Preface

My approach to hip dysplasia was influenced by my preceptor in bone pathology, LENT C. JOHNSON, who hypothesized that hip dysplasia was an example of a biomechanical disease representing a disparity between primary muscle mass and too rapid skeletal growth. He believed further that lag or failure of the muscle to develop and reach functional maturity at the same rate as the skeleton allowed a major joint such as the hip, which depends on muscle power for stability, to pull apart and thus trigger a series of events that ended in hip dysplasia and degenerative joint disease. His help and guidance during the first 3 years of this study are gratefully recognized.

Few students of biological science are trained to look beyond biochemistry and pathobiology to apply two principles fundamental to the architect and mechanical engineer; these are sound structure and sufficient bracing. The application of these principles seems essential to the investigation of hip dysplasia.

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Introduction

This study explores the growth and development of the normal canine hip and compares this with the development of spontaneous and induced canine hip dysplasia. Radiologic, gross, and histologic observations are compared.

Hip dysplasia, a condition in man and most domesticated animals, is a complex abnormality that has resisted a ready solution or easy elimination. This disease has been examined critically since Hippocrates first described it 3000 years ago [4]. Investigators have searched intensively for genetic, chemical or metabolic defects, but the cause has remained obscure. Genetically it is now considered polygenic (multifactorial) [5]. As in most polygenic diseases, there are both major and minor causative factors.

The general cause of hip dysplasia when defined must be broad enough to explain its development, not only in man, but also in all other affected animals. Many genetic and environmental factors can secondarily trigger events that bring about the condition [16–18, 22]. Hip dysplasia, therefore, is not one disease, but many diseases that result in common degenerative lesions of the hip joints [17].

Since neither metabolic nor endocrinologic errors have been found to be involved, the functional mechanisms influenced by biomechanics are impressive [8, 20, 21]. This is especially true in children who have been subjected to unusual environmental practices [13], and in dogs over a critical weight and height, particularly in those dogs with inadequate pelvic muscle mass for their skeletal size [9, 12]. From personal observations of the disease in man and other mammals, especially the dog, I now believe that the logical approach to understanding this musculoskeletal defect is to consider the disease from its broad aspects as one common to man and all domesticated animals and to fuse engineering principles of locomotion, construction, and

function with biology and physiology. 'Joints are, of course, bearings in mechanical systems marvellously specialized for accomplishing their locomotor functions' [17]. Although D'ARCY THOMPSON [21] introduced the field of biomechanics at the turn of the century, it is seldom considered when analyzing disease processes involving articulations.

In all mammalian embryos, the hip is laid down as a single unit from mesenchymal tissue, and it develops normally as long as the components are left in full congruity [17, 19]. The hip is normal at some time in the development of the mammal, and abnormal development occurs only when stresses pull the components apart [19]. In the dog, the hip is normal at birth [7, 11]. Intrauterine stresses are not sufficient to produce incongruity of the hip. The first time such forces are great enough is when the pup begins to take its position to nurse. In the child, however, because of the length and position of the legs and the tightness of the surrounding uterus, abnormal stresses are sometimes placed upon the soft tissues of the hip joint during intrauterine life. Thus, some children are born with unstable hips [2, 14, 16, 23].

The dog at birth is in a stage of transition from intrauterine to extrauterine existence. It is so poorly coordinated that it cannot walk or balance itself, and the powers of muscular detoxification and excretion of wastes are very poor. If the demands for musculoskeletal support exceed the strength at the time, the result is injury, fatigue, or stretching of the muscles and supporting connective tissue that hold the hip unit together [16, 19].

Furthermore, observations of the disease in man, dog, and a number of other mammals for many years have culminated in the conviction that the bony changes of hip dysplasia, regardless of species, occur because the soft tissues do not have sufficient strength to maintain congruity between the articular surfaces of the femoral head and the acetabulum [6, 15]. Few genes so far analyzed affect the osseous skeleton as such. The changes in bone merely reflect changes that occur in the cartilage and supporting connective tissues [3]. In man, coxofemoral instability (identified by the Ortolani click) has been recognized as a diagnostic sign of hip dysplasia [2, 14]. Similar instability has been observed by palpation of the hip joints of the dog at the age of 1–2 months [1]. The development of the bony lesions of hip dysplasia can be prevented in man by flexion-abduction splinting of the legs of the newborn after an unstable hip has been diagnosed [2, 8]. Bony changes characteristic of canine hip dysplasia can be prevented by confinement of young genetically conditioned dogs to small cages where the dogs sit most of the time on their haunches with their hind legs in flexion and abduction [10].

These observations and experiments have made it clear that in very young human and canine subjects with unstable coxofemoral joints, hip dysplasia can be prevented and the instability corrected if the congruity of the components of the hip joint is maintained and if femoral subluxation does not occur. If proper congruity cannot be maintained, the hip joint becomes malformed in a relatively short time.

The changes in the bones and cartilage of the hip are thus the indirect result of failure of the soft tissues to support full congruity of the bony components of the hip. There is no evidence that a primary osseous lesion exists other than the inherited configuration character of the acetabulum and the femoral head [6, 8, 17].

These observations substantiate the two premises upon which this study is based: (1) hip dysplasia occurs in the young child or animal only if hip joint instability and joint incongruity are present, and (2) the disease can be prevented if congruity of the joint can be maintained until ossification makes the acetabulum less plastic and the abductor muscles and supporting soft tissue becomes strong and functional enough to prevent subluxation of the femoral head.

This investigation was undertaken: (1) to establish the growth pattern of the normal canine hip from birth to maturity (1 year) by serial radiographs; (2) to compare the radiologic, gross, and histologic development of spontaneous canine hip dysplasia with that of the normal hip; and (3) to produce canine hip dysplasia surgically.

These experiments were used to study the ideas expressed above and to compare the effects of the various degrees of subluxation and instability of the femoral head with the lesions that developed.

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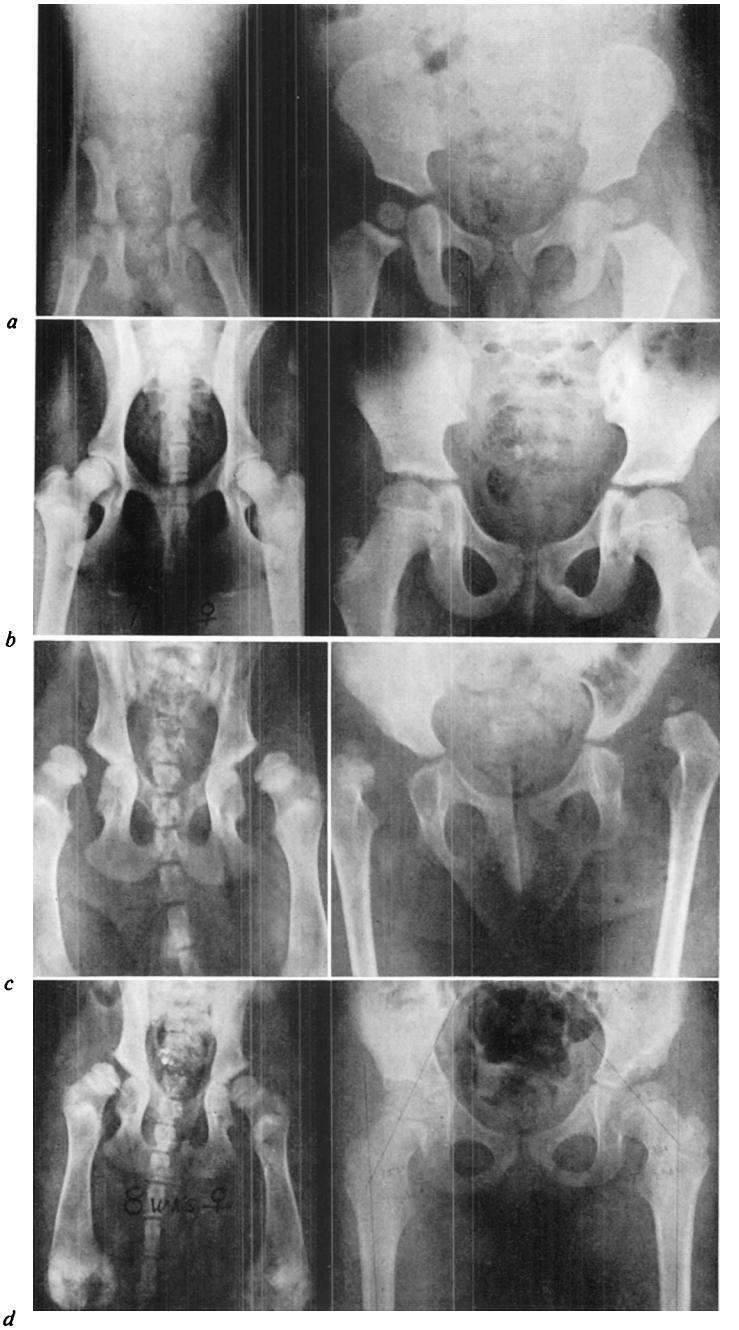
Observations and Research on Hip Dysplasia

Volumes have been written on hip dysplasia in the child, a comparable amount about the dog, and reports of the disease in other mammals are frequent. The literature is too extensive for a general review here. Only the pertinent observations and experiences will be summarized. The disease as it affects man and the dog will be compared where relevant from the viewpoint of prevalence and the genetic, environmental, and metabolic influences. The similarity of the radiographic appearance of both the normal and dysplastic hip joints of man and the dog is striking (fig. 1).

Prevalence

In the human infant in New York there is a frequency of hip dysplasia of 1.3 per 1000 births. This does not include those additional newborns with the Ortolani click as the only sign of hip dysplasia. An untreated population of Navajo Indians at Many Farms, Ariz., was estimated to have a prevalence of 10.9 hip dysplasias per 1000 births. In this Indian population it was highly significant that the occurrence of hip dysplasia decreased when the cradleboard was discarded and the children were diapered [44]. In Bantu tribes, hip dysplasia is relatively unknown [47].

Hip dysplasia has been observed in most breeds of dogs; however, it is a greater problem in some breeds [42]. Which breeds of dogs have the highest prevalence, and why is the disease more prevalent in some breeds? The true prevalence of hip dysplasia among breeds of purebred dogs is not known, but data from the Orthopedic Foundation for Animals on the first 36000 pelvic radiographs evaluated have given insight into answering these questions.



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Differences in the genetic composition of the dog are important in predisposing its shape, size, and general characteristics [53, 56]. The genetic code also controls the metabolic life and, to a great extent, the diseases a dog will have, especially the clinical and metabolic variations that are based on inherited traits. This is not a new concept. In man and dogs, it is well known that there are exaggerated deviations in types and constitutions [48, 49, 53]. These body types influence skeletal, physical, and behavioral characteristics to a noticeable extent. These body types or somatotypes have been classified into three general categories: the ectomorph – light type of body build; the mesomorph – intermediate or average body build (athletic), and the endomorph – heavy type of body build (short, stocky, rounded). In dogs as well as in man, all breeds and individuals exhibit a mixture of variation of specific somatotypes but, within limits, the general classification can be characterized and distinguished [24, 53].

In our early work with canine hip dysplasia, it was discovered in some breeds (July Foxhounds) that almost all were dysplastic, whereas in other breeds (Greyhounds) hip dysplasia was rare. Between these extremes the prevalence of hip dysplasia varied [41]. There was also a strong correlation between body form, size, growth rate, quantity of subcutaneous fat, type of connective tissue, pelvic muscle mass, and the general body type of the different breeds and the prevalence of hip dysplasia [24, 45, 46, 53].

Recently we have identified certain general characteristics of a breed that increase the risk of hip dysplasia [42]. Such correlation was also suggested previously [24]. Certain somatotypes are associated with a high prevalence of hip dysplasia in the dog.

The data used were tabulated from the first 36 000 radiographs received by the Orthopedic Foundation for Animals, which serves as a registry and

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Fig. 1. a Pelvic radiograph of a 4-week-old Greyhound (left) and a 2-year-old child (right). Both have normal hips. *b* Pelvic radiograph of a 7-month-old Greyhound (left) and a 10-year-old child (right). Both have normal hips. *c* Pelvic radiograph of an 8-week-old German Shepherd (left) and a 2⅔-year-old child (right). The hip joints of both are subluxated and dysplastic. *d* Pelvic radiograph of an 8-week-old German Shepherd (left) and a 5-year-old child (right). Both have dysplastic hips. In the dog there is subluxation of the femoral heads and retarded development of the acetabulums. The hip joints are unstable and characteristic of early, severe hip dysplasia. In the child, there is subluxation of the left femoral head and a lack of development of the acetabulum. This is characteristic of hip dysplasia in children.

diagnostic service to identify normal pelvic phenotypes with respect to hip dysplasia in dogs 1 year of age or older. Dog owners from all 50 states and the provinces of Canada have participated in the program, with more than 90 breeds represented. Each radiograph submitted was reviewed independently by at least three diplomates of the American College of Veterinary Radiology. By consensus, each radiograph was classified in one of four categories: normal, near-normal, dysplastic, or repeat study advised. Only breeds represented by more than 100 individual pelvic radiographs were included in the analysis (table I).

The percentage of 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 I). The rankings of the breeds are consistent with those obtained previously from smaller populations [24]. All breeds were screened in a similar manner.

The rankings of the 38 breeds represented were based on tabulation of 35 285 radiographs in breeds with more than 100 individual pelvic radiographs (137–5351 for each breed; table I). The prevalence of hip dysplasia varied from 4.2 to 43.2%, a spread of almost 40% [42]. Neither the prevalence of hip dysplasia nor the rank of the breeds changed appreciably from previous studies when tabulations were published for the first 5000 radiographs [24, 42].

Why dysplasia was more prevalent in some breeds was difficult to assess, but information was obtained by comparing a group of breeds having the highest percentage of hip dysplasia with a group having the lowest percentage. The two groups were compared for body size, body type, and growth pattern (table II).

Fifteen breeds, nine with a low percentage and six with a high percentage of dysplasia, which represented the breeds at the ends of the binominal curve (outside ± 1 SD), were studied as two groups. The mean percentage of dysplasia was 19.2%, and one standard deviation was ± 9.43%. Statistically, 68.26% of the population (representing 23 breeds) fell within ± 1 standard deviation.

The two groups varied widely in appearance and body type but resembled each other in the three categories studied. In the high percentage group, the three breeds at the top and the one that ranked sixth were of the giant type, with various characteristics of acromegalia. The two hunting breeds in this group were long-legged and rangy but had a low muscle mass index, which gave them a high probability toward hip dysplasia. The nine breeds with the

Table I. Pelvic radiographic diagnoses[1]

Breed	Evaluations	Dysplastic %	Normal %	Near normal %	Repeat study %
Saint Bernard	1453	43.2	49.5	2.6	4.7
Newfoundland	338	39.9	50.9	3.2	5.9
Bullmastiff	278	34.5	60.8	1.1	3.6
English Setter	327	32.1	59.0	2.4	6.4
Gordon Setter	218	31.2	62.4	0.4	6.0
Old English Sheepdog	2311	29.9	65.1	1.0	3.9
English Springer Spaniel	611	27.3	67.8	1.8	3.1
Akita	251	27.1	68.5	0.0	4.4
Chesapeake Bay Retriever	300	25.7	68.7	2.0	3.7
Golden Retriever	3028	25.7	68.6	2.1	3.6
Norwegian Elkhound	660	25.6	69.8	0.4	4.1
Rottweiler	660	25.4	70.2	0.4	3.9
German Shepherd Dog	5351	25.1	67.5	2.9	4.5
Giant Schnauzer	215	24.2	70.2	2.3	3.2
Standard Poodle	772	23.6	73.7	0.6	2.1
Brittany Spaniel	824	22.4	74.0	0.8	2.7
Rhodesian Ridgeback	317	19.6	77.0	0.0	3.5
Alaskan Malamute	1121	18.5	77.5	0.5	3.5
Weimaraner	995	18.0	78.7	1.2	2.1
Irish Setter	1614	16.9	79.7	0.7	2.7
Samoyed	1974	15.7	80.7	0.9	2.7
Labrador Retriever	4089	15.5	80.7	1.3	2.5
Vizsla	531	15.2	82.1	1.1	1.5
Great Dane	1325	15.2	81.4	0.2	3.2
Keeshond	244	15.2	82.0	0.8	2.0
Puli	175	13.1	85.1	0.0	1.7
Great Pyrenees	336	12.8	85.7	0.0	1.5
Standard Schnauzer	374	12.6	85.6	0.5	1.3
Australian Shepherd Dog	161	11.2	88.2	0.0	0.6
Wirehaired Pointing Griffon	160	10.6	87.5	0.0	1.9
Doberman Pinscher	377	10.6	86.2	1.3	1.8
Collie	137	8.0	89.8	0.0	2.2
Dalmatian	168	6.5	91.7	0.0	1.8
Irish Wolfhound	235	6.0	93.2	0.0	0.8
German Shorthaired Pointer	839	5.8	93.2	0.0	1.0
Afghan Hound	867	5.3	93.6	0.0	0.9
Belgian Turvuren	171	5.3	93.6	0.0	1.2
Siberian Husky	867	4.2	93.9	0.0	2.0
Total	35 285	19.2	77.2	0.8	2.8

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Table II. Analysis of phenotypes[1]

Low prevalence	High prevalence
Body size	
Weight and size of ancestral dog	Giant type, weight 2–3 times that of ancestral dog
Bones trim and small in diameter	Bones coarse and large in diameter
Head narrow and long	Head broad and oversized
Feet small and well arched	Feet oversized and splayed
Body type	
Slender ectomorphic (racing, hunting, fighting) type	Stocky acromegalic, endomorphic type
Thorax deep and narrow	Thorax barrel shaped
Skin trim and tight	Skin loose, thick, and wrinkled
Body fat limited	Body fat in excess
Muscles well developed and hard	Muscles deficient in quantity and tone
Joints stable, with well-developed ligaments and tendons	Joints unstable; ligaments and tendons weak
Gait well coordinated, fleet and light footed	Gait slow, awkward, heavy footed, poorly coordinated
Growth pattern	
Pups small at birth	Early rapid growth
Slow growing	Fat and heavy for age
Late physical and sexual maturity	Early physical and sexual maturity
Appetite good but self limiting	Appetite indulgent

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lowest percentage of hip dysplasia were similar in the three categories studied.

Body Size

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

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

Body Type

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

Of the high-risk group, the four breeds of the giant type were not only two to three times the size of the ancestral dog, but their body conformation was heavy, round, and stocky. Acromegalic characteristics were present to some extent in all four breeds. The skin was thicker than that in the other group; it lay in folds over the head and neck. At dissection, the thickened skin was infiltrated with fat. Fat was also abundant in the subcutaneous and fascial spaces and commonly accounted for 5–10% of the weight of the soft tissues of the hindquarters. In comparison with the other group, the muscles were less prominent and less developed. Fat also was infiltrated into the tendons and ligaments. The fibers of these two structures were smaller in diameter than those of the low-risk group. The gait of the giant breeds was less graceful and slower than that of the smaller breeds.

Growth Pattern

The group of breeds with the highest percentage of hip dysplasia grew and matured more rapidly than did those in the low-risk group. We have observed this in several studies. Starting at birth, this group gained rapidly. The pups of these breeds were aggressive eaters, both as they nursed and as they began to take supplemental food. In one study, the growth of four young Greyhounds was compared with that of four young German Shepherds. The growth curves of the Greyhounds maintained a uniform scope for the first 14–16 weeks. By the age of 24 weeks, the German Shepherds had gained more than 60% of their adult weight. In another study involving 222 German Shepherds, we found that 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. We have observed the same rapid rise in weight in other breeds of the high-risk group for dysplasia [37].

The 38 breeds, when ranked according to the highest prevalence for hip dysplasia, with few exceptions exhibited a gradual shift from the poorly muscled and poorly coordinated, acromegalic type giants at the top, to the lowest percentage of hip dysplasia at the bottom, characterized by the breeds that were sleek, tight skinned, highly coordinated and well muscled (table I, II). These correlations and observations support previous findings that the poorly muscled and coordinated breeds have a high percentage of hip dysplasia, whereas the well-muscled and highly coordinated types are relatively free of the disease [41].

As in man, in the breeds of dogs that have acromegalic characteristics, the muscular elements generally fail to keep pace in their development with the overgrowth of bone. Furthermore, some acromegalic breeds have long dense hair that conceals body conformation, and the lack of muscle mass in these dogs is not realized until comparisons are made with better muscled dogs.

Selection for acceleration in growth created dogs with excessive fat and weight at an early age. This has resulted in lowered dynamic and biomechanical efficiency of the hip joint [9]. The young dog that carries excessive weight runs the risk of over-extending the supporting soft tissues, and injury to these tissues results in pulling apart (subluxation) of the joint components. This results in changes that have been recognized as hip dysplasia. This is not a new concept. It was pointed out as long ago as 3 centuries that 'Muscles and bone are inseparably associated and connected, between muscle and bone there can be no change in one but it is correlated with changes within the other' [56, 57].

In 6313 pelvic radiographs representing 19 large breeds in Sweden, Finland, and Denmark, 2801 dogs (44.3%) were dysplastic [16].

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, their food came from nursing during the first few weeks. Then 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 [43]. 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 (for example, excessive body weight) [41, 42].

Genetic Influences and Heritability

Few genes so far analyzed directly affect osseous structures [10]. The shape of bones reflects changes by biomechanical stresses [9].

Among the Navajo Indians, multigenetic factors in the etiology of the disease were decidedly influenced by associated child-rearing environmental methods [44]. Hip dysplasia in this tribe has decreased in the past 2 decades when the cradleboard was discarded and the newborn were diapered. These changes allowed the legs of the infant to be free, flexed, and abducted. These positions are favorable to hip joint congruity and stabilization [44].

In the dog there has been no clear-cut pattern of inheritance recognized [16–19]. This meant many genes were affected, and polygenic traits were subject to environmental modifications. New data have substantiated these findings [18].

The spread of hip dysplasia centers around the genetic transmission and heritability of certain 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 the causes [41].

Critical evaluation of the heritability of hip dysplasia has been made in the German Shepherd, in 244 individual offspring from 54 full sub-families. In this 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' [18]. 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 disease [19].

The heritability of canine hip dysplasia is in the same range as milk yield for dairy cattle, pig growing and maturing weight at 180 days, and egg production in certain varieties of chickens [18, 19]. It was concluded, therefore,

that individual selection be considered a suitable and practical method for controlling hip dysplasia [18, 19].

Effects of Breeding Programs

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 [22].

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 [6, 31]. Similar decreases in prevalence have occurred in another controlled breeding program in a colony of guide dogs (Seeing Eye, Inc., Morristown, N. J.).

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 ½ years the incidence of hip dysplasia was lowered from 39% to less than 17% [38]. The male dogs in this colony had a wide variation in their ability to transmit normal hips to their progeny. For example, for one dog with radiographically normal hips at 2 years of age, only 8.7% of his progeny developed hip dysplasia, whereas for another dog with similar radiologic evaluation mated to the same bitches, 37.8% of his pups developed hip dysplasia [13].

In 1966 the Schäferhunde Club of Switzerland, cooperating with the veterinary schools of Berne and Zurich, developed a hip dysplasia control program for breeders. At the start, all dogs with greater than grade-2 dysplasia, and since 1970, grade-1 dysplasia, were refused breeding registration. During the control period, the number of dogs disqualified for breeding annually has declined appreciably [8].

In our work with breeders where small numbers of subjects were involved, every report, without exception, has indicated a decline in the number of affected dogs in successive generations when only dogs and bitches with normal hips were mated.

Environmental and Man-Made Influences

Embryologically, articular joints are differentiated as units *in situ* from a mass of skeletal mesenchyme [54]. 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 [51].

In man, intrauterine stress has been cited as contributing to hip dysplasia, particularly if the fetus is positioned with the legs in adduction and extension. In breech birth, 16% of neonates had an Ortolani click in one or both hips [33]. The practice of suspending an infant by its heels to induce breathing at birth may have contributed to stretching the supporting tissues of the hip. The Ortolani click was twice as frequent at birth in the first born as in successive siblings. In the first born, the uterus has greater muscular tone, and there is less placental fluid [47, 58]. The Ortolani click was less frequent when the birth was cephalic as such infants were carried in the uterus with the legs in flexion and abduction [58, 59]. The Ortolani click, when present, occurred unilaterally in 60% and bilaterally in 40% [33, 59].

Hip dysplasia in man was rarely associated with teratological abnormalities. Other hip abnormalities distinctive from dysplasia, however, were frequently associated with such deformities as club foot, hyperextension of the knees, spinal deformities, arthrogryposis multiplex, and chondroosteodystrophy [14].

In the young child, the position of the legs during infant care was found to be highly important to normal hip development [5, 45, 46, 49]. Abduction and flexion of the legs had a stabilizing effect on the hip joints. The square diaper favored greater abduction of the legs than did three-cornered diapers. 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 [45, 49]. 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 [44].

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 [30]. These observations show that both environmental and hereditary influences are important [17, 29].

In the dog, the hip joints are normal at birth [27, 40]. The long bones of the pup are short during prenatal life, and mechanical stresses that bring about dislocation of the femoral heads are minimal.

Teratological abnormalities of the joints are rare in the dog, except for congenitally dislocated elbows and an occasional club foot deformity. Congenital malformation of hips is also rare.

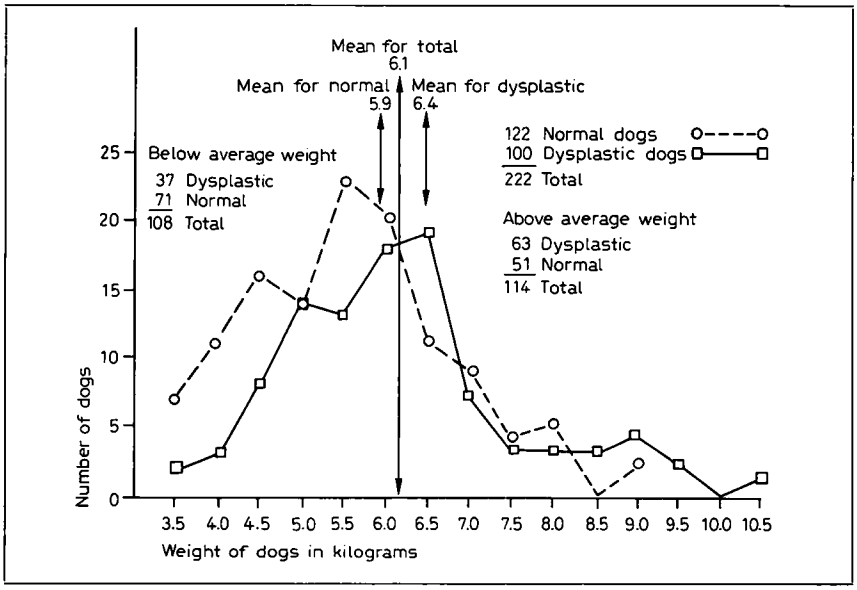

Fig. 2. Incidence of hip dysplasia in 222 German Shepherds. Data reproduced with permission from J. Am. vet. med. Ass. *145:* 661–668 (1964).

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 (fig. 2) [37].

The 222 pups at 60 days had a mean weight of 6.1 kg; the mean weight of 100 dysplastic pups was 6.4 kg, and the mean weight of the 122 pups with normal hips was 5.87 kg (fig. 2, table III). This correlation was statistically significant ($P>0.04$). Despite the averages, 37 out of 100 dysplastic dogs were under average weight; only five out of 122 normal dogs were over average weight [37].

The mean weight at 60 days of age according to sex for all males was 6.29 kg (fig. 2).

The weight of dysplastic males averaged 6.59 kg compared with 6.08 kg for normal males. The weight of the dysplastic females averaged 6.29 kg

Table III. Average weight (kg) of normal and dysplastic dogs

Sex	Normal pups	Dysplastic pups	Average
M	6.08	6.59	6.29
F	5.60	6.29	5.94
Both sexes	5.87	6.44	6.12

compared with 5.6 kg for normal females. This indicated that the heavier pups at 60 days within each sex group had a significant tendency to become dysplastic. In some instances, normal males were heavier than dysplastic females. Therefore, it was not simply weight *per se* but weight within each sex group that was associated [37].

These data suggested a number of indirect genetic aspects influencing the rate of hip dysplasia. The aggressiveness in nursing may be inherited as well as 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 [37].

The first subluxating stress on the hips occurs when the pup supports itself while nursing, and the hindlegs are in forceful adduction and extension [37]. The heaviest pups were the more aggressive, worked the hardest while nursing, and spent the most time feeding [37].

Pelvic Muscle Mass

Data indicate that there is a positive correlation between the amount of pelvic muscle mass and the prevalence of hip dysplasia. In three large breeds of dogs, the Greyhound is relatively free from hip dysplasia; over half of the German Shepherd Dogs are affected with hip dysplasia, and nearly all the July Foxhounds are dysplastic [41].

Ninety-five dogs (20 Greyhounds, 63 German Shepherds, 12 July Foxhounds) were radiographed, killed, skinned, and the pelves dissected to determine differences between these three breeds. These breeds were dissimilar in such characteristics as color, length of hair coat, and shape of ears, but similar in body type and skeletal structure, particularly in the hind quarters. All the dogs had attained most of their growth (aged 5 months to 10 years), were in good health and flesh, and none was extremely thin or fat.

At dissection, the right and left side of each pelvis muscle group was weighed separately in order to compare the total weight, the weight of the

Table IV. Dogs grouped by degree of hip dysplasia[1]

Group	Breed	Age range	Sex	Weight kg	Hip joints	Muscle mass index	
						minimum and maximum	mean
1	Greyhound	5 months to 10 years	12 F 8 M	12.7–29.5	normal	11.7–16.3	14.2±1.6
2	German Shepherd Dog	1 year to 3 years	4 F 3 M	17.7–30.0	normal	10.3–13.2	11.4±0.8
3	German Shepherd Dog	6½ months to 3 years	6 F 3 M	23.6–40.0	near-normal	10.3–11.4	11.0±0.1
4	German Shepherd Dog	5 months to 8 years	26 F 21 M	11.4–34.1	dysplastic	5.2–12.0	9.0±1.4
5	July Hound	6 months	6 F	13.6–19.1	dysplastic (subluxated)	7.4– 8.5	7.9±0.1
6	July Hound	6 months	1 F 5 M	13.6–17.3	dysplastic (luxated)	4.4– 6.5	5.6±0.2

[1] Reproduced with permission from Am. J. vet. Res. *124:* 769–777 (1967).

individual muscles, and the weight of each side of the pelvis. A 'pelvic muscle mass index' was then calculated for each dog from the following equation:

$$\frac{\text{Weight of pelvic muscles (kg)}}{\text{Total body weight (kg)}} \times 100 = \text{pelvic muscle index}$$

This equation took into account the differences in total body weight of each dog [41].

From the general observations it was determined that the Greyhound had a much larger muscle mass when compared with the German Shepherd, and, in turn, the pelvic musculature of the German Shepherd was considerably greater than that of the July Foxhound. The angles of the pelves and bone size and shape were similar in all three breeds. This suggested that significant differences lay in the muscle size and weight among these three groups [41].

The 95 dogs were first considered as a general population and secondly as allotted to six groups by breed and degree of hip dysplasia. The hips were classified as normal, near-normal, or dysplastic. Group 1 consisted of 20 Greyhounds. None was in racing condition at the time of the dissection, although 13 had raced, and seven had not raced or received training. All the hips were normal. Group 2 consisted of seven dogs, six German Shepherds and one half-breed Shepherd. Six dogs had received training as sentry and guide dogs (normal hips). Group 3 consisted of nine German Shepherds. These dogs had been selected for breeding but were discarded because of disposition. All had normal or near-normal hips. Group 4 consisted of 47

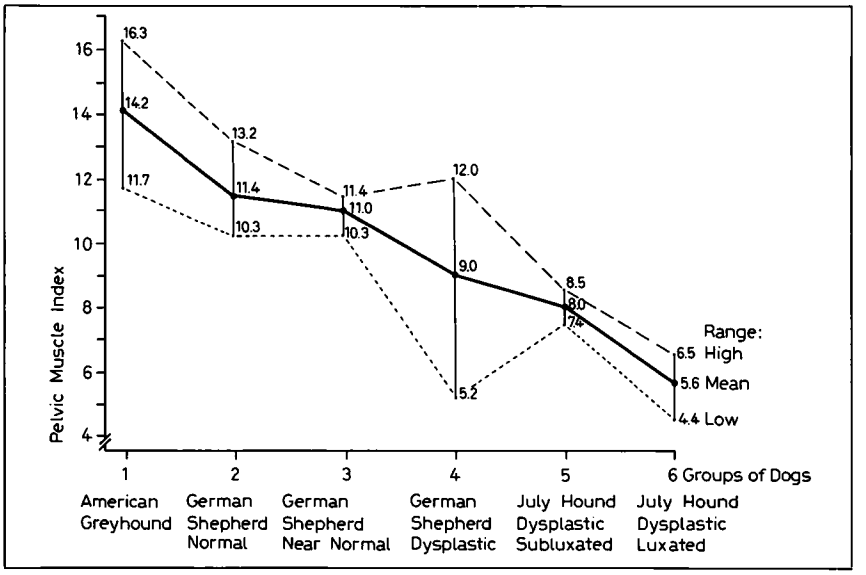

Fig. 3. Distribution of means and ranges of pelvic muscle mass indexes for six groups on the pelvic muscle mass index scale. Reproduced with permission from Am. J. vet. Res. *124:* 769–777 (1967).

German Shepherds. All were raised by us or given to the project, and all were dysplastic. Group 5 consisted of six July Foxhounds. All were raised in a kennel and were dysplastic (subluxated). Group 6 consisted of six July Foxhounds. All were raised in a kennel and were dysplastic (luxated). The data on these 95 dogs (six groups) are given in table IV and figure 3.

From these data, critical points on the pelvic muscle mass index scale have been established: a point above which hip dysplasia rarely occurs (10.89), and a point not far from this, below which hip dysplasia always occurs (9.0) (table V).

These data further emphasize that hip dysplasia encompasses biological 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 [56]. This basic rule pointed out many years ago strongly illustrates why a low foot stool fits solidly on the floor and the tall stool of the same area wobbles when supporting weight [56]. Similarly, it has been found that dogs less than 30.5 cm tall and less than 11.3 kg in weight (Dachshund) are relatively free from hip dysplasia. On the other hand, at least half the large dogs, 34 kg or more in weight and over 50.8 cm in height, are affected with dysplasia [39].

Table V. Expected incidence of hip dysplasia

Index	
14.2	All dogs have normal hips, and the disease was unreported
12.17	All dogs have normal hips, but the disease was reported in some siblings
11.63	Probability of 94% that dogs with this pelvic muscle mass would have normal hips
10.89	Probability of 86% that dogs with this muscle mass index would have normal hips
9.00	All dogs have some degree of dysplasia
8.00	All dogs have badly dysplastic hip joints
5.6	All dogs have hip joints that were luxated

The Greyhound is relatively free from hip dysplasia although he is over 50 cm in height and weighs from 22.7 to 34 kg. This dog was not bred specifically to have normal hips, but it has been rigidly selected for muscle power and speed for almost 3000 years. The Greyhound serves little purpose other than coursing, and those selected for breeding are those with the greatest pelvic mass and speed, less subcutaneous and fascial fat, and coarser tendon and ligament fibers. These are the ingredients that favor speed but also normal hips [39]. The lack of these qualities favors an unstable hip joint [24].

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, for example, the child, dog, and cat, to walk at birth [59].

There is evidence that the wide range of acetabular and femoral changes occurring in hip dysplasia are the consequences 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 [7, 27]. Consequently, the degree of subluxation in the young may be influenced by subnormal muscular function. In man, the possibility of iliopsoas muscle spasm in the infant has been explored [28, 30].

In the adult dog, we have used the light microscope 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 [39, 41].

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 acetabulums. This observer further suggested that if the pectineus were cut in the dog at an early age, the occurrence of hip dysplasia would be greatly lowered [4].

A causal relationship between the pectineus muscles and hip dysplasia could not be established in one experiment using the pelvic muscles from Labrador Retrievers, German Shepherds, Alaskan Malamutes, and Beagles [26]. 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 had 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, 7]. The available evidence does not support the concept that abnormal pectineus muscle behavior is a cause of hip dysplasia [25].

Developmental myopathy with type II fiber hypotrophy in the pectineus muscles of very young dysplastic German Shepherds has been described [7]. These investigators failed to establish a relationship between this muscle change, joint laxity, and dysplasia but have suggested this possibility. In their findings, the small fibers stained as type I (white) and the larger fibers as type II (dark) with an enzyme stain. 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 [7]. 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 [23]. The appearances of the atrophied muscle caused by a cut nerve and of the immature muscle are similar [23].

Another investigator working with intercostal muscles of young normal rats has reported a similar muscle fiber hypotrophy. This hypotrophy was considered normal, and it was believed to be a delay in maturation that was within the expected natural range of development [23].

Influence of Inheritance and Selection on Pelvic Muscle Mass

The question has been asked repeatedly whether in the Greyhound the potential for having greater pelvic muscle mass was inherited or whether the pelvic muscle mass was acquired through exercise associated with training for coursing.

Seven of the 20 Greyhounds reported in table IV had never been trained or exercised outside of their pens. These had a mean pelvic muscles mass index of 13.67 as compared with a mean of 14.2 for the Greyhound group. One Greyhound raised by us was confined to a cage a cubic meter in size from 30 days old until killed at 5½ months. His pelvic muscle index was 14.9 (fig. 4) [41]. This is good evidence that muscle mass is inherited.

Two German Shepherds with normal hips were put through the Sentry Dog Training Program of the Armed Forces and were then killed at the end of training because of sub-standard performance. Their pelvic muscle mass indices were 11.7 and 12.0, respectively. This was above average for the German Shepherds with normal hips, 11.4, but was lower than the range of the Greyhound group that had received no training (fig. 5) [41]. Although the rigid training of both the German Shepherd and the Greyhound was about equal, the German Shepherd never acquired the muscle development maintained by the Greyhound.

A significant finding relative to differences in muscle mass was also observed in differences in the pelvic tissues. The size of the individual muscles determined by weight has already been discussed. In addition, the fascia of the Greyhound was thicker and heavier than that of the German Shepherd and July Foxhound. The fibers of the tendons and ligaments were also larger and coarser. Relatively little or no fat was present in the fascial planes and under the skin. The absence of fat was also observed in the Greyhounds raised in the cage and in the pen. The consistent finding of these differences in tissues of the pelves in instances where exercise and training could not have been an influencing factor shows that inheritance and selection contribute to the mass and structure of the tissues and hence to occurrence of normal hips. In the selection of the German Shepherds and July Foxhounds, there has been less and sometimes no regard for the musculature, speed, or hindquarter power [37, 39].

Metabolic Influences

Sex

In humans, the female was affected with hip dysplasia four to eight times as often as the male [14]. In the dog as many females as males were affected.

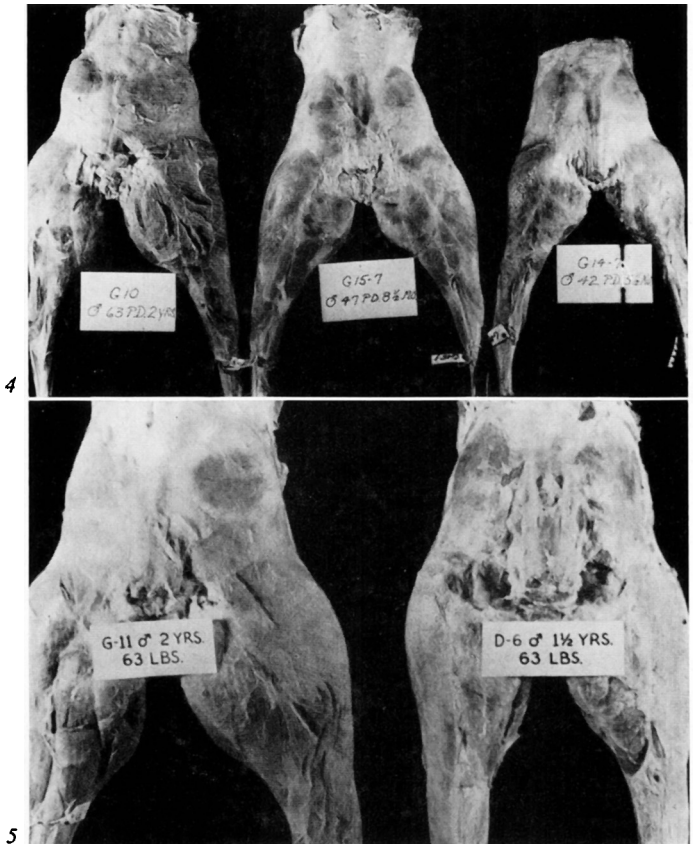

Fig. 4. Pelves of three Greyhounds illustrating influence of heredity compared with that of exercise on the quantity of muscle mass. Pelvis on the left from a dog trained for racing. Other two pelves from dogs of racing Greyhound parents. Pelvis in center from a dog that weighed 21 kg at 8½ months. The dog had been restricted to a small pen and not trained during life. Pelvis on the right from a dog that weighed 19 kg at 5 months and had been restricted to a 3 × 3 × 3 meter cage the last 4 months of life and not exercised. Muscle mass is more dependent on heredity than training. Reproduced with permission from Am. J. vet. Res. *124:* 769–777 (1967).

Fig. 5. Pelvis from a racing-trained Greyhound (left), weight 29 kg and the pelvis of a German Shepherd (right), trained for Army sentry duty, weight 29 kg. The German Shepherd has a limited amount of pelvic muscle mass compared with the Greyhound. The training of both dogs was extensive and rigorous. Reproduced with permission from Am. J. vet. Res. *124:* 769–777 (1967).

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 [37].

Chemical and Hormonal Influences

Pelvic tissue relaxation is a well-known physiological phenomenon 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 respond sufficiently to relax pelvic tissues on the hip joints [27, 36].

The urine of newborns was examined to see if there was a correlation of 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, 55]. The present conclusion is that hormonal influence is not associated with the development of congenital hip dysplasia in man or animals [1–3, 45, 55].

In the dog it has been possible to increase the occurrence of hip dysplasia by giving relaxin to newborn pups, and to produce hip dysplasia in the Greyhound [11, 27, 32, 36]. 'It does not prove, however, that estrogens have anything to do with etiology and pathogenesis of spontaneously occurring hip dysplasia' [12]. There is no evidence that estrogen levels within the biological range have a relationship to the incidence of hip dysplasia in dogs [12].

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 [25, 26].

Inborn metabolic errors of chemical or hormonal origin have not been found in hip dysplasia of either man or the dog [25, 26].

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 [39].

Production and Prevention

In addition to the production of hip dysplasia by the use of excessive doses of estrogens, hip dysplasia results from a number of mechanical and traumatic alterations.

In a group of mongrel pups, metal prostheses were substituted for the femoral heads; these were made in shapes that varied from normal femoral heads. Some were larger and round, some were larger and oval, some were larger and square. In each instance the acetabulum changed shape and size to accomodate the contacting femoral head. When the femoral head was removed the acetabulum cavity filled with new fiber bone [50].

Another group of investigators performed osteotomies on the femurs of young mongrel dogs and fixed the legs in extreme anteversion with intramedullary pins. The position of the femoral heads altered the congruity between the femoral heads and acetabulums by producing subluxation. This resulted in immediate remodelling changes and beginning degenerative disease that resembled hip dysplasia [15].

In newborn pigs, a hindleg was placed in extension and adduction with a plaster cast. Subluxation of the femoral head and dysplastic changes in the acetabulum resulted almost immediately. Dysplastic remodelling stopped, and immediate improvement in shape of the joint occurred when the plaster cast was removed and normal motion and position of the femoral heads was restored [15,49].

The same results occurred when a similar experiment was performed on young rabbits [58]. I have produced subluxation and luxation of the femoral head in both the rabbit and the dog by applying a cast to the leg that held the stifle in extension.

Traumatic coxofemoral luxation is followed by remodelling and degenerative joint disease. When healed, these changes are generally indistinguishable from spontaneous canine hip dysplasia. When traumatic coxofemoral luxation occurs the joint capsule and teres ligament are always ruptured. Even though the luxation is reduced, the joint never regains the former congruity and stability because of the torn supporting tissues. Fractures of the acetabulum, femoral head, and neck trigger similar remodelling and degenerative joint disease for the same reasons of incongruity and instability [39].

Acquired deformities of the femoral head bring about remodelling and degenerative joint disease, which when healed leaves changes indistinguishable from hip dysplasia [39].

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

remodelling 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 [50, 59].

In the young dog, confinement to a small cage (1 m^3) where the dog spends most of his time sitting on his haunches (abductor-flexed position) will prevent the development of hip dysplasia in the young dog genetically conditioned to develop hip dysplasia [39, 40].

Summary

Hip dysplasia affects man and all other domestic mammals. In man, 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 [5, 9, 20, 21, 26, 47, 50]. Consequently, the disease has been designated as polygenic or multigenic. 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 [20, 21].

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 [35]. No other malformations are associated with the disease [52], which signifies that hip dysplasia is 'many diseases that end up with common degenerative lesions of the hip joint' [51].

Inborn metabolic errors of chemical and hormonal origin are not evident [34, 58]. The quantity of hormones needed to produce hip dysplasia experimentally far exceeded those that could be produced by any biological system [34, 36, 57].

A causal relationship between muscles and soft tissue defects or pathological changes other than lack of muscle mass or strength has not been established [25, 26].

Experimentally, hip dysplasia may be produced in many ways [27, 36, 47, 50, 58, 59]. These include any circumstances that contribute to an unstable hip joint, that is, adductor forces, lack of muscle strength, chemical relaxation of the pelvic soft tissues, traumatic injury to the hip joint, and overload-

ing 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 remodelling and degenerative joint disease.

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Growth and Development of the Normal Canine Pelvis, Hip Joints and Femurs from Birth to Maturity: A Radiographic Study[1]

WAYNE H. RISER[2]

INTRODUCTION

This is a radiographic, gross and microscopic study of normal hip development from birth to maturity in the Greyhound. A clear understanding of the normal development pattern is imperative as a rational basis for comparison in evaluating radiographic changes associated with disease and injury of the hip.

LITERATURE REVIEW

There is a scarcity of reports dealing with the detailed prenatal development of the hip. The most thorough investigation describes how the limb buds are composed of mesoderm and appear as small protrusions on the lateral sides of the newly formed spine (12). This work stresses that it is possible for all parts of the hip to form within the joint enclosure from the designated primordia. Development of the hip proceeds normally as long as the structures differentiate within the range of balanced neutral forces and allow the acetabulum and femoral head to remain in full congruity (12).

More attention has been given to the development of joints and limbs after birth (14). In discussions of the canine skeleton, the Greyhound was the breed studied most frequently (2, 10, 11).

The anatomy of the adult dog has been described. The most extensive details are available in a recent American text (5).

MATERIALS AND METHODS

Four female purebred Greyhounds were studied. A 1-year-old male Greyhound was also included to complete the data on growth and development from birth to maturity.[3] The parent stock was retired from racing. The bitch whelped in our kennel. The pups were weaned at 30 days of age.

Four females from one litter were chosen for serial radiographs of the pelvis. Females were selected rather than males to avoid having the os penis interfere with the radiographic image of the pelvis. The pups were subjected to short caudectomies at birth for the same reason. One was designated at random for serial radiographic study. The other three served as controls for comparing weight, size and growth, and for replacement if the one being radiographed became ill or her development varied from the three controls. They were kept with the mother until weaning. The four females were then penned together away from the remainder of the litter.

Starting at the day of birth, radiographs were taken of the pelvis and femurs of the

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[3] None of the Greyhounds used in this study received training for racing, but all Greyhounds were from racing stock.

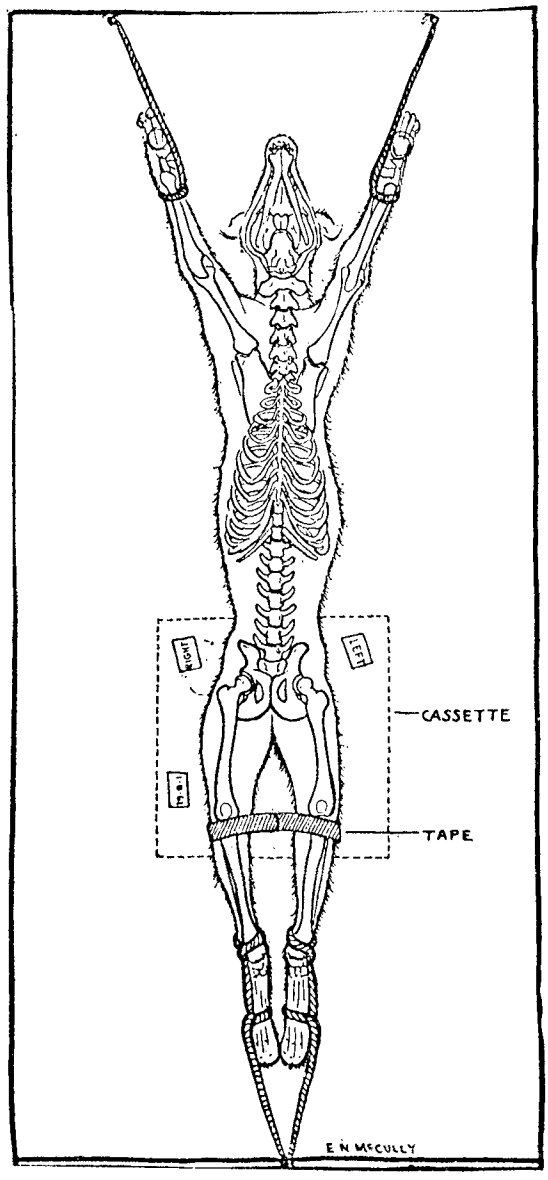

Fig. 1. Drawing of a dog secured in the extended ventro-dorsal position for radiography of the pelvis. (*This figure published with permission of The American Animal Hospital Association*).

selected dog twice daily (morning and evening) for the first 4 weeks of life, then daily for the next 26 weeks until the dog was 30 weeks old (seven months). At seven months, 95% of the growth in bone length was completed. A 1-year-old male Greyhound was included for comparison and to complete the data on the growth pattern of the pelvis and femurs.

Radiographs of the pelvis and femurs were taken with the hind legs extended to the maximum as described previously (4). In this position the pelvis and femurs were parallel to the long axis of the body and relatively close to the cassette when radiographed, thus minimizing bone magnification and distortion (Fig. 1).

The dogs were weighed at weekly intervals from birth to maturity at 40 weeks, after which they were euthanized.[4] The 1-year-old male Greyhound was also weighed and radiographed.

There were no significant differences in the growth rate, size, shape and weight of the four females (the one radiographed and the three controls).[5] At 10 weeks of age, however, the dog being radiographed developed an acute respiratory illness and was euthanized. She was replaced by a sibling. When the illness was noticed, the dog and her replacement were the same size and the pelvic radiographs were indistinguishable. The sibling was in good health and grew normally during the entire experiment. It is believed that the data obtained from these dogs represents an average normal growth, weight and size pattern for the Greyhound breed, and that the bone and body size of the male and female of this breed varied insignificantly (males 26 to 30 kg, females 23 to 27 kg). References to mean measurements for size and weight of this breed were not found.

Tracings of serial radiographs of the pelvis, femurs and hip joints were made to determine the changes in size and form during the growth period.

Weight Gain

The dogs made a steady weight gain from birth through the growth period (Fig. 2). A standard dry kennel food[6] was fed *ad lib* from the time the dogs began eating

[4] One dog died at 10 weeks of age.

[5] The Section of Radiobiology, Armed Forces Institute of Pathology, reported the amount of X-radiation exposure was well below the dose required to retard the rate of bone growth in the young dog.

[6] Ralston Purina Dog Meal, Ralston Purina Company, St. Louis, Mo.

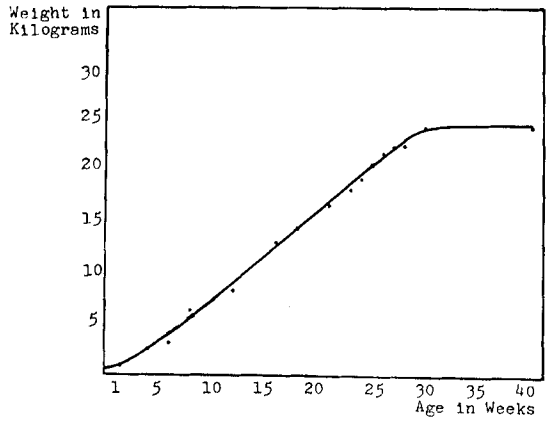

Fig. 2. Weight chart of the Greyhound dogs in kilograms per week.

until they were euthanized. The growth curve increased steadily for the first 28 weeks of life after which it leveled or straightened. The rise of the curve was the greatest between the 10th and 28th weeks. After the 28th week, the weight in the three remaining dogs was constant until they were euthanized at 40 weeks (Fig. 2). The weight recorded on the chart at each date was a mean weight of the four dogs for the first 10 weeks and for the three remaining dogs for the remainder of the experiment. The dogs were in good health and ate well during the experiment except for the illness of the one. The mean weight obtained was 22 kg when the weight

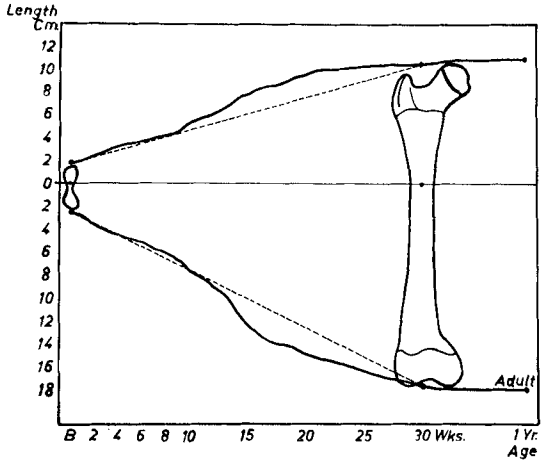

Fig. 3. Chart drawn from radiograph overlay tracings of the femur at intervals from birth to one year of age. The "O" point on the vertical axis is the level of the nutrient foramen.

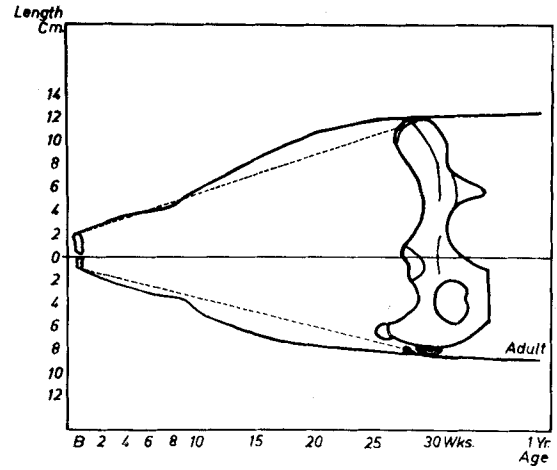

Fig. 4. Chart drawn from radiograph overlay tracings of one-half of the pelvis of a dog at intervals from birth to one year of age. The bipolar growth plate between the ilium and ischium served as the center point of growth. B refers to size at birth.

curve leveled by the 31st week. This represented approximately 90% of the total normal weight for the average adult female Greyhound. The growth in weight followed and correlated well with the increase in size of the femur and pelvis over the same period (Figs. 2–4). These increases were consistent for all dogs. The surviving dog that was radiographed and the two controls stayed in the same weight range.

Longitudinal Growth of the Femur

The nutrient artery around which the bony nutrient foramen later forms is at the center point of a long bone when the cartilage mold is laid down in the skeleton of the fetus (5, 6). 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 physes (growth plates)[7] is radiopaque. The cartilagenous epiphyses are radiolucent.

[7] The lucent line seen radiographically between the epiphysis and metaphysis is known as the physis.

In this study, at 12 days of age, the proximal and distal epiphyses were ossified sufficiently to be radiopaque. At 6 weeks the nutrient foramen was identified. In retrospect, with these landmarks, it was possible to identify the position of the nutrient foramen and the ends of the bone on the radiographs taken previously following birth.

At birth, the estimated total length of the femur was 3.5 cm with 1.5 cm proximal to the nutrient foramen and 2 cm distal to it. The femur maintained a uniform weekly length increase (Fig. 3). When the measurements were charted, the weekly growth as indicated on the chart for the proximal portion maintained an increase at a slope of approximately 20° until growth slowed at the 30th week. Between the 10th and 30th weeks the slope increased from approximately 25° to 30°. The distal portion of the femur grew faster. Its growth rate was at a slope of approximately 50°. A 1:1.5 growth ratio was maintained between the proximal and distal portions of the femur from birth to adulthood.

At 30 weeks of age the over-all length of the femur was 23.5 cm with 9.5 cm proximal to the nutrient foramen and 14 cm distal to it. This length represented 95% of the total adult femoral length and was within 0.9 cm of the length of the femur of the 1-year-old 26-kg male Greyhound whose femur measured 24.4 cm with 9.9 cm proximal and 14.5 cm distal to the nutrient foramen (a ratio of 1:1.5).

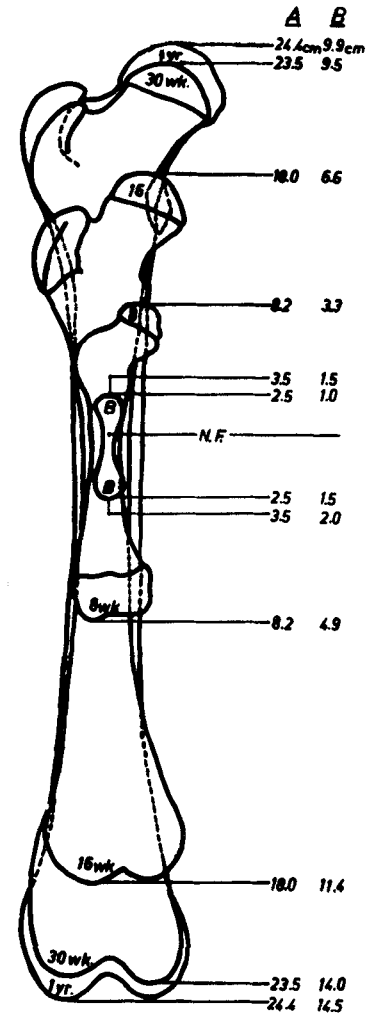

Fig. 5. Chart drawn from radiograph overlay tracings of a femur from birth to one year of age. The nutrient foramen (NF) served as the center point of growth from which measurements were made. (A) Total length of femur at the specified ages. (B) Length of proximal and distal ends of femur from the nutrient foramen (NF) at the specified ages.

Shape of the Femur

At birth, only the metaphyses and diaphyses of the femurs were visible radiographically and these were of hour-glass shape (Figs. 3, 5). The distal end was slightly larger in length and width than the proximal one. The ossification of the head (proximal epiphysis) and distal condylar epiphysis was sufficient to be visible at 12 days of age, and the shape of the head and condyles was evident by the 13th day.

Radiographically the center of ossification for the femoral head had the appearance of a small opaque circle after ossification began. By the fourth to fifth week the epiphysis appeared to be attached to the proximal end of the femur except for a thin radiolucent line which was the physis (Fig. 6). The proximal surface of the head was uniformly rounded initially. By the sixth to seventh week a notch appeared on the medial surface of the femoral head where the *ligamentum teres* attaches. The

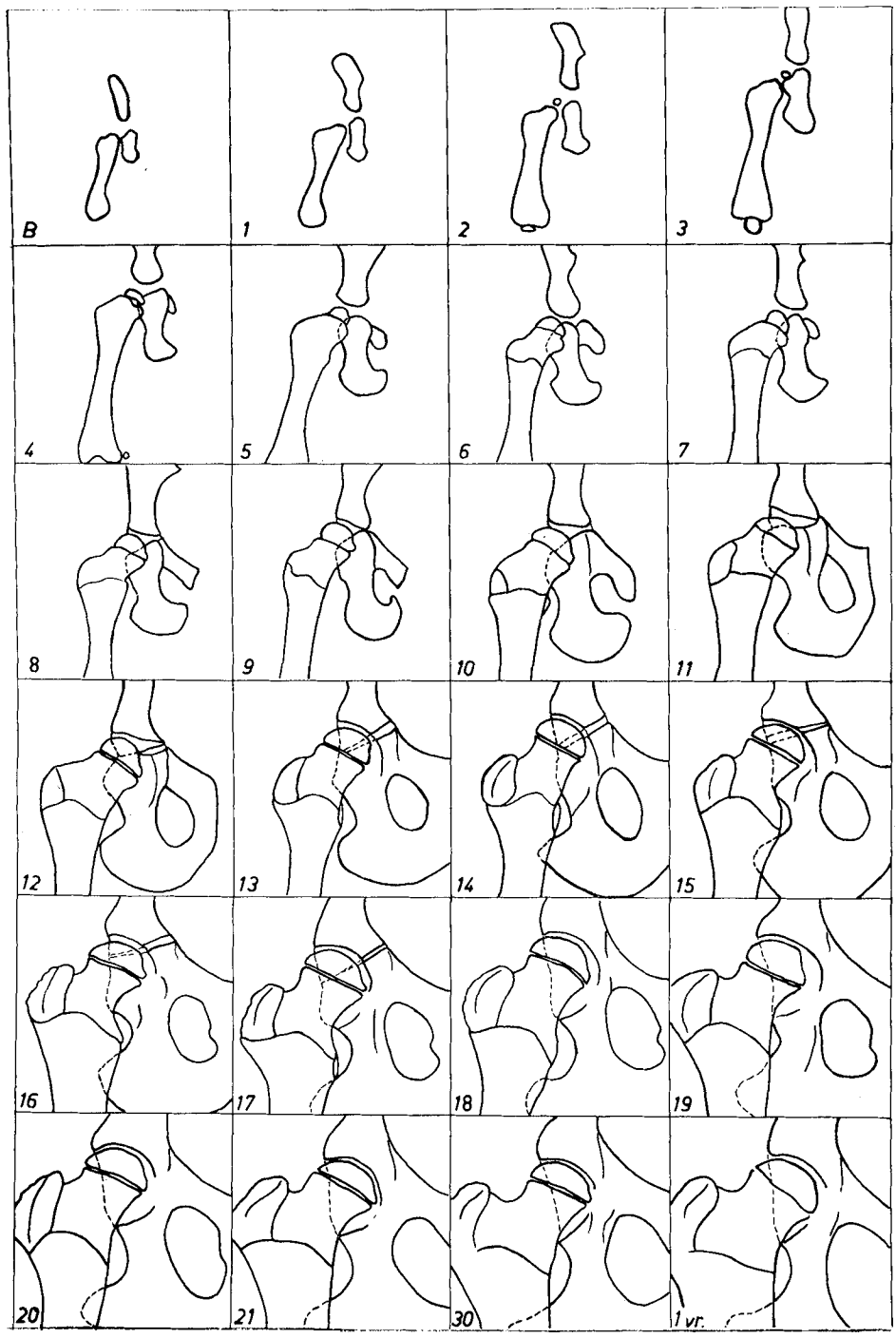

Fig. 6. Drawings copied from radiograph overlay tracings of normal hip joint growth and development from birth to one-year-old. The number with each drawing indicates the age of the dog in weeks.

diameter of the proximal physis was more than twice the height of the proximal epiphysis.

Physes (Growth Plates)

These radiolucent structures are composed of columns of cartilage which lie parallel to the long axis of the femur and the direction of forces (either compression or traction) exerted upon this bone.

The femur has three physes. The two proximal ones once were joined, but separated when the femoral head and greater trochanter moved away from each other as a result of the pulling forces of *gemelli*, the *obturator externus* and the *obturator internus* muscles (3). 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 five months of age. Then the width of the cartilage space narrows gradually. It disappeared radiographically between 11 and 14 months of age (Figs. 3, 5).

The physis at the distal end of the femur is modeled to withstand compression, shear and torsion forces. It has a stud-like surface with four prongs which insert into the epiphysis of the distal condyles. 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 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 on the ventrodorsal radiograph in this study was 135°. This neck angle was constant from birth through all ages of development (Figs. 3, 5).

The anteversion angle of the femoral neck[8] was 0° to 20° with a mean of 10°. It was obtained from the measurements of 32 macerated femurs of 16 mature Greyhounds. A number of other Greyhound pups were also examined from birth to adulthood. The anteversion angle at birth was 0°. It increased gradually with age.

The trochanter and femoral head in the embryological state 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*, *obturator externus* and *gemelli* muscles (2). The greater trochanter is extended by the dorsal and medial pull of the three pairs of gluteal muscles. In this study the trochanter became visible radiographically between the 8th and 9th weeks of life. The lesser trochanter on the medial side of the femur appeared as an apophysis and was composed of cartilage during the early weeks of life. It became identifiable radiographically at 10 to 12 weeks. When the physes closed (11 to 13 months) this process attached (as a single piece) to the diaphysis of the femur (Fig. 6).

Femoral Shaft

The metaphyses developed and extended proximally and distally as the columns of cartilage cells of the physes matured and distintegrated, new trabeculae ossified and the bones lengthened. Both the length and diameter of the femoral shaft increased in diameter by six to seven times during growth (from 3 to 17.5 mm). The length increased from 3.5 to 24.5 cm (Fig. 5). There was a direct correlation between growth in length and diameter.

Femoral Condyles

The shape of the condyles at the distal end of the femur varied little during growth. The increase in size was in proportion to the remainder of bone.

Growth of the Os Coxae (Innominate Bone)

The innominate bone is united with its contralateral fellow to form the pelvis.

[8] The anteversion angle is the angular relationship of the long axis of the femoral neck to a line drawn through the femoral condyles in a coronal plane.

This consists of the ilium, ischium, pubis and acetabular bones. Only portions of the ilum and ischium were visible radiographically at birth (Fig. 6).

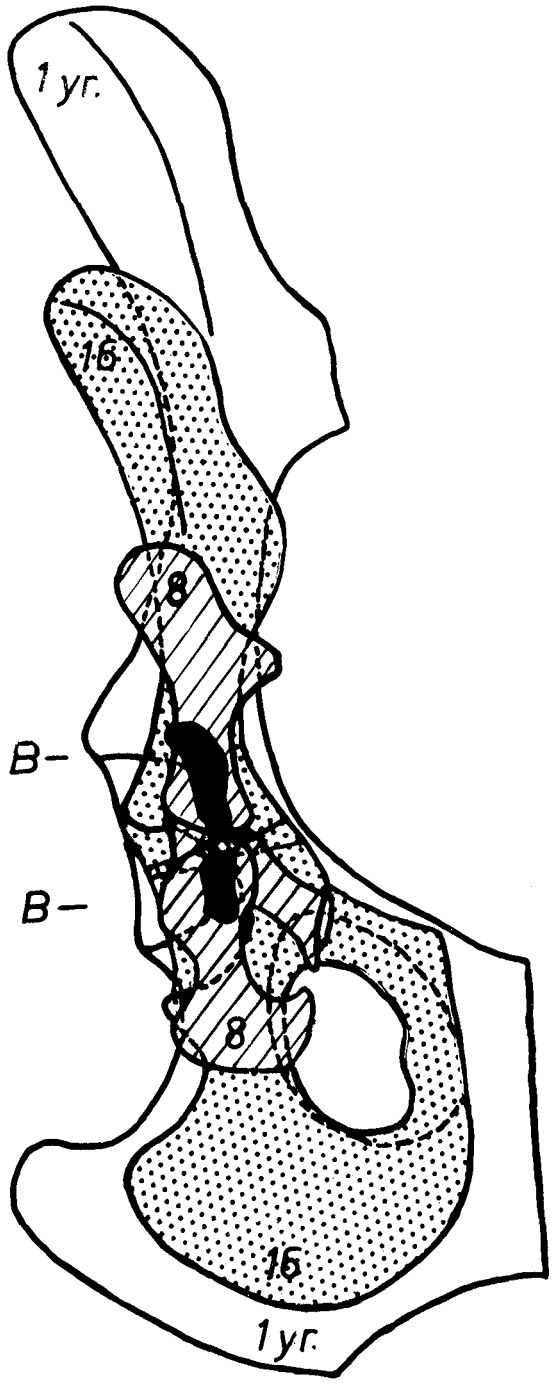

Fig. 7. Chart drawn from overlay tracings of radiographs of one-half of the pelvis of a dog at birth, eight weeks, 16 weeks and one year old. The bipolar growth plate between the ilium and ischium served as the center point of growth.

At birth all of the bones are present. The hip joint, with the acetabulum and femur, is functional and stable (Fig. 6). The bones constituting the hip joint are cartilaginous and radiolucent at birth except for portions of the ilium and ischium (Fig. 7).

Longitudinal Growth

Cartilagenous physes unite the ilium, ischium and pubis. These physes are functionally bipolar, *i.e.*, enchondral ossification takes place on both sides of the cartilage strip in the areas which unite the ilium, ischium and pubis. By the 28th week practically all of the enchondral growth of the pelvis has been completed. An additional increase of 2 cm in total length (1 cm at each end) is added as the tuber ilium and tuber ischium are stimulated by traction of the attached muscles (3).

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 size increase of the *os coxae* was uniform during the growth period. When plotted on a graph the ilium growth slope was 30° and the ischium was 20° (Fig. 4). Between the 10th and 30th weeks both slopes increased somewhat until the 29th week, when skeletal growth slowed abruptly. The growth patterns of the *os coxae* and the femurs are almost identical if the upper and lower curves of the *os coxae* are reversed (Figs. 3, 5).

Ilium

This scapula-shaped bone is divided into a wide cranial part known as the wing, and caudal compressed part which forms the cranial half of the acetabulum. Midway on the medial surface is a powerful synarthrosis which unites the pelvis with the sacrum. The growth lines, the shape of the ilium and the angle (slope) at which the ilium is secured to the sacrum vary 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 became visible rapidly within the next 20 days. Even by the first week the body had lengthened and the caudal end had become paddle-shaped and hooked as the tuberosity and ramus continued to ossify. The obturator foramen became evident when the pubis and ischium ossified at the seventh week.

Pubis

The pubis appeared radiographically at the fourth week and could be identified with its contralateral mate by the ninth week. The pubic symphysis and obturator foramen were visible by the 11th week (Fig. 6).

Acetabulum

The acetabulum is a cotyloid lunate cavity created by the fusion of the ends of three bones: ilium, ischium, 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 was masked by the head of the femur and could not be distinguished until the 12th week when the acetabular cavity had ossified sufficiently to give an adequate radiographic image (Fig. 6). A Y-shaped strip of endochondral cartilage united the ilium and ischium at the dorsal rim of the acetabulum. This was the last area to ossify. A secondary center of ossification located on the craniodorsal acetabular rim was visible sometimes on ventrodorsal pelvic radiographs of dogs between the 11th and 14th weeks of age (Fig. 4).

Relationship of the Femoral Head and Acetabulum

When the head was first recognized as a radiopaque dot at 12 days, it was positioned well within the visible bony boundaries of the acetabular cavity. As the bones of the cavity and head mineralized sufficiently to be recognized, approximately two-thirds of the globe of the femoral head lay within the acetabular cavity. This relationship did not change as the area ossified. As ossification progressed, the radiolucent non-ossified cartilaginous spaces narrowed (Fig. 6).

Gross and Histological Development of the Normal Hip

The hip joints of all dogs are normal at birth (6, 7). The joints continue to develop normally as long as full congruity is maintained between the acetabulums and femoral heads (12).

The most valuable insight into the appearance of the normal hip was 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. Figure 8 is a photograph of six normal hip joints. The first is from a dog 12 days of age, a time when the epiphyseal ossification begins. The others illustrate various stages of development at 4 weeks, 8 weeks, 16 weeks, 24 weeks and finally the hip from a dog over two years of age.

In these six photographs the dominant feature is the display of balance, symmetry and grace 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 present 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.

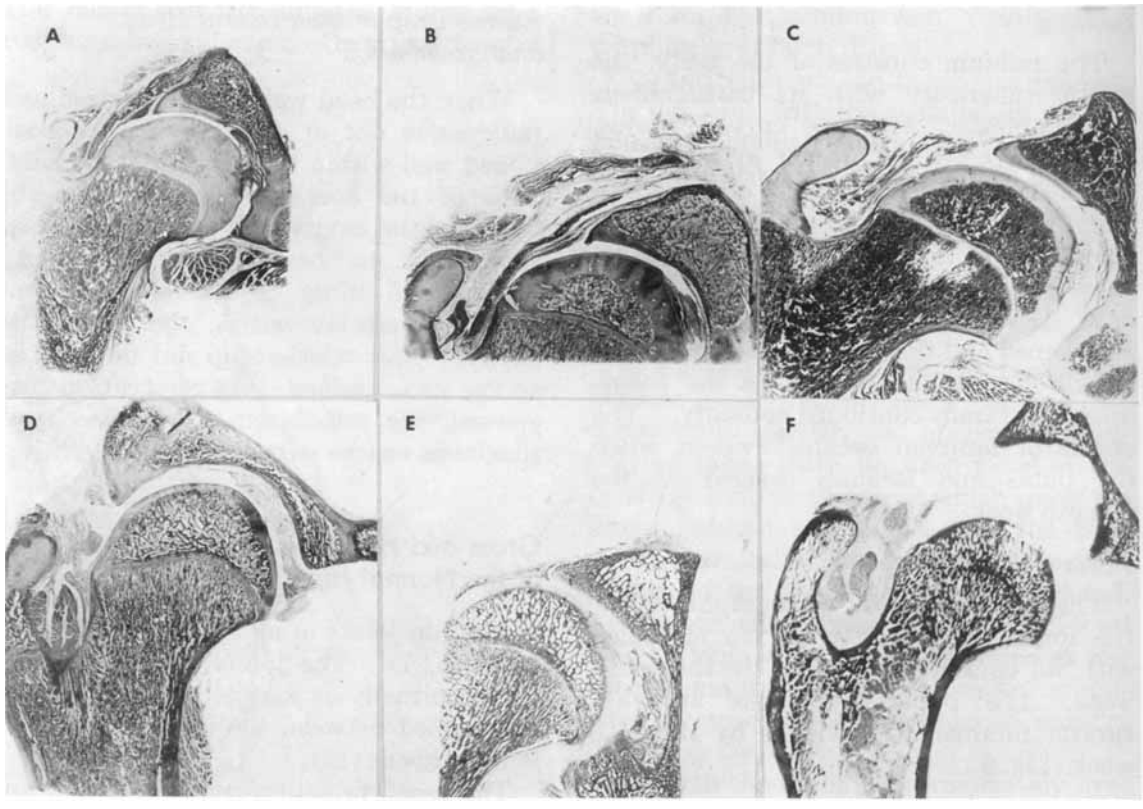

Fig. 8. Coronal histological preparations of normal hips of dogs, (A) 12 days, (B) four weeks, (C) eight weeks, (D) 16 weeks, (E) 24 weeks and (F) two years. These illustrate the normal development and maintenance of the hip joint. At all stages of development and maturity there is an achievement of functional balance, symmetry and congruity between the femoral head and the acetabular cavity. (Excessive separations between the femoral heads and acetabula are preparation defects.) The entire articular surface of the acetabulum and femoral head are maintained in full congruity wih 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, remodelling or wear.

In all six illustrations there is symmetry and grace in the outline of the bones. 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. Such joints have functioned within the parameters of neutral forces.

The effects of balanced forces are well illustrated in the hip joint section of the two-year-old racing Greyhound (Fig. 8F). 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. 8). Histologically, the bones composing the acetabular fossa are thin with a minimum of trabecular bone (Fig. 8). The dorsal portion of the acetabular cavity is extended sufficiently to cover two-thirds of the femoral head (Fig. 8). The cortices and trabeculae of the dorsal rim are thin. The acetabular rim (beak) is sharp and pointed (Fig. 8F).

Histologically, the proximal end 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. 8F). 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 was balanced sufficiently so there were 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 (Fig. 8F).

The long axis of the diaphysis and the neck form an angle of nearly 135°. The graceful 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.

In Figure 8 the acetabular rims (beaks) 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, grace 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 (13). Except for the racing and toy dog types, there is a great chance that the canine hip will develop abnormally (9).

The morphologic characteristics of the complex hip structure leave no serious question that biomechanical behavior is the prime influence in the growth of this joint (3). The display of symmetry, grace and balance in the femoral head and neck, the trochanter and the acetabulum of the normal hip is impressive when coronal histological sections are studied. The six photographs of this joint representing all ages from shortly after birth to late maturity depict how these qualities are maintained (Fig. 8). The photograph of a highly-trained racing dog retired after more than two years of grueling work displays no evidence of joint instability, wear, remodeling or degeneration (Fig. 8F).

The laws that control bone and soft tissue dynamics control the development of the hip. What are these laws? Newton's law of neutral forces when applied to biological tissues means that a joint is in functional equilibrium when all forces upon that joint mutually neutralize each other both in intensity and direction. Wolff's law introduces the concept of bone transformation in that changes in function of a bone are attended by alterations in its internal structure. It applies to cancellous and cortical bone (1).

In all six coronal sections of the hip (Fig. 8) there is no overgrowth of bone and the cortices are thin. Such joints are functioning within the parameters of neutral forces. There is no evidence of unbalanced forces having caused alterations in the cancellous or cortical structures of the bones making up the hip.

It has been stated previously that the hip joints of all dogs are normal at birth (6–9). They continue to develop normally as long as full congruity is maintained between the acetabula and femoral heads (12). If the joint components are pulled apart, development follows an abnormal course which correlates with the degree of incongruity (9). The biomechanical balance necessary for development of normal hips is demonstrated in this report.

SUMMARY

The body growth rate and weight gain, femoral shape and length, *os coxae* shape and length and shape of the normal hip joint as seen in the Greyhound have been recorded from birth to maturity. The relationship of the femoral head to the acetabulum is appraised radiographically. Since the growth of the pelvic skeleton of the Greyhound closely resembles the normal

radiographic appearance of the hip joints of many large and giant breeds, it provides a basis for comparison with abnormal hips.

The development of the normal hip is followed from birth to two years in histological coronal profile sections made through the proximal femur and acetabulum. The sections were taken from dogs at 12 days, 4 weeks, 8 weeks, 16 weeks, 24 weeks and 2 years of age. In all stages of development and maturity there is an achievement of functional biomechanical balance, symmetry and congruity between the femoral head and acetabulum.

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ZUSAMMENFASSUNG

Die Körperwachstumsrate und Gewichtzunahme, die femorale Form und Länge, die *oscoxae* Form, un Länge und Form des normalen Hüftgelenkes, wie sie beim Windhund gesehen werden, sind von Geburt bis zur Reife aufgezeichnet worden. Das Verhältnis des femoralen Kopfes zur Gelenkpfanne wird radiographisch ausgewertet. Da das Wachstum des Beckenskelettes des Windhundes sehr stark der normalen radiographischen Erscheinung der Hüftgelenke vieler grosser und Riesenzüchtungen ähnelt, ergibt es eine Basis für einen Vergleich mit abnormalen Hüften. Die Entwicklung der normalen Hüfte wird verfolgt von der Geburt bis zu zwei Jahren in histologischen Kronenprofilschnitten, die durch den obersten Teil des Femurs und der Gelenkpfanne gemacht wurden. Die Schnitte wurden entnommen von Hunden von 12 Tagen, vier Wochen, acht Wochen, 16 Wochen, 24 Wochen und zwei Jahren Alter. Bei allen Stadien von Entwicklung und Reife gibt es die Erreichung von einem funktionellen, bio-mechanischen Gleichgewicht, Symmetrie und Kongruität zwischen dem femoralen Kopf und der Gelenkpfanne.

RÉSUMÉ

On a enregistré chez le lèvrier, de sa naissance à sa maturité le taux de croissance du corps et le gain de poids, la forme et la taille du fémur, la forme et la taille de *l'os coxa* ainsi que la forme et la taille de l'articulation normale de la hanche. On a évalué radiographiquement la relation de la tête fémorale par raport à l'acétabule. Etant donné que la croissance de l'ossature pelvienne du lèvrier ressemble particulièrement à l'aspect normal radiographique des articulations de hanche de maintes races d'animaux de grande taille ou géants, cela nous fournit une base pour procéder à la comparaison avec des articulations anormales.

On a suivi de près, de la naissance à l'âge de deux ans le développement de l'articulation normale dans des sections de profil coronal histologique faites d'un bout à l'autre du fémur et de l'acétabule. Les sections ont été prises sur des chiens à l'âge de douze jours, quatre semaines, huit semaines, seize semaines, vingtquatre semaines et deux ans. A tous les stades de developpement et de maturité il se produit un équilibre biomécanique fonctionnel, une symétrie et une congruence entre la tête fémorale et l'acétabule.

Vet. Pathol. *12:* 279–305 (1975)

The Dysplastic Hip Joint: Radiologic and Histologic Development

Canine hip dysplasia is a complex disease. It is a concentration of factors from a pool of genetic weakness and environmental stresses that falls into a programmed pattern of progressive remodelling and degenerative joint disease. The degree of involvement varies from minute changes in the bone structure to total destruction of the hip joint. Theories regarding cause, heritability, treatment and control vary widely.

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

This study follows the preceding line of reasoning and is a description of the radiographic and anatomicopathological changes leading to hip dys-

plasia. In essence the pathogenic sequence of this disease as it occurs spontaneously is an experiment done by nature, and the description of the development of the normal hip serves as the control [11].

Literature Review

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

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

Materials and Methods

Four German Shepherds born to dogs with well-defined hip dysplasia were radiographed weekly from birth to maturity using methods described previously [13]. After birth they were subjected to short caudectomies to prevent the tails from interfering with the radiographic image of the pelvis.

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

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

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

Results

Birth to Thirty Days of Age

Eighty-seven dogs from birth to 30 days of age were dissected. Their appearance was compared to normal Greyhound dogs of the same age [11].

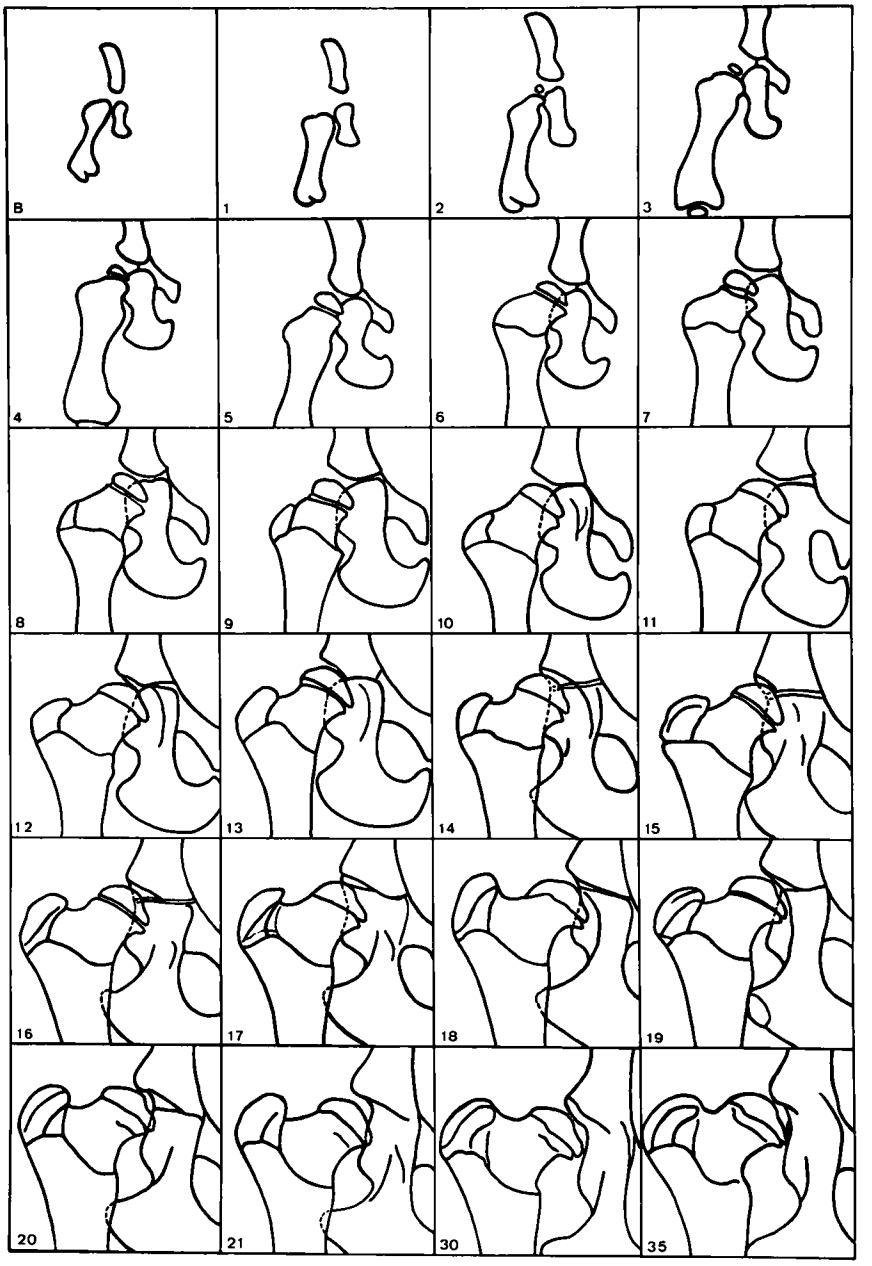


Fig. 1. Drawings copied from overlay tracings of hip joint radiographs of a dog during growth and development of hip dysplasia from birth to 35 weeks of age. The numbers with each drawing indicate the age of the dog in weeks.

Eighty-four of these appeared normal. In three that were 30 days of age, the teres ligaments of the hip joints were edematous, a few ligament fibers were torn, and capillary hemorrhage dotted the surface of the ligaments at the point of the tears. These changes were considered the first that might be linked to hip dysplasia.

From the dissection of the hips of these young dogs, it appeared that the teres ligament is largely responsible for holding the femoral head in place for the first month. For the first 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 1–2 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 were femoral head subluxation and a lag in the development of the craniodorsal acetabular rim. These were noted by the 7th week (fig. 1). At this time subluxation could be recognized by palpation. Grossly the joint capsule was stretched, but the tissue of the capsule had not changed in appearance. The teres ligament was also longer but did not appear to be stretched. Histologically the contour of the acetabulum was greater than that of the femoral head and the craniodorsal rim was no longer sharp.

Sixty to Ninety Days of Age

Radiographically, changes were dramatic. Femoral head subluxation increased. The head no longer fit deeply into the acetabulum. The lag of ossification at the craniodorsal acetabular rim was increased (fig. 1).

In affected, anesthetized dogs, the femoral head could be subluxated an estimated 0.5 cm. When the hips of these dogs were dissected and the muscles were removed, the joint capsules were slightly thickened and stretched to a width that allowed the femoral heads to be extended laterally to the acetabular rims. When the joint capsules were opened, the dorsal acetabular rim was rounded from the 10 to 2 o'clock position; the teres ligaments were pulled ventrally away from their attachment to the dorsal dome at the acetabular fossas; the teres ligaments were swollen; the articular cartilage on the dorsal surface of the femoral heads was worn and roughened where it contacted the acetabular rims when the femurs were subluxated laterally;

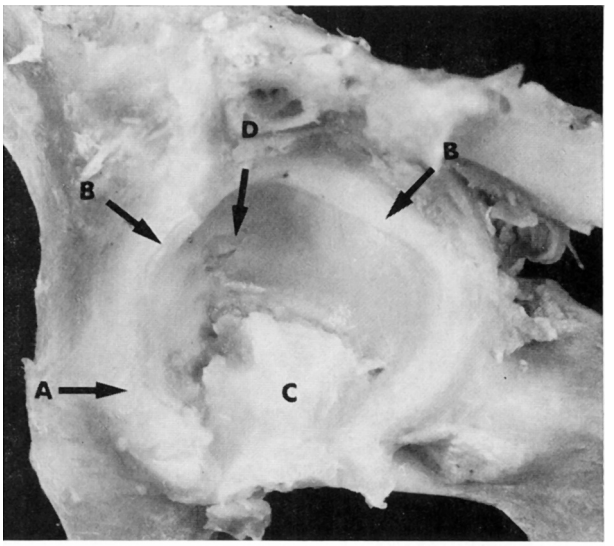


Fig. 2. The acetabulum in a 5-month-old dysplastic dog after the femoral head was removed by cutting the capsule and teres ligament. Early stages of severe hip dysplasia are evident. The joint capsule is thickened (A). The dorsal acetabular rim between 10 and 2 o'clock is rounded and uneven. The attachments of the joint capsule area are damaged and pulled away from the margin. The arrows (B) are placed at 10 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).

and flecks of unidentified 'coagulated' material were present in the joint cavities. These changes were the result of abnormal mechanical forces of traction and compression associated with developing subluxation.

Progressive changes were recognized as the dog became older. Radiographically, by 11–12 weeks the greater trochanter was bent medially in the subluxated hip (fig. 1, 3). Full significance of the medial bend was not realized until a profile view was seen of a coronal histologic section of the hip (fig. 3, 4). When the dysplastic leg luxated laterally, the femoral head rested on the dorsal rim of the acetabulum. This lateral displacement of the femoral head placed extra or unbalanced medial pull on the greater trochanter through the attachment of the powerful middle gluteal muscle to its dorsal tuberosity and by the deep and superficial gluteal muscles inserted on the lateral side of the same structure slightly distally (fig. 3, 5). The abnormal pull bent the trochanter medially.

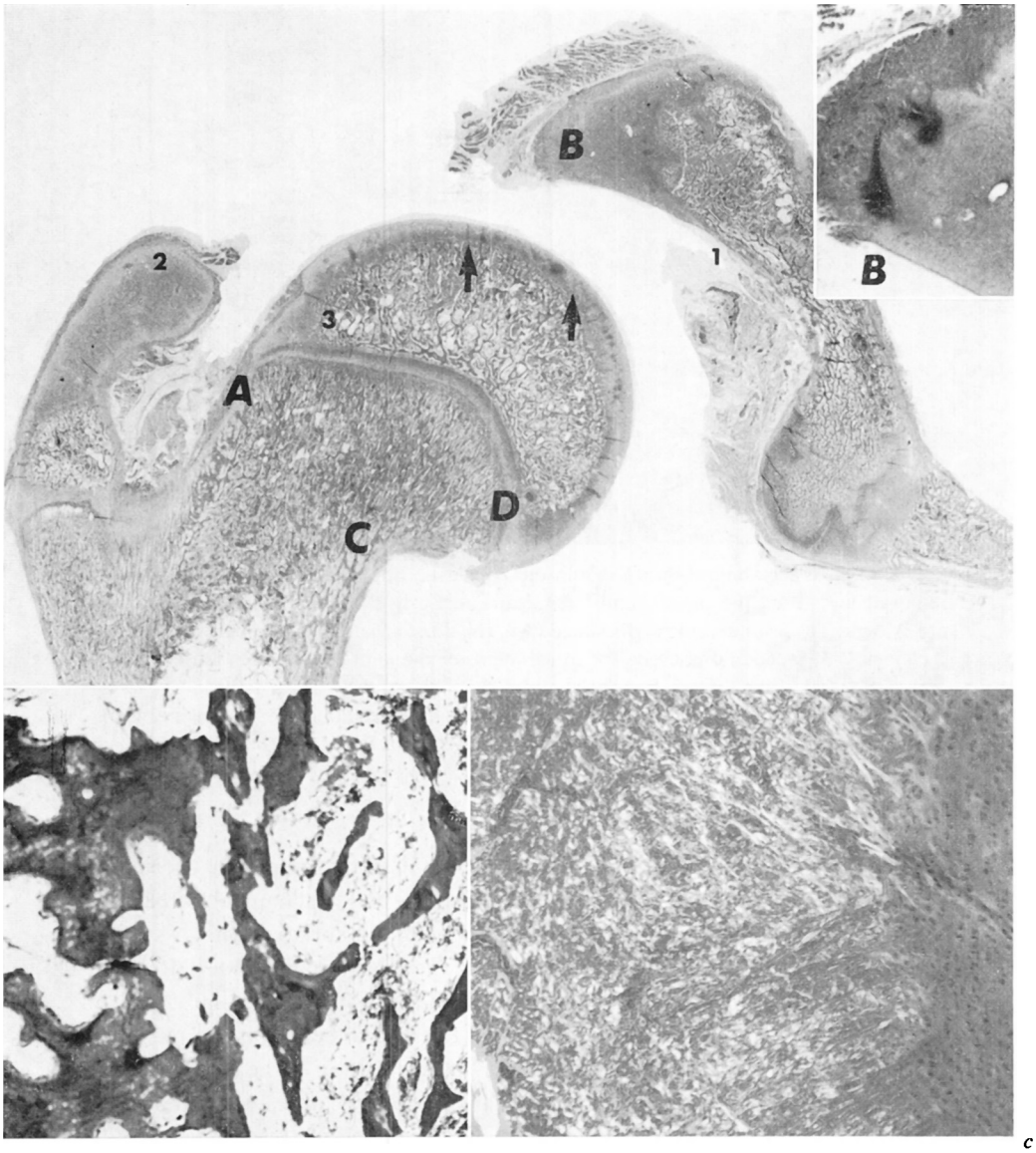
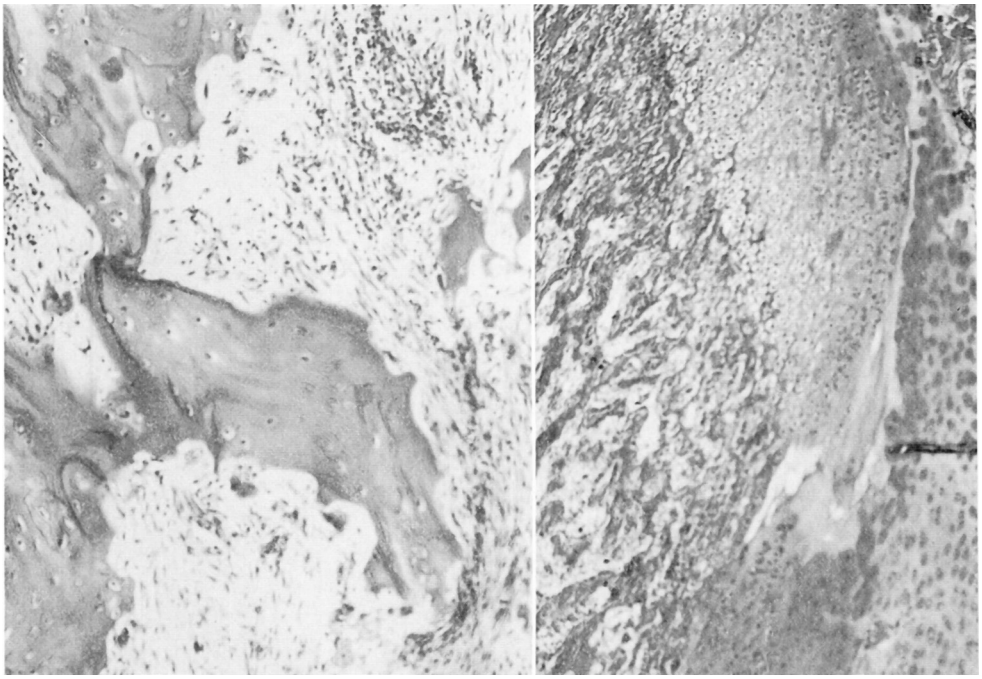


Fig. 3. Coronal section of the hip joint (a) of a 12-week-old dog with early dysplasia. In life, the femoral head of this dog was subluxated 6–8 mm. This subluxation caused an incongruous contact between the acetabulum and the femoral head and stretched the joint capsule and teres ligament. The contour of the acetabular cavity is widened (1). The greater trochanter is bent medially (2). The femoral head has drifted ventrally to a varus position (3). A section of the articular cartilage on the femoral head (arrows) is narrowed



d *e*

Fig. 3 d–e

Although the entire greater trochanter is cartilage at 12 weeks, histologically the cells and the matrix of the trochanter and the adjoining physis were normal and unaffected by the medial bending. In some instances, however, the rows of columnar cartilage of the physis were longer on the lateral side,

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by wear from overloading forces of the acetabular rim (B). The dorsal region on the femoral neck is being re-enforced by new fiber bone (A, and 3b), 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, and 3c). Varus drift of the femoral neck and epiphysis is accelerating the remodelling of the ventral neck area (C). Osteoclasts are resorbing bone at a rapid rate (3d). The epiphysis has drifted distomedially an estimated distance of 2 mm. This has resulted in epiphyseal lipping at the ventral margin. A fracture through the physis cartilage occurred as a result of the shift (D, and 3e). 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.

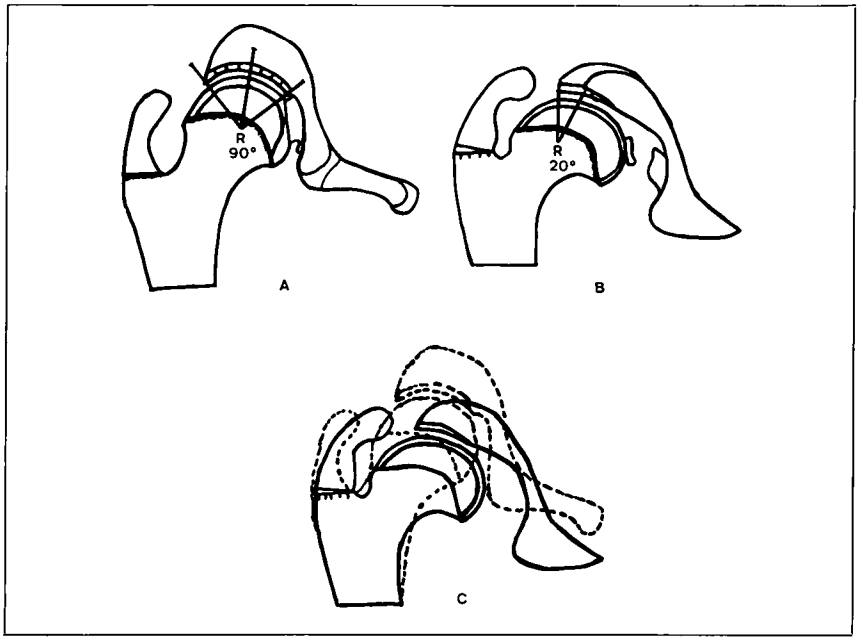


Fig. 4. A Histological sections of a normal hip. *B* Effects of 'drift' as the result of unbalanced load-forces upon the cartilage of the trochanter, acetabular cavity and physis. *C* Changes that have occurred when A is superimposed upon B.

thus making the physis wedge-shaped. Such changes in the columnar cartilage are known as 'cartilaginous drift' accomplished by 'fatigue-bending' [1].

The dysplastic hip joint becomes unstable as the acetabulum and femoral head pull away from each other. The dysplastic joint, the same size as the normal one, changes its shape and loses its grace and balance (fig. 3, 4, 6). 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 90–20°. The narrowed arc causes the compression forces to increase sharply on both the femoral head and the acetabular rim (fig. 4) [8].

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

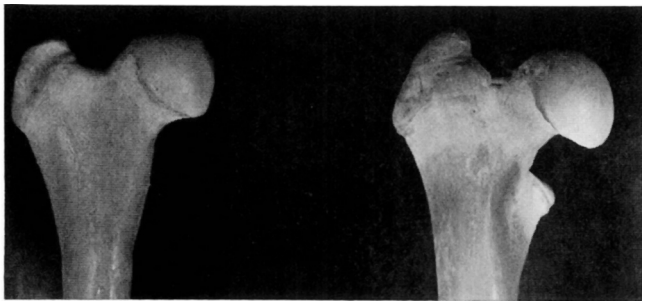


Fig. 5. Macerated proximal ends of femurs of young dogs in which the physes are still open. The femur (left) is from a normal dog. The femur (right) is from a dog with well-defined hip dysplasia and illustrates the effects of drift on the capital epiphysis. Drift caused the femoral neck to form a varus 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.

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 add new bone beneath the periosteum on the dorsal acetabular articular surface and stimulate the resorption of bone on the ventral surface of the acetabular cavity. The quantity and type of the new bone added or resorbed depend on the stress signals (Wolff's Law) [1].

The epiphysis of the femoral head changes its position on the physis. This change in position is called 'drift' and is accomplished either by plastic bending of the cartilage of the physis or by adding and resorbing bone in the metaphysis. There is drift and lipping of the epiphysis, bending of the physis, and a microfracture through the physis and damage to the adjoining tissue (fig. 3–5). In this instance, a microfracture through the physis accompanied a shift in the position of the cartilage columns (fig. 3–5). 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 (fig. 3).

The second method of changing shape is by osseous drift of the neck. The compression forces are concentrated in a small area on the dorsal articular surface of the acetabulum and are transmitted to a small area on the femoral head. The compression signals cause changes in bone deposition and

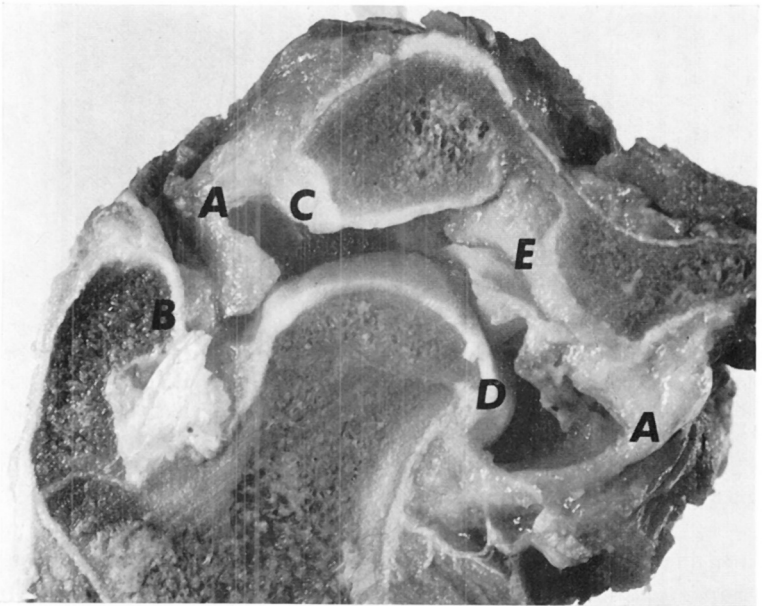


Fig. 6. Gross coronal section of a dysplastic hip illustrating the changes between the acetabulum and femoral head. There is a stretched and thickened joint capsule (A), medial bending of the greater trochanter (B), change in shape and lag in development of the acetabular rim (C) and the lipping or 'drift' of the ventral border of the femoral head (D). The teres ligament is swollen and frayed (E).

resorption that cause the head and neck to bend downward (fig. 3–5). 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' [1]. The osteoblastic activity on the dorsal or lateral surface and osteoclastic activity on the medial surface were intense at this time in a remodelling attempt to correct the exaggerated varus angle of the cantilever neck (fig. 3).

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

Twelve to Twenty Weeks of Age

Radiographically the subluxation of the femoral head, incongruity of the two joint surfaces, lag in development of the acetabular rim, and change in shape of the joint components continued to be the salient features of the dys-

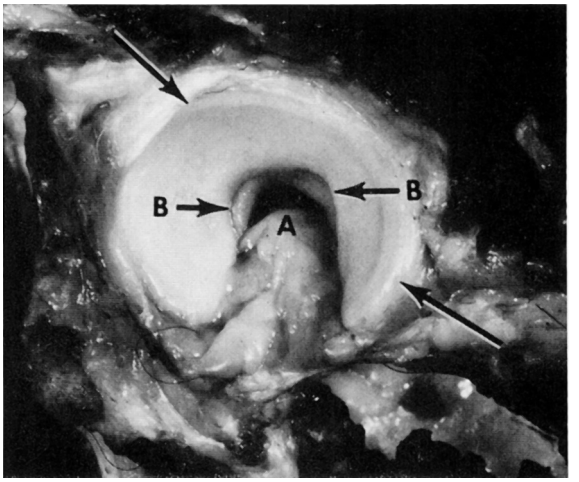
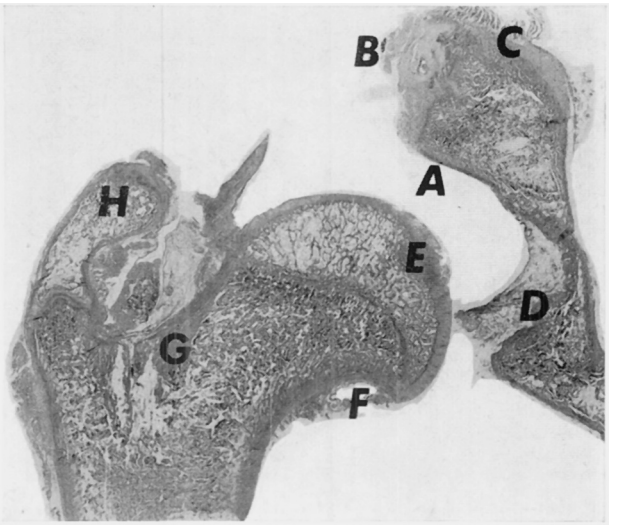


Fig. 7. A mildly dysplastic acetabulum after the femoral head had been removed. There is rounding of the dorsal and caudal margins of the acetabular rim between 11 and 5 o'clock (long arrows). The teres ligament (A) has been pulled away from the dome of the fossa, and new fiber bone has developed in the margin where the ligament was pulled away (B).

plastic joint (fig. 1). Grossly, rounding or rolling back of the acetabular rim became more prominent between the 10 and 2 o'clock positions (fig. 2, 6). Histologically, the increased localization of the compression forces in a restricted spot on the articular surface of the acetabular rim disturbed the developmental harmony of the area and fostered retardation of tissue maturation (fig. 3, 5, 6) [9]. 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 (fig. 2–4, 6). At this time the congruity between femoral head acetabulum is disrupted and there is interference with the normal cycle of hip development. This is in proportion to the degree of malposition or subluxation (fig. 2–4, 6).

Twenty to Thirty-Five Weeks of Age

The overloaded area on the dorsal acetabulum succumbed to wear, cartilage fibrillation, chondromalacia, and finally microfractures of the rim (fig. 7–9). The articulating surfaces stripped of cartilage became eburnated (fig. 9). The exposed subchondral bone assumed an ivory-like appearance as it condensed and became osteosclerotic, smooth, and highly polished. A corresponding area of eburnation occurred on the contacting femoral head surface (fig. 10).



a

Fig. 8. A coronal histological section of a hip joint from a 5-month-old dog with well-defined dysplastic changes. Compare these advanced changes with earlier changes illustrated in the 3-month-old dog (fig. 3). The contour of the articular surface of the acetabulum has changed from concave to convex (A). The greater trochanter is bent medially (H). The dorsal acetabular rim is fractured and displaced dorsally (B; 8a, c). The dorsal surface of the acetabulum is thickened greatly as the result of the proliferation of new bone (C; 8a, c). This was stimulated by the abnormal pull from the soft tissues attached to the dorsal margin (M; 8c). The trabecular formation is increased and is arranged in the direction of the pull from the dorsal area (M; 8c). Many of the damaged tissues on the rim are necrotic (N; 8e). Osteophytes have formed in the acetabular fossa (D; 8a). The effects of the epiphyseal drift are still present. The area of acetabular contact on the femoral head has been narrowed (arrows). The increased compression forces have worn away some of the articular cartilage in that area. The bony trabeculae beneath this area are increased and form a triangle that converges into the medial cortex (lines). Trabeculae also are increased beneath the attachment of the teres ligament (E; 8a). The cartilage at the attachment of the ligament is frayed and fragmented (E; 8d). New fiber bone has formed on the ventral aspect of the neck where remodelling was in progress at 3 months (F; 8a, b). New bone on the dorsal aspect of the neck (G; 8a, b) has become thicker.

The joint capsule in progressive hip dysplasia undergoes remarkable changes. The capsule of the normal joint is pale, semi-transparent, and 1–2 mm in thickness. The capsule of the dysplastic hip is thickened, stretched and traumatized from the stress and pull of the unstable, subluxated femoral head. The thickening of the capsule, which may measure up to 5–7 mm, was in response to the trauma caused by excessive pull from subluxation (fig. 6).

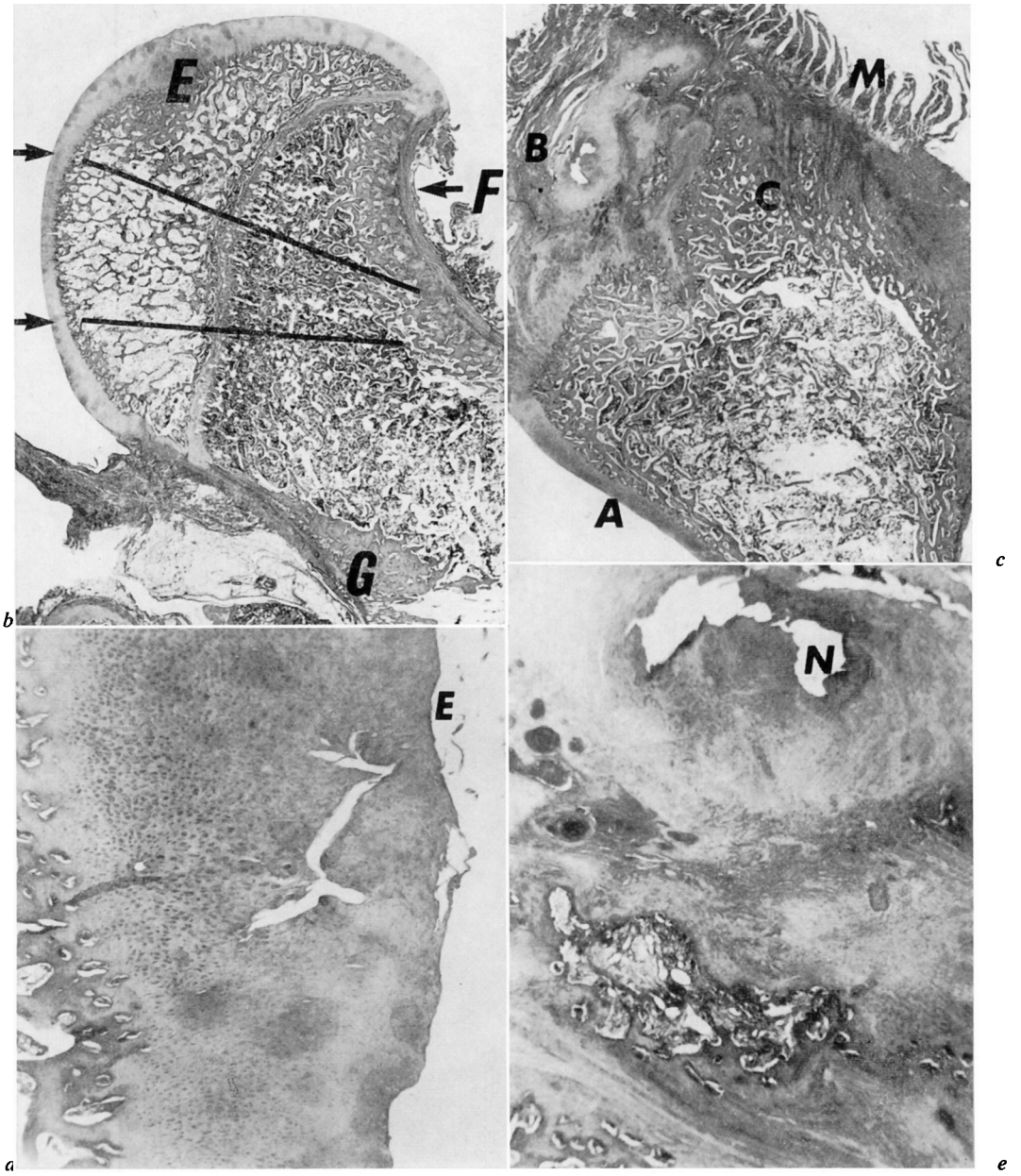


Fig. 8 b–e

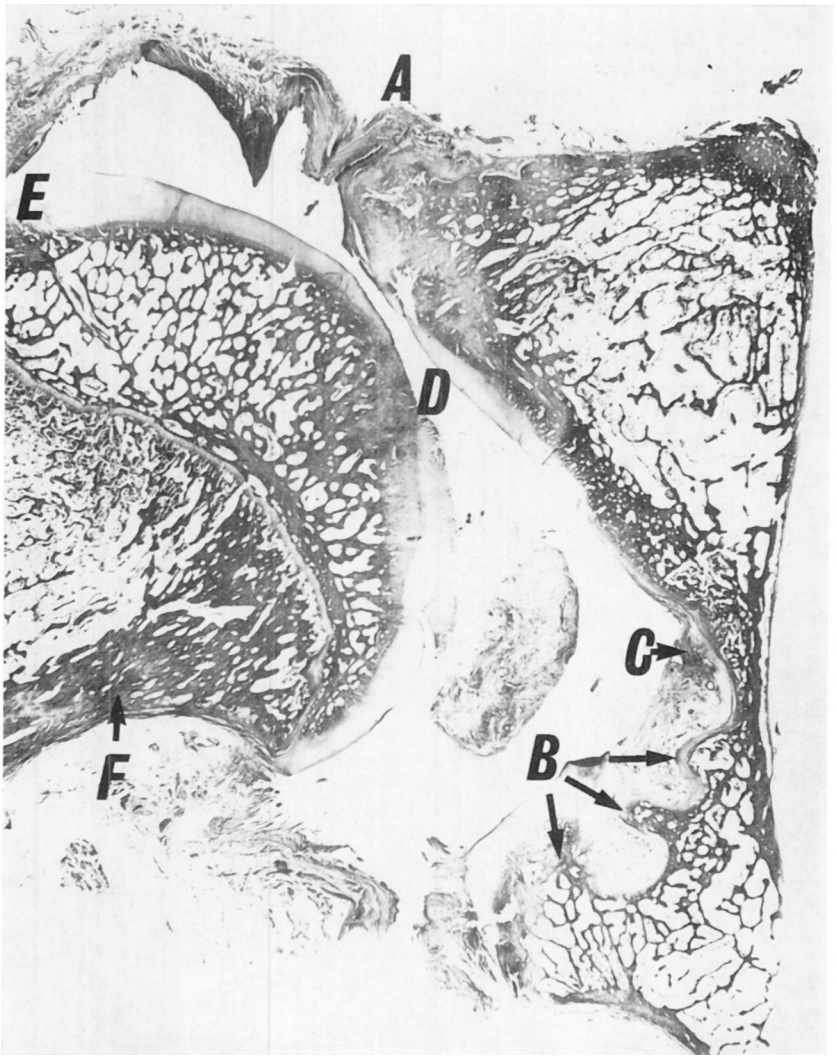


Fig. 9. Coronal histological section of a dysplastic hip joint from a 7-month-old dog. The subluxated femoral head has limited contact with the acetabulum restricted to an area at the rim (A). The increased compression forces have greatly altered the architecture of the entire joint. The contour of the articular surface of the acetabulum is changed from concave to convex. The rim has undergone various degrees of degeneration, cartilage fibrillation, malacia, necrosis, fracture and upward displacement (A). Osteophytes appear in the fossa (B). Histologically the dark shaded areas in the teres ligament were 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

New bone formed in the capsule along the dorsal capsular border. In the dysplastic joint the synovial fluid lost its adhesiveness, its specific gravity decreased, and the leukocyte count increased. Its ability to function as a lubricant was decreased [20].

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

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

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

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

Nine Months of Age and After

Bone and joint tissues were never static, whether functioning normally or not. In the dysplastic hip, progressive remodelling occurred at an accelerated

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head (F). The articular cartilage is eroded, and eburnation is underway (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.

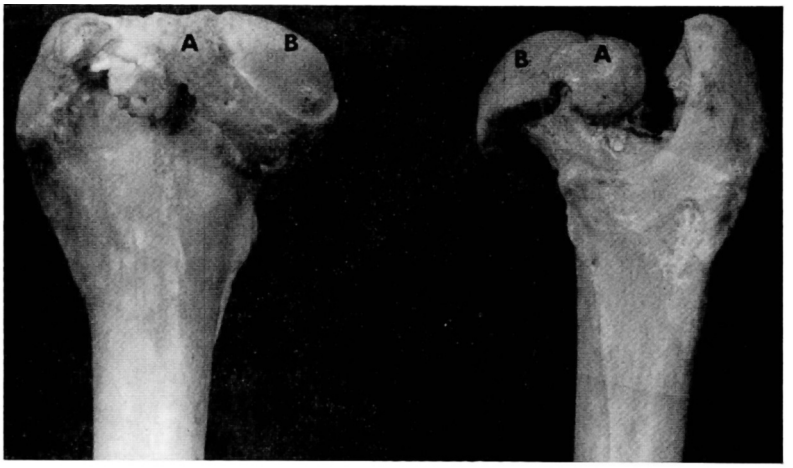


Fig. 10. The proximal ends of two femurs illustrating the effects of advanced hip dysplasia. New bone formation (exostoses) encircles the femoral necks at the junction of the head and neck (A). The femoral heads are shortened from wear and the cartilage surfaces are eroded and eburnated (B).

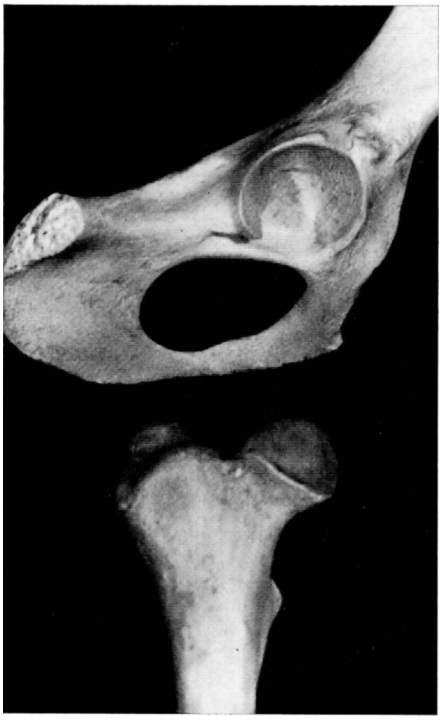


Fig. 11. Macerated normal hip joint of a young dog. Notice the smoothness of the bone contours, the sharp rim of the acetabulum, the concave acetabular cavity, and the sharp edges of the acetabular fossa. The femoral head is balanced and symmetrically formed.

rate. The entire shape of the hip was restructured. Femoral head subluxation contributed to mechanical imbalance of the hip. Abnormal compression, traction and tension forces created stresses that caused new bone to be laid down in one area and resorbed in another. At this stage of abnormal structure it was difficult to differentiate clearly the changes representing true hip dysplasia and those representing beginning secondary hip degenerative joint disease (fig. 10, 13, 16, 17).

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

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

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

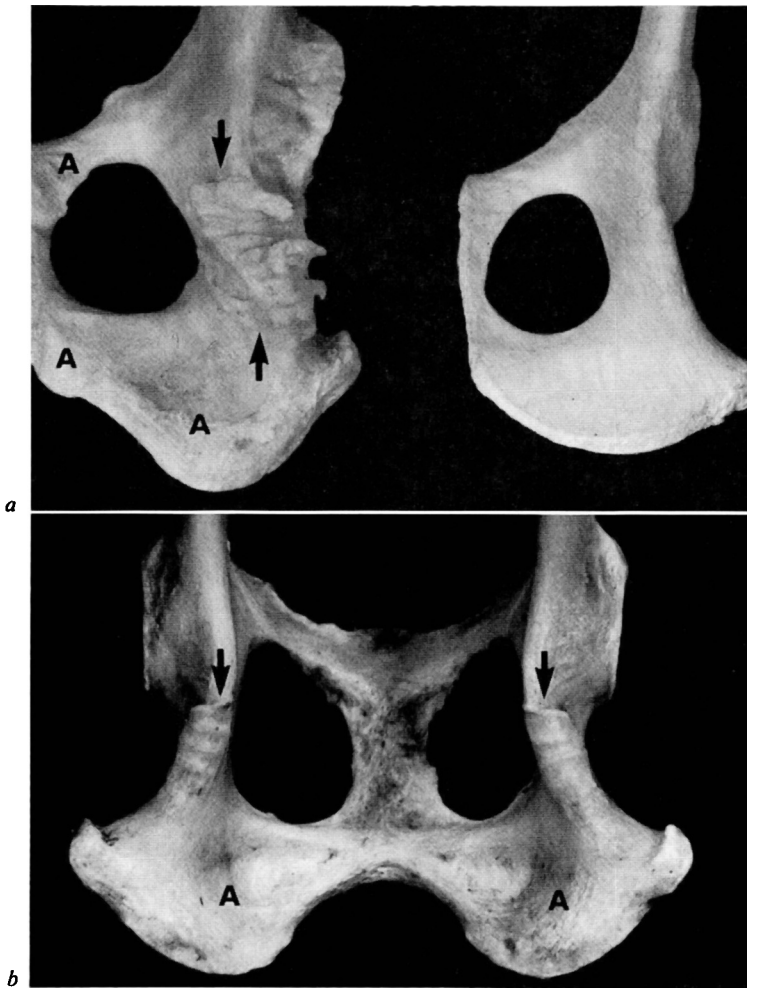


Fig. 12. a Photograph of a dysplastic (left) and normal (right) ischial part of the pelvis illustrating osseous changes associated with hip dysplasia. The lesser sciatic notch of the shaft of the ischium supports the synovial bursa, which serves as a pulley to facilitate the pull of the tendon of the 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). *b* The pelvis below 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 pubic bones are raised, grooved and roughened in response to excessive pull on the muscle attachments that were anchored there.

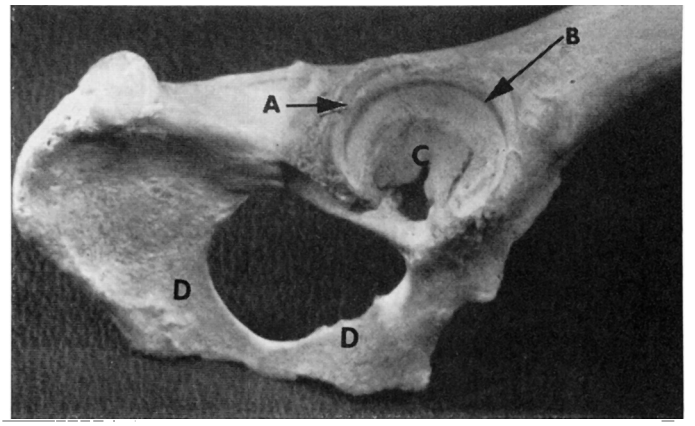


Fig. 13. Macerated pelvis of a dysplastic hip. The acetabular rim is displaced dorsally as the result of microfractures. The fracture lines are still visible (A). Below this there is a crescent area of eburnation (B). The acetabular fossa is filled with new bone (C). Ridges and exostoses present on the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

eburnated surface (fig. 13, 17). Occasionally the fossa was only partially filled with new fiber bone (fig. 7, 13).

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

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

In the normal articulating joint, the motion of the joint components with its efficient lubricating system keeps the shedding cartilage cells wiped off so

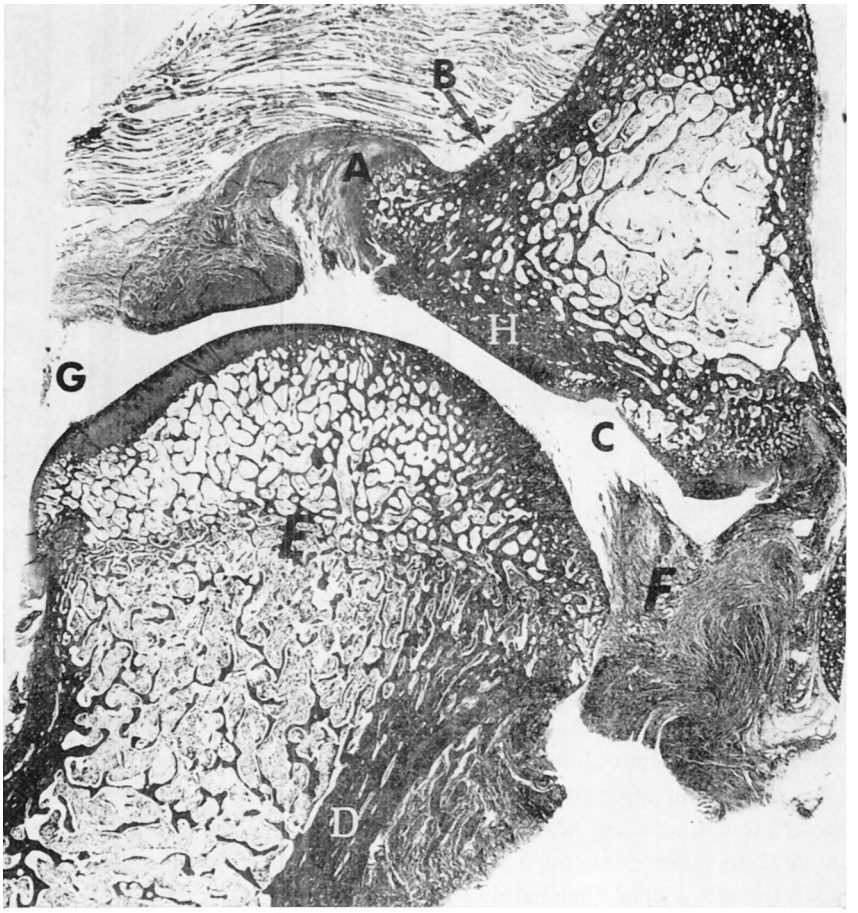


Fig. 14. Coronal histological section of a dysplastic hip from a 1-year-old dog. The femoral head is subluxated and the angle of the femoral neck is valgus (D). The acetabular rim now healed, had been fractured and displaced dorsally (A). A heavy trabecular cortex surrounds the dorsal aspect of the acetabulum (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 remodelled into a valgus position, and the ventral cortex is greatly thickened (D). The cartilage of the physis has disappeared, but the thickened trabeculae in the area of the subchondral plate remain (E). On the articular surface of the femoral head in the medial area the cartilage is worn away, and eburnation has developed in the narrowed area 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).

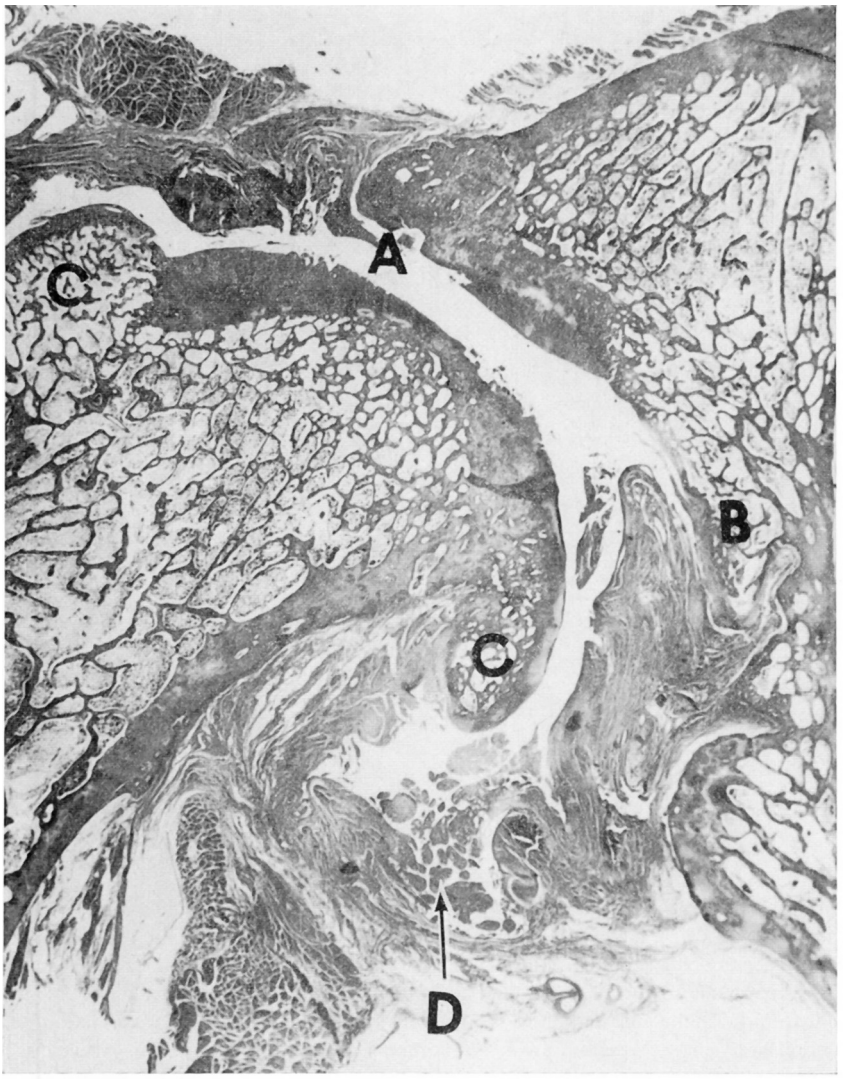
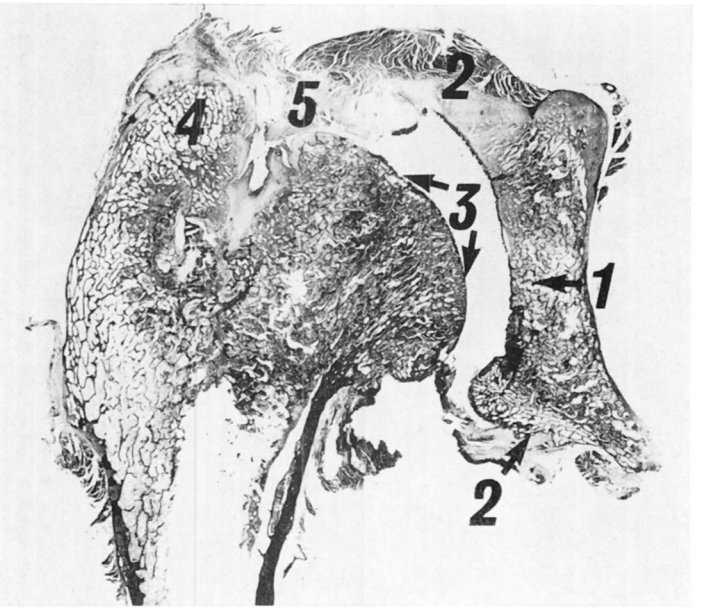


Fig. 15. Coronal histological section of the dysplastic hip joint from a 2-year-old dog. The dysplastic hip exhibits the effects of prolonged, unbalanced wear and remodelling applied to the weight-bearing areas. The acetabular rim has been fractured and displaced dorsally (A). The articular surface of 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).



a

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

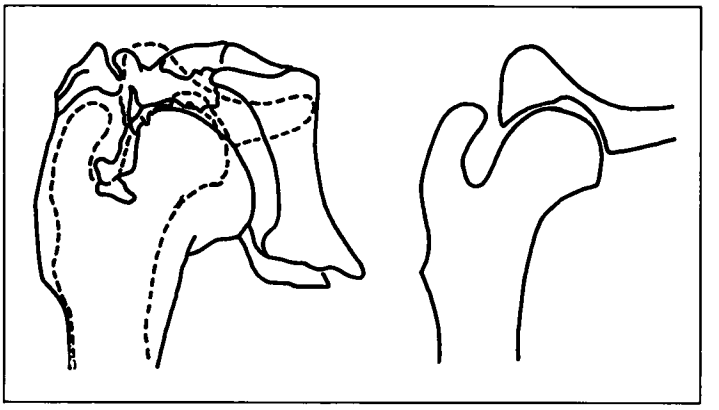


Fig. 16 b

the articular cartilage remains at a stable thickness. The normal articular cartilage is nourished by synovial fluid on the joint surface side and from diffusion of nutrients through the subchondral plate on the other side [1, 20].

In the unstable, subluxated joint, the width of the articular cartilage varies from nothing on the eburnated surface, where compression had been excessive, to the greatly thickened area of articular cartilage at the lateral border, where the incongruity did not allow the opposing surfaces to touch (fig. 10, 15–17). The synovial fluid supplies nourishment to articular cartilage for a depth of approximately 1 mm as it is forced in and out by the compression-hydraulic system. The subchondral plate on the attached side of 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 (fig. 15, 16) [1, 9, 18, 20]. This mechanism, tearing of the Sharpey's fibers, and stimulation of the periosteum resulted in osteophyte formation (fig. 10, 13, 15, 16).

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

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

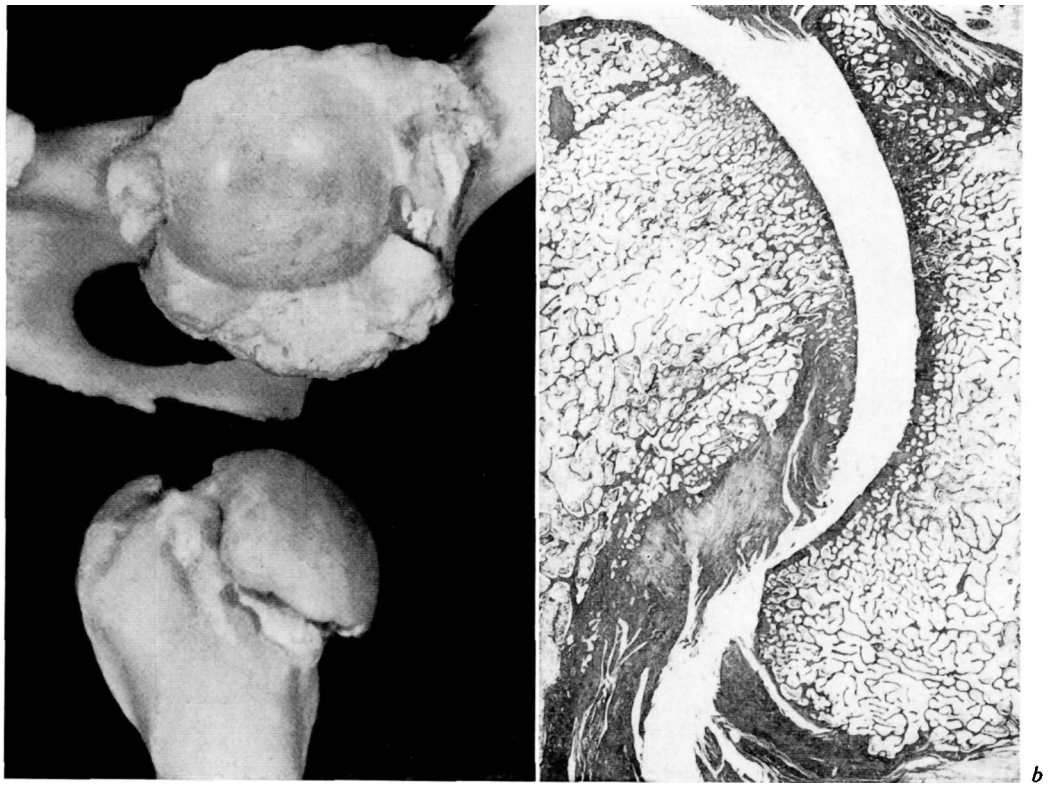


Fig. 17. a Macerated hip joint of an old dog with extensive degenerative joint disease and hyperostoses secondary to hip dysplasia. The articular cartilage has disappeared. The contacting surfaces of the femoral head and acetabulum are eburnated. *b* Histological coronal section of the hip joint of the opposite hip, which had similar anatomical changes. Notice the valgus angle of the femoral head, the osteophyte lipping, the eburnated surfaces, and the hyperostoses of the acetabulum.

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

Discussion

The locomotion of the young dog with severe hip dysplasia may be unsteady, but very little or no pain is exhibited. Suddenly, at 5–6 months of

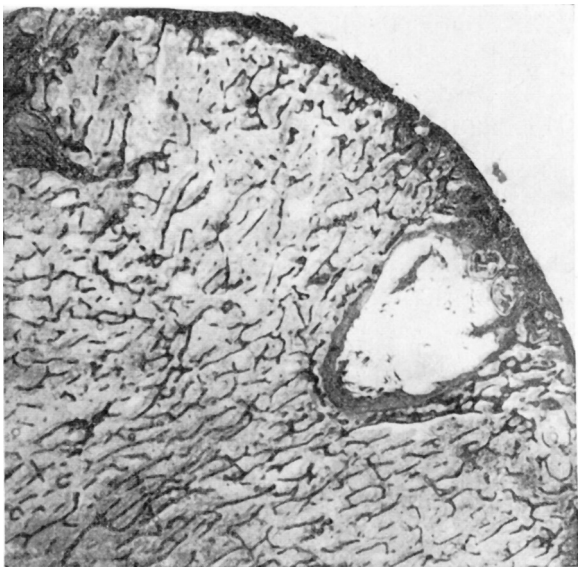


Fig. 18. Coronal histological section of part of the femoral head of a man with degenerative joint disease. Articular cartilage is gone and a large subchondral cyst is present. Such cysts are common in humans with degenerative joint disease but are rare in the dog.

age, pain of the hindquarters appears, especially when the dog rises to his feet and after exercise.

This is the age when microfractures of the dorsal acetabular rim appear. These changes were difficult to demonstrate radiographically, even though the extent of the microfractures varied widely. Since a fracture is a painful lesion, it is logical to consider that the microfracture of the dorsal acetabular rim is a possible cause of the early pain. This pain subsides when the fractures are healed, usually by the 8th–11th month. These signs do not recur because the bone has ossified sufficiently to withstand further trauma and fracture.

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

Abnormal remodelling and hypertrophic osseous development are continually present in and around the dysplastic hip. Even though these mecha-

nisms are functional during the disease, there is commonly poor correlation between the clinical signs of the patient and the radiographic signs of the involvement unless the dog is worked hard. Often the radiographic involvement is extensive, but there are no physical signs of involvement. In a few instances radiographic lesions are mild but signs of pain are severe.

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

Summary

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

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Surgically Induced Hip Dysplasia

It has been hypothesized that hip dysplasia results from a disparity between primary pelvic mass and too rapid skeletal growth. The deficiency of soft tissue support results in a pulling apart of the femoral head from the acetabulum. This subluxation or luxation of the femoral head then breaks or disturbs the normal developmental cycle of the hip joint and leads to hip dysplasia and degenerative joint disease [1, 2].

This hypothesis has been tested positively when hip dysplasia was induced in Greyhounds, a breed normally free of the disease, by administering estrogens in doses many times greater than biological levels [3, 5].

The purpose of the present experiment was to test the hypothesis by surgically creating a subluxation between the femoral head and acetabulum by the attachment of the obturator internus, obturator externus and gemelli muscles in the trochanteric fossa.

The normal action of these three muscles of the hip is to rotate the limb outward and stabilize the femoral head in the acetabulum. The outward rotation action is counter-balanced by the inward rotation and abduction of the three gluteal muscles [4]. After cutting the three muscles at the insertion, the femoral head is freely movable in a lateral direction to the extent of the teres ligament and joint capsule, which allows it to come to the dorsal rim of the acetabulum (1 cm). The femoral head subluxates to the dorsal acetabular rim when the dog walks. The condition created by this operation mimics those that spontaneously occur as a result of soft tissue failure and stretching in hip dysplasia.

Materials and Methods

Five 4-month-old, well-nourished, healthy male German Shepherds, weighing from 9 to 16 kg were used. These dogs were known as D88, D89, D90, D91, and D93. They

came from a colony where at least 50% of the dogs, when mature, had some degree of bilateral hip dysplasia. The dogs received prophylactic protection from canine distemper and leptospirosis, and they were wormed. They were kenneled together in inside runways, which allowed for adequate exercise and freedom. The diet was of commercial dog food *ad libitum*. When the dogs were accustomed to the new environment, ventrodorsal pelvic radiographs were made. The tendons of the obturator internus, obturator externus, and gemelli muscles were severed surgically in the right hip at the muscle insertions in the trochanteric fossa. The left hip served as the control.

Results

The right hips were compared regularly with the left control hip until the dogs were killed. This provided an unusual opportunity to study and follow the development of the normal hip joint, the mildly dysplastic hip joint, and grades of more severe hip dysplasia as it occurs spontaneously and as it was produced surgically (fig. 1–5).

At surgery (4 months of age) the hip joints of the five dogs were considered radiographically within normal ranges, except for dog D93 (fig. 5). The hip joints of dog D93 were considered radiographically questionable at that time but not positively dysplastic.

During surgery, as soon as the tendons of the obturator externus, obturator internus and gemelli were severed on the right side at the trochanteric fossa, the femoral head could be lifted and freely luxated laterally as far as the dorsal rim of the acetabulum, where the subluxation was restricted by the joint capsule and teres ligament. The hip joint on the operated side, when radiographed after surgery, was subluxated noticeably compared with the control side. In two dogs, approximately 10 ml of serum were removed from the surgical site on the third postoperative day, after which the wounds healed promptly.

For the first 10 postoperative days, the dogs favored the operated limb slightly. During the remainder of the experiment, no difference in the gait of the hind legs was detected. All the dogs grew normally and were in good flesh and health. The femoral heads of the operated legs subluxated to the dorsal acetabular rim when the dogs walked. When the operated leg was flexed, the stifle (knee) rotated slightly medially and the hock rotated laterally to a similar degree. This occurred because of a lack of stabilization from the three severed muscles. Pain and restricted activity of the leg were not evident.

For the first 3 postoperative weeks it was possible to replace the luxated femoral heads in the acetabulums; after that, they remained permanently

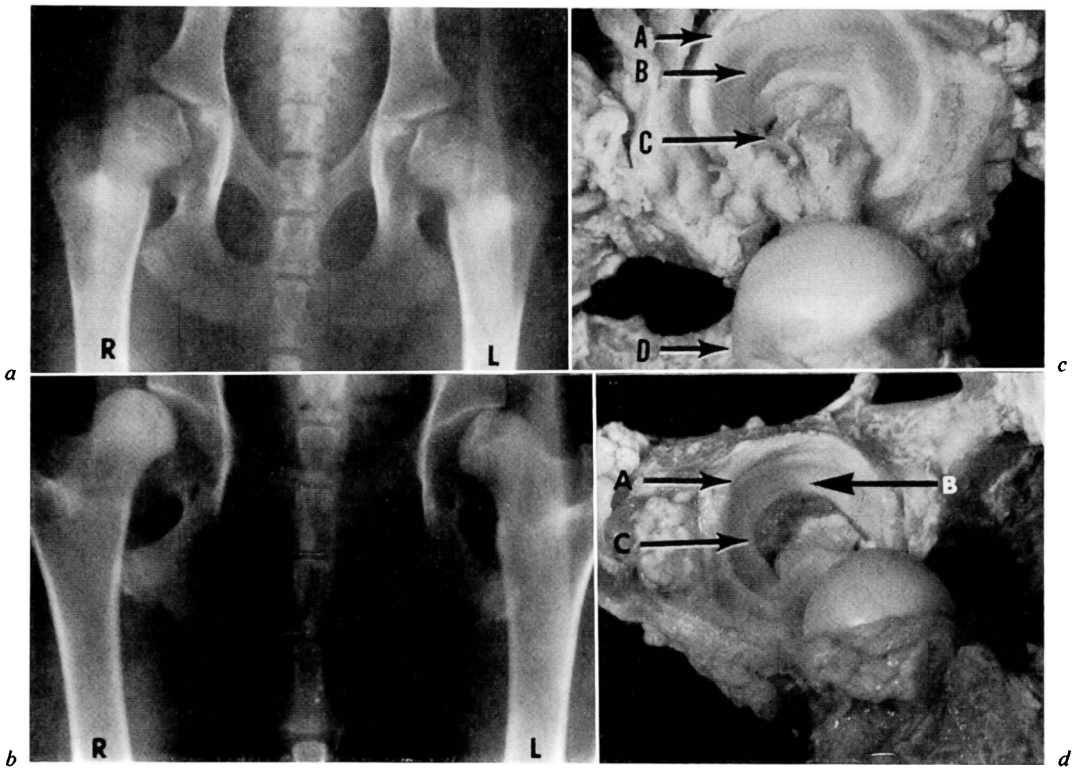

Fig. 1. a Radiograph of the hip of dog D88, 4 months old at beginning of experiment. The hip joints are normal for age. *b* Radiograph at end of experiment when the dog was 10 months old. The (right) operated hip has subluxated extensively (grade 3). The left hip is mildly subluxated. *c* The operated side (right) after the muscles were removed and the joint capsule was opened. The joint was markedly dysplastic as a result of previously severing the three supporting muscles. The dorsal beak (rim) was rounded from 10 to 2 o'clock (A). There was wear and fissuring of the articular cartilage on the acetabulum, and the concavity of the acetabular surface had become convex (B). The teres ligament was edematous and torn away from the dome of the fossa (C). New fiber bone had begun to form across the dome of the fossa. The femoral head has remodelled, and an osteophyte was forming at the junction of the femoral head and neck (D). *d* The left hip joint after the muscles were removed and the joint capsule was opened. Mild changes characteristic of spontaneous hip dysplasia are present. The dorsal beak (rim) is slightly rounded from 10 to 2 o'clock (A). There is a small fissure line in the acetabular articular cartilage over the dome of the fossa (B). The teres ligament is pulled down from the dome of the fossa, and it is also edematous (C).

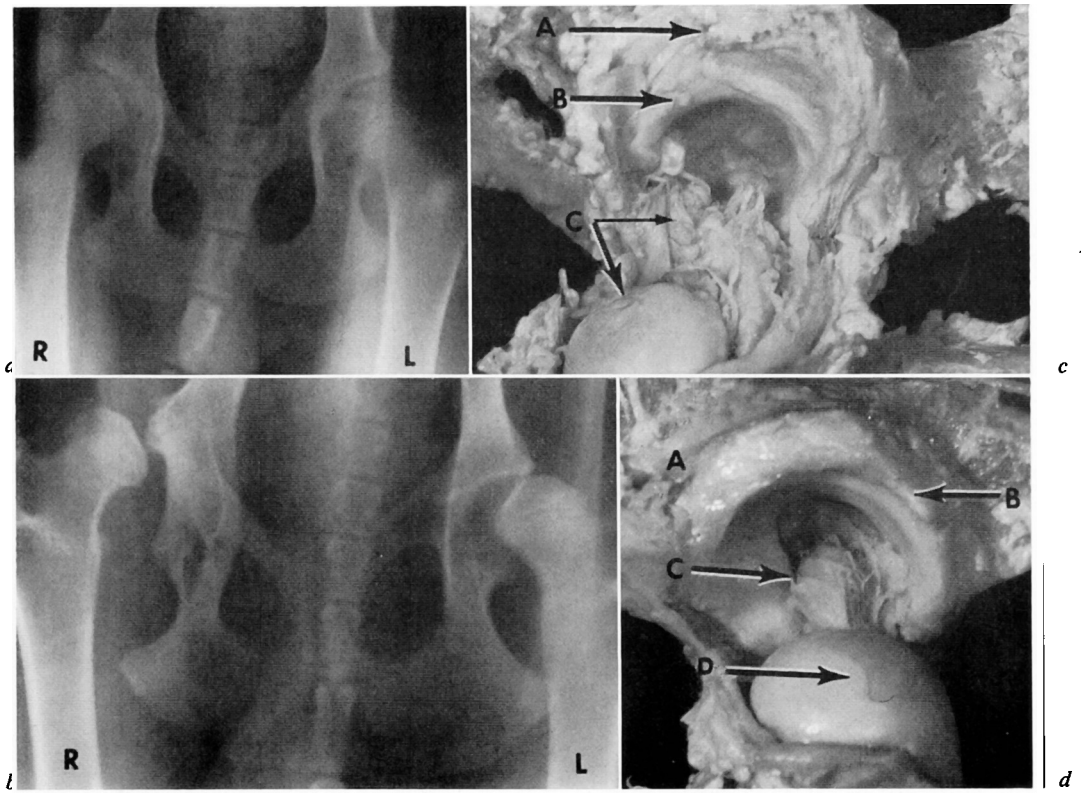

Fig. 2. a Radiograph of the hips of dog D89, 4 months old at beginning of the experiment. The hip joints are in normal position for age. *b* Radiograph at the end of the experiment when the dog was 10 months old. The operated hip (right) is luxated, and marked dysplastic changes have occurred (grade 4). The left hip has undergone spontaneous dysplastic changes (grade 2). *c* The operated hip (right) after the muscles were removed and the joint capsule was opened. Severe dysplastic changes have occurred. The capsule has thickened greatly (A). The beak (rim) is fractured and displaced dorsally (B). The teres ligament is frayed, swollen and completely severed from the fovea of the femoral head (C). *d* The left hip joint after the muscles were removed and the joint was opened. The joint capsule was thickened (A). The dorsal beak (rim) is rounded from 10 to 3 o'clock (B). The teres ligament is frayed and torn away from the dome of the fossa (C). The femoral head is remodelling, the articular cartilage is damaged from unusual forces, and an osteophyte has developed (D).

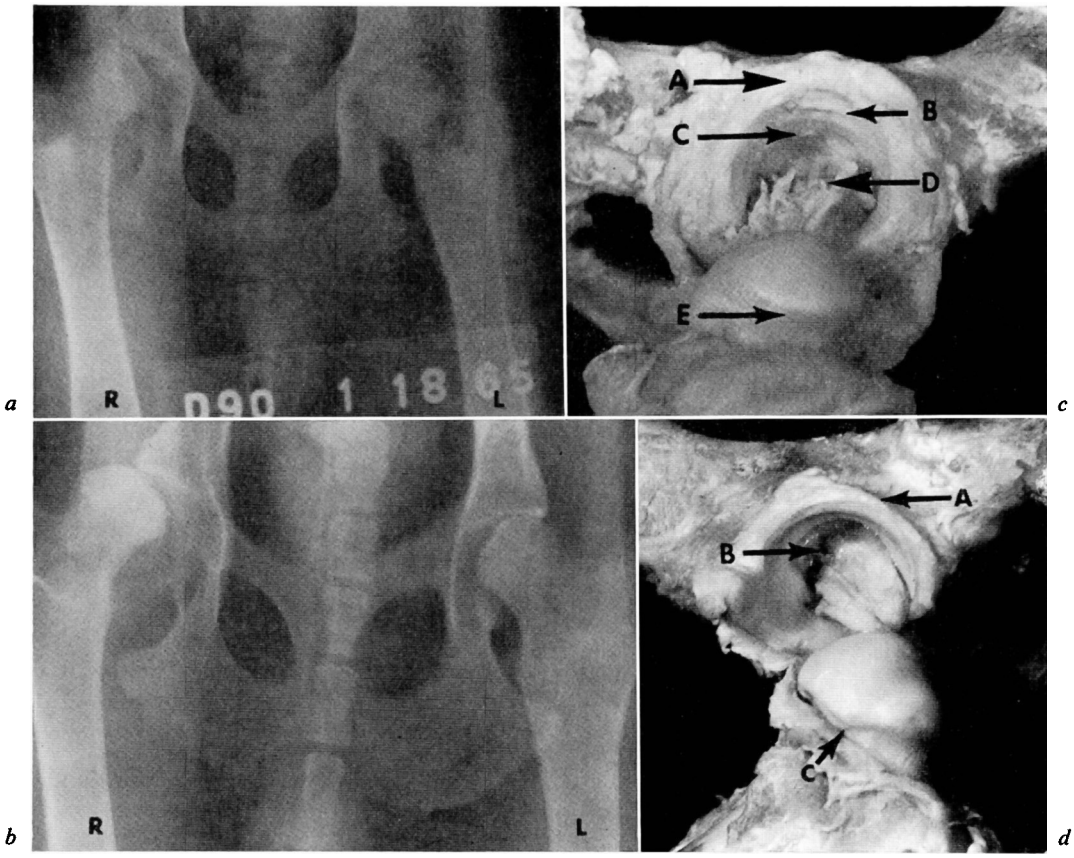

Fig. 3. a Radiograph of the hip of dog D90, 4 months old at the beginning of the experiment. The hip joints were considered in normal position for age. *b* Radiograph of the hip joints at the end of the experiment when the dog was 10 months old. The operated hip (right) is luxated, and well-defined dysplastic changes have occurred (grade 3). The left hip appeared normal radiographically. *c* The right operated hip after the muscles were removed and the joint capsule was opened. Well-defined dysplastic changes have occurred. The capsule was thickened (A). The beak (rim) was fractured and displaced dorsally (B). The articular cartilage has degenerated, fibrinated and worn (C). The acetabular articular surface has changed from concave to convex. The teres ligament was frayed and swollen (D). The femoral head has undergone remodelling and has become flattened (E). *d* The hip joint radiographically appeared normal, but grossly the joint capsule was slightly thickened, and the dorsal beak (rim) was rounded from 10 to 3 o'clock (A). The teres ligament was pulled down from the dome of the fossa (B). The femoral head had remodelled slightly, and a ridge was present where the dorsal acetabular beak (rim) had worn (C).

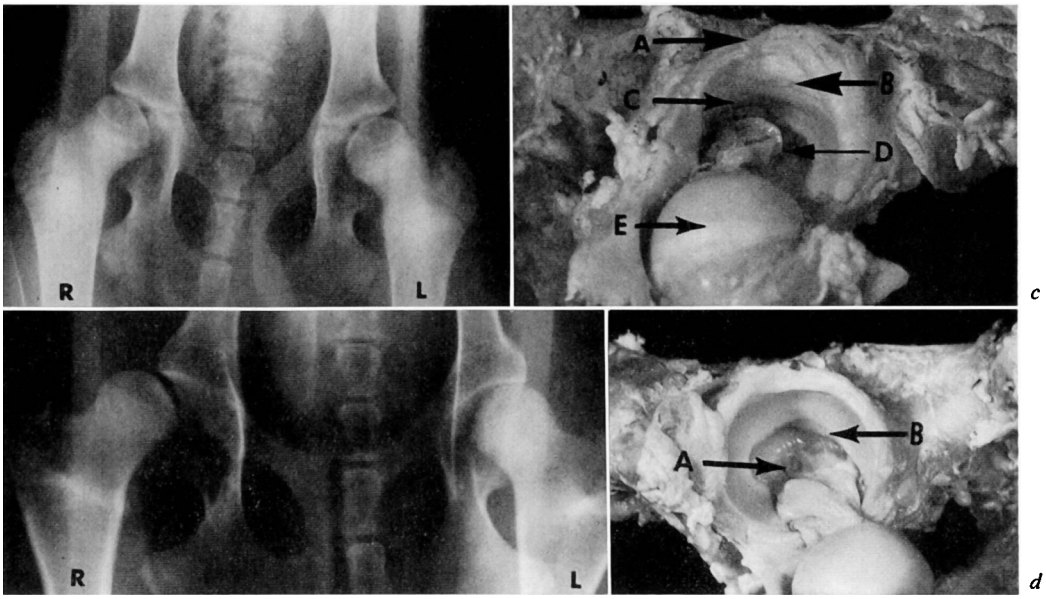

Fig. 4. a Radiograph of the hip of dog D91, 4 months old at the beginning of the experiment. The hip joints were considered in normal position for age. *b* Radiograph at the end of the experiment when the dog was 10 months old. The operated hip (right) was luxated, and well-defined dysplastic changes had developed (grade 3). The left hip appeared normal radiographically. *c* The operated hip (right) after the muscles were removed and the joint capsule was opened. Well-defined dysplastic changes were present. The joint capsule was thickened (A). The dorsal beak (rim) was fractured and displaced dorsally, and there was wear of the cartilage between 10 and 3 o'clock (B). There was wear and fissuring of the articular cartilage of the acetabulum, and the concavity of the acetabular surface had become convex (C). The teres ligament was edematous and torn away from the dome of the fossa (D). The femoral head was remodelling (E). *d* This hip joint appeared normal except that the teres ligament was torn from the dome of the fossa (A), and new bone was forming in the fossa where the teres ligament was torn (B).

subluxated. Radiographically, the pelvis of each dog was observed immediately after surgery, at 10 days, 45 days, and at each 6 weeks until the end of the experiment when the dogs were killed at 10 months of age. Each time the radiographs were taken, the femoral heads were subluxated more than when examined previously, until at 90 days after surgery, when the dogs were 7 months of age, the femoral heads rested on the dorsal rim of the acetabulums. This position remained constant from the 90th postoperative day until the experiment was concluded. In D93 (fig. 5), however, the right

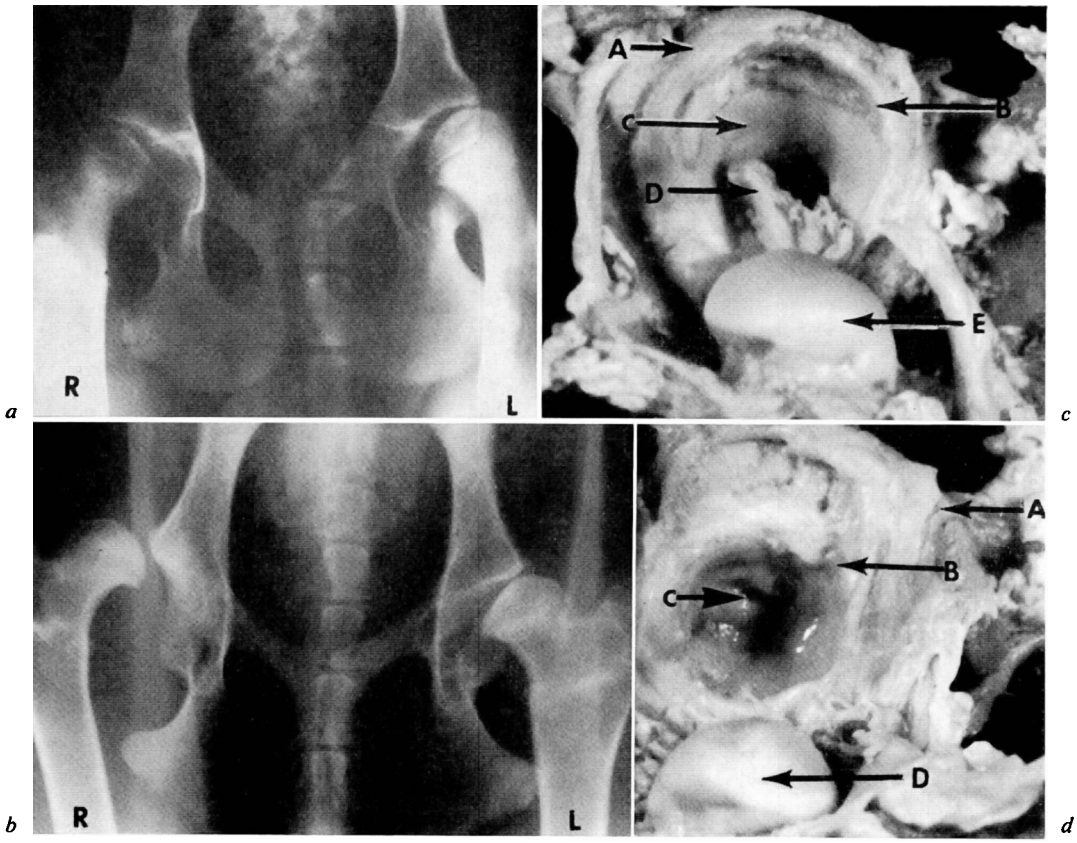

Fig. 5. a Radiograph of the hip of dog D93, 4 months old at the beginning of the experiment. Both femoral heads fit poorly in the acetabulums, and it was predicted that these hip joints would become dysplastic. *b* Radiograph at end of experiment with the operated hip (right) luxated. The acetabulum was filling with new bone, and the femoral head was remodelling. The left hip had spontaneously become severely dysplastic (grade 3), and both the acetabulum and femoral head had undergone dysplastic changes. *c* The operated hip joint (right) after the muscles had been removed and the joint capsule was opened. The predisposition to dysplasia as seen in radiograph 5a and the severing of the muscles allowed for femoral head to luxate extensively. The capsule is greatly thickened and stretched to allow for the luxation (A). The dorsal rim is flattened (B). The acetabular cavity is degenerated and is filling with new fiber bone (C). The femoral head has remodelled and flattened. The teres ligament was severed. The articular surface was roughened (D). *d* The left hip has well-defined changes characteristic of severe spontaneous hip dysplasia. The joint capsule is thickened (A). There is wear and fissuring of the articular cartilage of the acetabulum, and the acetabular beak is fractured and displaced dorsally (B). The concavity of the acetabular surface has become convex (C). The teres ligament is edematous and is torn away from the dome of the fossa (D). The femoral head has undergone remodelling changes and is mushroom shaped (E).

(operated) femoral head luxated radiographically a distance of over 1 cm lateral to the acetabular rim.

In the final radiographs of the control hips of the five dogs, two hips were radiographically normal, D90 (fig. 3) and D91 (fig. 4); two were mildly dysplastic, D88 (fig. 1) and D89 (fig. 2); the fifth, D93 (fig. 5), subluxated severely until the femoral head rested on the dorsal acetabular rim.

At necropsy, four of the operated hips (D88, D89, D90, D91) had the same gross changes (fig. 1–4). The capsules were thickened to 3–5 mm and were stretched; the femoral heads were unstable and luxated as far as the acetabular rims, which was the length of the teres ligament. The synovial fluid had increased in amount and appeared to be of low density. In D90, the teres ligament of the operated hip was ruptured and only a few fibers remained attached to the femoral head fovea. The teres ligaments in the operated hips of the other three dogs (D88, D89, D91) were torn down from their attachments in the dome of the acetabular fossa for a distance of 3–4 mm. The ligaments were swollen and edematous but still intact (fig. 1, 2, 4). Small hemorrhages were present, and many of the fibers were frayed.

The acetabular changes were characteristic of well-defined hip dysplasia, remodelling, and beginning degenerative joint disease. The concave curvature of the acetabular cavity was flattened between the acetabular rim and fossa (fig. 1). Various degrees of chondromalacia were present at the rim between 10 and 2 o'clock (fig. 2). Microfractures as indicated by fissures parallel to the margin of the rim were present. The fractured rims had turned dorsally (fig. 3, 4) in the same manner as described in the spontaneously severe dysplastic hip. These were not visible radiographically in the live dogs. New fibrous bone was forming around the rims of the fossas.

In D93 the operated hip had undergone dislocation and more marked destruction of the acetabulum than the other four (fig. 5). The femoral head was dislocated 1 cm lateral to the acetabular rim, and it was doubtful whether there was contact between the two parts when the dog walked. The capsule was greatly stretched and thickened to 5 mm. The specific gravity of the synovial fluid appeared less than normal.

Grossly, in D93 it was difficult to imagine that a normal acetabulum had ever existed (fig. 5). The acetabular cavity was flattened and more than half filled with new fibrous and osseous tissue. The articular cartilage was not recognizable, and fresh blood clots were present in the acetabular cavity. Only shreds of tissue from the injured teres ligaments remained attached to the fovea. The acetabular rim had been displaced dorsally and was surrounded by fibrous reactive tissue. Chondromalacia of the femoral head

was extensive, and osteophytes were present on the ventral portion of the femoral head, extended around the junction of the head and neck. The femoral head was flattened and mushroom-like. Brown pigment adhered to the villi of the inner capsular surface.

The control hips (left) of the five dogs varied from what was believed to be radiographically normal to severely dysplastic. Two of the hip joints were within normal limits radiographically (fig. 3, 4); however, when the capsules were opened at dissection, the edges of the dorsal acetabular rims were rounded and worn in the areas between 10 and 2 o'clock. This was interpreted as evidence of excessive pressure on the rim as a result of mild subluxation of the femoral head when the dogs walked. In both dogs D90 and D91 the teres ligaments were pulled away from the dorsal aspects of the acetabular fossa for a distance of 3 mm. A few fibers of the teres ligament were torn and edematous. The femoral heads had not as yet undergone change, but well-defined changes were present in the acetabular cavities of these two dogs (fig. 3, 4).

In the left or control hip of dogs D88 and D89 the articular surfaces of the acetabulums were remodelling consistent with spontaneous hip dysplasia. The acetabular rims had rounded between 10 and 3 o'clock ((fig. 1, 2). The concave articular contour of the acetabulum was transformed to a convex surface. In dog D93 (fig. 5) the control hip had developed dysplasia spontaneously to a degree comparable to four of the operated hips (D88, D89, D90, D91).

At necropsy, the 21 pairs of muscles of the hip and thigh were individually dissected, weighed, and the pelvic muscle index was calculated for each dog as described previously [6]. Histological sections of each muscle were made.

The pelvic muscle index of all five dogs was near 9. Such an index is low and carries a high probability that these dogs would have developed hip dysplasia under normal circumstances if the dogs were allowed to grow and develop (table I).

In the three muscles (obturator externus, obturator internus, and gemelli) that were severed, disuse atrophy caused a mean weight reduction of 34.1%. When the operated side was compared with the side that was not operated, the muscle weight loss for the operated side was 12.5%. The weight loss varied from 48–180 g, as indicated in table I. No reason was detected for the variation among the five dogs (table I).

The individual muscles were examined histologically, and the three cut muscles had undergone extensive fibrosis, generalized atrophy, and were infiltrated by fat. The unoperated muscles were normal, and no evidence of fibrosis, generalized atrophy or necrosis was found.

Summary

The dysplastic changes exhibited by the five dogs in both the control and operated sides were consistent with those seen in spontaneous hip dysplasia. Joint instability triggered a series of events that followed a definite pattern of joint destruction. The degree of joint destruction depended on the degree of joint instability and damage to the associated tissues.

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Hip Dysplasia as a Disease

Range of the Lesion

It is extremely difficult to describe and illustrate the wide range of changes and lesions that occur in hip dysplasia. The lesions may be mild and barely distinguishable either radiographically or after dissection, or the entire joint may become remodelled and destroyed almost beyond recognition.

Hip dysplasia is not an 'all or none' phenomenon. Even in the hip of the adult dog that is radiographically normal, the joint components vary in conformation [3]. Nine grades of variation in congruity and fit between the femoral head and acetabulum are recognized[1]. The top three are considered within the range of normal.

1 Excellent conformation
2 Normal conformation for age and breed
3 Less than ideal, but within normal radiographic limits
4 Near-normal, minor hip joint abnormalities
5 Borderline minimal dysplastic change
6 Grade 1 dysplasia, subluxated 25%
7 Grade 2 dysplasia, subluxated 50%
8 Grade 3 dysplasia, subluxated 75%
9 Grade 4 dysplasia, femoral head luxated out of acetabulum

Effects of Overloading the Joint

In the dog, the hip joint is normal at birth [5, 10]. The most critical time in the development and stability is from birth to 60 days of age. At that time the muscles and nerves of the hip are immature, and their function is limited

[1] The Hip Dysplasia Registry, Orthopedic Foundation for Animals, Columbia, Mo.

[4]. The tissues are soft, plastic, elastic, and they have an 'elastic limit', a point on its strain curve beyond which the material does not return to its original shape and size [2]. Once ossification occurs, change in shape, except for fracture, is achieved only by adding new bone in one area and taking it away in others. If weight and stress placed upon the hip by the dog exceed the strength of the supporting soft tissues and the plasticity limits of the joint tissues, the components of the hip pull apart and change shape [19]. At this immature age, incongruity of the acetabulum and femoral head triggers a series of events that retard normal development and pull the joint out of its natural shape [9].

The effects of overloading and stress may start shortly after birth and be mild or severe. They may occur in later developmental stages and again may vary in severity. The changes that occur seem to correlate with the degree and length of time of the biomechanical imbalance [11]. If the imbalance is corrected and congruity is reestablished before a certain stage in the development of the hip, progression of the dysplasia stops and the hip returns to normal development [1, 10, 14].

If full congruity can be maintained until the muscles and nerves are fully functional, muscle power is sufficient to maintain biomechanical balance so that full congruity between the parts is maintained [9]. If hip dysplasia does not occur by 6 months of age, ossification of the joint is 90% complete. At that time the tissues are too hard to respond to plastic bending, and changes in shape are restricted to adding new bone in one area and resorbing it in another [2].

Occurrence and Triggering Factors

These facts and the discussion to follow provide an opportunity to consider the validity of two premises: (1) hip dysplasia occurs only if hip joint instability and joint incongruity are present in the young child or animal, and (2) 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. At 6 months of age, function, tissue strength, and ossification have progressed sufficiently to prevent the disease under the usual circumstances.

The radiographic, gross, and histological descriptions of hip dysplasia outlined the changes for the first 9 months of life and are those of the severely

involved hip (grade 3–4) [16]. Once the scope of these severe changes is fully appreciated and recognized, the minor changes can be distinguished and correlated.

Embryonically, in all vertebral life the hip joint emerges as a unit from the mesenchyme in the region of the proximal posterior limb but with the femoral head articulating in congruity with the acetabulum. Development proceeds normally as long as there is full congruity between the acetabulum and the femoral head. At birth, the unit is composed of soft, plastic hyaline cartilage, a tissue that has high water and mucopolysaccharide content and low collagen and mineral proportions. The growth of the two parts, the acetabulum and femoral head, is synchronized and dependent on good mechanical function, lubrication, full congruity, and neutral or balanced forces for continued normal growth [19]. Any changes in biomechanical balance, stress, compression, traction, muscle pull, lubrication or congruity between the femoral head and acetabulum affect the programmed pattern of normal hip joint development [19].

Biomechanical Stress

It has been difficult for the clinician and investigator to look beyond biochemistry and pathobiology and into mechanical environmental factors for mechanisms that initiate the hip dysplasia cycle. But no evidence has been found that faulty congenital conformation or a metabolic defect is present in any mammal that develops hip dysplasia. As indicated above, development of the hip proceeds normally until some mechanical force or forces pull apart the two main components of the hip, the acetabulum and the femoral head [19]. In man, uterine compression in the first-born, breech birth, large full-term babies, and suspension of the newborn by the heels to start breathing have been cited as examples of unusual factors of stress that could contribute to the unstable hip that is associated with a high prevalence of hip dysplasia [15, 21, 22]. Postnatally, the cradle board, which binds the legs parallel to the body, has been associated with an increased prevalence of hip dysplasia [13, 15]. Conversely, in countries where it is the practice to carry an infant bound around the mother's waist with the legs in abduction, hip dysplasia is almost nonexistent [15].

In the dog, the hind legs are short while *in utero*, and the pups are born with normal hips [9]. Stress on the hip joints starts when the pup begins to force itself to the mother's mammary glands and to walk [5, 10].

Anatomical Differences Among Breeds

In considering the dog as a model for studying hip dysplasia there are several features of anatomical variation that have influenced the prevalence rate. The large breeds vary as to color, hair length, length of nose and tail. The anatomical features of the hind legs and pelvis also vary significantly [7]. Hip dysplasia occurs in all large breeds in spite of the fact that once a breed is established there is relatively no intermating between breeds [9]. The condition is characteristic of large dogs in general, therefore, and not specific to a certain breed. Height, weight, rate of growth, and pelvic muscle mass seem to be the most critical anatomical features that have influenced the rate of occurrence. The prevalence decreases markedly in those breeds with short leg bones and light body weight. The dividing point is near 30 cm in height and 10 kg in weight [9].

In our observations, unstable hip joints were also found frequently in the toy dog, cat, and chinchilla, but the bone changes were mild and did not resemble those described in the large dog. The bone changes characteristic of hip dysplasia as seen in the heavier dogs do not develop in most of the small dogs with unstable hips. It is hypothesized that in small dogs there is not enough weight, and the levers (leg bones) are too short to produce fatigue fractures, change in acetabular shape, and tears in the Sharpey fibers, the factors that trigger the dysplastic changes and degenerative joint disease [2, 17].

Structural Factors

It was pointed out as long as three centuries ago that, 'Muscle and bone... are inseparably associated and connected; they come into being together and act and react together. Between muscle and bone there can be no change in the one but it is correlated with changes in the other... This brings the similitude of engineering to biology, that is, the strength of a girder varies with the square of its linear dimension but the weight of the structure varies with the cubic of its linear dimensions. It follows at once that two bridges geometrically similar, the larger is the weaker of the two... If we tried building ships, palaces or temples of enormous size, the keels, beams and bolts would cease to hold together. Nor can Nature grow a tree nor construct an animal beyond a certain size while retaining the proportions and employing the materials which suffice in the case of a smaller structure. The thing will fall to pieces of its own weight unless we either change its relative pro-

portions... or else we must find new material, harder and stronger than used before... A tall homogenous body must increase as the $^3/_2$ of its height. Put another way, the bracing must triple as the height doubles' [20].

It has been difficult for the medical scientist who has been trained to search for defects with ultrastructural techniques to observe gross anatomical structures for variations. It becomes rather obvious, however, after examining the pelvic radiographs of 6000 large dogs that those with the greater pelvic muscle mass have the more normal hip joints [8].

This observation has been documented by dissecting and recording the weights of the pelvic muscles and setting up a muscle index for each dog dissected, an index based on the dog's weight and pelvic muscle weight. The Greyhound, a breed relatively free from hip dysplasia, has a much higher pelvic muscle mass than the breeds commonly affected with hip dysplasia. Not only was there greater muscle mass, but the fasciae, tendons, and ligaments were larger and stronger. This was true whether the Greyhound had been trained for racing previously or raised in a restricted area. The data collected from the dissections of the pelves of a large number of dogs were compared with the soundness of the hip joints. It was found that there was a positive correlation between a high pelvic muscle mass index and normal hip joints. These data lent themselves to statistical analysis of the considerations of probability. The probable occurrence of hip dysplasia could be predicted by the index of the pelvic muscle mass [11].

Control and Prophylactic Measures

'If routine examination of the hips in every newborn human infant can be nationally established, the problem of congenital dislocation of the hip which has plagued orthopedists in times past can be almost abolished' [6].

A reduction in the incidence of hip dysplasia in the Navajo Indians occurred when diapers replaced the cradle board. The cradle board held the femurs in adduction; the diapers held the legs in abduction and forced the femoral heads into the acetabulums [13]. These findings detract from the disease being genetically conditioned in this inbred population.

I believe that if dogs were selected and produced with as much pelvic muscle mass and strength as the Greyhound possesses, hip dysplasia would no longer be a major problem. Breeders who have bred only dogs with radiographically normal hips have, without exception, lowered the prevalence of hip dysplasia in the offspring [12].

It is SOKOLOFF's [17] conclusion that, 'The balance of the present evidence shows that a genetic predisposition to congenital dislocation of the hip is real, and that multiple genes are involved. Environmental factors are also of importance, presumably according to the genetic susceptibility of the individual'. It should not be forgotten that bone, cartilage, and tendons and ligaments have fatigue limits, the same as in the materials and bearings of machines. Failure in machines keeps the motor car off the highway, and it is logical that similar failures are related to dysfunction of the hip joints.

Conclusions

The literature review and report of our work presented here have revealed a basis for optimism in controlling and reducing the prevalence of hip dysplasia in both man and animals. A number of the possibilities for coping with the disease have been explored, and clinicians and researchers have gathered many good prophylactic tools. In children, it is basically the test for hip laxity at birth, and for the dog it is restriction of breeding only those animals with radiographically normal hips. Both in children and dogs, hip dysplasia is, for the most part, a 'man-made' problem and can be controlled if man will use the tools at his disposal.

The essays in this monograph are a detailed description of radiographic, gross and histological lesions of spontaneous and surgically acquired canine hip dysplasia. The significant changes are recorded and compared with the rate and pattern of normal development of the hip joint from birth to maturity. These findings uphold the validity of our two premises that (1) hip dysplasia occurs only if hip joint instability and joint incongruity are present in the young child or animal; and (2) 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.

In the dog at birth and for several weeks to follow the structures of the hip are immature in function and strength. When forces exceed the strength of these tissues, hip dysplasia occurs. At 6 months of age, function, tissue strength, and ossification have progressed sufficiently to prevent this disease under the usual circumstances. Likewise in the child, as ossification progresses and the soft tissues mature, hip dysplasia no longer occurs.

Table I. Weights of experimental subjects

Subject	At surgery (4 months), kg	10 months, killed 6 months after surgery, kg	Pelvic muscle weight, g, hip soundness[1]		Difference in pelvic muscle weight, g	Pelvis muscle index corrected for muscle atrophy[2]
			Left	Right		
D88	10.9	23.6	986 G 1	945 G 3	41	8.5
D89	8.6	24.1	1126 G 2	956 G 3	170	9.0
D90[3]	10.9	21.4	984 N	804 G 3	180	9.3
D91[3]	11.3	19.0	815 N	766 G 3	49	8.6
D93	13.1	22.3	1077 G 3	916 G 4	161	9.5

[1] Schnelle grading system for canine hip dysplasia: G 1 = femoral head subluxated one quarter way out of the acetabulum; G 2 = femoral head subluxated half way out of the acetabulum; G 3 = femoral head subluxated three quarter way out of the acetabulum; G 4 = femoral head luxated out of the acetabulum.

[2] All large dogs with a pelvis muscle mass index of 9.00 and below have a probability of developing canine hip dysplasia in both hips.

[3] D90 and D91 had radiographically normal hips, but at dissection mild dysplastic changes were present.

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