called the joint capsule. The joint capsule itself is composed of an inner layer called the synovial membrane and another consisting of a fibrous outer covering. Thus most of the pathologic changes associated with hip dysplasia and subsequent degenerative joint disease can be attributed to the various chemical changes in the synovial fluid and the articular cartilage.

No toxic effects from the use of polysulfated glycosaminoglycans in dogs have been reported in the literature, but caution should be taken for use in those breeds with known blood coagulation problems such as von Willebrandâs disease (vWD) or hemophilia.[14] Furthermore, this drug should not be used in conjunction with other drugs that interfere with normal blood clotting mechanisms. Other studies have shown that it can inhibit the complement cascade (part of the secondary immune response), and suppress neutrophil activity.[15] Neutrophils are white blood cells that surround and digest foreign substances, including bacteria and viruses. So its use would be proscribed if the dog had an active infection, especially joint sepsis.

Two nutritional products are now being suggested for management of degenerative joint disease as possible alternatives or adjuncts to the drug Adequan, Glyco-Flex and Cosequin. These two products have the advantage of being administered orally, and so far the data supports their manufacturersâ claim that absorption readily occurs from the gastrointestinal tract. [16]

Glyco-Flex is a freeze-dried preparation of the New Zealand green-lipped mussel, *Perna canaliculus*, to which brewerâs yeast and alfalfa have been added to reduce the marine odor and increase palatibility. The end result is a complex mixture of proteins, mixed glycosaminoglycans, amino acids, chelated minerals, enzymes and vitamins. The activity of the *Perna* mussel is probably the effect of several ingredients working in combination.

Cosequin [17,18] is a patented nutraceutical sold only to vets which has numerous clinical studies currently under way at veterinary universities. The active ingredients in Cosequin are glucosamine HCL (hydrochloride), purified chondroitin sulfate and manganese ascorbate. Currently this product is being evaluated by veterinary orthopedic surgeons for use in dogs and the results are encouraging. Other studies are looking at Cosequinâs ability to stabilize articular surfaces of the joint and improve the jointsâ overall function.[19]

Physical Therapy and Exercise

Owner-conducted physical therapy is an indispensable component of treatment. Heat, followed by range of motion exercises, may provide temporary relief. Often favorable results are obtained by gently moving the affected joint through a full range of motion several times daily. This may prevent capsular contraction and its increased pressured on the articular cartilage. A variety of forms of heat are available., ranging from the unsophisticated heating pad to ultrasound and diathermy. Simplicity, availability and cost are considerations. A heating pad under the bedding is often appreciated as may be seen by the dog resting with the most affected hip placed over the heating pad. Where possible, refraining from weight-bearing on affected joints may help. Similarly, vertical load reduction on joints may help. Thus in some cases of CHD, the dog should be prevented from going up or down stairs, from jumping up or jumping down from a height.

Muscle atrophy can cause increased stress on the affected joint. Graduated exercise may be effective to correct this muscle imbalance so characteristic of CHD (overdeveloped shoulder girdle; weak hips). In any case, weight loss, even if it means a "lean and hungry" look in old age, often pays large dividends in quality of life for the animal. Simple measures such as bedding changes can make a difference. Many an older dog, which in younger days would refuse a bed, preferring instead hard concrete or linoleum floors, may accept and be helped by a piece of plush pile carpet or a pad of some kind.

Warning: medical management of a degenerative joint disease, such as canine hip dysplasia, is simply management, not cure. Both you and your animal have to learn to live with the condition and to adjust your lifestyles accordingly. In mild cases, especially of the insidious

form of CHD, little adjustment may be required, other than to precede bouts of increased activity with a "pre-dose" of aspirin. Be very careful that you do not fall in the trap that many human patients and their dogs fall into: When the pain is gone and the inflammation is reduced there is an extreme tendency to overdo it. The pain will come back to visit if the animal gives in to temptation to romp until it drops.

Conclusions: For many animals, canine hip dysplasia is a manageable condition, and they can lead relatively normal and active lives given that caution is exercised. Every dog is different in its response to pain, and the treatment protocol needs to be tailored specifically to the particular animal. *Only* aspirin and phenylbutazone ("bute") are FDA-approved drugs for use in dogs, and they are not without serious side effects. Corticosteroids are dangerous and may require experimenting to find proper dosage levels and intervals. Favorable results have been reported from chiropractic, physical, drug and nutritional therapy.

The final two articles in this series will cover surgical intervention in the management of canine hip dysplasia. Surgical measures are measures of last choice. We hope however, to make the case that surgery may be a viable choice, and even an economically sensible choice, especially for companion dogs for the elderly, assistance, drug-sniffing, search-and-rescue and other specially trained dogs where costs and time associated with training and replacement are high.

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NEXT SEGMENT