Canine Hip Dysplasia Part III

The authors assess the pros and cons of standard diagnostic methods for hip dysplasia

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This article is the third in an eight-part series on canine hip dysplasia (CHD).

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 either is 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.

This third article deals with the abnormal hip and how to diagnose it. Though CHD can afflict all breeds, it is more common in the large and giant breeds. There is far more to a proper diagnosis than first meets the eye. Anecdotal evidence has shown that canine hip dysplasia is one of the most over-diagnosed and misdiagnosed problems afflicting dogs. Many clinicians may depend too often on only subjective radiographic interpretation in the diagnosis of CHD. Physical examination techniques are helpful, and one can often pick up on concurrent conditions that could be otherwise overlooked. Initially, this article will focus on the clinical signs of hip dysplasia, the specific methods used by the experienced practitioner to make the diagnosis and the problems associated with the classic hips-extended, Orthopedic Foundation for Animals-approved X-ray positioning for radiographic study. The latter part of the article will be devoted to important new developments that hold promise for predicting the probability of phenotypic expression of CHD.

In the second article in the series, we said that canine hip dysplasia can be conveniently categorized into two major types. The first is severe and is seen early in the afflicted dog's life. The second, and far more common type, is the insidious chronic form that develops over a period of time. It is therefore useful to separate dogs by age classification when describing the clinical signs of hip dysplasia. A reasonable classification that takes into account maturity, puberty and attaining adult height, if not near adult weight, would be dogs less than one year in age and those more than one year in age. This gives time for atrophy and extraordinary musculature to develop as clinically recognizable signs. In the young dog, the first symptoms appear to be decreased activity, sometimes accompanied by joint pain.[1] If a young dog is found to have a swaying or unsteady gait, or runs with both hind legs moving together - often referred to by breeders as the "bunny hop" - it is worth further investigation. Acute

episodes of lameness with both or only one side affected can also occur after exercise or minor trauma. These signs can also be the result of infections in joints, lack of synovial fluid or the result of trauma. As CHD progresses, the dog may also have difficulty rising from a lying or sitting position and will frequently balk at going up or down stairs.

TYPE OF MOVEMENT	RANGE IN DEGREES
Flexion	From Neutral to 70 to 80
Extension	From Neutral to 80 to 90
Adduction	From Neutral to 30 to 40
Abduction	From Neutral to 70 to 80
Internal Rotation	From Neutral to 50 to 60
Internal to External	From Neutral to 80 to 90

Two clinical signs that most often appear together in the older dog are well-developed muscles in the forelimbs and shoulders due to shifting weight forward. As the disease progresses, hypertrophy (over-development) of the front end is accompanied by symmetrical or non-symmetrical atrophy of the pelvic muscles. Such animals appear weak in the pelvic region, are reluctant to exercise, generally prefer sitting to standing and exhibit extreme discomfort when their forelimbs are lifted off the ground.

RADIOGRAPHIC METHOD	SCORES						TYPE OF SCORING	TYPE OF SCALE
7 Point Scale(OFA)	Excellent	Good	Fair	Borderline	Mild-HD	Severe-HD	Subjective	Oridinal
3 Point Scale	Normal			Borderline	Dysplastic		Subjective	Oridinal
Norberg Angle (NA)	Tight hip > 105 degrees				Loose Hip <90 degrees		Quantitative	Interval
DJD Score	DJD Absent			NA	DJD Present		Subjective	Oridinal
Distraction Index	Index = 0 Tight Hip			NA	Index = 1 Loose Hip		Quantitative	Interval

Remember also that the affected dog may exhibit none of these symptoms. A substantial number of dogs with radiographic signs of hip dysplasia show no clinical signs of the disease. Explanations of this phenomenon are as varied as they are controversial. Quite a few practitioners believe that a dog

radiographically positive for hip dysplasia but clinically negative for signs is just a dog in an intermediate stage of the disease progression. This period may last for months, even years, until the onset of substantial degenerative joint disease. It is not uncommon for an afflicted (genetically predisposed) dog to die of old age before any non-radiographic signs develop.

We repeat again the warning issued in the preceding articles: You cannot tell if a dog is genetically predisposed to hip dysplasia by its movement. Reject the false wisdom of the old-time breeder who emphatically states that if his or her dogs had hip dysplasia he or she would be able to see it. Hip dysplasia is a polygenic, multifactorial disease.

Before a definitive diagnosis of CHD can be made, other problems must be ruled out.[3] Thorough medical, orthopedic and neurological examinations must be made in order to rule out other disorders of the hip and spine. Multiple joint involvement may be the case. The following is a condensed list of some of the more common conditions that mimic or may be concurrent with canine hip dysplasia:

- Physical disorders of the stifle-ruptured or torn cranial cruciate ligaments; luxating patellae; meniscus tears in the knee.
- Diseases of the joints-rheumatoid arthritis; metabolic bone disease; polyarthritis from Lyme and other infectious disease; panosteitis (bone inflammation).
- Nutritional bone disease-chronic subclinical scurvy.
- Spinal disorders-ruptured vertebral disease; degenerative spinal disease; lumbosacral instability.
- Neurological conditions-trauma; poisoning (lead, etc.); infections; neural lesions; proprioception (posture sense).

An example of another condition masquerading as hip dysplasia is the all-too-common spinal degenerative myelopathy in German Shepherd Dogs. After reading the preceding list, you should realize that CHD is not an easy condition to diagnose with great surety unless a full examination is conducted. If you do not find radiographic signs, that still does not preclude some of the problems mentioned above.

Dr. William Inman a clinician in Washington state feels that canine hip dysplasia is the most over-diagnosed and misdiagnosed condition in the veterinary medical practice.[4] While he feels that hip dysplasia is genetically predisposed, he remains puzzled by finding in his practice clinically dysplastic dogs with radiographically normal hips and symptom-free dogs with coxofemoral joints that look "like a bomb went off in them." Inman states, "Curiously, in all the young dogs we see with hip dysplasia signs in the 5 to 18-month range, we always find a subluxation at T8-T10 [dislocation of the Thoracic vertebra 8 through Thoracic vertebra 10]." This is a potentially important finding because the T8 to T10 area "innervates the peraspinal muscles and the iliopsas muscle, which attaches to the femoral head and pulls it forward. Subluxation leads to muscle spasming, which causes continued anterior traction of the femur on the hip socket, flattening the joint…reduction of this subluxation reverses the progression of hip dysplasia by curing the musculo-skeletal dysfunction." Inman has relieved the symptoms of more than 3,500 dogs with his procedure.

The conclusion that Inman has drawn from his practice is that the T8-T10 subluxation is a physical condition that, unless dealt with immediately, will progress to the joint capsular fibrosis and muscle stricture associated with decreased range of motion. The subsequent skeletal changes that follow can only be addressed surgically. He recommends early intervention in dogs thus afflicted to halt this insidious process.

Inman’s theory appears radical, but it is not contrary to the concepts previously presented. He does not maintain that a genetic disease is not associated with hip dysplasia, only that a misdiagnosed

physical condition mimics the disease process. Thus, the incidence of CHD may be lower than previously thought by other researchers.

Given that many other processes may be at play, the following are some of the physical techniques used in the diagnosis of CHD. While a tentative diagnosis can be made on the basis of history, clinical signs and the various palpation methods, standard veterinary practice requires radiographic signs of CHD. Diagnostic methods fall into two general categories: subjective and quantitative. We have found no method, subjective or quantitative, that is without its detractors or without serious controversy.

Subjective Methods of Diagnosis

Observation. The first step in the diagnosis of a suspected case of CHD is orthopedic examination, which should include observation of the dog at rest, walking, running and a re-examination of the dog the day following vigorous exercise.[5, 6] Observation and neurologic examination should be conducted before administering any drugs, and especially before sedation or general anesthesia, which can significantly alter the dog's neurologic status.

Range of motion. In an anesthetized dog, the coxofemoral joint's range of motion is approximately 110 degrees.[7] With pathology, this range of motion can be reduced to as little as 45 degrees. When following a chronic patient, the clinician uses changes in the range of motion to quantify the progress of the disease and as an aide when determining treatment options. Figure 1 is a table of the clinical categories by range of motion.

Changes in gait patterns. A shortened length of stride is associated with a loss in range of motion. There is a considerable variance among animals, but as a general rule, shortened stride length does not appear until fully extended movement is painful for the dog. This is the case with severe degenerative joint disease. Similarly, this type of gait abnormality can occur if the joint capsule has become fibrous. The many shapes and sizes of dogs make it impossible to describe all the potential gait changes. However, the bunny hop, left to right shift of the pelvis or an elliptical swing of the leg and hip are common gait problems encountered.

Forced extension. Affected dogs will not only exhibit discomfort with forced extension of the hip, but will try to return the limb to a more relaxed position. Depending on the temperament of these animals, they may also vocalize or exhibit aggressive behavior in response to pain. Be aware that the fighting dogs and the Northern breeds tend to have high pain tolerance levels and are generally stoic with respect to pain.

Downward pressure on the rear limb. When force is applied to the hips of a standing animal, the affected animal will show little or no resistance to the pressure, and will assume a sitting position. Several factors may simultaneously be involved and interrelated, such as pain, muscle weakness or atrophy.

Palpation. In humans, the most popular and reliable palpation maneuver used to identify congenital dislocation of the hip determines the presence or absence of the Ortolani sign. "A positive Ortolani sign confirms the diagnosis of coxofemoral subluxation in newborns prior to development of clinical signs or radiographic changes."[8] Many veterinarians feel that the techniques have too much subjectivity and variance to be of much use. Nonetheless, the Ortolani sign still figures prominently in the literature.[9-14] Animals to be examined must be anesthetized past the point where there is still a palpable response. Two basic approaches are used: dorsal recumbency and lateral recumbency, with dorsal recumbency being preferred for large dogs. Downward pressure is applied down the axis of the femur until the femoral head subluxates. The leg is slowly abducted while holding the stifle firmly. If the joint is loose, a distinct clicking may be felt and in some cases will be audible.

Other palpation methods have been proposed by Barlow and Bardens.[15,16] Barlow's Sign is essentially the first half of the Ortolani Test. Downward axial pressure is applied on the femur without abducting the leg. The Bardens' Test places the dog on its side, and the leg is held perpendicular to the spine. Lifting pressure is applied to the femoral shaft without abduction. The examiner's finger is placed on the greater trochanter. Any movement of the finger by more than one-fourth inch is considered a positive sign for a loose joint. Palpation has shown diagnostic use in human neonates, but is controversial and may have little diagnostic or prognostic utility in the dog. A caution: In human infants, it has been suggested that repetitive Barlow tests, and presumably Ortolani and Bardens as well, are capable of making infant hips unstable, thus giving a false-positive result.[17]

The Neurologic exam. During a normal physical examination, the clinician will observe both the posture and movement of the dog. Of the two observations (gait and posture), how the animal stands or its ability to return to a normal stance tells more about the neurological status. Some breeds have been selectively bred for a characteristic gait. Thus gaits may vary tremendously among breeds. A Borzoi moving as a Bulldog would be one sick Borzoi. A poor postural response may indicate a proprioceptive deficit.

Proprioception, or posture sense, is the ability to recognize the location of limbs in relation to the rest of the body without visual clues. An abnormally wide stance is one indication of a possible problem. The simplest method of evaluation is to bend the paw so the back of the foot is bearing the dog's weight. The normal response is to immediately reposition the paw correctly. A problem in proprioception positioning is often an early indication of neurological problems, and most often precedes motor dysfunction (gait anomalies).

When evaluating the dog specifically for hip dysplasia, one needs to rule out deficits in the spinal-reflex arc. An example of the spinal-reflex arc where the neural response is not transmitted to the brain but returned (arcs back) is the familiar tap on the knee with a rubber hammer. (The neural response travels from the muscle to the spine and returns to the muscle, without traveling to the brain.) The absence of an involuntary response or an exaggerated response are indications of neurologic problems. Some variance among breeds is noted, as large dog responses tend to be less rapid than those in smaller breeds.

Routinely, the "knee jerk" (quadricep reflex) is tested first with the normal reaction being a single quick extension of the stifle. Next, the flexor reflex is evaluated by gently pinching the toes. The normal dog should pull the entire limb (hip, stifle and hock) up toward the belly. Although not strictly analogous, the extension toe reflex has been compared to the Babinski reflex in humans. The examiner will hold the hock and gently stroke the back surface from the hock down toward the pad. The normal animal will either exhibit no response or a slight flexion of the toes. The abnormal reaction is the extension and spreading of the toes. These tests, by no means comprehensive or exhaustive, constitute the minimal examination to rule out spinal problems in a dog being evaluated for hip dysplasia.[18]

Subjective Diagnostic Radiographic Methods

Hip-extended radiographic method. This traditional X-ray position has been the standard position, which has the dog sedated, on its back, with legs fully extended and patella facing upward, became the standard of the American Veterinary Medical Association Panel on Hip Dysplasia in 1961, and was adopted by the Orthopedic Foundation for Animals in 1966. University of Pennsylvania studies have been conducted that show interpretations are not highly consistent among radiologists, and are not highly consistent when the same radiologist reads the same deck of X-rays in shuffled order.[19] OFA scores (excellent, good, fair, borderline, mild, moderate and severe) have wide acceptance but as subjective interpretations not readily repeatable with the same animal, nor likely to be interpreted

consistently by different radiologists. At first it appeared that the seven-point scale was more discrete than diagnostic protocol warranted. When the seven-point scale was collapsed to a three-point scale (normal, borderline, dysplastic) agreement improved. The hips-extended positioning has come under criticism because it masks joint laxity. This positioning masks joint laxity in two ways both involving the joint capsule. With the hip extended, the fibers of the joint capsule tighten in such a way as to push the femoral head into the acetabulum. This position also leads to a lowering of the intra-articular pressure, which combined with the fixed synovial fluid volume causes invagination of the joint capsule. These two conditions limit the amount of sideways movement of the femoral head. Similarly, unsedated positioning may further mask joint laxity.

Norberg Angle method. The Norberg Angle radiographic method of determining joint laxity (subluxation) has been used more in Europe than in the United States. The standard OFA hip-extended radiographic projection is used (see figure 3). Norberg angles typically range from 55 degrees to 115 degrees, with the smaller numbers representing looser hips. Unfortunately, there is no common agreement as to what constitutes a normal angle, though 105 degrees may be used as a point estimate for normal joint laxity. Correlation with OFA interpretations is poor, which is one reason the Norberg Angle method is not well accepted as a diagnostic tool and is considered subjective at this time.

Quantitative Diagnostic Radiographic Method

Compression/Distraction method. This new stress radiographic method originated at the University of Pennsylvania School of Veterinary Medicine and is currently marketed by PennHIP®. What started as a look at the role of passive hip laxity in CHD has become a quantitative diagnostic protocol referenced to an extensive data base. In recent years joint laxity has been established in the literature as prognostic for degenerative joint disease. Initially, however, little statistical evidence supported this contention. Now that a major data base has been developed for purposes of comparison and for determining probabilities, joint laxity can be used as an indirect variable with which to predict the probability of eventual phenotypic expression of CHD.

Unfortunately for breeders, deep sedation is required in the compression/distraction method. The traditional OFA positioning was found inadequate. In the stress radiographic method, the dog is laid on its back with its hips at a neutral flexion/extension angle. A compression view is taken with the femoral heads seated tightly in the acetabula congruency between the two joint surfaces. A second, or distraction, view is taken showing the maximum separation distance of the femoral head center from the acetabular center. A special device is used to force the femoral head away from the acetabulum for the distraction view. This protocol has been shown at University of Pennsylvania to reveal 2.5 times more joint laxity than the standard hip-extended radiograph.

The power of this method lies both in the new positions and in the statistical significance of the compression index (CI) and the distraction index (DI) as supported by a data base.[20] The indices range from 0 to 1, with "0 being a fully congruent hip (as seen in the compression radiographic view) and 1 representing the most extreme joint laxity as might be seen in the distraction view of hips that are virtually luxated."[21] The OFA scoring method is an ordinal scale, the Norberg Angle method is an interval scale and the DI is a ration scale. Thus the DI is intuitive in its meaning: A hip with a DI of 0.5 has twice the laxity of a hip with a DI of 0.25. Similarly a DI of 0.5 can be thought of as a hip 50 percent luxated. The DI ratio scale is far more useful a rating than the Norberg Angle. See figure 2 for a comparison of scales.

Breeders are always looking for earlier detection of CHD, the earlier the better for determining which animals to keep and classify as show and breeding hopefuls. Compression and distraction evaluations have been done on a sample of 8-week-old German Shepherd Dog puppies without the

results being conclusive. At 16 weeks, this method becomes useful. Dr. Gale Smith, et. al., at the University of Pennsylvania Hip Improvement Program (PennHIP) recommended that dogs not be evaluated before 16 weeks and that follow-up radiography should be done at 6months or 1 year of age.[22] In later articles in this series we will address the utility of the PennHIP protocol for prognosis.

Genetic (blood-based) diagnostic test. At this time, no biomechanical or metabolic differences have been identified in the dysplastic dog. Extensive work continues for an early blood marker for the condition. Finding such a marker would be ideal, as it would both allow the breeder to definitively screen breeding stock, and help the clinician identify appropriate treatment protocols. Parallel work is being done in determining genetic factors in humans for rheumatoid arthritis and osteoarthritis. Restriction Fragment Length Polymorphism (RFLP) linkage analysis has been used to identify genes associated with those diseases. Since there appears to be a strong genetic base for CHD, restriction fragments in the white blood cell DNA should correspond to the dysplastic phenotype.[23, 24]

Conclusions: Canine Hip Dysplasia can be difficult to diagnose. Other orthopedic, neurological, autoimmune/infection and metabolic problems may mimic CHD or may be concurrent with CHD. Numerous palpation techniques (Ortolani, Bardens, Barlow) have been proposed; however, they remain subjective nonquantitative methods that rely heavily on the skill of the clinician. The standard in current veterinary practice is to confirm CHD radiographically. The traditional American Veterinary Medical Association and Orthopedic Foundation for Animals hip-extended radiographic view distorts the amount of joint laxity present by forcing the femoral head deeper into the acetabular cup, thus understating the amount of laxity present. University of Pennsylvania (PennHIP) protocols for stress radiography are coming to the forefront as a more definitive way of visualizing hip joint laxity. Canine hip dysplasia remains a polygenic, multifactorial disease.

The next article in this series will discuss the various hip dysplasia registries, their approaches to the problems of canine hip dysplasia and the importance of having a "tamper-proof" identification system.

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