The pathogenesis and diagnosis of canine hip dysplasia: A review

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Abstract

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.

Animals must be 2 years old before they can be certified as normal, but the disease may be diagnosed earlier. Earlier diagnosis of the condition would be very useful for the selection of breeding stock, but palpation techniques and the standard extended view radiographs have unacceptably high rates of error in young puppies. Stress radiography techniques may improve the accuracy of early diagnosis in the future.

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Résumé

La pathogenèse et le diagnostic de la dysplasie de la hanche chez le chien : une étude

La dysplasie de la hanche est une maladie de croissance rencontrée chez le chien. Malgré des recherches approfondies, plusieurs questions demeurent sans réponse et plusieurs idées fausses persistent chez le grand public. Cet article a pour objectif de réviser les connaissances actuelles sur le développement de la dysplasie de la hanche, les facteurs modifiant sa progression et les moyens de diagnostic. Une étude de la documentation a été effectuée sur ordinateur à partir des bases de données de «MEDLINE» et de CAB sur «Silver Platter» de 1983 à 1995; les mots clés utilisés étaient : dysplasie de la hanche, hanche, chien, canin. D'autres articles publiés avant 1983, provenant, dans la mesure du possible, de recherches originales ont aussi été révisés. Les animaux qui présentent de la dysplasie de la hanche naissent avec des hanches normales, mais développent rapidement une subluxation de la tête fémorale. Une dégénérescence articulaire s'ensuit. La dysplasie de la hanche a un caractère héréditaire, polygénique et complexe.

La seule méthode recommandée pour diminuer l'incidence de la dysplasie de la hanche dans la population canine est d'effectuer des accouplements sélectifs à partir de chiens normaux provenant de portées dont les animaux ont des hanches normales et dont les parents et les grands-parents sont exempts de dysplasie.

L'expression génétique des sujets atteints peut être modifiée par plusieurs facteurs environnementaux. Ces facteurs ne causent pas la maladie, mais altèrent la manifestation de son caractère et de sa sévérité. La nutrition est un facteur environnemental considérable. L'ingestion d'un excès d'énergie alimentaire augmente l'incidence et la sévérité de la dysplasie chez les chiens prédisposés génétiquement. L'alimentation doit être ajustée de façon à maintenir une silhouette élancée. Les côtes et la colonne vertébrale dorsale doivent être palpables mais non visibles. Un excès de calcium et de vitamine D contribue à l'expression de la dysplasie chez les animaux prédisposés et devrait être évité. L'administration de supplément de vitamine C à forte dose, chez les animaux en croissance, ne prévient pas l'apparition de la dysplasie de la hanche et cette pratique devrait être abandonnée. Les animaux peuvent être certifiés exempts de dysplasie à l'âge minimal de deux ans. Toutefois, la maladie peut être diagnostiquée plus tôt. Un diagnostic précoce serait avantageux pour la sélection des chiens de reproduction, mais les techniques actuelles de palpation et la position radiologique normale avec les membres en extension donnent un taux trop élevé de résultats erronés chez les chiots. Les techniques radiologiques en

position de stress pourraient améliorer dans le futur l'efficacité du diagnostic précoce.

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Introduction

Canine hip dysplasia is a complex developmental, not congenital, abnormality. The term means abnormal formation of the hip joint (1). It is a biomechanical disease where hip instability in the young dog alters the concentration of forces on the growing femoral head and acetabulum. This affects bone growth and remodeling, resulting in abnormal joint conformation and secondary degenerative joint disease (2–7).

Hip dysplasia is a common problem in veterinary practice, accounting for up to 30% of orthopedic cases (8). The frequency of the disease varies among breeds from as high as 70.5% in bulldogs and 48.2% in St. Bernards to a low of 1.9% in borzois (9). Males and females are affected with equal frequency, in contrast to the disease in humans, where 80% of cases are female (1). Despite expensive screening and breeding programs, the disease continues to have a major economic and emotional impact on dog breeders and owners. While few diseases have been studied as intensively, the disease is complex, with many questions unanswered and numerous myths that continue to be propagated. This paper reviews the current knowledge on the development of the disease, factors modifying its development, and current diagnostic techniques. This review is based on a computerized literature search using the CAB and MEDLINE on Silver Platter (US National Library of Medicine, Boston, Massachusetts, USA) database from January 1983 to April 1995, using the keywords hip dysplasia, hip, dog, and canine. Selected articles, wherever possible original research articles, written prior to this date were also reviewed. It is hoped this will provide a current update for practitioners and serve as a source for client education.

Development of hip dysplasia

Pups genetically predisposed to hip dysplasia, unlike humans, are normal at birth (3,10–13). Stretching of the joint capsule and ligament of the femoral head is observed as early as 2 wk of age (3,12,14,15). Mild proliferative, nonsuppurative synovitis, edema, and fibroplasia of the ligament of the femoral head, as well as joint effusion, are present at 4 wk. By 12 wk, affected individuals have changes in both the synovium and the articular cartilage (16). Grossly, there is flaking and fissuring of the surface cartilage. Microscopically, surface chondrocytes are lost and changes in the matrix's proteoglycan content and collagen fibril network have occurred (3,12,13,16,17).

Joint effusion and progressive stretching of the joint capsule and ligament of the femoral head are associated with increasing joint laxity (3,5,16,17). This allows the femoral head to subluxate during weight bearing and changes the forces acting on the plastic, immature skeleton. Compressive weight-bearing forces are concentrated on the medial aspect of the femoral head and dorsal rim of the acetabulum, delaying their ossification (3,5,11). Less force is transmitted to the lateral aspect of the femoral head and ventromedial aspect of the acetabulum, thereby reducing remodeling and speeding ossification in these regions (4). A convex or flatter dorsal rim and shallower acetabulum (3,18) that result further compromise joint stability.

Abnormal weight bearing forces cause microfractures in the subchondral bone of the dorsal acetabular rim and femoral head. With healing, the bone becomes harder and less able to absorb shock. More force is transmitted to the overlying cartilage, increasing its degeneration at these sites (3). Cartilage on the medial aspect of the femoral head and dorsal acetabular rim is gradually worn away, exposing the subchondral bone (4,5). The subchondral bone becomes sclerotic and eburnated (3,11,13). Sharpey's fibers tear, causing osteophytes to form along the joint capsule's attachment to the acetabulum and femoral neck (3).

The cycle of degenerative joint disease and bone remodeling continues. Cartilage degeneration, joint capsule thickening, stretching or rupture of the ligament of the femoral head, proliferation of the dorsal acetabular rim, thickening of the femoral neck, and atrophy of local muscle characterize advanced hip dysplasia (12,13,17). At this point, joint stability may improve, or progress to complete luxation. The rate and degree of disease progression vary with the individual and the amount of joint instability present (11). Synovitis and articular cartilage degeneration and abrasions have been observed in the shoulder, stifle, elbow, vertebral, and mandibular joints of 30% of dogs genetically predisposed to hip dysplasia (1,17). The hip, because of its inherent instability, may just be the most significant site of a generalized condition (1).

The cause of hip dysplasia: genetics

Hip dysplasia is an inherited disease (3,10,14,19). Exceptions, like neonatal trauma, are so rare that they should be assumed not to exist (14, 20–22). Hip dysplasia is a polygenic trait caused by the interaction of hundreds of genes, each contributing a small part to the disease (12,23). At least 1 pair of these genes is believed to be recessive (14). It is an additive trait where the severity of an individual's disease is determined by the number of "affected" genes present (4). The genotype determines the genetic blueprint for the hip's shape, size, anatomical relationships, musculature, and innervation, and a program for its growth and remodeling (3,7).

The expression of these genes may be modified by a number of environmental factors. Environmental factors do not cause hip dysplasia (14), but they may determine whether the trait is manifested and to what degree (20). This means that an animal's phenotype does not necessarily equal its genotype. Two individuals with the same "dysplastic" genotype may have very different phenotypes (1 dysplastic, 1 normal) because of environmental differences (11).

Heritability is an estimate of the extent genes are influenced by the environment (14,22). A heritability index of 1.0 means that the occurrence of a trait is controlled entirely by the presence or absence of genes and that environment does not play a role. An index of 0.0 means the trait is not genetically influenced. Heritability estimates for hip dysplasia vary from 0.2 to 0.6, with most falling between 0.4 and 0.5 (4,14,23,24).

Estimates differ because the value is influenced by the breed, the population studied, the degree of inbreeding, and environmental factors, like diet, feeding regime, and exercise (23,25). Selection pressure has decreased heritability estimates over the last 10 y (4). Heritability may also be linked to different phases of the disease in different breeds. Heritability is closely linked to joint laxity in golden retrievers, in contrast to German shepherds, where it is more closely linked to the degree of degenerative changes that develop (26).

While the genetics of hip dysplasia is complex, selective breeding has been valuable in reducing the frequency within the general population. When a large number of dogs were studied, 85% of the offspring were dysplastic if both parents had hip dysplasia, compared with 52% if only 1 parent was dysplastic, and 37.5% if both parents were normal (18). Breeding successive generations of normal dogs in a closed colony of German shepherds decreased the incidence from 39% to 17% over 3.5 years (15). There is a wide variation in the ability of sires to transmit normal hips to offspring (10,15,27). Any degree of hip dysplasia, including mild, increases the frequency in the offspring (10). Current breeding recommendations are to breed only normal dogs whose parents and grandparents are normal. At least 75% of the parents' littermates should be normal. The sire should have a good record of producing normal offspring. Replacement bitches should have better conformation than their parents (14). Littermates can be used to measure each other's genotype and to reflect the parents' genotypes, especially if they are raised in different environments. Progeny testing is slow and expensive, but necessary for continuing to reduce the frequency of hip dysplasia within the population. It is recommended that sires not be extensively bred until at least 18 puppies from more than 3 litters have been evaluated (4).

Factors affecting the development of hip dysplasia

Joint laxity

Joint laxity is accepted as a significant factor in the pathogenesis of hip dysplasia. It is present before the characteristic remodeling and degenerative changes take place (1,4,15,19,21,28). The disease can be prevented by maintaining joint congruency until 6 mo of age, when the skeleton is less plastic and the supporting structures are strong enough to prevent subluxation (3,12,14,15). Genetically predisposed puppies did not develop the disease when confined to small cages that forced them to remain sitting (15,18). Subluxation caused by cutting the internal and external obturator and gemelli muscles resulted in classical hip dysplasia (18).

Despite this evidence, joint laxity remains a controversial issue. It can be argued that all joints in an immature dog are more lax than in a mature dog (29). One problem is in determining what is normal "puppy" joint laxity and how much laxity is sufficient to initiate the remodeling and cartilage changes associated with hip dysplasia. Severe joint laxity typically results in development of hip dysplasia, and stable hip joints usually develop normally. What happens in between these extremes is less clear (9). In addition, cases of dogs with severe joint laxity developing normal hip conformation and ones with tight hips developing degenerative joint disease have been observed. It is well accepted that joint laxity increases the environmental stress on the joint and significantly increases the frequency and severity of hip dysplasia, but other environmental factors must modify the influence of joint laxity on hip development (9). The specific cause of the joint laxity remains unknown, but postnatal trauma, hormonal imbalances, and neuromuscular immaturity have all been speculated on (1,13,30). It has been postulated that genetics plays an important role in the degree of hip joint laxity present in an individual, but this remains to be proven (21).

Body type

A number of trends regarding body type have been noted in dysplastic dogs. In general, the smallest breeds have a low frequency, while larger breeds have a higher frequency of hip dysplasia (15). It tends to be less common in breeds with tight skin, prominent full-bellied muscles, and little fat in the skin, subcutaneous tissue, and fascia. Those with the highest frequency have loose skin, a heavy, rounded, stocky conformation, less developed muscles, and more than 5% to 10% subcutaneous fat (15). Affected dogs have a narrower pelvis than normal dogs of the same breed. A wide, flat pelvic conformation seems to tolerate more hip joint laxity than does a narrow, sloping one (31).

The prevalence of hip dysplasia has been reported to correlate with pelvic muscle mass. Pelvic muscle mass indices (PMMI = total postmortem mass of pelvic muscle [kg]/live weight [kg] $\times$ 100%) correctly predicted hip dysplasia 94% of the time. The disease was not observed if the index was greater than 12.0 and was almost always present if the index was below 9.0 (32). While adequate conditioning between 3 and 6 mo of age may help to prevent hip dysplasia (13) and pelvic muscles may atrophy with disease (30), these factors did not significantly alter the index. It was thought to be genetically determined (12,18,32).

Dysplastic dogs tend to be those that grow and gain weight rapidly (15). The hip muscles may not develop sufficient relative strength to prevent subluxation during weight bearing (3,11,12,15,16,18,33). Neuromuscular immaturity has been speculated on as a cause for the delayed development of muscle strength, but this remains unproven (7,12).

The biomechanical, environmental, and genetic factors associated with a certain body conformation and size should be considered significant factors in the development of hip dysplasia (15). They are likely interrelated and linked to joint instability and its progression to abnormal bone remodeling and cartilage degeneration (12).

Nutrition and rapid growth

Nutrition is a major environmental factor influencing the development of hip dysplasia. It may change the frequency and severity in genetically predisposed individuals, but it does not cause hip dysplasia. No dietary deficiencies are known to influence the development of hip dysplasia, but current research suggests that dietary excesses are important contributing factors (5,8).

Variations in dietary protein and carbohydrate levels do not affect the development of hip dysplasia

(8,34,35). There is no difference in plasma amino acid concentrations between normal and dysplastic dogs (36). As long as sufficient protein and amino acids for growth are supplied in the diet, the actual amounts are less important (8).

Young dogs do not have a protective mechanism against excess dietary calcium. High dietary levels increase the amount of calcium absorbed from the gastrointestinal tract (37). High calcium decreases osteoclastic activity, delaying endochondral ossification and skeletal remodeling (1,5,15,38–40). The absolute amount of calcium rather than the calcium:phosphorus ratio is more important (35,37). Since vitamin D increases intestinal calcium absorption and renal resorption, excess vitamin D has an effect similar to that of excess calcium (5). Excess dietary calcium and vitamin D may contribute to the development of hip dysplasia in genetically predisposed individuals and should be avoided in young, rapidly growing dogs.

Vitamin C is necessary for collagen synthesis, but dogs do not have a dietary requirement, as they synthesize sufficient amounts. Feeding high doses of vitamin C to pregnant bitches and their offspring until 2 y of age was reported to eliminate hip dysplasia (41), but lack of radiographic evaluation and follow-up, and inability to reproduce the results in controlled clinical trials (42), make these results questionable. Other studies show that excess vitamin C in puppies causes hypercalcemia and may delay bone remodeling and cartilage maturation (8,43). There is no scientific evidence that supplementing the diet of growing puppies with high doses of vitamin C prevents hip dysplasia. As this practice is potentially harmful, it should be discontinued.

A higher frequency of hip dysplasia is associated with rapid growth rates and relative overloading of the skeleton (3,8,11,16,18,30). Excess energy consumption, whether in the form of fat, protein, or carbohydrate, in immature animals will, within genetic limits, increase skeletal growth and body weight compared with those fed normal or restricted amounts (2,5,8,10,11,22,23,28,44). This increases the frequency and severity of hip dysplasia in genetically predisposed dogs. "Overnutrition" is most critical in the first 6 mo of life (10), with the more rapidly growing individuals of a breed at the highest risk (8,15,30).

Matched litters of pups genetically predisposed to hip dysplasia were used to study the effect of overfeeding a balanced diet. Dogs with unlimited food access showed rapid growth and weight gain that correlated with the severity of hip dysplasia at 2 y of age. Limiting food intake to 75% of what these dogs ate in 15 min significantly reduced body weight, decreased hip laxity, and reduced by 38% the incidence of animals classified as dysplastic according to the Orthopedic Foundation for Animals (OFA) (20).

Overeating a balanced ration may result in excess energy and calcium, as well as other nutrients. Many commercial diets are extremely palatable and should not be fed free choice to large, rapidly growing breeds. Most dogs eat at least enough food to meet their caloric requirements. Other nutrients in the diet are balanced to the caloric density to ensure that these nutrient requirements are met. Quantities of all nutrients per 1000 kcal of metabolizable energy are generally higher in adult diets than in growth diets (45). Therefore, recommending that a large breed dog be fed an adult diet does not ensure a significant decrease in consumption of nutrients that contribute to skeletal disease (45).

Current recommendations for large, growing dogs are to feed 15 g of protein, 0.7 g of calcium, and 30 IU of vitamin D per 1000 kJ of metabolizable energy (5). Food intake should be regulated to maintain a slender figure with the ribs and dorsal vertebral spines easily palpable, but not visible. Dogs fed for rapid growth that develop normal hip conformation may have a better genotype than those fed restricted amounts. Some suggest that feeding of potential breeding dogs for rapid growth may allow easier detection of genotypes suitable for breeding (30).

Dietary anion gap

Synovial fluid volume has been implicated in the pathogenesis of hip dysplasia through its effect on joint laxity. When normal synovial fluid volumes are present, displacement of the femur creates negative intraarticular pressure that tends to pull the femoral head back into the acetabulum. This mechanism is lost when joint effusion is present (19). Dysplastic dogs had higher synovial fluid osmolalities than did normal dogs, due to differences in synovial fluid electrolyte concentrations of sodium, potassium, and chloride (28). Maintaining a dietary anion gap (DAG [meq/100g of food] = $Na^+ + K^+ - Cl^-$) below 20 meq/100g of food in an otherwise nutritionally balanced diet resulted in less subluxation at 30 wk of age and better hip scores at 2 y of age (28). Modification of the dietary anion gap may decrease synovial fluid volume, thereby improving joint stability and minimizing hip dysplasia. Further studies are needed to determine if the differences in synovial osmolality are primary or secondary changes with hip dysplasia, and to evaluate the effect of modification of the dietary anion gap on systemic acid-base status, skeletal mineral content, and bone growth (5).

Exercise

Exercise has not been shown to specifically contribute to the development of hip dysplasia, but it has not been studied as intensively as nutrition (46). Intuitively, if hip dysplasia is a biomechanical disease caused by overstressing the immature skeleton, exercise may speed the development of degenerative changes in dogs with hip instability. Exercise may have a protective effect against overnutrition by decreasing the amount of energy available for growth (47). It may also improve muscle strength. Further research is needed to answer these questions.

Hormonal influences

A number of hormones, including estrogen, relaxin, growth hormone, parathyroid hormone, and insulin, have been investigated as potential causes or contributing factors in hip dysplasia. Bitches in season may demonstrate joint laxity not present during anestrus (14). Abnormal estrogen metabolism in humans causes joint laxity (12), and estrogen given to puppies can induce hip dysplasia (30), but estrogen levels in dysplastic pups are not higher than in normal pups (15). Relaxin levels are increased in postpartum bitches with

hip dysplasia, and relaxin given to pups can influence the development of hip dysplasia (1,15). There is, however, no evidence that estrogen, relaxin, or any other hormones play a role when present in normal biological amounts (15,30).

Diagnosis

The clinical presentation of dogs with hip dysplasia is very variable. Some dogs are normal, while others are very lame. The amount of pain and associated clinical signs do not correlate with the changes in joint morphology (4,30,48). Progression of the disease also varies. A history of a stilted or abnormal gait that worsens with exercise or causes difficulties in jumping or sitting is common. In general, there are 2 ages at which animals present with clinical hip dysplasia: dogs younger than 1 y of age with pain caused by microfractures and older than 12 to 16 mo with chronic pain from degenerative joint disease (48). On examination, joint manipulation causes pain, especially during extension or abduction and internal rotation. Pelvic muscle atrophy, restricted range of motion, subluxation, and crepitus are also common findings. The clinical diagnosis is confirmed by radiographs.

Other joints should be examined and a complete neurological examination should be performed to localize the lameness. In young dogs, multiple joint abnormalities, like concurrent elbow dysplasia, are not uncommon. It is important to remember that many older dogs have subclinical hip dysplasia, so care should be taken to ensure that other common conditions, like degenerative myelopathy or cranial cruciate ligament rupture, are not missed.

The diagnosis is usually straightforward in animals clinically affected with hip dysplasia. Animals must be 2 y old before they can be certified by the OFA to have normal hip conformation. Breeders often select breeding stock at an early age, so veterinarians are faced with the challenge of trying to determine which individuals will develop the disease. The 2 methods currently used for early screening of hip dysplasia are palpation and radiography. Both techniques have an unacceptable level of error when used in 8- to 12-week-old puppies. Research is ongoing to develop techniques with improved accuracy.

Palpation

When young dogs are palpated for hip dysplasia, the examiner is trying to determine if significant joint laxity is present. The Ortolani sign (49) is performed by placing the dog in dorsal recumbency with the examiner standing behind the dog facing the pelvis (Figure 1). The stifles are grasped with the palm of each hand over the patella. The hip joint is placed in a standing angle with the femurs perpendicular to the long axis of the pelvis. Pressure is applied gently, but firmly, down the femoral shaft. This subluxates the femoral head when excessive joint laxity is present. The femurs are slowly abducted, maintaining the downward pressure, until an audible or palpable click, associated with reseating the femoral head within the acetabulum, is detected. This is a positive Ortolani sign. It indicates joint laxity and is considered to be an early indicator of hip dysplasia. The test can be performed in awake dogs but is not to be considered to be negative unless the patient is anesthetized. False negatives are also observed with improper technique, in large patients, and in chronic disease, where marked capsular fibrosis, destruction of the dorsal acetabular rim, or complete femoral head luxation is present (48,49). As a result, a positive Ortolani sign indicates significant joint laxity, but a negative sign does not mean that the hips are normal.

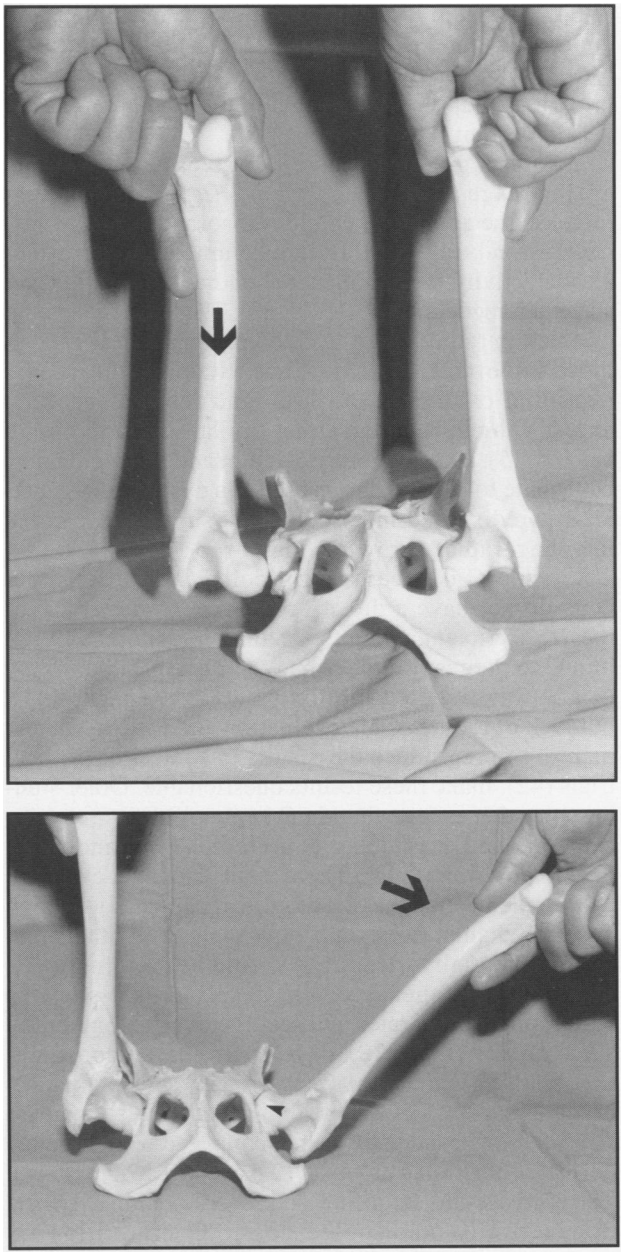

Figure 1. The Ortolani sign is performed with the dog in dorsal recumbency and the examiner standing behind the dog facing the pelvis.

A) The stifles are grasped with the palm of each hand over the patella. The hip joint is placed in a standing angle with the femurs perpendicular to the long axis of the pelvis. Pressure is applied down the femoral shaft (arrow). This subluxates the femoral head when excessive joint laxity is present.

B) The femurs are slowly adducted (arrow), maintaining the downward pressure, until an audible or palpable click, associated with reseating the femoral head within the acetabulum, is detected (arrowhead). This is a positive Ortolani sign.

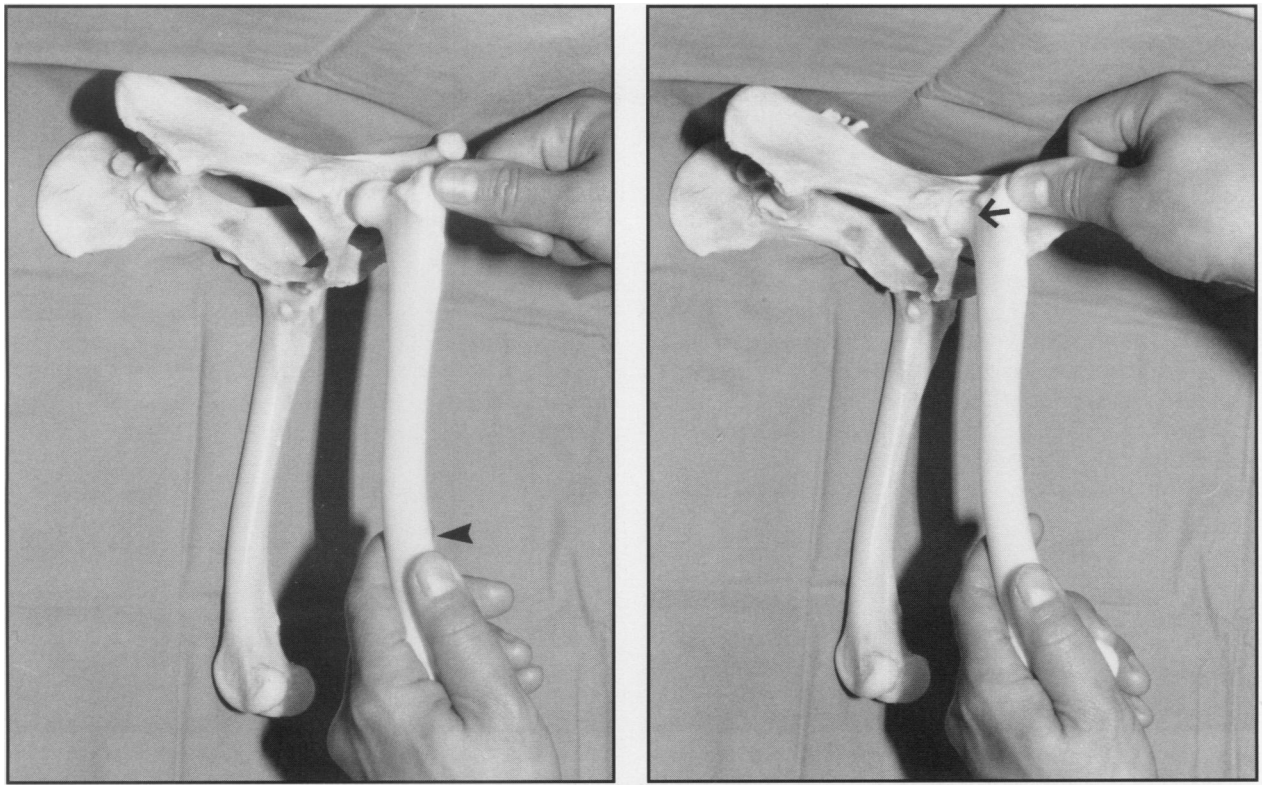

Figure 2. The Barden's test is performed with the patient in lateral recumbency.
A) The examiner grasps the upper femur in the left hand, so that the fingers lie along the medial aspect of the femoral shaft. Upward pressure is applied by these fingers to elevate the femur horizontally (arrowhead).
B) The right index finger or thumb is placed on the greater trochanter and alternately applies downward pressure (arrow) to determine if the femoral head can be "bounced" in and out of the acetabulum.

Barden's hip lift is another test to evaluate joint laxity (49,50). With the patient in lateral recumbency, the examiner grasps the upper femur in the left hand, so that the fingers lie along the medial aspect of the femoral shaft (Figure 2). Upward pressure is applied by these fingers to elevate the femur horizontally. The right index finger or thumb is placed on the greater trochanter and alternately applies downward pressure to determine if the femoral head can be "bounced" in and out of the acetabulum. A positive test is diagnostic of significant joint laxity, but a negative test is of little diagnostic significance. In Barden's original work, lateral displacement of greater than 1 mm was considered diagnostic for early hip dysplasia (50), but other researchers failed to repeat these findings and palpation is considered as a subjective test (17,30).

The diagnostic challenge with puppies is to determine the significance of the laxity present, as most dogs under 1 y have some degree of measurable laxity (51). The accuracy of predicting hip dysplasia with these techniques varies with the patient's age. It is poor at 4 to 8 wk of age but steadily improves by 6 mo of age. It also improves with the examiner's experience and when combined with radiographs, but it is never 100% (4,11,21,30,49). Most, but not all, dogs with extreme joint laxity will develop hip dysplasia, and most, but not all, with tight joints will develop normal hips (13,17,30,49,51). Palpation is best used as part of a diagnostic workup and should not be used alone as a criterion for the diagnosis of hip dysplasia (11). It may be prudent to warn owners of young dogs with significant laxity on palpation that their dogs are at risk for hip dysplasia, even if radiographic changes are absent.

Radiography

The standard radiographic position involves placing the dog in dorsal recumbency with the rear limbs extended parallel to each other and the spine. The stifles are adducted and internally rotated. Care is taken to ensure that the pelvis is not rotated (1,52). The paws should be 10 to 12 cm off the table in large breed dogs to reduce the pull of the soft tissues and prevent masking of subluxation (52,53). If the hip conformation is normal, the center of each femoral head lies medial to the cranial edge of the acetabulum with more than 50% of the head shadowed by the dorsal acetabular rim (1) (Figure 3A). The joint is considered dysplastic if the femoral head conforms poorly to the acetabulum, the joint space is increased or subluxation is present, structural abnormalities are detected in the femoral head or acetabulum, or osteophytes are present (17) (Figure 3B). Unilateral hip dysplasia has a frequency of 3% to 30%, depending on the breed; it is generally considered to be genetic, not traumatic, in origin (14).

The need for anesthesia or heavy sedation for radiography is controversial. It facilitates accurate positioning (4), but is unnecessary in many cases (54). Muscle relaxation accompanying anesthesia may affect the degree of subluxation (4,17,26,51), although this has been disputed (55). Dogs that subluxate under general anesthesia, but not when awake, will likely show conclusive evidence of hip dysplasia at later evaluations (51).

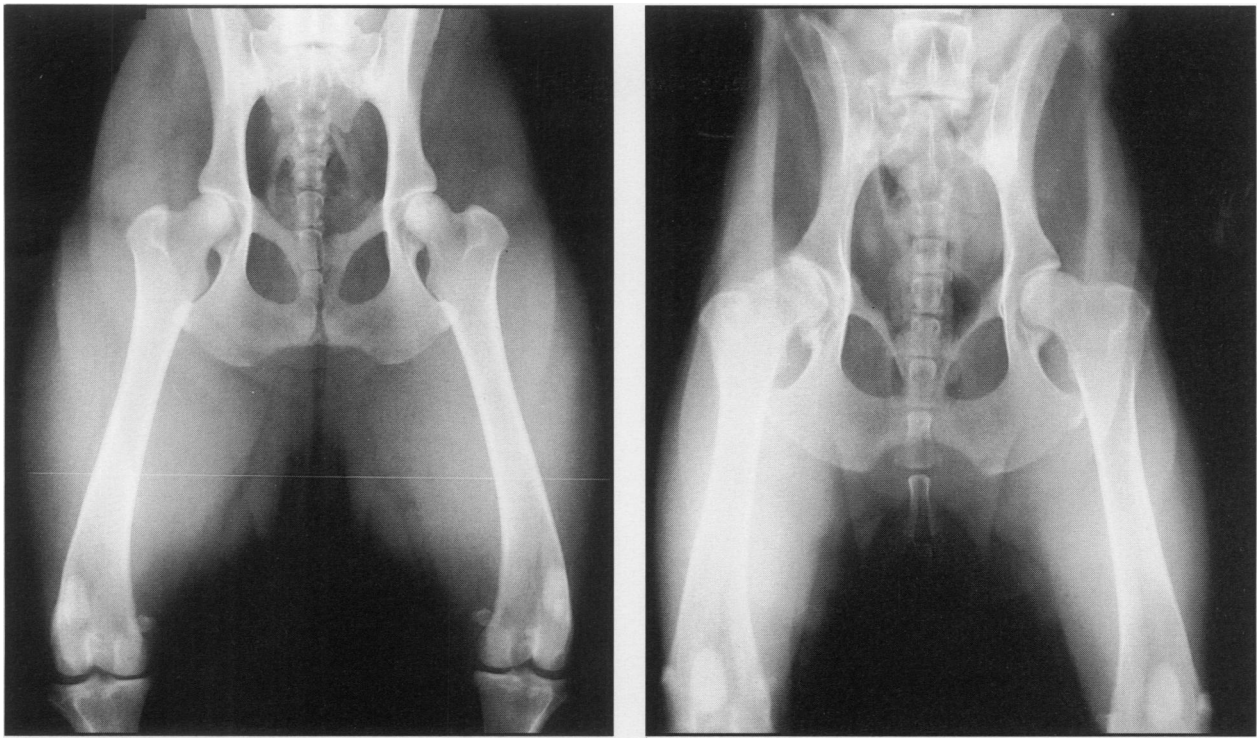

Figure 3.
A) If the hip conformation is normal, the center of each femoral head lies medial to the cranial edge of the acetabulum with more than 50% of the head shadowed by the dorsal acetabular rim.
B) The joint is considered dysplastic if the femoral head conforms poorly to the acetabulum, the joint space is increased or subluxation is present, structural abnormalities are detected in the femoral head or acetabulum, or osteophytes are present.

The reliability of radiographs in diagnosing hip dysplasia improves with age. Accuracy at 24 mo is 85% to 95% (11,51,54). It is less accurate at younger ages, although recently, the OFA reported a 89% reliability in preliminary evaluations at 4 to 23 mo of age (51).

Stress radiography

The conventional extended view has been criticized because the position tightens the joint capsule, the ligament of the femoral head, and associated muscles. This is thought to lessen the accuracy for early detection of joint laxity (1). Attempts to detect and quantitate joint laxity have been reported (19,29). One technique uses 2 views taken with the dog in dorsal recumbency with the hips at a neutral flexion/extension angle: a compression view with the femoral heads maximally seated in the acetabula, and a distraction view with the femoral heads maximally displaced laterally (19). The 2 views are used to calculate a distraction index to quantitate the amount of joint laxity present. The procedure is aimed at detecting hip dysplasia at an earlier stage than is possible with current radiographic schemes. Preliminary studies report an 88% accuracy in predicting normal hips in 4-month-old pups, but only a 57% accuracy in determining dysplastic individuals (12% false negatives and 48% false positives) (21). This technique revealed an average of 2.5 times more joint laxity than is apparent on the standard hip extended view (56). Four- to 8-month-old pups from parents with normal hip conformation had significantly lower distraction indexes than those from dysplastic parents, so the amount of joint laxity may have a genetic basis (21). Early heritability estimates calculated for German shepherds and Labrador retrievers were 0.61 and 0.45, respectively (56). A highly significant difference was found between the mean distraction index for borzois, a breed with a low incidence of hip dysplasia, and German shepherds, a breed with a much higher incidence of hip dysplasia (19). Evaluation of this technique is continuing in a large multi-institutional study.

This stress radiography technique is based on the premise that joint laxity is the earliest sign of hip dysplasia. Joint laxity is necessary, but not sufficient, for the development of hip dysplasia, as not all dogs with significant joint laxity develop degenerative joint disease (57). It is hypothesized that dogs with increased laxity that do not develop degenerative joint disease may be carriers for hip dysplasia (56). Currently, studies are also being undertaken to determine the heritability of the distraction index for different breeds affected by hip dysplasia, and its effectiveness as a screening test in breeding programs.

Other diagnostic tests

The accuracy of ultrasonography is being evaluated for early diagnosis of hip dysplasia (58). In addition, some work has been done to find a marker for hip dysplasia that could be measured in young dogs. Procollagen type III aminoterminal peptide is a molecule liberated into extracellular fluid during active capsular fibrosis. Serum levels increase in humans with active arthritis. While levels were higher in the synovial fluid of dysplastic dogs, serum levels did not become abnormal until a positive Ortolani sign was detected (59), so its

usefulness is limited. Isolation of a genetic marker for the disease is being attempted (44).

In conclusion, hip dysplasia is a complex developmental condition commonly affecting large breed dogs. It is a hereditary disease, but the genetics is not simple. Hip conformation is determined by the number of affected genes present and a number of environmental factors influencing their expression. The frequency of hip dysplasia can be reduced by breeding dogs with normal hip conformation over successive generations. Feeding a good-quality diet, so that puppies grow slowly and do not become overweight, lessens the incidence and severity of hip dysplasia. Current diagnostic techniques are most accurate in later stages of the disease. Potential breeding dogs may be screened at 6 to 18 mo by palpation and radiographs, but errors will occur. Newer diagnostic imaging techniques and blood screening may allow more accurate early diagnosis in the future. CVJ

References

1. Lust G. Other orthopedic diseases: hip dysplasia in dogs. In: Slatter D, ed. Textbook of Small Animal Surgery, 2nd ed., vol 2. Philadelphia: WB Saunders, 1993: 1938–1944.
2. Dammrich K. Relationship between nutrition and bone growth in large and giant dogs. J Nutr 1991; 121: S114–S121.
3. Alexander JW. The pathogenesis of canine hip dysplasia. Vet Clin North Am Small Anim Pract 1992; 22: 503–511.
4. Brass W. Hip dysplasia in dogs. J Small Anim Pract 1989; 30: 166–170.
5. Hazewinkel HA. Skeletal diseases. In: Willis JM, Simpson KW, eds. The Waltham Book of Clinical Nutrition of the Dog and Cat. Frome, England: Butler and Tanner, 1994: 395–423.
6. Weigel JP, Wasserman JF. Biomechanics of the normal and abnormal hip joint. Vet Clin North Am Small Anim Pract 1992; 22: 513–528.
7. Frost HM. Pathogenesis of congenital hip dysplasia (CDH): A proposal. Vet Comp Orthop Trauma 1989; 1: 1–10.
8. Richardson DC. The role of nutrition in canine hip dysplasia. Vet Clin North Am Small Anim Pract 1992; 22: 529–540.
9. Corley EA, Keller GG. Hip Dysplasia: A Progress Report and Update. Columbia, Missouri: Orthopedic Foundation of Animals 1993 (suppl).
10. Hedhammar A, Olsson SE, Andersson SA, *et al.* Canine hip dysplasia: study of heritability in 401 litters of German shepherd dogs. J Am Vet Med Assoc 1979; 174: 1012–1016.
11. Fox SM, Burns J, Burt J. The dysplastic hip: a crippling problem in dogs. Vet Med 1987; 82: 684–693.
12. Shepherd J. Canine hip dysplasia: Aetiology, pathogenesis and eradication. Aust Vet Pract 1986; 16: 71–78.
13. Morgan SJ. The pathology of canine hip dysplasia. Vet Clin North Am Small Anim Pract 1992; 22: 541–550.
14. Corley EA, Keller GG. Hip Dysplasia: A Guide for Dog Breeders and Owners, 2nd ed. Columbia, Missouri: Orthopedic Foundation of Animals, 1989.
15. Riser WH, Rhodes WH, Newton CD. Hip dysplasia. In: Newton CD, Nunamaker DM, eds. Textbook of Small Animal Orthopedics. Philadelphia: JB Lippincott, 1985: 953–980.
16. Lust G, Summers BA. Early, asymptomatic stage of degenerative joint disease in canine hip joints. Am J Vet Res 1981; 42: 1849–1855.
17. Lust G, Rendano VT, Summers BA. Canine hip dysplasia: concepts and diagnosis. J Am Vet Med Assoc 1985; 187: 638–640.
18. Riser WH. A half century of canine hip dysplasia. Semin Vet Med Surg (Small Anim) 1987; 2: 87–91.
19. Smith GK, Biery DN, Gregor TP. New concepts of coxofemoral joint stability and the development of a clinical stress-radiographic method for quantitating hip joint laxity in the dog. J Am Vet Med Assoc 1990; 196: 59–70.
20. Kealy RD, Olsson SE, Monti KL, *et al.* Effects of limited food consumption on the incidence of hip dyplasia in growing dogs. J Am Vet Med Assoc 1992; 201: 857–863.
21. Lust G, Williams AJ, Burton-Wurster N, *et al.* Joint laxity and its association with hip dysplasia in Labrador retrievers. Am J Vet Res 1993; 54: 1990–1999.
22. Lanting F. Canine hip dysplasia and the breeder: a layman's point of view. Vet Clin North Am Small Anim Pract 1992; 22: 739–743.
23. SA Mackenzie, Oltenacu EA, Leighton W. Heritability estimate for temperament scores in German shepherd dogs and its genetic correlation with hip dysplasia. Behav Genet 1985; 15: 475–482.
24. Lingaas F, Klemetsdal G. Breeding values and genetic trend for hip dysplasia in the Norwegian Golden Retriever population. J Anim Breed Genet 1990; 107: 437–443.
25. Mackenzie SA. Canine hip dysplasia: why heritability estimates differ. Canine Pract 1985; 12: 19–22.
26. Lavelle RB. Advances in hip dysplasia schemes in the dog. Vet Ann 1991; 31: 154–158.
27. Rendano VT, Ryan G. Canine hip dysplasia evaluation: A positioning and labeling guide for radiographs to be submitted to the orthopedic foundation for animals. Vet Radiol 1985; 26: 170–186.
28. Kealy RD, Lawler DF, Monti KL, *et al.* Effects of dietary electrolyte balance on subluxation of the femoral head in growing dogs. Am J Vet Res 1993; 54: 555–562.
29. Belkoff SM, Padgett G, Soutas-Little RW. Development of a device to measure canine coxofemoral joint laxity. Vet Comp Orthop Trauma 1989; 1: 31–36.
30. Wallace LJ. Canine hip dysplasia: past and present. Semin Vet Med Surg (Small Anim) 1987; 2: 92–106.
31. Olsson SE. Canine hip dysplasia: Pathology. Vet Comp Orthop Trauma 1993; 6: 61.
32. Riser WH, Shirer JF. Correlation between canine hip dysplasia and pelvic muscle mass: a study of 95 dogs. Am J Vet Res 1967; 28: 769–777.
33. Lust G, Craig PH, Ross GE, Geary JC. Studies on pectineus muscles in canine hip dysplasia. Cornell Vet 1972; 62: 628–645.
34. Nap RC, Hazelwinkel HA, Voorhout G, *et al.* The influence of dietary protein content on growth in giant breed dogs. Vet Comp Orthop Trauma 1993; 6: 1–8.
35. Nap RC, Hazewinkel HA, Voorhout G, Van den Brom WE, Goedegebuure SA, Van't Klooster AT. Growth and development in Great Dane pups fed different levels of protein intake. J Nutr 1991; 121: S107–S113.

36. Hansen I. Plasma amino acids in dogs with osteochondrosis or hip dysplasia. Acta Vet Scand 1989; 30: 111–112.
37. Hazewinkel HA, Van den Brom WE, Van't Klooster AT, Voorhout G, Van Wees A. Calcium metabolism in Great Danes fed diets with various calcium and phosphorus levels. J Nutr 1991; 121: S99–S106.
38. Hazewinkel HA. Calcium metabolism and skeletal development in dogs. In: Burger IH, Rivers JP, eds. Nutrition of the Dog and Cat. Cambridge: Cambridge Univ Pr, 1989: 293–302.
39. Hazelwinkel HA, Goedegebuure SA, Poulos PW, Wolvekamp W. Influences of chronic calcium excess on the skeletal development of growing Great Danes. Am Anim Hosp Assoc 1985; 21: 377–391.
40. Madsen JS, Reimann I, Svalastoga E. Delayed ossification of the femoral head in dogs with hip dysplasia. J Small Anim Pract 1991; 32: 351–354.
41. Belfield WO. Chronic subclinical scurvey and canine hip dysplasia. Vet Med Small Anim Clin 1976; 74: 1399–1401.
42. Teare JA, Krook L, Kallfelz FA. Ascorbic acid deficiency and hypertrophic osteodystrophy in the dog. Cornell Vet 1979; 69: 384–401.
43. Bennett D. Hip dysplasia and ascorbate therapy: fact or fancy? Semin Vet Med Surg (Small Anim) 1987; 2: 152–157.
44. Lust G. Studies on a genetic marker for canine hip dysplasia. Vet Comp Orthop Trauma 1993; 6: 62.
45. Remillard RL. Practical nutritional and dietary recommendations: minimizing clinical aspects of orthopedic diseases. Proc Western Vet Conf 1995: 25–28.
46. Lust G, Geary JC, Sheffy BE. Development of hip dysplasia. Am J Vet Res 1973; 34: 87–91.
47. Lavelle RB. The effects of the overfeeding of a complete balanced commercial diet to a group of growing Great Danes. In: Burger IH, Rivers JP, eds. Nutrition of the Dog and Cat. Cambridge: Cambridge Univ Pr, 1989: 303–315.
48. Fry TR, Clark DM. Canine hip dysplasia: clinical signs and physical diagnosis. Vet Clin North Am Small Anim Pract 1992; 22: 551–558.
49. Chalman JA, Butler HC. Coxofemoral joint laxity and the Ortolani sign. J Am Anim Hosp Assoc 1985; 21: 671–676.
50. Bardens JW, Hardwick H. New observations on the diagnosis and cause of hip dysplasia. Vet Med Small Anim Clin 1968; 63: 238–245.
51. Corley EA. Role of the orthopedic foundation for animals in the control of canine hip dysplasia. Vet Clin North Am Small Anim Pract 1992; 22: 579–593.
52. Henry GA. Radiographic development of canine hip dysplasia. Vet Clin North Am Small Anim Pract 1992; 22: 559–578.
53. Burns J, Fox SM, Burt J. Diagnostic radiography: the only definitive determination of CHD. Vet Med 1987; 82: 694–700.
54. Farrow CS, Back RT. Radiographic evaluation of nonanesthetized and nonsedated dogs for hip dysplasia. J Am Vet Med Assoc 1989; 194: 524–526.
55. Aronson E, Kraus KH, Smith J. The effect of anesthesia on the radiographic appearance of the coxofemoral joints. Vet Radiol 1991; 32: 2–5.
56. Smith GK. Current concepts in diagnosing coxofemoral joint instability. Proc Annu Meet Am Coll Vet Surg 1994: 59–64.
57. Smith GK, Gregor TP, Rhodes HW, Biery DN. Coxofemoral joint laxity from distraction radiography and its contemporaneous and prospective correlation with laxity, subjective score and evidence of degenerative joint disease from conventional hip-extended radiography in dogs. Am J Vet Res 1993; 54: 1021–1042.
58. Greshake RJ, Ackerman N. Ultrasound evaluation of the coxofemoral joints of the canine neonate. Vet Radiol Ultrasound 1993; 34: 99–104.
59. Madsen JS, Jensen LT, Strom H, Horslev-Petersen K, Svalastoga E. Procollagen type-III aminoterminal peptide in serum and synovial fluid of dogs with hip dysplasia and coxarthrosis. Am J Vet Res 1990; 51: 1544–1546.

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CVMA Conventions/ Congrès de l'ACMV

1996

Charlottetown, Prince Edward Island, July/Juillet 3–6

SEPTEMBER/SEPTEMBRE 1995

World Veterinary Congress 1995. September 3–9, 1995, at the Pacifico Yokohama, in Yokohama, Japan. Theme: Advancing the Veterinary Profession in a Changing World. Contact: World Veterinary Congress Secretariat, c/o Sankei Convention, Sankei Building 10F, 1-7-2, Otemachi, Chiyoda-ku, Tokyo 100, Japan; tel.: 81-3-3273-2084; fax: 81-3-3273-2439.

7th International Conference on Human-Animal Interactions. September 6–9, 1995, in Geneva, Switzerland. Theme: Animals, Health and Quality of Life. Includes the Delta Society (USA) 14th Annual Conference. Contact: Delta Society, P.O. Box 1080, Renton, Washington 98057-9606, USA; tel.: (206) 226-7357, fax: (206) 235-1076.

Canadian Embryo Transfer Association Annual General Meeting and Scientific Conference. September 10–13, 1995, at the Hotel Radisson Gouverneurs in Quebec City. Contact: Dr. M.S. Mills, Secretary-Manager, Canadian Embryo Transfer Association, P.O. Box 2000, Kemptville, Ontario K0G 1J0; tel.: (613) 258-5944; fax: (613) 258-3719.

American Holistic Veterinary Medical Association 12th Annual Conference. September 16–19, 1995, in Snowmass, Colorado. Contact: American Holistic Veterinary Medical Association, 2214 Old Emmorton Road, Bel Air, Maryland 21015, USA; tel.: (410) 569-0795; fax: (410) 515-7774.

21st Annual International Veterinary Acupuncture Congress. September 21–23, 1995, in Snowmass, Colorado. Contact: David H. Jaggar, Executive Secretary, International Veterinary Acupuncture Society, 1750-1 30th Street, Box 142, Boulder, Colorado 80301, USA; tel.: (303) 449-7936; fax: (303) 449-8312.

British Columbia Veterinary Medical Association Conference and Annual General Meeting. September 23–24, 1995, in Harrison Hot Springs, British Columbia. Contact: British Columbia Medical Association, 1200 West 73rd Street, Suite 155, Vancouver, British Columbia V6P 6G5; tel.: (604) 266-3441; fax: (604) 266-8447.

International Conference on the Care and Use of Fish, Amphibians and Reptiles in Research. September 28–29, 1995, in Toronto, Ontario. Contact: Canadian Council on Animal Care, 315-350 Albert Street, Ottawa, Ontario K1R 1B1; tel.: (613) 238-4031; fax: (613) 238-2837; e-mail: CCAC@Carleton.CA.

Fourth World Veterinary Dental Congress. September 28–October 1, 1995, at the Hotel Vancouver in Vancouver, British Columbia. The program includes basic and intermediate-level scientific lectures for veterinarians who are developing their dentistry skills, plus hands-on laboratory courses. Contact: Dr. Ed Eisner, tel.: (303) 757-8481; fax: (206) 542-2101; e-mail: Dog 2thdoc@aol.com.