Hip Dysplasia

Chapter 83

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Section One: Theories of Pathogenesis

Canine hip dysplasia is a complex disease. It is a concentration of factors from a pool of genetic weaknesses and environmental stresses that fall into a programmed pattern of progressive remodeling and degenerative joint disease. The degree of involvement varies from minute changes in bone structure to total destruction of the hip joint. Investigators have searched intensively for genetic, chemical, and metabolic defects, but the cause has remained obscure.

Hip dysplasia affects humans and all other domestic mammals. In humans, 1.3 children in 1000 are affected. In dogs the prevalence may run over 50% in large dogs if control measures have not been practiced. Few data are available on the prevalence of hip dysplasia in other mammals, but it is thought to be low. The disease is undoubtedly rare in undomesticated animals.

No specific genetic pattern of inheritance has been demonstrated in this variable disease. It has been demonstrated that both genetic and environmental influences contribute to development, regardless of the species affected.(15,31, 32,40,74,76) Consequently, the disease has been designated as polygenic or multigenic.(28) As in most polygenic diseases, there are both major and minor causative factors. There is no evidence that a primary defect of bone exists but rather the disease is a failure of the muscles and other soft tissues to hold the hip joint in full congruity.(31,32) This is further supported by the fact that bony dysplasia can be increased, decreased, or prevented by controlling the degree of joint instability and incongruity.(53) No other malformations are associated with the disease.(79) A causal relationship between muscles and soft tissue defects or pathologic changes other than lack of muscle mass or strength has not been established. (40,41)

Experimentally, hip dysplasia may be produced in many ways.(43,56,74,76,87,88) These include any circumstances that contribute to an unstable hip joint, namely, adductor forces, lack of muscle strength,

chemical relaxation of the pelvic soft tissues, traumatic injury to the hip joint, and overloading of the joint by weight. Hip dysplasia is a concentration of factors from a pool of genetic weaknesses and environmental stresses that fall into a programmed pattern of progressive remodeling and degenerative joint disease.

The general cause of hip dysplasia, when defined, must be broad enough to explain its development, not only in dogs, but also in all other affected animals. Many genetic and environmental factors can trigger events that bring about the condition secondarily.(74,77,79,88) Hip dysplasia, therefore, is not one disease but many diseases that result in common degenerative lesions of the hip joints.(77)

Hip dysplasia has been observed in cats (27,35) and in most breeds of dogs; however, it is a greater problem in some breeds(65) than in others. The true prevalence of hip dysplasia among breeds of purebred dogs is not known, but data from the Orthopedic Foundation for Animals (OFA) on the first 36,000 pelvic radiographs evaluated has given insight into answering this question (Table 83-1).

The percentage of dogs of various breeds affected by hip dysplasia is not a true representation of the prevalence of the disease in these breeds because radiographs depicting obvious dysplasia were screened by referring veterinarians and not submitted. Therefore, the overall prevalence of hip dysplasia is higher than that represented in the analysis (Table 83-1). The rankings of the breeds are consistent with those obtained previously from smaller populations.(36) All breeds were screened in a similar manner.

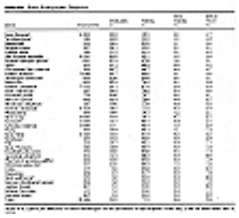

Table 83-1 Pelvic Radiographic Diagnosis

Body Size

The breeds with the lowest prevalence of hip dysplasia are near the size of the ancestral dog. The bones are small in diameter and smooth, the feet are small and well arched, and the shape of the head is long and narrow.

The giant breeds with the highest prevalence of hip dysplasia are two to three times larger than the ancestral dog. Their bones are coarse and large in diameter, with prominent protrusions and depressions. The feet are large and splayed, and the head is wide and oversized.

Body Type

In general, the body conformation of the breeds with the lowest prevalence of hip dysplasia is slender and trim. The skin is thin, smooth, and stretched tightly over the underlying tissues. The muscles are prominent, hard, and full-bellied. At dissection in these breeds, the skin and subcutaneous tissues and fascia rarely contain over 1% to 2% fat by weight. The joint ligaments are well developed; the fibers are coarse, closely packed, and relatively free of fat. The well-formed pelvic and thigh muscles are attached to broad, coarse tendons that are attached securely to the bones. These dogs are fleet-footed and well-coordinated in their movements.

Of the high-risk group, the four breeds of the giant type are not only two to three times the size of the ancestral dog, but their body conformation is heavy, round, and stocky. Acromegalic characteristics are present to some extent in all four breeds. Fat is abundant in the subcutaneous and fascial spaces and commonly accounts for 5% to 10% of the weight of the soft tissues of the hindquarters. In comparison with

the low-dysplasia group, the muscles are less prominent and less developed. Fat is infiltrated into the tendons and ligaments. The fibers of these two structures are smaller in diameter than those of the low-risk group. The gait of the giant breeds is less graceful and slower than that of the smaller breeds.

Growth Pattern

Breeds with the highest prevalence of hip dysplasia grow and mature more rapidly than those in the low-risk group. Starting at birth, this group gains rapidly. The pups of these breeds are aggressive eaters, both as they nurse and as they take supplemental food. In a study involving 222 German shepherds, 63% of the dogs that weighed more than the mean of this group at 60 days of age were dysplastic at 1 year of age, whereas only 37% of those less than the mean became dysplastic. The same rapid rise in weight in other breeds of the group at high-risk for dysplasia has been observed.(63)

Hip dysplasia has not been reported in the wild undomesticated carnivorous animals, such as wolves and foxes. A study of their pattern of growth found that the pups were slow-growing and late maturing. The young pups were whelped in dens. As newborns, they received their nourishment by nursing during the first few weeks. When more food was required, the mother killed rodents and either brought them to the den or ate the animal where it was killed and then returned to the den where the ingested rodents were regurgitated for the young to eat.(61) Young carnivores were quite mature and 6 to 10 months old before they began to hunt. The amount of food available for the growing members of a litter was limited. This caused the young to mature slowly and remain thin and light for their body size. Such an environment favored the completion of ossification and developmental maturity of the joint before the hips could be subjected to possible injury, incongruity, or subluxation from excessive extrinsic forces (e.g., excessive body weight) (65,69)

Genetic Influences and Heritability

Few genes analyzed thus far directly affect osseous structures.(17) The shape of bones reflects changes by biomechanical stresses.(15)

In the dog no clear-cut pattern of inheritance has been recognized.(23,28,30) This means that many genes are affected, and polygenic traits are subject to environmental modifications. New data have substantiated these findings.(29)

The spread of hip dysplasia centers around the genetic transmission and heritability of a particular body size, type, conformation, movement, growth pattern, and temperament. This conclusion is based on the facts that the prevalence of hip dysplasia is approximately the same in a number of breeds with similar body characteristics and there is no gene flow between these purebred breeds. Since these facts must be respected, biomechanical and environmental factors associated with certain body conformation and size must be considered as causes.(69)

Critical evaluation of the heritability of hip dysplasia has been made in the German shepherd in 244 offspring from 54 full subfamilies. In one report, "heritability was defined as a property not only of the character (trait) but also the population and the environmental circumstances to which individuals are subjected. Heritability, because it represents the proportion of the total phenotypical variance, receives the attributes of a positive number which may range from 0 to 1.0 in magnitude".(29) On this scale and based on evaluations of radiographs from 2 year-old dogs, the heritability was given an average estimate of 0.25. The conclusions were that canine hip dysplasia be termed a moderately heritable diseased.(30)

In a study involving 236 German shepherds, it was demonstrated that the most reliable way to eliminate canine hip dysplasia was through the establishment of "pedigree depth," that is, by the use of ancestral lines of dogs radiographically free of hip dysplasia.(33)

Results of controlled breeding programs in Sweden further indicated that the prevalence of hip dysplasia in the German shepherd was substantially reduced by mating only dogs with radiographically normal hips.(7,50) Similar decreases in prevalence have occurred in another controlled breeding program in a colony of guide dogs (Seeing Eye, Inc. Morristown, NJ).

In another account, with 584 progeny in a closed colony of German shepherds, it was shown that the prevalence of hip dysplasia was noticeably reduced by selectively breeding dogs proved radiographically to have normal hips at 1 year of age or older. In 3-1/2 years the incidence of hip dysplasia was lowered from 39% to less than 17%.(64) The male dogs in this colony had a wide variation in their ability to transmit normal hips to their progeny. For example, only 8.7% of the progeny of one dog with radiographically normal hips at 2 years of age developed hip dysplasia, whereas 37.8% of the pups of another dog with similar radiologic evaluation mated to the same bitches developed hip dysplasia.(20)

Environmental and Man-Made Influences

Embryologically, articular joints are differentiated as units in situ from a mass of skeletal mesenchyme.(90) Development progresses normally in each joint as long as there is full congruity between the parts. The congruity remains as long as the supporting tissues are strong enough to withstand the mechanical or physiological factors that tend to pull them apart.(77)

In humans, intrauterine stress has been cited as contributing to hip dysplasia, particularly if the fetus is positioned with the legs in adduction and extension.

Hip dysplasia in humans is rarely associated with teratology abnormalities. Other hip abnormalities distinctive from dysplasia, however, are frequently associated with such deformities as clubfoot, hyperextension of the knees, spinal deformities, arthrogryposis multiplex, and chondro-osteodystrophy.(22)

In the young child, the position of the legs during infant care is found to be very important to normal hip development.(71,73,75) Abduction and flexion of the legs has a stabilizing effect on the hip joints. The square diaper favors greater abduction of the legs than does the three cornered diaper. The Bantu baby, who is carried with its front side bound to the mother's back with its legs in acute abduction and flexion, seldom has abnormal hip joints.(71,75) In contrast, the Navajo Indian baby, who spends its first years of life strapped to a cradleboard with the legs in abduction and extension, has a high rate of hip joint instability.(70)

Other factors such as femoral anteversion and spastic shortening of the psoas muscle have been shown to favor acetabular dislocation when the leg was extended.(44) These observations indicate that both environmental and hereditary influences are important.(28,42)

In the dog, the hip joints are normal at birth.(43,68) The long bones of the pup are short during prenatal life, and mechanical stresses that bring about dislocation of the femoral heads are minimal. Teratologic abnormalities of the joints are rare in the dog, except for congenitally dislocated elbows and an occasional clubfoot deformity. Congenital malformation of the hips is also rare.

Extrauterine Influences

EARLY WEIGHT GAIN

In 222 German shepherds born consecutively, 100 were dysplastic, and the prevalence of hip dysplasia at 1 year had a direct correlation with their weight at 60 days of age. The heavier dogs, that is, the heaviest males and heaviest females at 60 days of age, had the highest incidence of hip dysplasia at maturity.(63) (See Fig. 83-2.)

These data suggested a number of indirect genetic factors influencing the rate of hip dysplasia. The aggressiveness in nursing may be inherited, as may be the quality and quantity of the supporting tissues around the hip joint. It was concluded that when growth, gain in weight, and nursing aggressiveness exceeded the strength of the supporting tissues, subluxation and hip dysplasia occurred.(63)

The first subluxating stress on the hips occurs when the pup supports itself while nursing, and the hindlegs are in forceful adduction and extension. The heaviest pups were the more aggressive, worked the hardest while nursing, and spent the most time feeding.(63)

PELVIC MUSCLE MASS

Data indicate that here is a positive correlation between the amount of pelvic muscle mass and the prevalence of hip dysplasia. Of three large breeds of dogs, the greyhound is relatively free of hip dysplasia; over half of the German shepherds are affected with hip dysplasia, and nearly all the July foxhounds are dysplastic.(69)

These data further emphasize that hip dysplasia encompasses biologic height, weight, and muscle bracing. The builder, before architecture was a science, learned that when the height of a structure was doubled, the bracing had to be tripled or the structure would fall of its own weight.(82) This basic rule, learned many years ago, illustrates clearly why a low foot stool fits solidly on the floor and the tall stool of the same area wobbles when supporting weight.(82) Similarly, it has been found that dogs less than 30.5 cm in height and less than 11.3 kg in weight (dachshund) are relatively free of hip dysplasia. On the other hand, at least half the large dogs, those 34 kg or more in weight and more than 50.8 cm in height, are affected with dysplasia.(66)

MUSCLE MYOPATHIES

All newborn mammals, including human infants, undergo many metabolic changes during their transition from intrauterine to extrauterine life. The muscle tissues are relatively immature both anatomically and biochemically at birth. Lack of muscular maturation in the newborn influences the manner in which the newborn responds to function. This immaturity accounts for the failure of many mammals, including the human, dog, and cat, to walk at birth.(88)

There is evidence that the wide range of acetabular and femoral changes occurring in hip dysplasia is the consequence of joint laxity. The possibility that this may be associated with or influenced by the rate of muscle maturation has not been explored. The rate of muscle maturation may be an inherited factor.(12,43) Consequently, the degree of subluxation in the young may be influenced by subnormal muscular function. In humans, the possibility of iliopsoas muscle spasm in the infant has been explored. (41,44)

In the adult dog, the light microscope was used to examine histologically the individual pelvic muscles associated with hip joint motion. Evidence of muscle disease was not recognized. In dogs with advanced hip dysplasia and associated osteoarthritis, atrophy of the pelvic muscles was present but changes such as muscular necrosis, inflammation, and extensive fibrosis were not found.(66,69)

One observer suggested that in young dogs with developing dysplasia, the pectineus muscles were in spasm and contained a degenerative lesion.(4) The pectineus muscle (an adductor), when in spasm, was thought to favor forcing the femoral heads out of the acetabula. This observer further suggested that if the pectineus were cut in the dog at an early age, the occurrence of hip dysplasia would be drastically lowered.(4)

A causal relationship between the pectineus muscles and hip dysplasia was not established in an experiment using the pelvic muscles from Labrador retrievers, German shepherds, Alaskan malamutes, and beagles.(40) Pectineus muscles in these dogs with both normal and dysplastic hips were examined and compared. The relationship between pectineus muscle abnormality and hip dysplasia remains undefined. The pectineus muscles from some young pups showed both hypotrophic and hypertrophic changes. It was suggested that the

alterations seen in the pectineus muscles of dysplastic dogs probably represented secondary manifestations associated with a disease of developing hip joints (hip dysplasia).(4,12) The available evidence does not support the concept that abnormal pectineus muscle behavior is a cause of hip dysplasia. (39)

Developmental myopathy with type II fiber hypotrophy has been described in the pectineus muscles of very young dysplastic German Shepherds These investigators failed to establish a relationship between this muscle change, joint laxity, and dysplasia but have suggested the possibility of such a relationship. In their experiments using an enzyme stain, the small fibers stained as type I (white) and the large fibers as type II (dark). They considered the differentiation between small and large fibers in young dogs to be a myopathy. No myopathies were present in either the normal or dysplastic adult dogs in their study.(12) This change in the young dog resembles muscle fiber hypotrophy, which follows the cutting of the nerve to a muscle. These hypotrophied muscles become functional again and the fibers become normal in size when the nerve unites and use is restored.(34) Atrophied muscle due to a severed nerve and immature muscle are similar in appearance. (34)

Metabolic Influences

SEX

In humans, the female is affected with hip dysplasia four to eight times more often than the male.(22) In the dog an equal number of females and males are affected. The reasons for this difference have not been explained. Of 100 dysplastic German shepherds at the Armens Hund Skula (Sweden), 49 were males and 51 were females.(63)

CHEMICAL AND HORMONAL INFLUENCES

Pelvic tissue relaxation is a well-known physiological phenomenon that occurs during the terminal phase of pregnancy in mammals. This reaction has been associated with the female hormone, estrogen. Experimentally, this reaction has been studied by injecting ovarian extracts into dogs to produce pelvic tissue relaxation resembling that seen at the termination of pregnancy. The specific polypeptide hormone that is commonly used is called relaxin. Male and spayed and virgin females when "primed" with estrogen before relaxin was administered responded sufficiently to relax pelvic tissues around the hip joints.(43,55)

The urine of newborns was examined to see if there was a correlation between high estrogen levels and the unstable hip. From the first tests, it appeared that such a correlation existed, but the use of more refined tests failed to verify these findings. (1,3,81) The conclusion is that hormonal influence is not associated with the development of congenital hip dysplasia in humans or animals.(1,3,71,81)

In the dog it has been possible to increase the incidence of hip dysplasia by giving relaxin to newborn pups and to produce hip dysplasia in the greyhound. (18,43,51,55) "It does not prove, however, that estrogens have anything to do with etiology and pathogenesis of spontaneously occurring hip dysplasia."(19) There is no evidence that estrogen levels within the biologic range have a relationship to the incidence of hip dysplasia in dogs.(19,52,55,81)

Defective protein biosynthesis of collagen was suggested as a cause for increasing articular cartilage degradation in osteoarthritic joints. Soluble collagen was reported to be found in the acetabular cartilage of dysplastic dogs, while predominantly insoluble collagen was present in dogs with normal hip joints. It was not possible to relate these changes to hip dysplasia or to osteoarthritis.(39,40)

Inborn metabolic errors of chemical or hormonal origin have not been found in human or canine hip dysplasia.(39,40,52,87)

DIET

A variety of nutritional and mineral supplements have been used in attempts to alter or prevent the course of hip dysplasia in the dog. Diet has not affected the occurrence or course of the disease other than the mechanical effect of increased or decreased weight upon the hip joint.(66)

Prevention

In the child the development of hip dysplasia can be stopped and the condition can be reversed to a stable normal hip if it is discovered early before remodeling has begun. The key to treatment is the restoration of full congruity between the femoral head and acetabulum by placing the legs in an abductor-flexed position.(76,88)

In the young dog genetically conditioned to develop hip dysplasia, confinement to a small cage (1 m3) where the dog spends most of his time sitting on his haunches (abductor-flexed position) will prevent the development of hip dysplasia.(66,68) Surgical improvement of joint congruity can also be very beneficial.

Section Two: Growth and Development of the Normal Canine Pelvis, Hip Joints, and Femur from Birth to Maturity

A clear understanding of the normal development of the hip joints, pelvis, and femur is imperative as a basis for comparison in evaluating change associated with disease and/or injury of the hip. The American racing greyhound is the model used to obtain normal growth patterns because of the low incidence of hip disease, particularly hip dysplasia, in this breed. Furthermore, the shape, size, and rate of growth and development of the bones of the pelvis, hindlegs, and caudal half of the spine are similar to other medium-large dogs, some of which have a high prevalence of hip dysplasia.

Longitudinal Growth of the Femur

The nutrient artery around which the bony foramen later forms is at the center of a long bone when the cartilage mold is laid down in the skeleton of the fetus.(45) By identifying the nutrient artery, it is possible to measure the growth at the two ends of a long bone. At birth, it is difficult to measure the length of a long bone radiographically because only the diaphysis between the proximal and distal growth plates is radiopaque. The cartilaginous epiphyses are radiolucent.

At birth, the estimated total length of the femur is 3.5 cm, with 1.5 cm on the proximal end of the femur and 2 cm on the distal end. The femur maintains a uniform weekly length increase (Fig. 83-1). When measurements are charted, the weekly growth as indicated on the chart for the proximal part maintains an increase at the slope of approximately 20° until growth slows at the 30th week. Between the 10th and 30th weeks, the slope increases to approximately 25° and 30°. The distal part of the femur grows more quickly. Its growth rate is at a slope of approximately 50°. A 1: 1.5 ratio is maintained between the proximal and distal parts of the femur from birth to adulthood.

At 30 weeks (7 months) of age the overall length of the femur is 23.5 cm, with 9.5 cm at the proximal part and 14 cm at the distal end. This length represented 95% of the total adult femoral length and is within 0.9 cm of the length of the femur of the 1-year-old, 26-kg (56-lb), male greyhound, whose femur measured 24.4 cm, with 9.9 cm in the proximal and 14.5 cm in the distal parts (a ratio of 1:1.5 between the proximal and distal ends).

Shape of the Femur

At birth, only the metaphyses and diaphyses of the femur are visible radiographically, and these are of hour-glass shape (Figs. 83-1 and 83-2). The distal end is slightly longer and wider than the proximal end. The ossification of the head and distal condylar epiphysis is sufficient to be visible at 12 days of age, and the shape of the head and condyles is evident by the 13th day.

Radiographically, the center of ossification of the femoral head has the appearance of a small opaque dot or circle after ossification begins. By the fourth or fifth week the epiphysis appears to be attached to the proximal end of the femur except for a thin radiolucent line that is the physis (Fig. 83-3). Initially the proximal surface of the head is uniformly rounded. By the sixth or seventh week a notch appears on the medial surface of the femoral head where the ligamentum teres attaches. The diameter of the proximal physis is more than twice the height of the proximal epiphysis.

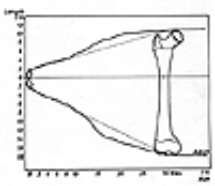

FIG. 83-1 Chart drawn from radiographic overlay tracings of the femur at intervals from birth to one year of age. The 0 point on the vertical axis is the level of the nutrient foramen. (Reproduced with permission from Riser WH: Growth and development of the normal canine pelvis. JAVRS 14:24, 1973)

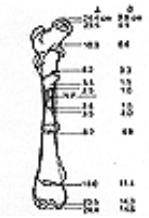

FIG. 83-2 Chart drawn from radiographic overlay tracings of a femur from birth to one year of age. The nutrient foramen (NF) served as the center point of growth from which measurements were made. (A, total length of femur at the specified ages; B. length of proximal and distal ends of femur from the nutrient foramen at the specified ages.) (Reproduced with permission from Riser WH: Growth and development of the normal canine pelvis. JAVRS 14:24, 1973)

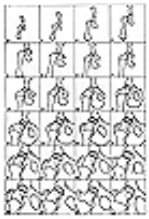

FIG. 83-3 Drawings copied from radiographic overlay tracings of normal hip joint growth and development from birth to one year of age. Number of each drawing indicates age, in weeks, of the dog. (Reproduced with permission from Riser WH Growth and development of the normal canine pelvis. JAVRS 14:24, 1973)

PHYSES (GROWTH PLATES)

The growth plates are radiolucent structures composed of columns of cartilage that lie parallel to the long axis of the femur and the direction of forces (either compression or traction) exerted upon the bones.

In the femur there are three physes. The two proximal ones were once joined but separated when the femoral head and greater trochanter moved away from each other as a result of the pulling forces of the gemelli, the obturator externus, and the obturator internus.(32) These physes are distinct and relatively the same width from the time of their appearance when ossification of the head is first visible until 80% of the growth is completed at 5 months of age. Then the width of the cartilage space narrows gradually. It disappears radiographically between 11 and 14 months of age (Figs. 83-1 through 83-3).

The physis at the distal end of the femur is modeled to withstand compression, shear, and torsion forces. It has a studlike surface with four prongs that insert into the epiphysis of the distal Condoles. This physis remains active and appears radiolucent until the 10th to 13th month.

FEMORAL NECK

The diameter of the femoral neck narrows slightly directly below the head. The joint capsule attaches around the neck immediately distal to the area where the femoral head unites with the neck. The trochanteric fossa appears as a deep cavity. The angle of the neck with the diaphysis as viewed from the ventrodorsal radiograph is 135°. This neck angle is constant from birth through all stages of development (Figs. 83-1 through 83-3).

The anteversion angle of the femoral neck is 0° to 20°, with a mean of 10°. It has been obtained from the measurements of 32 macerated femora of 16 mature greyhounds.

In the embryologic state the trochanter and femoral head are composed of a single piece of cartilaginous tissue. With the development and contraction of the muscles of the pelvis, the trochanteric fossa is formed to allow for the insertion of the obturator internus, the obturator externus, and the gemelli muscles.(21,45) The greater trochanter is extended by the dorsal and medial pull of the three pairs of gluteal muscles. The trochanter becomes visible radiographically between the eighth and ninth weeks of life. The lesser trochanter on the medial side of the femur appears as an apophysis and is composed of cartilage during the early weeks of life but is identifiable radiographically at 10 to 12 weeks. When the physis closes (11-13 months), this process attaches as a single piece to the diaphysis of the femur (Fig. 83-3).

FEMORAL SHAFT

The metaphyses develop and extend proximally and distally as the columns of cartilage cells of the physes mature and disintegrate, and new trabeculae ossify and the bones lengthen. Both the length and diameter of the femoral shaft increase to six to seven times their size (Fig. 83-4). There is a direct correlation between growth in length and increase in diameter.

FIG. 83-4 Chart drawn from overlay tracings of radiographs of one half of the pelvis of a dog at birth, 8 weeks, 16 weeks, and one year. The bipolar growth plate (B) between the ilium and ischium served as the center point of growth. (Reproduced with permission from Riser WH: Growth and development of the normal canine pelvis JAVRS 14:24, 1973)

FEMORAL CONDYLES

The shape of the condyles at the distal end of the femur varies very little during growth. The increase in size is in proportion to that of the rest of the bone.

Growth of the Os Coxae (Innominate Bone)

The innominate bone is united with its contralateral fellow to form the pelvis, which comprises the ilium, ischium, pubis, and the acetabular bone. Only parts of the ilium and ischium are visible radiographically at birth. (See Fig. 83-3.)

At birth all the bones are present, and the hip joint with the acetabulum and femur is functional and stable. The parts making up the hip joint are cartilaginous and radiolucent, except for parts of the ilium and ischium (Figs. 83-3 and 83-4).

Growth of the Pelvis

Cartilaginous physes unite the ilium, ischium, and pubis. These physes are functionally bipolar; for example, enchondral ossification takes place on both sides of the cartilage strip in the area that unites the ilium,

ischium, and pubis. By the 28th week practically all the enchondral growth at the pelvis has been completed. An additional increase of 2 cm in total length (1 cm at each end) occurs as the tuber-ilium and tuber-ischium are stimulated by traction of the attached muscles.(57) Growth in these two areas is completed by the 30th week.

At birth the ilium is approximately one third longer than the ischium. These two parts maintain a 3:2 growth ratio during the growth period and throughout life. The total weekly length increase of the os coxae is uniform during the growth period. When plotted on a graph, the growth slope of the ilium is 30° and that of the ischium is 20° (Fig. 83-5). Between the 10th and 30th weeks both slopes increase somewhat until the 29th week, when skeletal growth slows abruptly. The upper and lower curves of the growth patterns of the os coxae and the femora are almost identical.

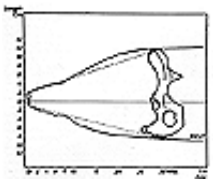

FIG. 83-5 Chart drawn from radiographic overlay tracings of one-half of the pelvis of a dog at intervals from birth to one year of age. The bipolar growth plate between the ilium and ischium served as the center point of growth. (B. size at birth.) (Reproduced with permission from Riser WH Growth and development of the normal canine pelvis JAVRS 14:24, 1973)

ILIUM

The ilium is a scapula-shaped bone that is divided into a wide cranial part known as the wing and a caudal compressed part that forms the cranial half of the acetabulum. Midway on the medial surface is a powerful synarthrosis that unites the pelvis and hindquarters with the sacrum. The growth lines, the shape of the ilium, and the angle (slope) at which the ilium is secured to the sacrum change relatively little from birth to maturity.

ISCHIUM

The ischium consists of the body, the sciatic tuberosity with its cartilaginous caudal border, and the curved ramus. These parts are all present at birth, but only a small rectangular piece of the ischial body is visible radiographically. The shape of the ischial body and ramus becomes visible rapidly within the next 20 days. Even by the first week the body lengthens and the caudal end becomes paddle-shaped and hooked as the tuberosity and ramus continue to ossify. The obturator foramen becomes evident when the pubis and ischium ossify at the seventh week.

PUBIS

The pubis appears radiographically by the fourth week and is identified with its contralateral mate by the ninth week. The pubic symphysis and obturator foramen are visible by the 11th week. (See Fig. 83-3.)

ACETABULUM

The acetabulum is a cotyloid lunate cavity created by the fusion of the ends of three bones: the ilium, the ischium, and the pubis. These encircle a fourth bone, the acetabular bone. The articular surface of the acetabular cavity is horseshoe-shaped and open ventrally. There is a central acetabular fossa. It is estimated that the ilium and ischium each contribute two fifths of the acetabulum and the pubis and acetabular bone together contribute one fifth.

Radiographically the union of these bones is masked by the head of the femur and cannot be distinguished until the 12th week when the acetabular cavity has ossified sufficiently to give a good radiographic image. (See Fig. 83-3.) A Y-strip endochondral cartilage unites the ilium and ischium at the dorsal rim of the

acetabulum. This is the last area to ossify. A secondary center of ossification located on the craniodorsal acetabular rim is sometimes visible on ventrodorsal pelvic radiographs of dogs between the 11th and 14th weeks of life. (See Fig. 83-3.)

Relationship of the Femoral head and Acetabulum

When the femoral head is first recognized, as a radiopaque dot, it is positioned well within the visible boundaries of the acetabular cavity. As the bones of the cavity and head mineralize sufficiently to be recognized, approximately two thirds of the globe of the femoral head lies within the acetabular cavity. (See Fig. 83-3.) This relationship does not change as the area ossifies. As ossification progresses, the radiolucent, nonossified cartilaginous spaces narrow.

Gross and Histologic Development of the Normal Hip

The hip joints of all dogs are normal at birth.(43,62) The joints continue to develop normally as long as full congruity is maintained between the acetabulum and the femoral head.(80)

The most valuable insight into the appearance of the normal hip is gained by following the development of the coronal profile by the use of histologic sections taken through the proximal femoral shaft and heads and acetabulum of the intact hip joint. Fig. 83-6 shows six normal hip joints. The first is from a dog 12 days of age, when the epiphyseal ossification begins. The others illustrate various stages of development at 4 weeks, 8 weeks, 16 weeks, 32 weeks and finally the hip from a dog over 2 years of age. In these photographs the dominant feature is the display of balance and symmetry in each joint. Each evidences the same angles of balanced cantilever support for the body weight through the dorsal curvature of the acetabulum and onto the femoral head and shaft, with lines of force converging into the medial femoral cortex.

The trabeculae in the acetabulum and femoral head and neck serve as a series of radiating "studs" and "braces" arranged in linear fashion as dictated by compression and tensile stresses. The supporting trabeculae are distributed evenly. The increase in size with growth is uniform and orderly. No overgrowth of bone occurs, and the cortices are thin.

The effects of balanced forces are well illustrated in the hip joint section of the 2-year-old racing greyhound (Fig. 83-6). In spite of the extensive training and hard work of this dog, the hip joint shows no evidence of wear, hypertrophic ossification, or deformation remodeling. In this hip the same lines of design exist after extensive abuse that are seen in the 12-day-old dog (Fig. 83-6). Histologically, the bones composing the acetabular fossa are thin with a minimum of trabecular bone (Fig. 83-6). The dorsal part of the acetabular cavity is extended sufficiently to cover two thirds of the femoral head. The vortices and trabeculae of the dorsal rim are thin. The acetabular rim (beak) is sharp and pointed.

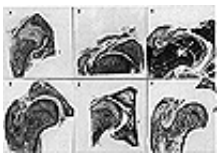

FIG. 83-6 Coronal histologic preparation of normal hips of dogs: (A) 12 days, (B) 4 weeks (C) 8 weeks; (D) 16 weeks; (E) 32 weeks; (F) 2 years. The illustrations show the normal development and maintenance of the hip joint At all stages of development and maturity there is an achievement of functional balance, symmetry and congruity between the femoral head and the acetabular cavity. (Excessive separations between the femoral heads and acetabula are preparation artifacts.) The entire articular surfaces of the acetabulum and femoral head are maintained in full congruity with each other while in a full range of motion The body weight is distributed evenly over the contacting articular surfaces. The hip joint functions with the forces about the hip neutralized and balanced. In these illustrations there is

no evidence of abnormal development, remodeling, or wear. (Reproduced with permission from Riser WH Growth and development of the normal canine pelvis. JAVRS 14:24, 1973)

Histologically, the proximal ends of the femur and trochanter appear almost osteoporotic. There is very little bone at the subchondral plate beneath the articulating surface of the femoral head and an absence of extra bone beneath the attachment of the teres ligament. The trabeculae supporting the femoral head are few and thin in diameter, but they form a triangle converging into the medial cortex (Fig. 83-6). The medial cortex is dense and thin, indicating that stress forces are minimal. In the trochanteric fossa, the site where the gemelli, obturator externus, and obturator internus muscle insert, the trabecular response is minimal, indicating that the hip has balanced sufficiently that there are no unusual forces on these attachments. In the trochanter major there is also a scarcity of both cortical and trabecular bone. The cortex is slightly thicker at the dorsal rounded tip of the trochanter and at the point where the trochanter joins the femur. These are the sites of attachment for the three gluteal muscles.

The long axes of the diaphysis and the neck form an angle of nearly 135°. The structure of the joint and adjacent bones reflects neutral stresses (compression, tension, and torsion), sufficient lubrication, and balanced muscle pull upon all of the bony components in the joint.

The acetabular rims are stimulated to grow by mild traction applied by the joint capsule and gluteal muscles attached along their dorsal borders, and from pressure by the femoral heads upon the articular surfaces. The diameters of the acetabular cavities are only slightly larger than the femoral heads.

Discussion

A normal hip must be an example of symmetry and balance as it develops and maintains itself from birth through adulthood. The hip joint is composed of specialized tissues, all of which participate in a programmed chain of development.(33) Except in the racing and toy dog types, there is a great chance that the hip will develop abnormally.(60)

The morphologic characteristics of the complex hip structure show that biomechanical behavior is the prime influence in the growth of this joint.(32) The symmetry in the femoral head and neck, the trochanter and the acetabulum of the normal hip, is impressive when coronal histologic sections are studied. The photographs of this joint from dogs of all ages-from shortly after birth to late maturity-illustrate how these qualities are maintained. The photograph of a highly trained racing dog retired after more than 2 years of grueling work displays no evidence of joint instability, wear, remodeling, or degeneration. (See Fig. 83-6.)

The laws that control bone and soft tissue dynamics control the development of the hip. Newton's law of neutral forces when applied to biologic tissues means that a joint is in functional equilibrium when all forces upon that joint mutually neutralize one another both in intensity and direction. Wolffs law introduces the concept of bone transformation in that changes in function of a bone are attended by alterations in its internal structure. This law applies to cancellous and cortical bone.(14)

Section Three: The Dysplastic Hip Joint: Radiologic and Histologic Development

Chronologic Changes Seen in Hip Dysplasia

Birth to 30 Days of Age

Eighty-seven dogs from birth to 30 days of age were dissected. Their appearance was compared with normal greyhounds of the same age.(51) Eighty-four of these appeared normal. In three dogs 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 findings that might be linked to hip dysplasia.

From the dissection of the hips of these young dogs, it appears that the teres ligament is largely responsible for holding the femoral head in place for the first month. For the first 2 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 2 weeks, the teres ligament begins to lengthen very slowly. After the first 4 weeks the femoral head may be subluxated laterally 1 mm 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

Radiographically the first signs of hip dysplasia are femoral head subluxation and a lag in the development of the craniodorsal acetabular rim. These are noted by the seventh week (Fig. 83-7). At this time subluxation can be recognized by palpation. Grossly the joint capsule is stretched, but the tissue of the capsule has not changed in appearance. The teres ligament is also longer but does not appear to be stretched. Histologically the contour of the acetabulum is greater than that of the femoral head, and the craniodorsal rim is no longer sharp.

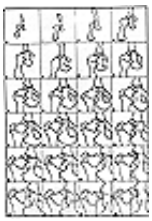

FIG. 83-7 Drawings copied from overlay tracings of hip joint radiographs of a dog during growth and development of hip dysplasia from birth to 35 weeks of age. The numbers within each drawing indicate the age of the dog in weeks.

SIXTY TO NINETY DAYS OF AGE

Radiographic changes are dramatic. Femoral head subluxation increases. The head no longer fits deeply into the acetabulum. The lag of ossification at the craniodorsal acetabular rim is increased (Fig. 83-7).

In affected, anesthetized dogs, the femoral head can be subluxated an estimated 0.5 cm. When the hips of these dogs are dissected and the muscles are removed, joint capsules are slightly thickened and stretched to a width that allows the femoral heads to be extended laterally to the acetabular rims. When the joint capsules are opened, the dorsal acetabular rim is rounded from the 10 o'clock position to the 2 o'clock position; the teres ligaments are pulled ventrally away from their attachment to the dorsal dome at the acetabular fossae; the teres ligaments are swollen; the articular cartilage on the dorsal surface of the femoral heads is worn and roughened where it contacts the acetabular rims when the femora are subluxated laterally. These changes are the result of abnormal mechanical forces of traction and compression associated with developing subluxation.

Progressive changes are recognized as the dog becomes older. Radiographically, by 11 to 12 weeks the greater trochanter is bent medially in the subluxated hip (Fig. 83-7). The full significance of the medial bend is not realized until a profile view is seen of a coronal histologic section of the hip (Fig. 83-8). When the dysplastic leg luxates laterally, the femoral head rests on the dorsal rim of the acetabulum. This lateral displacement of the femoral head places 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. 83-8 and 83-9). The abnormal pull bends the trochanter medially.

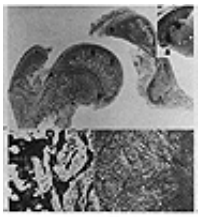

FIG. 83-8 Coronal section of the hip joint (a) of a 12-week-old dog with early dysplasia. In life, the femoral head of this dog was subluxated 6 mm to 8 mm, which caused an incongruous contact between the acetabulum and the femoral head and stretched the joint capsule and teres ligament The contour of 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 reinforced by new fiber bone (A, b), which is being laid down adjacent to the lamellar bone (right). The rim (B) is rounded as the result of the delay of chondrogenesis and osteogenesis. Overloading forces on the acetabular rim have delayed the conversion of fibrocartilage to hyaline cartilage and then to bone (inset, c) Varus drift of the femoral neck and epiphysis is accelerating the remodeling of the ventral neck area (C). Osteoclasts are resorbing bone at a rapid rate. The epiphysis has drifted distomedially an estimated distance of 2 mm This has resulted in epiphyseal tipping at the ventral margin. A fracture through the physis cartilage occurred as a result of the shift (D). This 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. (Reproduced with permission from Riser WH: The dysplastic hip joint: Its radiographic and histologic development. JAVRS 14:35, 1973)

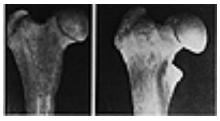

FIG. 83-9 Macerated proximal ends of femora of young dogs in which the physes are still open. The femur on the left is from a normal dog. The femur on the right is from a dog with well-defined hip dysplasia and illustrates the effects of drift on the capital epiphysis. Drift caused the femoral neck to form a varus angle. The lesser trochanter is enlarged as the result of abnormal pull from the iliopsoas muscle. Muscles about a dysplastic hip usually develop spasm when forces are unbalanced. The lesser trochanter of the normal femur is only slightly visible. (Reproduced with permission from Riser WH: The dysplastic hip joint: Its radiographic and histologic development. JAVRS 14:35, 1973)

Although the entire greater trochanter is cartilage at 12 weeks, histologically the cells and the matrix of the trochanter and the adjoining physis are normal and unaffected by the medial bending. In some instances, however, the rows of columnar cartilage of the physis are 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. "(15)

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 balance (Figs. 83-8 and 83-10). The congruous union between the femoral head and the acetabulum is disrupted, and the contacting area that forms an arc on the femoral head is narrowed from a normal of 90° to 20° The narrowed arc causes the compression forces to increase sharply on both the femoral head and the acetabular rim.(54)

FIG. 83-10 Gross coronal section of a dysplastic hip illustrates the changes between the acetabulum and femoral head. There is a stretched and thickened joint capsule (A), medial bending of the greater trochanter (B.), change in shape and lag in development of the acetabular rim (C), and tipping of "drift"

of the ventral border of the femoral head (D). The teres ligament is swollen and frayed (E). (Reproduced with permission from Riser WH: The dysplastic hip joint: Its radiographic and histologic development. JAVRS 14:35, 1973)

The excessive compression and overloading at the acetabular rim stretch 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. 83-8, 83-10, and 83-11).

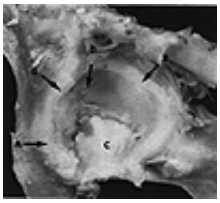

FIG. 83-11 The acetabulum in a 5-month-old dysplastic dog after the femoral head was removed by cutting the capsule and teres ligament. Early stages of severe hip dysplasia are evident. The joint capsule is thickened (A). The dorsal acetabular rim between 10 o'clock and 2 o'clock is rounded and uneven. The attachments of the joint capsule area are damaged and pulled away from the margin. The arrows (B.) are placed at 10 o'clock and 2 o'clock. The teres ligament is pulled downward from the dorsal dome of the acetabular fossa. The ligament is swollen, and some of the fibers are torn (C). There is a fracture through the articular cartilage at 11 o'clock (D). (Reproduced with permission from Riser WH: The dysplastic hip joint: Its radiographic and histologic development. JAVRS 14:35, 1973)

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 stretch signals that in turn adds new bone beneath the periosteum on the dorsal acetabular articular surface and stimulates the resorption of bone on the ventral surface of the acetabular cavity. The quantity and type of the new bone added or resorbed depend on the stress signals (Wolffs law). (15)

The epiphysis of the femoral head changes its position on the physic This change in position is referred to as "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 tipping of the epiphysis, bending of the physis, and a microfracture through the physis and damage to the adjoining tissue (Figs. 83-8 and 83-9). In this instance, a microfracture through the physis accompanied a shift in the position of the cartilage columns. The position of the femoral head upon the physis immediately undergoes a change so that the long axis of the enchondral cartilage columns remains parallel to the lines of forces and the 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. (See Fig. 83-8.)

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 cause changes in bone deposition and resorption that in turn cause the head and neck to bend downward. (See Figs. 83-8 and 83-9.) Since the cantilever arm forming the head and neck is composed of bone, it changes shape by adding new bone on one side and resorbing it on the other. Such change in shape is known as osseous drift.(15) The osteoblastic activity on the dorsal or lateral surface and the osteoclastic activity on the medial surface are intense at this time in a remodeling attempt to correct the exaggerated varus angle of the cantilever neck. (See Fig. 83-8)

The direction of the physis also changes to remain parallel with the compression surface of the acetabulum. 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.(83)

TWELVE TO TWENTY WEEKS OF AGE

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 continue to be the salient features of the dysplastic joint. (See Fig. 83-7.) Grossly, rounding or rolling back of the acetabular rim becomes more prominent between the 10 o'clock and 2 o'clock positions. Histologically, the increased localization of the compression forces in a restricted spot on the articular surface of the acetabular rim disturbs the developmental harmony of the area and fosters retardation of tissue maturation.(57) The effect 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. At this time the congruity between the femoral head and the acetabulum is disrupted and there is interference with the normal cycle of hip development in proportion to the degree of malposition or subluxation (Figs. 83-8, 83-10, and 83-11).

TWENTY TO THIRTY-FIVE WEEKS OF AGE

The overloaded area on the dorsal acetabulum succumbs to wear, cartilage fibrillation, chondromalacia, and finally microfractures of the rim (Figs. 83-12 and 83-13). The articulating surfaces, stripped of cartilage, become eburnated (Fig. 83-13). The exposed subchondral bone assumes an ivory-like appearance as it condenses and becomes osteosclerotic, smooth, and highly polished. A corresponding area of eburnation occurs on the contacting surface of the femoral head (Fig. 83-14).

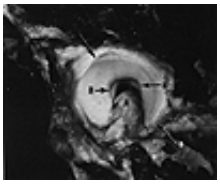

FIG. 83-12 A mildly dysplastic acetabulum after the femoral head had been removed. There is rounding of the dorsal and caudal margins of the acetabular rim between 11 o'clock and 5 o'clock (long arrows). The teres ligament (A) has been pulled away from the dome of the fossa, and new fiber bone has developed in the margin where the ligament was pulled away (B). (Reproduced with permission from Riser WH: The dysplastic hip joint: Its radiographic and histologic development. JAVRS 14:35, 1973)

FIG. 83-13 Coronal histologic section of a dysplastic hip joint from a 7-month-old dog. The subluxated femoral head has limited contact with the acetabulum restricted to an area at the rim (A). The increased compression forces have greatly altered the architecture of the entire joint. The contour of the articular surface of the acetabulum is changed from concave to convex. The rim has undergone various degrees of degeneration, cartilage fibrillation, malaria, necrosis, fracture, and upward displacement (A). Osteophytes appear in the fossa (B). Histologically the dark shaded areas in the teres ligament was hemorrhage (C). Trabecular bone is increased under the area of femoral head contact with the acetabulum (D). Unbalanced forces have caused ventral lipping (drift) of the femoral head (F). The articular cartilage is eroded, and eburnation is under way (D). The articular cartilage is increased in depth on the femoral head as contact is diminished at the lateral margin (E). Increased trabecular formation is present in the ventral area of the femoral neck just below the epiphyseal lipping (drift) (F. arrow). There is also increased trabecular buttressing over this area above the physis. (Reproduced with permission from Riser WH: The dysplastic hip joint: Its radiographic and histologic development. JAVRS 14: 35, 1973)

FIG. 83-14 The proximal ends of two femora illustrate the effects of advanced hip dysplasia. New-bone formation (exostoses) encircles the femoral necks at the junction of the head and neck (A). The femoral heads are shortened from wear and the cartilage surfaces are eroded and eburnated (B). (Reproduced with permission from Riser WH: The dysplastic hip joint: Its radiographic and histologic development. JAVRS 14:35, 1973)

The joint capsule in progressive hip dysplasia undergoes remarkable changes. The capsule of the normal joint is pale, semitransparent, and 1 mm to 2 mm thick. 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 mm to 7 mm, occurs in response to the trauma caused by excessive pull from subluxation. (See Fig. 83-10.) New bone forms in the capsule along the dorsal capsular border. In the dysplastic joint the synovial fluid loses its adhesiveness, its gravity decreases, and the leukocyte count increases. Its ability to function as a lubricant is decreased.(89)

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

As the subluxation becomes greater, the teres ligament continues to stretch and detaches from the dome of the acetabular fossa. (See Fig. 83-12.) Individual ligament fibers are ruptured as they are pulled away and become edematous. Swelling of the ligament is so great that it prevents the return of the femoral head to its normal position in the acetabulum. (See Figs. 83-l0 and 83-12) The ligament is completely destroyed and the entire acetabular fossa fills with fiber bone (Figs. 83-13, 83-15 through 83-17).

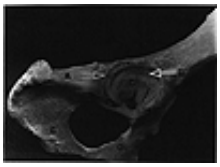

FIG. 83-15 Macerated pelvis of a dysplastic hip. The acetabular rim is displaced dorsally as the result of microfractures. The fracture lines are still visible (A). Below this there is a crescent-shaped area of eburnation (B). The acetabular fossa is filled with new bone (C). Ridges and exostoses present on the wing of the ischium are due to tears at the attachment of the muscle fibers (D). (Reproduced with permission from Riser WH: The dysplastic hip joint: Its radiographic and histologic development. JAVRS 14:35, 1973)

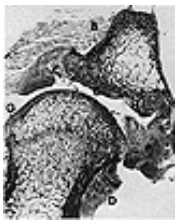

FIG. 83-16 Coronal histologic section of a dysplastic hip from a one-year-old dog. The femoral head is subluxated and the angle of the femoral neck is valgus (D). The acetabular rim, which is now healed, had been fractured and displaced dorsally (A). A heavy trabecular cortex surrounds the dorsal aspect 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 neck is remodeled 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 that 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). (Reproduced with permission from Riser WH:

The dysplastic hip joint: Its radiographic and histologic development. JAVRS 14:35, 1973)

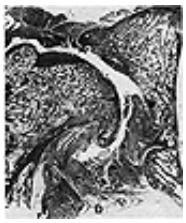

FIG. 83-17 Coronal histologic section of the dysplastic hip from a 2-year-old dog. The dysplastic hip exhibits the effects of prolonged, unbalanced wear and remodeling applied to the weight-bearing areas. The acetabular rim has been fractured and displaced dorsally (A). The articular surface of the acetabular rim is fractured and eburnated (A). The teres ligament has been pulled away from the fossa, and new-bone formation has filled the dome of the fossa (B). The femoral head has been shortened and eburnated, and osteophyte formation has occurred at both the medial and lateral margins of the femoral head (C). Synovial villi have formed in the distal margin of the joint capsule (D). (Reproduced with permission from Riser WH: The dysplastic hip joint: Its radiographic and histologic development. JAVRS 14:35, 1973)

Radiographically, at this stage in severe dysplasia a shallow acetabulum is observed. The shallowness results 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. 83-15).

There is marginal lipping of the femoral head with a rim of newly formed fiber bone that encircles the neck at the junction of the head and neck. (See Fig. 83-14.) It should be emphasized that the first bony changes of hip dysplasia appear in the acetabulum and that the changes in the femoral head follow and are not as pronounced as those in the acetabulum. Later, in advanced hip dysplasia, however, the changes in the femoral head are sometimes as pronounced as those in the acetabulum. This is the result of progressive remodeling and degenerative joint disease stimulated by joint instability.

NINE MONTHS OF AGE AND AFTER

Bone and joint tissues are never static, whether functioning normally or not. In the dysplastic hip, progressive remodeling occurs at an accelerated rate. The entire shape of the hip is restructured. Femoral head subluxation contributes to mechanical imbalance of the hip. Abnormal compression, traction, and tension forces create stresses that cause new bone to be laid down in one area and resorbed in another. At this stage of abnormal structure it is difficult to differentiate clearly the changes representing true hip dysplasia from those representing beginning secondary degenerative joint disease of the hip (Figs. 83-14, 83-15, and 83-18).

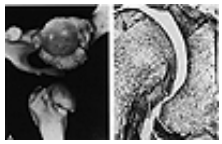

FIG. 83-18 (A) Macerated hip joint of an aged dog with extensive degenerative joint disease and hyperostoses secondary to hip dysplasia. The articular cartilage has disappeared. The contacting surfaces of the femoral head and acetabulum are eburnated. (B) Histologic coronal section of the hip joint of the opposite hip, which had similar anatomical changes. Notice the valgus angle of the femoral head, the osteophyte tipping, the eburnated surfaces and the hyperostoses of the acetabulum. (Reproduced with permission from Riser WH: The dysplastic hip joint: Its radiographic and histologic development. JAVRS 14:35, 1973)

Comparing a macerated hip joint of a dysplastic dog with that of a normal dog illustrates the effects Of remodeling. 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. 83- 19). 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 depth

of the fossa is very thin.

FIG. 83-19. Macerated normal hip joint of a young dog. Notice the smoothness of the bone contours, the sharp rim of the acetabulum, cavity, and the sharp edges of the acetabular fossa. The femoral head is balanced and symmetrically formed.

In the dysplastic hip, wear, exostoses, and distortion of bony surfaces are present in various degrees. The effects of abnormal or unbalanced stresses dominate the structure. The surface of the wing of the ilium is roughened to a greater degree than normal where Sharpey's fibers of the gluteal muscles are torn away from their attachment to the periosteal surface. In the ischium, bone surfaces are roughened and grooved as a result of attrition of bone due to prolonged irritation to the periosteum by unbalanced, excessive forces and pull from the obturator internus muscle (Fig. 83-20). In some instances exostoses are especially prominent over the synovial bursa at the sciatic notch. The external and internal surfaces of the pubic bones are raised, grooved, and roughened. The apophysis for the attachment of the iliopsoas muscle on the lesser trochanter of the femur is abnormally large and roughened. These changes are scars resulting from abnormal stresses on the unbalanced hip.

In the severely dysplastic hip, the scars of the previous fracture line on the polished, eburnated acetabular surface remain visible. (See Figs. 83-13, 83-15 through 83-17.) This line persists throughout life in dogs with dysplastic hips. (See Fig. 83-18.) The dorsal or periosteal surface above the acetabular rim is roughened by mounds of osteophytes. The formation of these exostoses is triggered when subluxation causes unusual stress to the supporting tissues. In many instances the roughened osseous surface of osteophytes circles the entire acetabulum. The contour of the eburnated surface of the acetabular cup is continuous across the fiber bone that fills the fossa; however, the latter never assumes the smooth, dense texture of the eburnated surface. (See Figs. 83-15 and 83-18.) Occasionally the fossa is only partially filled with new fiber bone.

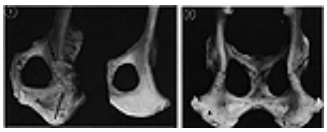

FIG. 83-20. (1). Photographs of dysplastic (left) and normal (right) ischial parts of the pelvis illustrate osseous changes associated with hip dysplasia. The lesser sciatic notch of the shaft of the ischium supports the synovial bursa, which serves as a pulley to facilitate the pull of the tendon of 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 (A). (2). Pelvis illustrates typical roughened and osseous change at the bursal areas (arrows) and on the floor of the dysplastic pelvis (A). The external and internal surfaces of the pubis bones are raised, grooved, and roughened in response to excessive pull on the muscle attachments that were anchored there.

The femoral head becomes eburnated except for its periphery. In many cases as much as 5 mm of the femoral head is worn away. (See Figs. 83-14 and 83-18.) When subluxation first occurs at 12 weeks and extra compression is applied to the head, the head and neck "drift" downward into a varus angle as the head remodels. (See Fig. 83-9.) As wear shortens the eburnated femoral head, the stress lines change and the femoral neck gradually shifts from the varus angle, when the drift appears, to a short-necked valgus angle or position. (See Fig. 83-18.) The lipping at the ventral border of the femoral head, caused by an accumulation of osteophytes at the junction of the head and neck, remains. These osteophytes, composed of new fiber bone,

extend around the entire femoral head at the junction of the head and neck. (See Figs. 83-14 and 83-18.)

During the development of eburnation, after the subluxation is stabilized, the cartilage surface of the femoral head at the nonarticulating margin of the head and neck becomes thickened owing to lack of contact with the opposing acetabular surface. (See Fig. 83-17.)

In the normal articulating joint, the motion of the joint coupled with its efficient lubricating system ensures that shedding cartilage cells are wiped off; thus, the articular cartilage remains at a stable thickness. The normal articular cartilage is nourished by synovial fluid on the joint surface side and by diffusion of nutrients through the subchondral plate on the other side.(15,89)

In the unstable, subluxated joint, the width of the articular cartilage varies from nothing on the eburnated surface, where compression has 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. (See Figs. 83-14, 83-17, and 83-18.) 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 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. (See Fig. 83-17.)(15,57,83,89) This mechanism, tearing of the Sharpey's fibers, and stimulation of the periosteum result in osteophyte formation. (See Figs. 83-14, 83-15, and 83-17.)

In the subluxated hip, osteophytes rim the femoral head at the junction of the head and neck. (See Fig. 83-16.) 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 where the capsule attaches. Osteophytes also form in the area where Sharpey's fibers are torn.

Subchondral cysts of the femoral head are observed rarely; in humans their occurrence is common in osteoarthritic hips (Fig. 83-21).

FIG. 83-21 Coronal histologic section of part of the femoral head of a man with degenerative joint disease. Articular cartilage is gone and a large subchondral cyst is present. Such cysts are common in humans with degenerative joint disease but are rare in the dog.

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

Discussion

The locomotion of the young dog with severe hip dysplasia may be unsteady, but 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 its feet and after exercise.

It is at this age that microfractures of the dorsal acetabular rim appear. These changes are difficult to demonstrate radiographically, even though the extent of the microfractures varies widely. Since a fracture is a painful lesion, it is logical to assume 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 remodeling and hypertrophic osseous development are continually present in and around the dysplastic hip. Even though these mechanisms are functional during the disease, frequently there is 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, yet there are no physical signs of involvement. In a few instances radiographic lesions are mild but signs of pain are severe.

The significant changes described above are recorded for comparison with the rate and pattern of normal development of the hip joint from birth to maturity.(60) 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.(74)

Section Four: Clinical Manifestations

History

Hip dysplasia is a disease of young animals. Most dogs will be between the ages of 5 months and 12 months at first presentation. A much smaller population will present with first signs as late as 36 months. Young dogs present with joint instability, which may result in abnormal hindlimb gait, thigh muscle atrophy, pain, low exercise tolerance, inability to climb stairs, an audible "click" when walking, or walking with an arched back. Any one (or several) of the above signs may be present. Past pedigree history is of little relevance, since many generations may be normal and still produce dysplastic progeny.

Physical Examination

Physical examination may or may not produce definitive information. Palpation of the hips may result in pain on extremes of range of motion. Pressure on the hip may cause discomfort. Attempts to subluxate the hip should be made either by lifting the hip laterally or by adducting with axial pressure. The latter may force the hip to subluxate. When the limb is then abducted, the femoral head will drop again into the acetabulum and make a "popping" sound referred to as the Ortolani sign.

Many severely dysplastic dogs will subluxate their hips on each step. This can be monitored by walking behind the dog with a hand over each hip. Radiography is needed to produce definitive diagnosis. Old dogs do not present with hip dysplasia. They present with degenerative joint disease of the hips, which is secondary to hip dysplasia as a young animal. Any subsequent workup or treatment is directed toward treatment of the resultant arthritis rather than the dysplasia.

Radiography

Radiographs are the definitive visible diagnostic evidence of hip dysplasia. The diagnostic value of hip radiographs depends upon their quality, that is, proper patient positioning, proper centering of the x-ray beam, proper exposure technique, and proper darkroom technique. Positioning faults and technical deficiencies may

result in nondiagnostic radiographs or the illusion of hip disease where it does not exist. It is unlikely that all of the possible abnormalities associated with hip dysplasia will appear in any single radiograph. An awareness of these changes will, however, aid in perceiving their presence and understanding their significance.

POSITION FOR RADIOGRAPHY

The ventrodorsal position is recommended for hip radiographs (Fig. 83-22). The dog is placed on its back. The hindlegs are extended fully in a caudal direction, held parallel, and rotated fully in a medial direction. When this position has been achieved, the cranial midline of the femora will face directly up toward the x-ray tube.

Symmetric pelvic positioning may be achieved by determining that major palpable anatomical sites, such as the iliac crest and greater trochanter, are equidistant from the cassette surface. Sedation or anesthesia may be required to achieve this position in all but the most cooperative dogs.

An exposure technique should be selected that will produce clearly visible structural detail of the hipbone in the finished radiograph. Radiographic evaluation of the hip may be severely limited by exposure technique inadequacies.

THE NORMAL HIP

The hip is a ball-and-socket joint formed by the convex, hemispherical femoral head and a deep, concave acetabulum. The normal femoral head has a localized surface irregularity where the femoral head ligament is attached. The acetabular articular surface is a peripheral rim that is incomplete ventrally. The acetabular fossa, the second site of femoral head ligament attachment, extends medially and caudally from the articular surface.(45) Hip bone detail should be clearly visible in good quality radiographs.

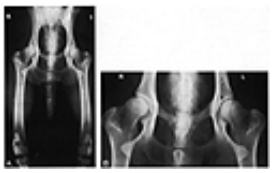

FIG. 83-22 Ventrodorsal hip radiographs of a normal canine pelvis and femora positioned symmetrically. (A) All parts of the pelvis appear bilaterally symmetric. The legs are fully extended and parallel with the patellae appearing over the femoral midline. (B) An enlargement of A. The hemispherical femoral heads fit snugly into the deep, concave acetabulae. The flattened central area of the femoral head is the site of femoral-head ligament attachment. Cranial to this the femoral head contours parallel that of the cranial acetabular walls. This is the normal hip component positional relationship. (cranial acetabular wall -; dorsal acetabular wall ....; joint space J)

The femoral head and acetabular articular cartilage is not discernible as a discrete radiodensity. Articular cartilage thickness and condition can, however, be inferred from hip joint space width and shape and subchondral bone structure.

The hip joint capsule attaches on the pelvis adjacent to the acetabulum and on the femoral neck.(45) The joint capsule is similar in density to the soft tissues surrounding it and does not appear as a discrete radiodensity.

The growth and development of the normal canine pelvis, hip joints, and femora from birth to maturity are described elsewhere in this chapter. A thorough understanding of this description is imperative for assessing radiographically the many variations within the limits of normal hip conformation and differentiating these variations from hip disease. The coronal histologic preparations of normal dog hips (See Fig. 83-6) are of special value in gaining this understanding, since much of what appears in these coronal preparations can be

perceived radiographically. A macerated normal hip joint is shown in Figure 83- 19.

The correct position for hip radiography has been described. Strict adherence to this position is a prerequisite for accurate radiographic assessment of hip structures. Major problems caused by variations from it include distortions of acetabular depth and contours, the illusion of femoral head subluxation, and distortion of the femoral neck-diaphyseal angle. Each of these variations can mimic signs of disease and lead to diagnostic errors (Figs. 83-22 and 83-23).

The normal hips shown in Figures 83-24 through 83-26 should be examined in detail after reviewing Figures 83-22 and 83-23. The reader should correlate the anatomical landmarks and contours of the macerated bone specimens and the coronal histologic preparations with each of the radiographs.

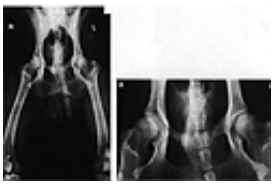

FIG. 83-23 The same dog as shown in Figure 83-22 but positioned asymmetrically. The pelvis and femora are rotated about their long axes, and the femora are pulled laterally. Pelvic rotation in this figure is characterized by the wing of the ilium appearing wider and the obturator foramen less rounded on the right side than the left side. The right acetabulum appears less deep and the left acetabulum more deep than in the same pelvis positioned symmetrically (Fig. 83-22). Lateral femoral rotation causes the femoral necks to appear shorter and the femoral neck-diaphseal angles more obtuse than normal. The distal femora appear bowed laterally, the patellae appear lateral to the femoral midlines, and the patellae appear more medial than normal. In both A and B. lateral femoral rotation has caused the joint space between the cranial acetabular wall and the femoral head to appear wedge shaped (subluxated) rather than of even width as in Figure 83-22 The apparent abnormalities seen in this figure mimic hip dysplasia but are illusions created by faulty positioning of a normal dog. (cranial acetabular wall; dorsal acetabular wall ....; joint space J)

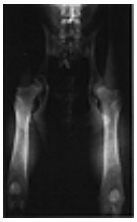

FIG. 83-24 Excellent hip conformation in a 27-month-old female German shepherd. (Courtesy of the Orthopedic Foundation for Animals)

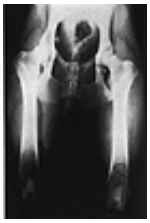

FIG. 83-25 Good hip conformation in a 32-month-old female German shepherd. (Courtesy of the Orthopedic Foundation for Animals)

The coronal histologic preparations and specimen photographs are of special value, since much of what appears in these figures can be perceived radiographically.

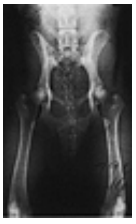

FIG. 83-26 Fair hip conformation in a 24-month-old female German shepherd. (Courtesy of the Orthopedic Foundation for Animals)

THE DYSPLASTIC HIP

The primary radiographic signs of hip dysplasia are a shallow acetabulum and a small, flattened femoral head. The secondary radiographic signs of hip dysplasia are femoral head subluxation and degenerative joint disease of the hip.

The cranial acetabular border becomes less concave; its dorsal border recedes medially. The femoral head becomes less hemispherical than normal; and its size diminishes. The anatomical abnormalities are those of an incompetent ball-and-socket joint, namely a structure inadequate for weight bearing and predisposing to craniolateral and craniodorsal femoral head subluxation, the direction of maximum weight-bearing stress.

The earliest radiographic sign of femoral head subluxation is a wedge-shaped joint space between the femoral head and the cranial acetabular wall. As the femoral head shifts laterally, the joint space widens medially and narrows laterally at the point of maximum head weight-bearing stress against the acetabular rim. (See Fig. 83-29,B.)

Femoral head subluxation is considered a secondary change of hip dysplasia because subluxation may have other causes, including muscle atrophy, traumatic injuries, and leg axial deformities. Indeed, if femoral head subluxation from any cause persists, the acetabulum and femoral head may remodel to resemble the primary abnormalities of hip dysplasia.

Degenerative joint disease is secondary to femoral head subluxation and the associated joint tissue abnormalities. Frequently degenerative joint disease is first recognizable radiographically as a crescent-shaped line of hypertrophic bone at the site of joint capsule attachment to the femoral neck (See Fig. 83-30,B.) Hypertrophic bone spurs may also be seen at the femoral head border, where the joint capsule attaches to the pelvis, and at the craniolateral acetabular border. An increase in width and density of the craniolateral acetabular subchondral bone is a sign of degenerative joint disease that reflects excessive stress placed upon that region by a subluxated femoral head. (See Figs. 83-16 and 83-29, B.) The gross bony proliferations and deformities associated with hip dysplasia and secondary degenerative joint disease are seen in Figures 83-15, 83-18 through 83-20.

Degenerative disease may result from any stresses or diseases that adversely affect joint biomechanical behavior or tissue. The presence of degenerative joint disease of the hip does not by itself constitute hip dysplasia and must not be so construed.

The radiographic and histologic development of the dysplastic hip is described elsewhere in this chapter. A thorough understanding of this description is imperative for assessing the early radiographic signs of hip dysplasia.

THE ORTHOPAEDIC FOUNDATION FOR ANIMALS

Consensus reports on pelvic radiographs from the OFA contain information for serious breeders and concerned owners. The consensus is the result of independent review by three veterinary radiologists and is reported as one of the following categories. The first three are considered within normal radiographic limits for age and breed and are eligible for assignment of an OFA breed number if the dog is 24 months of age or older at the time of radiography. The last four (Nos. 4-7) are not eligible for an OFA breed number.

1. Excellent hip joint conformation (Fig. 83-24): superior hip joint conformation as compared with other animals of the same breed and age.

2. Good hip joint conformation (Fig. 83-25): well-formed hip joint conformation as compared with other animals of the same breed and age.

3. Fair hip joint conformation (Fig. 83-26): minor irregularities of hip joint conformation as compared with other animals of the same breed and age.

4. Borderline hip joint conformation (Fig. 83-27): marginal hip joint Conformation of indeterminate status with respect to hip dysplasia at this time. A repeat study is recommended in 6 to 8 months.

5. Mild hip dysplasia (Fig. 83-28): radiographic evidence of minor dysplastic change of the hip joints.

6. Moderate hip dysplasia (Fig. 83-29): well-defined radiographic evidence of dysplastic changes of the hip joints.

7. Severe hip dysplasia (Fig. 83-30): radiographic evidence of marked dysplastic changes of the hip joints.

Hip joint conformation (phenotype) is the radiographic appearance of the hip joints and is the outcome of interaction between a dog's hereditary makeup (genotype) and its environment. Hip joint conformation can be represented as a range from excellent to very dysplastic with shades in between these two ends of the spectrum. Traits such as this are referred to as "quantitative characters" and are thought to depend upon the interaction of many genes (polygenic).

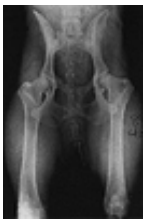

FIG. 83-27 Borderline hip conformation in a 24-month-old female German shepherd. (Courtesy of the Orthopedic Foundation

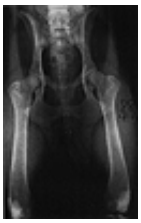

FIG. 83-28 Mild hip dysplasia in a 26-month-old female German shepherd. (Courtesy of the Orthopedic Foundation for Animals)

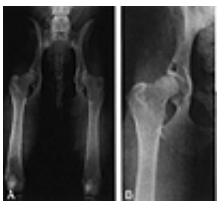

FIG. 83-29 Moderate hip dysplasia in a 24-month-old female German shepherd. (A) The femoral head is subluxated. The craniolateral shift of the femoral head creates a wedge-shaped joint space with maximum weight-bearing stress on the craniodorsal acetabular articular surface. The subchondral acetabular bone at this site has increased in width and density (14), a sign of degenerative joint disease (B). Compare this figure with Figure 83-22. (Courtesy of the Orthopedic Foundation for Animals)

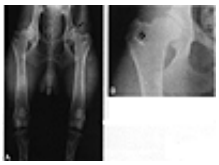

FIG. 83-30 Severe hip dysplasia in a 30-month-old German shepherd. (A) Degenerative joint disease is seen as a crescent-shaped rim of new bone farmed at the site of joint capsule attachment to the femoral neck (Arrows). (B) A hypertrophic bone spur is present on the cranial border (arrow)(A). (Courtesy of the Orthopedic Foundation for Animals)

Modern breeds of dogs vary widely in body size and shape and in pelvic conformation. Because of these

differences, OFA classifications are based on comparisons among other animals of the same breed and age. Knowledge of a dog's pelvic phenotype can be a valuable guide for the breeder in selection against hip dysplasia; it can also be a useful means of estimating an individual dog's potential for an active working life.

Treatment

MEDICAL

Hip dysplasia can be managed in its early stages with enforced rest or analgesics.(67) Enforced inactivity, namely, cage rest, decreases the pressure on the hip, lessens the constant subluxation and rubbing, and may make dogs more comfortable for a short time.

Analgesics or anti-inflammatory drugs decrease the animal's discomfort. Aspirin, buffered aspirin, phenylbutazone, or other noncorticosteroid anti-inflammatory drugs are useful. Dosage must be titered to the individual patient. Most dogs will require medical analgesia for the 6 to 10 weeks of hip remodeling, which results in the greatest amount of discomfort. Chronic administration is rarely necessary. The use of steroidal anti-inflammatory drugs is rarely indicated.

SURGICAL

Many reasons exist for performing surgery on dysplastic animals. One reason is to correct a bony abnormality and thus correct joint incongruity; this can be accomplished by pelvic osteotomy,(26,74) acetabuloplasty, (9,10) derotational femoral osteomy(46,47) or varus femoral osteotomy.(48) Another reason is to totally resect or replace the abnormal bone; this is accomplished by femoral head and neck resections (13,16,37,56,58) or total hip replacement. Finally there are pain-relieving procedures such as myotomy and myectomy.(24,35,58,88) Each of these procedures has specific indications and contraindications that will be discussed below. None of these procedures cures hip dysplasia.

Corrective Osteotomy

Many corrective osteotomy procedures have been developed to treat canine hip dysplasia.(9,10,26) The specific procedures mentioned are described in chapters 42 and 43. All presuppose that by correcting anatomical abnormalities of the hip, a more stable hip joint will result. All osteotomy procedures are aimed at improving joint depth or congruity by placing the femoral head more deeply into the acetabulum or by rotating the acetabulum more laterally to cover more of the femoral head.

Corrective osteotomy must be performed on an immature animal. It is preferable that no major radiographic evidence of bony remodeling be present. If remodeling has occurred, the benefits of osteotomy will be limited, since the nonfitting components of the hip will again remodel into smooth surfaces.

The benefits of osteotomy are varied. In follow-up studies of pelvic osteotomy, it has been found that while the animals improve, the hips become progressively arthritic such that by 2 years after surgery the hip that has been operated upon mimics the untreated side.* No data have been published on long-term follow-up of acetabuloplasty .

Femoral osteotomy procedures have produced very good results that appear to have lasting benefits to the animal; however, no long-term follow-up data have been published. The reformed hip is deeply placed, remodels to a relatively smooth interface, and appears stable enough to allow near normal function. A young dog with hip dysplasia may benefit from femoral varus or derotational osteotomy.(48)

* DeAngelis M: Personal communication, 1973.

Total Hip Replacement

Total hip replacement (Chapter 44) seems to be an ideal procedure.(25,38,49) It provides for better joint stability and alleviates pain. Technically, the success rate of the procedure is improving with time, as surgeons gain more experience. Small and medium sized dogs seem to be better candidates for hip replacement than large breed dogs. The shallow acetabulae, small pelvic muscle mass, and body weight of large breed dogs can lead to prosthesis dislocation. For a more complete discussion see Chapter 44.

Pain-Relieving Procedures

The following procedures are designed for the amelioration of pain. Any improvement in gait results from the relief of pain. These procedures do not increase hip stability and do not cure hip dysplasia.

MYOTOMY. Myotomy was introduced in 1956 by Voss(85) for symptomatic relief of pain due to osteoarthritis of the hip in humans. The Voss procedure involved release of the gluteal muscles by trochanteric osteotomy, tenotomy of the proximal insertion of the adductor muscles, and myotomy of the tensor fasciae latae. This technique has been modified to include section of the iliopsoas muscle as well, with or without gluteal release, known popularly as a "hanging-hip" procedure. A modified version of this procedure, pectineal myotomy, is used in dogs.

What occurs when myotomy is performed around a painful joint? We know from experience in humans that pain disappears immediately after surgery. The same is probably true in dogs. Of greater concern is why pain is gone and will it return as spontaneously as it disappeared.

Since 1956, orthopaedists have postulated and argued as to why a hanging-hip procedure works. Possibly it relieves pressure within the joint; however, preoperative and postoperative pressure readings can be shown to be identical. Blood supply is surely altered, but collateral circulation can be re-established very quickly. Presently, the best answers are that myotomy involves nerve section, which, at least temporarily, can help alleviate pain. Of importance here is the mention of temporary relief of pain, because in humans this procedure is usually of temporary benefit and pain returns in 6 to 9 months. Similarly, the relief seems to be temporary in dogs.

There are explicit reasons for performing a myotomy in the hip region. One of these is symptomatic relief of pain in osteoarthritis. Animals demonstrating gait anomalies due to dysplasia but who have no pain are not candidates for the procedure. These animals will respond as dramatically to aspirin as to surgery. No animal under 12 months of age, unless afflicted by severe osteoarthritis, should undergo a myotomy. Myotomy, specifically pectineal myotomy, does not alter the progression of the disease and does not cure the disease. (8,11,84)

Myotomy can also be beneficial in painful hips following excision arthroplasty. For the same reasons as in osteoarthritis, there will be symptomatic relief of pain; however, there is no guarantee that pain will not return.

The long-term results as well as the current status of the hanging-hip procedure in humans indicate that this is a generally unsatisfactory procedure that has been superseded by intertrochanteric osteotomy and total hip arthroplasty. Interest in this simple procedure in the dog should be tempered by the long experience of similar procedures in humans.

Although sectioning muscles may relieve pain, overzealous use of multiple myotomies may have a detrimental effect on the stability of the hip. It has been postulated that active muscle mass is related directly to the incidence of hip dysplasia,(59,69) and therefore sectioning multiple muscles in dogs with limited muscle mass would not seem advantageous.

PECTINEAL MYO'I'OMY, MYECTOMY, AND TENOTOMY (24,58,85) All refer to the same basic principle: section of a functional pectineal muscle. The pectineal muscle is one of the many adductors of the hip, and its section (high, low, midsection or complete resection) yields the same result as does section of any hip adductor-temporary relief of pain and improvement in gait. It is important to note that the pectineal muscle is not a magic muscle the section of which in itself is going to relieve pain; it is simply one of many adductors, and its release may give results that are no different than those produced by release of another adductor.

ILIOPSOAS MYOTOMY. The iliopsoas muscle is the continuation of the iliac muscle and psoas major. Its insertion on the lesser trochanter of the femur makes it a flexor and external rotator of the hip. It too can be sectioned to produce temporary relief of pain due to osteoarthritis. Better results are obtained if it is sectioned in conjunction with some of the adductors; however, section of only the iliopsoas has a desirable effect.

GLUTEAL MYOTOMY. Release of parts of the gluteal mass will also result in temporary relief of pain. Usually the deep gluteal and deep half of the middle gluteal muscle must be sectioned to obtain an effect.

FEMORAL HEAD AND NECK RESECTION. Removal of the femoral head and neck is an excellent method of pain relief in dogs with hip dysplasia.(78) This technique (described in Chapter 43) effectively removes the cause of pain by eliminating one component of the painful, arthritic joint. However, this procedure is useful only when pain and discomfort are a major problem for the animal. It will not improve gait abnormalities, and it has no place in the dog with totally luxated hips. The end-stage dysplastic dog with dislocated hips has effectively provided its own method of pain relief.

Femoral head and neck removal should be used as an end-point salvage procedure when medical management is no longer effective. Most animals will be vastly improved following the surgery, will function well as pets, and may still be able to hunt on a limited basis. The need for analgesia may remain, but the amount of drug necessary will be greatly decreased.

If bilateral femoral head and neck resections are needed, it is preferable to allows 4 to 6 weeks between the two surgeries. This will permit rehabilitation of the first surgery to begin prior to the second surgery. While bilateral simultaneous surgery is possible, the complication rate is much higher than with two separate surgeries.

CONCLUSIONS

Canine hip dysplasia can cause great discomfort to the animal being considered for treatment and can present the veterinarian with many problems in choice of treatment. The wide indications for various procedures, as quoted in the literature, must be tempered with the knowledge of their long-term results. In many cases these results have not been uniformly good. The best procedures for one animal or clinic may not work in all situations; therefore, a formula should be developed that will allow each case to be judged on its own.

In general, a dog is better with its hip joint than without it, provided it has stability and an absence of pain. Medical management is very successful in most dogs.

Reconstructive osteotomies should be designed to completely stabilize and improve hip conformation. These procedures are best done early but with the full realization that future problems (i.e., degenerative joint disease) may still occur.

Artificial joints will find a place in our armamentarium for those dogs who do not respond to medical management or who are not candidates for osteotomy procedures.

Myotomy procedures should be used only as pain-relieving procedures in dogs with proven degenerative joint disease of the hips. Myotomy is inappropriate in immature dogs or as a proposed "cure" for hip dysplasia.

Femoral head and neck resection, a good tool for relief of pain, should be regarded only as a salvage procedure.

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