The Dysplastic Hip Joint: Its Radiographic and Histologic Development

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INTRODUCTION

Canine hip dysplasia continues to be a complex and important problem to both the dog fancy and the veterinary profession. Theories regarding cause, heritability, treatment and control vary widely. The pathogenesis of hip dysplasia is the subject of this study.

Hip dysplasia is not well understood. To make this report meaningful, however, a number of observations that seem valid have been accepted. "The balance of the present evidence shows that a genetic predisposition to congenital dislocation of the hip is real, and that multiple genes are involved. Environmental factors also are important, presumably according to the genetic susceptibility of the individual" (15). Few genes so far analyzed affect the osseous skeleton primarily. The changes in bone merely reflect changes that occur in the cartilage, supporting connective tissue and muscles (2). Hip dysplasia is an example of a biomechanical disease representing a disparity between primary muscle mass and disproportionately rapid skeletal growth. Hip dysplasia begins with a normal hip at birth (6, 12, 17). The lag or failure of the muscle to develop and reach functional maturity at the same rate as the skeleton results in joint instability (3). Abnormal development is induced when the acetabulum and femoral head pull apart and trigger a series of events that end in hip dysplasia and degenerative joint disease (16). The evidence is strong that the bony changes of hip dysplasia occur because the soft tissues do not have sufficient strength to maintain congruity between the articular surfaces of the femoral head and the acetabulum (3).

This study follows the preceding line of reasoning and is a description of the radiographic and anatomicopathological changes that bring about hip dysplasia. In essence the pathogenic sequence of this disease as it occurs spontaneously and recorded in this paper is an experiment done by nature and the description of the development of the normal hip serves as the control (11).

LITERATURE REVIEW

Descriptions of the radiographic and histologic serial changes associated with hip dysplasia were not found. A committee from the American Veterinary Medical Association described the radiographic appearance of the normal hip and the varying degrees of dysplasia in the mature dog in 1961 (19). This remains the most complete of any description to date.

The radiographic appearance of the disease was defined and adopted by the Orthopedic Foundation for Animals Symposium Workshop Panel in 1972 (7). Limited descriptions of the histologic appearance were found in three reports (4, 5, 10). No reports were found of the path-
ologic changes encountered from the normal hip at birth through the breakdown of the hip joint in the various stages of dysplasia.

MATERIALS AND METHODS

Four German Shepherd Dogs born to parents with well-defined hip dysplasia were radiographed weekly from birth to maturity using methods described previously (13). At birth they were subjected to short caudectomies to prevent the tails from interfering with the radiographic image of the pelvis.

All four dogs became markedly dysplastic. Weekly radiographic tracings of the hip joint of one dog (Fig. 1) are representative of the lesions in all four dogs.

To visualize the neonatal hip, 87 neonatal dogs were euthanized and the hips dissected. Of these, 79 were purebred German Shepherd Dogs and 8 were cross-bred Golden Retriever-German Shepherd Dogs. Seventy were from birth to 10 days of age, 7 were 2 to 3 weeks of age, and 10 were 1 month old.

Coronal histologic sections were made of the hips of more than 300 dogs at all ages from birth to advanced age. The dogs illustrated in this paper are either the German Shepherd Dog or July Foxhounds.

RESULTS

Birth to Thirty Days of Age

Eighty-seven dogs from birth to 30 days were dissected. Their appearance was compared to normal Greyhound dogs of the same age (11). Eighty-four of these appeared normal. In three that were 30 days of age, the teres ligaments of the hip joints were edematous, a few ligament fibers were torn and capillary hemorrhage dotted the surface of the ligaments at the point of the tears. These changes were considered the first changes that might be linked to hip dysplasia.

From the dissection of the hips of these young dogs, it appeared that the teres ligament is largely responsible for holding the femoral head in place for the first month. For the first two weeks the teres ligament is so short that the femoral head attachment fractures at the fovea when luxation of the femoral head is forced. After the first two weeks, the teres ligament begins to lengthen very slowly. After the first four weeks the femoral head may be subluxated laterally 1 to 2 mm. In the normal adult dog the ligament is lengthened sufficiently to permit femoral head subluxation to the edge of the acetabular rim after the muscles are removed.

Thirty to Sixty Days of Age (1–2 months)

Radiographically the first signs of hip dysplasia were femoral head subluxation and a lag in the development of the craniodorsal acetabular rim. These were noted by the seventh week (Fig. 1). At this time subluxation could be recognized by palpation. Grossly the joint capsule was stretched, but the tissue of the capsule had not changed in appearance. The teres ligament was also longer but did not appear to be stretched. Histologically the contour of the acetabulum was greater than the femoral head and the craniodorsal rim was no longer sharp.

Sixty to Ninety Days of Age (2–3 months)

Radiographically during this period changes were dramatic in the German Shepherd Dog. Femoral head subluxation increased. The head no longer fit deeply into the acetabulum. The lag of ossification at the craniodorsal acetabular rim was increased (Fig. 1).

In German Shepherd Dogs and July Foxhounds of comparable age and degree of radiographic subluxation, when anesthetized, the femoral head could be subluxated an estimated 0.5 cm. When the hips of these dogs were dissected and the muscles were removed, the joint capsules were slightly thickened and stretched to a width
which allowed the femoral heads to be extended laterally to the acetabular rims. When the joint capsules were opened, 1) the dorsal acetabular rim was rounded from birth to 35 weeks of age.

Fig. 1  Drawings copied from overlay tracings of hip joint radiographs of a dog during growth and development of hip dysplasia from birth to 35 weeks of age. The numbers with each drawing indicate the age of the dog in weeks.
Progressive changes were recognized as the dog became older. Radiographically by 11 to 12 weeks the greater trochanter was bent medially in the subluxated hip (Figs. 1, 3). Full significance of the medial bend was not realized until a profile view was seen of a coronal histologic section of the hip (Figs. 3, 4). When the dysplastic leg luxated laterally, the femoral head rested on the dorsal rim of the acetabulum. This lateral displacement of the femoral head placed extra or unbalanced medial pull on the greater trochanter through the attachment of the powerful middle gluteal muscle to its dorsal tuberosity and by the deep and superficial gluteal muscles inserted on the lateral side of the same structure slightly distally (Figs. 3, 5). The abnormal pull bent the trochanter medially.

Although the entire greater trochanter is cartilage at 12 weeks, histologically the cells and the matrix of the trochanter and the adjoining physis were normal and unaffected by the medial bending. In some instances, however, the rows of columnar cartilage of the physis were longer on the lateral side, thus making the physis wedge-shaped. Such changes in the columnar cartilage are known as "cartilaginous drift" accomplished by "fatigue bending" (1).

The dysplastic hip joint becomes unstable as the acetabulum and femoral head pull away from each other. The dysplastic joint, the same size as the normal one, changes its shape and loses its grace and balance (Figs. 3, 4, and 6). The congruous union between the femoral head and acetabulum is disrupted and the contacting area that forms an arc on the femoral head is narrowed from a normal of $90^\circ$-$20^\circ$. The narrowed arc causes the compression forces to increase sharply on both the femoral head and acetabular rim (Fig. 4) (8).

The excessive compression and overloading at the acetabular rim stretches the plastic cartilage. Once it stretches beyond its fatigue limit, it takes a permanent set and the contour of the acetabular articular surface changes from concave to convex leaving the acetabular rim rounded as observed grossly (Figs. 2-4, and 6).

Since all but the acetabular rim is bone and not subject to plastic bending, the change in compression lines or stresses produces a new set of stress signals which in turn add new bone beneath the perios-
teum on the dorsal acetabular articular surface and stimulate the resorption of bone on the ventral surface of the acetabular cavity. The quantity and type of the new bone added or resorbed depend upon the stress signals (Wolff's Law) (1).

The epiphysis of the femoral head changes its position on the physis. This change in position is called "drift" and is accomplished either by plastic bending of the cartilage of the physis or by adding and resorbing bone in the metaphysis. There is drift and lipping of the epiphysis, bending of the physis, and a microfracture through the physis and damage to the adjoining tissue (Figs. 3–5). In this instance, a microfracture through the physis accompanied a shift in the position of the cartilage columns (Figs. 3–5). The position of the femoral head upon the physis immediately undergoes a change in order that the long axis of the enchondral cartilage columns remains parallel to the lines of forces and physis itself remains parallel to the contacting or supporting surfaces of the acetabulum. This type of drift accomplishes plastic bending and a permanent set of the hyaline cartilage. Such plastic drift can be demonstrated histologically (Fig. 3).

The second method of changing shape is by osseous drift of the neck. The compression forces are concentrated in a small area on the dorsal articular surface of the acetabulum and are transmitted to a small area on the femoral head. The compression signals caused changes in bone deposition and resorption that caused the head and neck to bend downward (Figs. 3–5). Since the cantilever arm forming the head and neck is composed of bone, it changed shape by adding new bone on one side and resoring it on the other. Such change in shape is known as "osseous drift" (1). The osteoblastic activity on the dorsal or lateral surface and osteoclastic activity on the medial surface were intense at this time in a remodelling attempt to correct the exaggerated varus angle of the cantilever neck (Fig. 3).

The direction of the physis also changed to remain parallel with the compression surface of the acetabulum (Figs. 3, 4, 6, and 8). A physis is subject to injury and possible fracture if the compression forces are not applied parallel to the long axis of the enchondral columns (Fig. 3) (18).

**Twelve to Twenty Weeks of Age (3–5 months)**

Radiographically the subluxation of the femoral head, incongruity of the two joint surfaces, lag in development of the acetabular rim, and change in shape of the joint components continued to be the salient features of the dysplastic joint (Fig. 1). Grossly, rounding or rolling back of the acetabular rim became more prominent between the 10 and 2 o'clock positions (Figs. 2, 6). Histologically, the increased localization of the compression forces in a restricted spot on the articular surface of the acetabular rim disturbed the developmental harmony of the area and fostered retardation of tissue maturation (Figs. 3, 5, and 6) (9). The effects of abnormal or increased compression is demonstrated as the dorsal articular surface of the acetabular cup is forced out of shape and the contour changes from concave to convex (Figs. 2–4, and 6). At this time the congruity between femoral head and acetabulum is disrupted and there is interference with the normal cycle of hip development. This is in proportion to the degree of malposition or subluxation (Figs. 2–4, and 6).

**Twenty to Thirty-Five Weeks of Age (5–9 months)**

The overloaded area on the dorsal acetabulum succumbed to wear, cartilage fibrillation, chondromalacia, and finally microfractures of the rim (Figs. 7–9). The articulating surfaces stripped of cartilage became eburnated (Fig. 9). The exposed subchondral bone assumed an ivory-like appearance as it condensed and became osteosclerotic, smooth and highly polished. A corresponding area of eburnation oc-
Fig. 3 A coronal histological section of the hip joint (E center illustration) of a 12-week-old dog with early dysplasia. In life, the femoral head of this dog was subluxated 6 to 8 mm. This subluxation caused an incongruous contact between the acetabulum and the femoral head and stretched the joint capsule and teres ligament. The contour of the acetabular cavity is widened (1). The greater trochanter is bent medially (2). The femoral head has drifted ventrally to a varus position (3). A section of the articular cartilage on the femoral head (arrows) is narrowed by wear from overloading forces of the acetabular rim (B). The dorsal region on the femoral neck is being re-enforced by new fiber bone (Box A), which is being laid down adjacent to the lamellar bone (right) (enlargement A). The rim (Box B) is rounded as the result of the delay of chondrogenesis and osteogenesis. Overloading forces on
Vol. XIV

The Dysplastic Hip Joint

The joint capsule in progressive hip dysplasia undergoes remarkable changes. The capsule of the normal joint is pale, semi-transparent and 1 to 2 mm in thickness. The capsule of the dysplastic hip is thickened, stretched and traumatized from the stress and pull of the unstable, subluxated femoral head. The thickening of the capsule, which may measure up to 5 to 7 mm, was in response to the trauma caused by excessive pull from subluxation (Fig. 6). New bone formed in the capsule along the dorsal capsular border. In the dysplastic joint the synovial fluid lost its adhesiveness, its specific gravity decreased, and the leucocyte count increased. Its ability to function as a lubricant was decreased (20).

The acetabular rim (Box B) are delaying the conversion of fibrocartilage to hyaline cartilage and then to bone (enlargement B). Varus drift of the femoral neck and epiphysis is accelerating the remodelling of the ventral neck area (Box C). Osteoclasts are resorbing bone at a rapid rate (enlargement C). The epiphysis has drifted distomedially an estimated distance of 2 mm. This has resulted in epiphyseal lipping at the ventral margin. A fracture through the physis cartilage occurred as a result of the shift (Box D, enlargement D). The physis has been repositioned to remain parallel to the dorsal contact of the acetabulum and the direction of columns of cartilage cells has been altered to be parallel to the load forces.
Fig. 7. Photograph of a mildly displastic acetabulum after the femoral head had been removed. There is rounding of the dorsal and caudal margins of the acetabular rim between 11 and 5 o'clock (long arrows). The teres ligament (A) has been pulled away from the dome of the fossa and new fiber bone has developed in the margin where the ligament was pulled away (B).

The thickened joint capsule restricted the range of hip motion. In the normal hip the craniocaudal movement is 110° in the anesthetized dog and 180° after the muscles have been removed in the euthanized dog. Motion in the dysplastic hip may be restricted to a range of only 45° in the anesthetized or euthanized dog.

As the subluxation becomes greater the teres ligament continued to stretch and become detached from the dome of the acetabular fossa (Fig. 7). Individual ligament fibers were ruptured as they were pulled away and became edematous. Swelling of the ligament was so great that it prevented the return of the femoral head to its normal position in the acetabulum (Figs. 6-8). The ligament was completely destroyed and the entire acetabular fossa filled with fiber bone (Figs. 8, 9, 13–15).

Radiographically at this stage in severe dysplasia a shallow acetabulum is observed. The shallowness has resulted from the filling in of the acetabular fossa with new fiber bone and from the fracturing, bending back and wear of the acetabular rim. These events take away the deep contours of the acetabular cup and are the result of excess and unbalanced mechanical pressures (Fig. 13).

There is marginal lipping of the femoral head with a rim of newly formed fiber bone encircling the neck at the junction of the head and neck (Fig. 10). It should be emphasized that the first bony changes of hip dysplasia appeared in the acetabulum and that the changes in the femoral head followed and were not as pronounced as those in the acetabulum. Later in advanced hip dysplasia, however, the changes in the femoral head were sometimes as pronounced as those in the acetabulum. This was the result of progressive remodelling and degenerative joint disease stimulated by joint instability.

Nine Months of Age and After

Bone and joint tissues were never static, whether functioning normally or pathologically. In the dysplastic hip, progressive remodelling occurred at an accelerated rate. The entire shape of the hip was restructured. Femoral head subluxation contributed to mechanical imbalance of the hip. Abnormal compression, traction and tension forces created stresses that caused new bone to be laid down in one area and resorbed in another. At this stage of abnormal structure it was difficult to differentiate clearly the changes representing true hip dysplasia and those representing beginning secondary hip degenerative joint disease (Figs. 10, 13, 16, and 17).

Comparing a macerated hip joint of a dysplastic and normal dog illustrates the effects of remodelling. In the normal joint, the ridges, grooves and indentations of the pelvis and femur are smooth and the rim of the acetabulum is sharp (Fig. 11). The articular surface of the acetabulum is concave. In the macerated pelvis the acetabular fossa is smooth when the teres ligament is removed and the bone at
the depths of the fossa is very thin (Fig. 11).

In the dysplastic hip, wear, exostoses and distortion of bony surfaces were present in varying degrees. The effects of abnormal or unbalanced stresses dominated the structure. The surface of the wing of the ilium was roughened to a greater degree than normal where Sharpey’s fibers of the gluteal muscles were torn away from their attachment to the periosteal surface. In the ischium, bone surfaces were roughened and grooved from attrition of bone resulting from prolonged irritation to the periosteum from unbalanced, excessive forces and pull from the obturator internus muscle (Fig. 12). Exostoses were especially prominent in some instances over the synovial bursa at the sciatic notch. The external and internal surfaces of the pubic bones were raised, grooved and roughened. The apophysis for the attachment of the iliopsoas muscle on the lesser trochanter of the femur was abnormally large and roughened (Fig. 5). These changes are scars resulting from abnormal stresses on the unbalanced hip.

On the articular surface of the acetabulum in the severely dysplastic hip, the scars of the previous fracture line on the polished eburnated acetabular surface remained visible (Figs. 8, 9, and 13-16). This line persisted throughout life in dogs that had lived a normal life span with dysplastic hips (Figs. 16, 17). The dorsal or periosteal surface above the acetabular rim was roughened by mounds of osteophytes. The formation of these exostoses was triggered when subluxation caused unusual stress to the supporting tissues. The roughened osseous surface of osteophytes in many instances circled the entire acetabulum. The contour of the eburnated surface of the acetabular cup was continuous across the fiber bone which filled the fossa. The surface of that fiber bone filled the fossa but never assumed the smooth dense texture of the eburnated surface (Figs. 13, 17). Occasionally the fossa was only partially filled with new fiber bone (Figs. 7, 13).

The femoral head became eburnated except for the periphery. As much as 5 mm of the femoral head was worn away in many instances (Figs. 10, 16, 17). When subluxation first occurred at 12 weeks and extra compression was applied to the head, the head and neck “drifted” downward into a varus angle as the head remodelled (Figs. 5, 8). As wear shortened the eburnated femoral head, the stress-lines changed and the femoral neck gradually shifted from the varus angle when the “drift” appeared to a short-necked valgus angle or position (Figs. 16, 17). The lipping at the ventral border of the femoral head caused by an accumulation of osteophytes at the junction of the head and neck remained. These osteophytes, composed of new fiber bone, extended around the entire femoral head at the junction of the head and neck (Figs. 10, 17).

During the eburnation development after the subluxation was stabilized, the cartilage surface of the femoral head at the nonarticulating margin of the head and neck became thickened because of lack of contact with the opposing acetabular surface (Figs. 15, 16).

In the normal articulating joint, the motion of the joint components with its efficient lubricating system keeps the shedding cartilage cells wiped off so the articular cartilage remains at a stable thickness. The normal articular cartilage is nourished by synovial fluid on the joint surface side and from diffusion of nutrients through the subchondral plate on the other side (1, 20).

In the unstable, subluxated joint, the width of the articular cartilage varies from nothing on the eburnated surface where compression had been excessive to the greatly thickened area of articular cartilage at the lateral border where the incongruity did not allow the opposing surfaces to touch (Figs. 10, and 15-17). The synovial fluid supplies nourishment to articular cartilage for a depth of approximately 1 mm as it is forced in and out by the compression-hydraulic system. The subchondral plate on the attached side of
Fig. 8. A coronal histological section of a hip joint from a 3-month-old dog with well-defined dysplastic changes (1). Compare these advanced changes with earlier changes illustrated in the 3-month-old dog (Fig. 3). The contour of the articular surface of the acetabulum has changed from concave to convex (1A). The dorsal acetabular rim is fractured and displaced dorsally (1B, 3B). The increased compression of new bone (3C). This was stimulated by the abnormal pull from the soft tissues attached to the dorsal margin (3C). The trabecular formation is increased and is arranged in the direction of the pull from the dorsal area (3C). The bony trabeculae beneath this area are increased and form
the cartilage allows nourishment to diffuse to about the same distance. When the articular cartilage becomes too wide for adequate nutrient diffusion, the undernourished cartilage cells hypertrophy and new capillaries enter and induce ossification (Figs. 15, 16) (1, 9, 18, and 20). This mechanism, tearing of the Sharpey's fibers and stimulation of the periosteum re-

a triangle which converges into the medial cortex (lines). Trabeculae are increased beneath the attachment of the teres ligament (2E). The cartilage at the attachment of the ligament is frayed and fragmented (4E). New bone has formed on the ventral aspect of the neck where remodeling was in progress at 3 months (2F). New bone on the dorsal aspect of the neck (2G) is increasing its diameter.
Fig. 12. Photograph of a dysplastic (left) and normal (right) ischial part of the pelvis illustrating osseous changes associated with hip dysplasia (1). The lesser sciatic notch of the shaft of the ischium supports the synovial bursa which serves as a pulley to facilitate the pull of the tendon of the obturator internus muscle. Extensive exostoses (arrows) are present on the bursal surface (left) as the result of prolonged irritation to the periosteum from unbalanced excessive forces and pull. Also of note are the roughened areas of the pelvic surfaces of the ischium where this muscle attached (1A). The pelvis below (2) illustrates typical roughened and osseous change at the bursal areas (arrows) and on the floor of the dysplastic pelvis (2A). The external and internal surfaces of the pubic bones are raised, grooved and roughened in response to excessive pull on the muscle attachments that were anchored there (2B).

Fig. 13. Macerated pelvis of a dysplastic hip. The acetabular rim is displaced dorsally as the result of microfractures. The fracture lines are still visible (A). Below this is a crescent area of eburnation (B). The acetabular fossa is filled with new bone (C). Ridges and exostoses present on the wing of the ischium are due to tears at the attachment of the muscle fibers (D).

Fig. 14. Coronal histological section of a dysplastic hip from a one-year-old dog. The femoral head is subluxated and the angle of the femoral neck is valgus (D). The acetabular rim now healed has been fractured previously and displaced dorsally (A). A heavy trabecular cortex surrounds the dorsal aspect of the acetabulum (B). A narrow contact point articulates with the femoral head (C). New fiber bone formation filled the fossa and changed the contour of the acetabular cup (H). Articular cartilage has disappeared except for a small area on the dome of the fossa (C) and the rim which is turned upward. The cantilever arm of the femoral head is remodelled into a valgus position and the ventral cortex is greatly thickened (D). The cartilage of the physis has disappeared but the thickened trabeculae in the area of the subchondral plate remain (E). On the articular surface of the femoral head in the medial area the cartilage is worn away, and eburnation has developed in the narrowed area which contacted the acetabulum (C). The articular cartilage is thicker on the lateral side and an osteophyte has appeared at the extreme lateral edge (G). The joint capsule is greatly thickened (F).
sulted in osteophyte formation (Figs. 10, 13, 15, and 16).

In the subluxated hip, osteophytes rim the femoral head at the junction of the head and neck (Fig. 14). Osteophytes also develop on the dorsal surface of the neck and extend laterally to the trochanter and into the concave fossa between the trochanter and femoral neck (Fig. 14). This is where the capsule attaches and osteophytes form in the area where Sharpey's fibers are torn.

In our material, subchondral cysts of the femoral head were observed only twice compared to their common occurrence in the femoral head in man with osteoarthritis of the hip (Fig. 18).

The shape of the femoral shaft changed into a mild "S" curve to align the leg after lateral subluxation of the femoral head had occurred. Frequently, the stifle underwent degenerative joint disease as the result of malalignment in support of the subluxated hip.

Fig. 16. Coronal histological section of a hip joint from a middle-aged German Shepherd Dog with severe hip dysplasia and degenerative joint disease (1). The joint has undergone extensive remodelling and hypertrophic osseous formation. Lines and scars are still present and serve as clues to the changes that had occurred during the remodelling of this hip joint from normal conformation to the one illustrated here. The articular surfaces of the femoral head and acetabulum are fully eburnated and devoid of articular cartilage (3). The series of changes that occurred in this hip can best be understood by superimposing a drawing of a coronal section from a normal hip joint of a similar dog (2). In the affected hip new osteophyte bone fills the acetabular fossa (1) and acetabular cavity and nodules of new bone project from the dorsal and ventral acetabular surfaces (2). The femoral head is shortened from wear and widened by a collar of osteophytic new bone. The entire articular cartilage surface is lost as eburnation occurs (3). The angle of the femoral neck had become valgus, the trochanter major is remodelled and new bone is formed around the areas of muscle attachments (4). The joint capsule is greatly thickened (5). This restricted the cranial to caudal motion of the leg, even after the muscles were dissected away.
Fig. 17. Macerated hip joint of an old dog with extensive degenerative joint disease and hyperostoses secondary to hip dysplasia. The articular cartilage has disappeared. The contacting surfaces of the femoral head and acetabulum are eburnated. Below is a histological coronal section of the hip joint of the opposite hip which had similar anatomical changes. Notice the valgus angle of the femoral head, the osteophyte lipping, the eburnated surfaces, and the hyperostoses of the acetabulum.

DISCUSSION

The locomotion of the young dog with severe hip dysplasia may be unsteady but very little or no pain is exhibited. Suddenly, at 5 to 6 months of age, pain of the hindquarters appears, especially when the dog rises to his feet and following exercise. This is the age that microfractures of the dorsal acetabular rim appear. These changes were difficult to demonstrate radiographically even though the extent of the microfractures varied widely. Since a fracture is a painful lesion, it is logical to consider that the microfracture of the dorsal acetabular rim is a possible cause of the early pain. This pain subsides when the fractures are healed, usually by the 8th to 11th month. These signs do not recur because the bone has ossified sufficiently to withstand further trauma and fracture.

By the 9th to 11th month, the pain gradually subsides until the dog is seemingly pain-free in rising and in locomotion. The range of motion of the hindlegs is restricted, but this is usually not detectable from the dog’s normal gait because the dog does not swing the legs more than 45°
in normal walking. Occasionally the balance of the hindquarters is unstable, but frequently even when the radiographic signs are severe, the gait is normal, except for a low threshold of fatiguability.

Abnormal remodelling and hypertrophic osseous development are continually present in and around the dysplastic hip. Even though these mechanisms are functional during the disease, there is commonly poor correlation between the clinical signs of the patient and the radiographic signs of the involvement unless the dog is worked hard. Often the radiographic involvement is extensive but there are no physical signs of involvement. In a few instances radiographic lesions are mild but signs of pain are severe.

The investigation reported here is a detailed description of radiographic, gross and histologic lesions of spontaneous canine hip dysplasia. The significant changes are recorded for comparison with the rate and pattern of normal development of the hip joint from birth to maturity (13). These findings support the theory that hip dysplasia occurs only if hip joint instability and joint incongruity are present in the young dog. It is also believed that the disease can be prevented if hip joint congruity can be maintained until ossification makes the acetabulum less plastic and the abductor muscles and supporting soft tissues become sufficiently strong and functional to prevent femoral head subluxation (14).

In conclusion, hip dysplasia is a concentration of factors from a pool of genetic weaknesses and environmental stresses that fall into a programmed pattern of progressive remodelling and degenerative joint disease.

SUMMARY

The pelvic growth in dogs which developed hip dysplasia was followed radiographically, grossly and histologically from birth to maturity to obtain clues to the earliest signs of the disease. The growth and development in the length and diameter of the pelvic bones and femurs were at a normal rate in the dysplastic dog. The bony malformations were confined to the hip joint except for minor adjustments in shape. In the dogs described, hip dysplasia became severe. There was no recognizable evidence of the disease until the 7th week, at which time the femoral head became unstable and subluxated from the cavity of the acetabulum. In other less severe instances (mild dysplasia), the femoral head subluxation usually was not evident until the 5th to 6th month of age. In even milder cases, the dogs were 12 to 14 months of age before hip dysplasia was apparent radiographically. The degree of the maldevelopment depended upon the extent of femoral head and acetabular separation and the age of the dog when congruity was interrupted by subluxation. In this report the radiographic, gross and histologic changes were described in depth.

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REFERENCES


RÉSUMÉ

La croissance pelvienne des chiens qui ont développé la dysplasie de la hanche a été suivie radiologiquement, macroscopiquement, et histologiquement, à partir de la naissance jusqu'à la maturité pour obtenir des indices des signes primitifs de la maladie. On a constaté que la croissance et le développement de la longueur et du diamètre des os pelviens et des fémurs se faisaient à un taux normal chez le chien dysplastique. Les malformations des os ont été limitées à l'articulation de la hanche, sauf pour des petites modifications de forme. Chez les chiens décrits, la dysplasie de la hanche est devenue instable et a subluxé de la cavité de l'acétabule. Dans d'autres cas moins sévères (dysplasie bénigne), d'habitude la subluxation de la tête fémorale n'était pas évidente avant l'âge de 5 ou 6 mois. Dans sept cas plus sévères, les chiens avaient douze ou quatorze mois avant que la dysplasie de la hanche soit devenue apparente radiographiquement. Le degré du mauvais développement se faisait en fonction de la mesure de séparation de la tête fémorale et de l'acétabule, ainsi que de l'âge du chien quand la congruence a été interrompue par la subluxation. Dans cette étude on a décrit les modifications radiographiques, macroscopiques, et histologiques à fond.

ZUSAMMENFASSUNG
