This is to certify that the

thesis entitled

The Measurement of Canine Coxofemoral Joint Laxity

presented by

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has been accepted towards fulfillment
of the requirements for

M.S. degree in Mechanics

Major professor

Date 11/14/86

# THE MEASUREMENT OF CANINE COXOFEMORAL JOINT LAXITY

By

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A THESIS

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

MASTER OF SCIENCE

Department of Metallurgy, Mechanics, and Material Science

1986

ABSTRACT

THE MEASUREMENT OF CANINE COXOFEMORAL JOINT LAXITY

By

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The purpose of this research is to develop an instrument to measure laxity in the coxofemoral joints of anesthetized canines. Animals of various sizes and ages were tested and a quantitative measurement of the laxity in each hip joint was obtained for each animal. The measurements were then converted to a laxity index which would allow the clinician to compare joint laxity for normal, bilaterally lax and unilaterally lax animals regardless of their age, sex, breed or size. The test protocol is non-invasive and non-injurious and the results obtained from the test were reproducible.

The effectiveness of present joint laxity diagnostic techniques is considered and a correlation is drawn between joint laxity and morphological abnormalities of the joint connective tissue.

To my parents, for their love and support.

ACKNOWLEDGEMENTS

The author wishes to express his deepest appreciation to the following people for making the completion of his Master of Science degree possible:

To Dr. Robert Wm. Soutas-Little, his major professor, for his encouragement, friendship, and invaluable assistance in the preparation of the manuscript.

To the Orthopedic Foundation for Animals, for their generous funding of the project.

To Dr. Ulreh Mostosky, for his assistance in the project.

To Robert E. Schaeffer, for his amazing technical assistance and his invaluable instruction in the finer points of the design process.

To Jane Walsh, for her assistance with the project and her warm friendship.

To Lisa Stewart and Mark Jones for their assistance with the project and, more importantly, their invaluable friendship.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

## LIST OF SYMBOLS

| Symbol | Definition |
|---|---|
| $\varnothing_l$ | Femoral head diameter, left |
| $\varnothing_r$ | Femoral head diameter, right |
| D | Distance between the left and right cranial effective acetabular rims |
| $d_l$ | Overlap distance, left |
| $d_r$ | Overlap distance, right |
| $\delta_l$ | Overlap ratio, left |
| $\delta_r$ | Overlap ratio, right |
| $\mu_l$ | Laxity index, left |
| $\mu_r$ | Laxity index, right |
| $P_l$ | Normalized load, left |
| $P_r$ | Normalized load, right |
| L | Applied load |
| $K_l$ | Joint stiffness, left |
| $K_r$ | Joint stiffness, right |

# I. INTRODUCTION

Canine hip dysplasia is thought to be a non-congenital, polygenetically inherited and usually progressive disease of the hip. It was first reported in veterinary medicine, in 1935, and it is still considered to be one of the major orthopedic problems in many breeds of dog. In some breeds as many as 47% of the animals (1) may be affected by it. There is no known cure, and the only accepted means of controlling the disease is through selective breeding.

The term hip dysplasia literally means any malformation of the hip. When used in reference to small animals, particularly canines, the term denotes an abnormality characterized radiographically by remodeling of the femoral head and neck, a shallow acetabulum, coxofemoral subluxation and secondary degenerative joint disease.

In order to understand the abnormality, one must be familar with both the anatomical and biomechanical functions of the hip joint. The joint is joined by the ligament of the femoral head, a strong strand of collagenous tissue which runs from the acetabular fossa to the fovea capitis and the articular capsule (see Fig. 1). Early in life, the ligament of the femoral head appears to stabilize the joint as well as to supply blood to the femoral head. After 3 - 4 months of age, however, the ligament of the femoral head lengthens and the articular capsule, which encloses the joint space, assumes the role of joint stabilizer. If the tissues of the articular capsule are lax or overly compliant, the femoral head will probably move laterally or subluxate

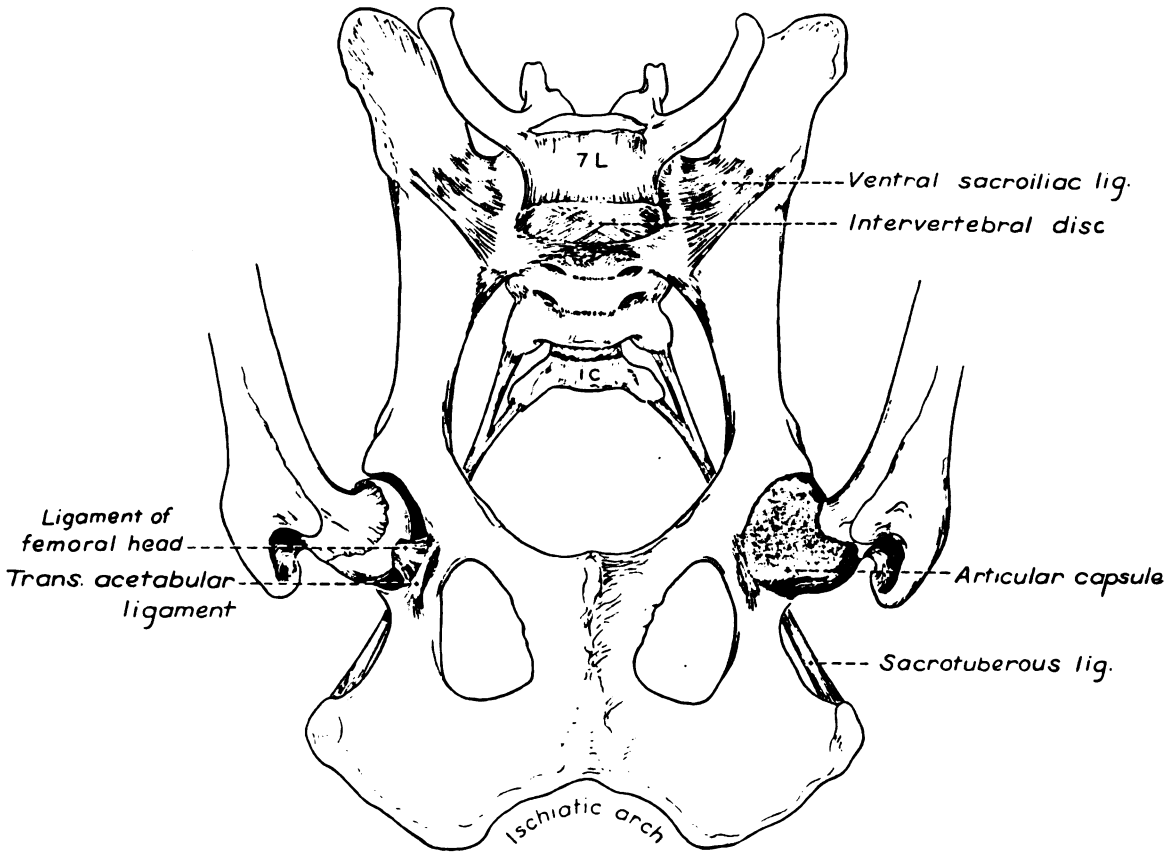


**Figure 1. Coxofemoral Joint**
**(Reproduced by courtesy of the W.B. Saunders Company, Phil.)**

from the acetabulum. When the femoral head is in this subluxated position, it no longer lies in intimate contact with the cranial acetabular edge and the load paths between the femoral head and the acetabulum become altered. These altered load paths may induce joint remodeling, especially in young developing animals. The abnormal remodeling is characterized by osteoarthritic spurs on the cranial effective acetabular rim, and flattening of the cranial acetabular edge.

Radiographs are the most acceptable means of diagnosing hip dysplasia, but they become limited as a tool for diagnosing the condition in young animals. Although dysplastic changes have been evident in animals as early as 14 days of age in post mortem examinations, these changes are not normally radiographically apparent before 6 months of age. For animals which eventually develop dysplasia, the changes are usually apparent by two years of age. For this reason, the Orthopedic Foundation for Animals (OFA), which analyzes radiographs for dysplasia, will not certify an animal as having normal hips before the animal is 2 years old. The use of radiographs for the diagnosis of hip dysplasia is totally subjective. Even though the OFA uses a minimum of three veterinarians to review the radiographs for certification, conflicting diagnostic opinions often occur in borderline cases.

In order to incorporate joint laxity into the diagnostic process, a palpation technique and "stress radiography" have been employed. The amount of lateral displacement of the femoral head was determined by palpation, and, although the method is still used by some clinicians, subjectivity made it less than desirable for widespread use. Stress radiography is a method to exhibit the joint laxity by forcing laterally both femoral heads from the acetabula by means of a medial force at the

stifles. During this loading configuration, a fulcrum is placed between the femurs, distal to the hip joints, the femurs are loaded medially at the stifle joint, and a radiograph is taken. Although the displacement of the femoral heads from the acetabula can be measured from the radiographs, the applied load remains unknown. Without knowing the effective load at the joint, the test cannot be controlled and is therefore limited as a diagnostic tool.

In human medicine a luxation examination, similar to the palpation technique in veterinary medicine, has a large degree of acceptance. This acceptance is due, however, to a large extent to the fact that human medicine does not have to contend with the wide variety in breed and patient size.

To measure joint laxity as an aid in the diagnosis of hip dysplasia, an instrument and accompanying test protocol were developed. This would enable the clinician to quantitatively measure connective tissue laxity of the coxofemoral joint and to objectively compare test results with other animals regardless of size and breed.

## II. LITERATURE SURVEY

Although Hippocrates (460-370 BC) reported hip dysplasia in humans, more than 2000 years elapsed before hip dysplasia was first observed in canines. Since this first reporting by Schnelle (2), in 1935, hip dysplasia has become perhaps the most common and frustrating problem facing modern veterinary orthopedics. During the ensuing decades, several investigations were undertaken in search of the cause of the disorder while simultaneous efforts were made to develop more accurate diagnostic techniques.

The first major contribution to diagnosing hip dysplasia came in 1955 when Schnelle (3) classified canine hip dysplasia into four catagories according to its radiographic appearance. His criteria for diagnosing dysplasia were the femoral head fit with the acetabulum and whether the acetabulum showed any shallowness or flattening. These criteria were later redefined and reevaluated. In 1961 the American Veterinary Medical Association asked a panel of ten veterinarians to report on hip dysplasia (4). The panel suggested that the radiographic positioning be standardized. They also suggested a grading system ranging from slight deviation from the norm (grade I) to flat acetabulum and dislocation of the femoral head (grade IV). This grading system was not changed until several years later. In 1972, the Orthopedic Foundation for Animals held a symposium on hip dysplasia (5), which resulted in a redefinition of the radiographically apparent characteristics of hip dysplasia. This definition included a shallow

acetabulum, femoral head flattening, coxofemoral subluxation and secondary degenerative joint disease. These characteristics have remained the radiographic diagnostic criteria of hip dysplasia to date.

The diagnostic criteria were made from observations developed mainly from older dogs whose joints had already undergone some secondary degenerative changes. Young dogs examined radiographically may not have developed degenerative changes and as a result may be mistakenly diagnosed as normal. Therefore, for young animals, the radiographic examination may be inconclusive, and other diagnostic methods are needed.

Bardens and Hardwick (6) suggested a palpation technique to predict hip dysplasia in young animals. The technique depends upon the assumption that hip dysplasia is caused by lax hip joint connective tissue. With this technique, the puppy is placed under deep anesthesia and laid on its side. Standing directly behind the patient, the clinician places the first joint of the right index finger on the ischiatic tuberosity with the thumb of the same hand placed lightly on the greater trochanter. With the left hand, the femur is approached caudally, grasped in the middle third and lifted upward while the puppy is held down with the index finger on the ischiatic tuberosity. The amount of displacement at the greater trochanter is indicative of the degree of laxity, and allegedly predictive of dysplasia. Wright and Mason (7) used this technique and reported in 1977 that he found a definite relationship between laxity diagnosed by palpation and later development of hip dysplasia. His claim was further supported by Bardens in 1979. By this time, Bardens (8) had palpated 6,000 puppies and claimed a diagnostic accuracy of 85%. He reported that all the

puppies he predicted would develop hip dysplasia did and 15% of those that he felt were normal later developed hip dysplasia. None of the puppies predicted to be dysplastic, however, ever became normal. Some of the subjects radiographed in the standard position appeared normal yet when palpated showed pronounced joint laxity, Bardens devised the wedge technique to show this laxity on the radiograph. This was done by placing a fulcrum, such as a roll of cotton, between the femurs and as close to the rectum as possible. The tibias were then grasped distal to the stifles and a medial force was applied forcing the femoral heads to translate laterally, at which time the subject's joint was radiographed. Bardens reported that those animals who were radiographically normal (OFA radiograph) but had dysplastic offspring had been determined previously to be lax using stress radiography.

To supplement the radiographic diagnosis, radionuclide joint imaging was used by Allands, et al. (9). The major advantage of this method was the information gained about the bone metabolic activity unattainable using normal radiographic procedures. It did not, however, aid significantly in diagnosing hip dysplasia.

A comprehensive study on the developments in diagnosing hip dysplasia, by Van Der Velden in 1983 (10), claimed that all of the present methods of diagnosis were too subjective and could not be used as a selective criteria for breeding. His opinion underscored the need for the development of an objective quantitative diagnostic method.

Paralleling the development of diagnostic techniques was the investigations of the cause of hip dysplasia. These investigations began in 1956 when Schales (11), using information on human hip

dysplasia as a model for canines, speculated that hip dysplasia was a dominant genetic trait.

Henricson and Olsson (12) examined some 750 German Shepherd dogs during the years 1957 and 1958. Their criterion for radiographically diagnosing hip dysplasia was abnormal shallowness of the acetabula. They did not consider subluxation or luxation to be correlated with acetabular dysplasia and doubted that "acetabular dysplasia should be only a sequela to luxation or subluxation caused by an insufficient ligament and joint capsule." They also reported secondary changes such as spur formations to be credited as sequelae to subluxation. Based on their investigation, dysplasia did not appear to be inherited as a regular monogenic characteristic, thus contradicting the speculations of Schales.

Smith et al (13), contrary to Henricson and Olsson, hypothesized that the ligament of the femoral head and articular capsule played an important role in maintaining the integrity of the hip joint. For their study, the investigators used 15 four-week-old puppies. In seven of the puppies (Group I) they excised the posterior, anterior and superior portions of the capsule of the right hip and left the contralateral hip intact as a control. In the other eight, (Group II) a similar capsulectomy was performed and a complete surgical removal of the ligament of the femoral head was performed. The animals in group I showed slight lateral displacement and mild acetabular obliquity within seven weeks. Within four weeks, five of the group II puppies showed complete dislocations. Six of the animals, including the five with dislocations, demonstrated acetabular dysplasia. The ligament of the femoral head appeared to play a vital role in joint integrity in animals

at a very young age. These investigators also demonstrated that "acetabular dysplasia is the result of the dislocation rather than the cause. Secondary changes in the head and neck of the femur such as varus deformity of the neck, flattening of the head and some reduction of the angle of anteversion were also observed as to result from experimental dislocation". This result was supported by research done by Riser and Shirer (14) who felt that the femoral head ligament served to stabilize the femoral head.

In 1966, Henricson et al. (15) compared congenital hip dysplasia in man to that in canines and found that canine hip dysplasia cannot be diagnosed at birth, is apparently non-congenital, and the primary cause is joint laxity occurring very early in life.

As it became generally accepted that joint laxity was associated with hip dysplasia, researchers began to investigate the cause of joint laxity. Riser and Shirer, in 1967 (16), correlated pelvic muscle mass in canines with the incidence of hip dysplasia. He found that the more muscle mass the animal had, the lesser the incidence of hip dysplasia. Other investigators, Pierce et al. (17), found a higher occurrence of hip dysplasia in dogs which had been injected with estrogen in early life.

In 1973, Lust (18) embarked on an investigation monitoring the growth rate in puppies. He found that puppies which were Cesarean delivered and reared at a reduced growth rate exhibited a lower incidence of canine hip dysplasia than normally born puppies which were allowed to grow at optimal and suboptimal rates.

In 1974, Cardinet et al. (19), performed a study to see if pectineal myectomy had any effect on the development of canine hip

dysplasia. They found that there was no significant difference between the side to which the pectineal myectomy had been performed and the contralateral control side.

Lust et al. (20,21) also investigated the relationship between the synovial fluid volume and joint stability. He found that increased synovial fluid volume, increased volume of the ligament of the femoral head, degenerative cartilage lesions, and mild non-supportive synovitis accompanied canine hip dysplasia. He first thought that the increased synovitic volume might have been the cause of the joint instability, but he later concluded that it was probably a secondary result of joint laxity. His study also showed that mild intraarticular abnormalities found on the necropsied animals did not show up on the radiographs, which emphasizes the limitation of radiographic diagnoses.

In 1984, Schoenecter et al. (22) did a dynamic study on growing puppies in which one hind leg was cast in extension and the other was left uncast as a control. In the cast leg they observed decreased blood flow to the femoral head through the ligament of the femoral head, progressive subluxation of the femoral head, and eventual dislocation. The continuous pressure resulted in acetabular dysplasia. There was a clear correlation between load distribution on the joint and dysplastic changes.

# III. EXPERIMENTAL METHODS

It has become generally accepted there is a correlation between joint laxity and hip dysplasia. Although several subjective methods have been developed to determine joint laxity, they are less desirable than quantitative measures. In order to quantify joint laxity measurements, a special device and an accompanying test protocol had to be developed.

Before laxity testing could occur, it was necessary to determine the load level required to cause the hip joint to exhibit abnormal laxity, but a level low enough not to damage connective tissue. Initial testing used a lateral load between 6 and 10 lbs. applied to several medium sized (40-50 lbs.) animals in order to place the connective tissue of the hip joint in tension. A 15 lb. load was reported (13) to be necessary to begin tearing of the joint capsule, so the 6 lb. load was initially chosen as a safe upper limit for medium sized animals.

During the preliminary testing, animals appeared to be in two distinct groups based on their hip joint's response to loading. Figure 2 shows typical results for this preliminary stage of testing. For ease in locating the no load and positive load data points for each dog, a line was drawn between them. It is important to note that this line does not describe the relationship between the normalized displacements and normalized load, but it is simply an aid for displaying the data end points. The normalized displacement is the distance between the medial most surfaces of the femoral heads divided by the normalizing distance

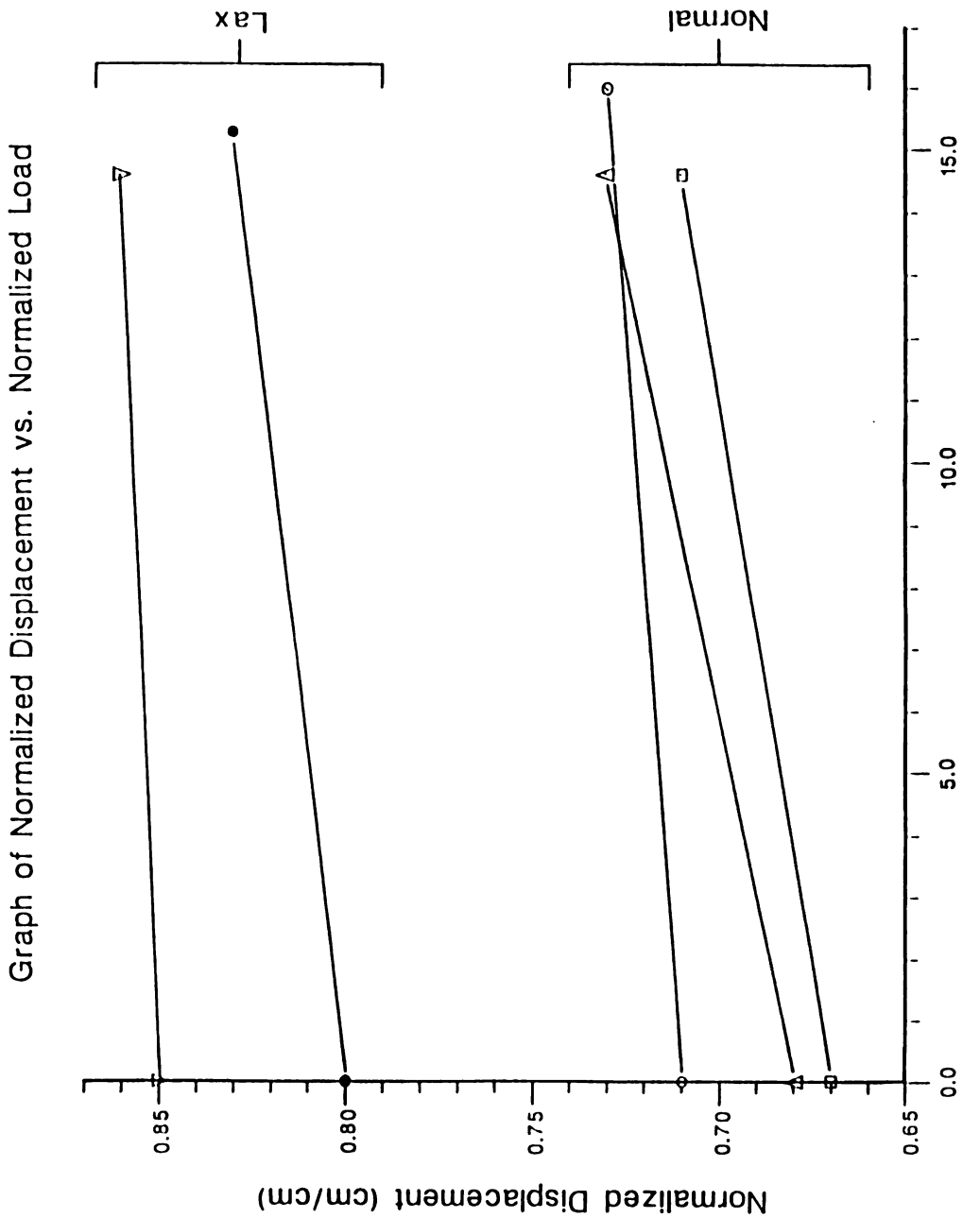


Figure 2. Preliminary Test Results

between the cranial effective acetabular rims. The normalized load is the 6 lbs. applied load converted into Newtons and divided by the femoral head diameter in centimeters. The normal group showed the femoral heads seated well within the acetabular fossa, indicating normal or desired coxofemoral articulation, whereas the femoral heads of the lax group were subluxated from the acetabula.

As preliminary testing progressed, animals outside of the medium sized group were used, and it became apparent that a scaling of applied load would have to be made to account for the variation in animal size. It was assumed that the ability of the hip joint to resist load was directly proportional to the cross-sectional area of the articular capsule. The capsule thickness could not be determined non-invasively but the circumference of the capsule was assumed to be related to the diameter of the femoral head. Therefore, the normalized load $P_1 = L/\emptyset_1$ where $P_1$ is the normalized load, L is the applied load and $\emptyset_1$ is the femoral head diameter. The subscript 1 denotes the left side. Once the data were normalized, the results for animals of different sizes could be compared. There was a reduction in data scatter, which also supported using the femoral head diameter as the scaling factor. It was noted that in all cases tested thus far, a distinct division existed between the lax and normal animals at a normalized load of 10 N/cm; that is, no further significant laxity information was obtained when the animals had greater than 10 N/cm loads applied. Thus, the 10 N/cm load became the maximum allowable applied load for the test protocol.

The following test protocol was used on all further experiments. All animals were tranquilized and anesthetized until palpebral reflexes

were lost. The animal was then placed in dorsal recumbancy on the device platform and a standard OFA radiograph was taken as shown in Figure 3. Still in dorsal recumbancy, leg cuffs were attached to the

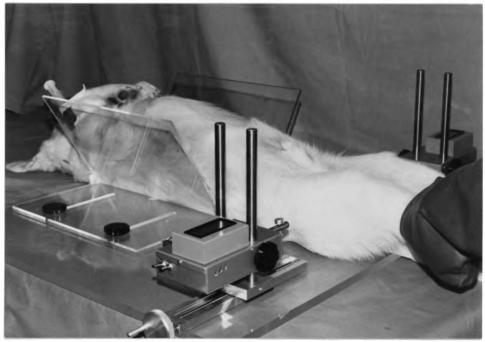

Figure 3. OFA Radiographic Position

animal's thighs and then connected to the load measuring instrument. By translating the load measuring instruments laterally, the connected leg cuffs pulled the animal's legs laterally, thereby placing the connective tissue of the hip joint in tension. The load applied to the animals legs was transmitted into the load measuring instruments via a length of Thompson* ball-groove shaft to a Sensotec model 11 ($\pm$ 25 lbs.) load cell. The load cell signal was processed using a Sensotec** SA-4 amplifier and displayed digitally. Once the load reached the normalized 10 N/cm magnitude, a ventral dorsal radiograph was taken of the joint

* Thompson Industries, Inc., Port Washington, NY
** Sensotec, 1200 Chesapeake Ave., Columbus, OH 43212

area. This radiograph was referred to as the radiograph taken under load. The animal was then released from the device and returned to the kennel. Figure 4 shows the test set-up and the load measuring instrument attached to the animal.

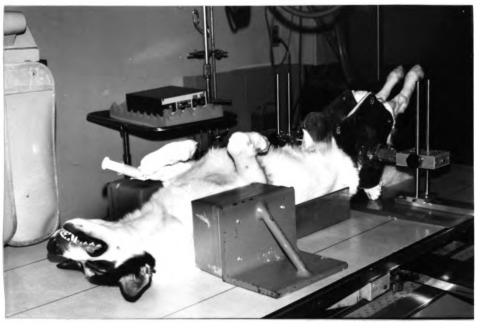

Figure 4. Testing Setup

Each animal tested was assigned a case number for identification and reference purposes. Those animals which were tested more than once were assigned an additional letter. For example, case 10b was the tenth case tested and was tested at least twice, b signifying the second time.

Several measurements were taken from the radiographs to analyze the extent of joint laxity and are shown in Figure 5. The left and right femoral head diameters, $\phi_l$ and $\phi_r$, were measured along the epiphyseal line of the femoral head. These values are used to normalize the applied load. The width of the hip was defined as the distance between the left and right cranial effective acetabular rims, and was

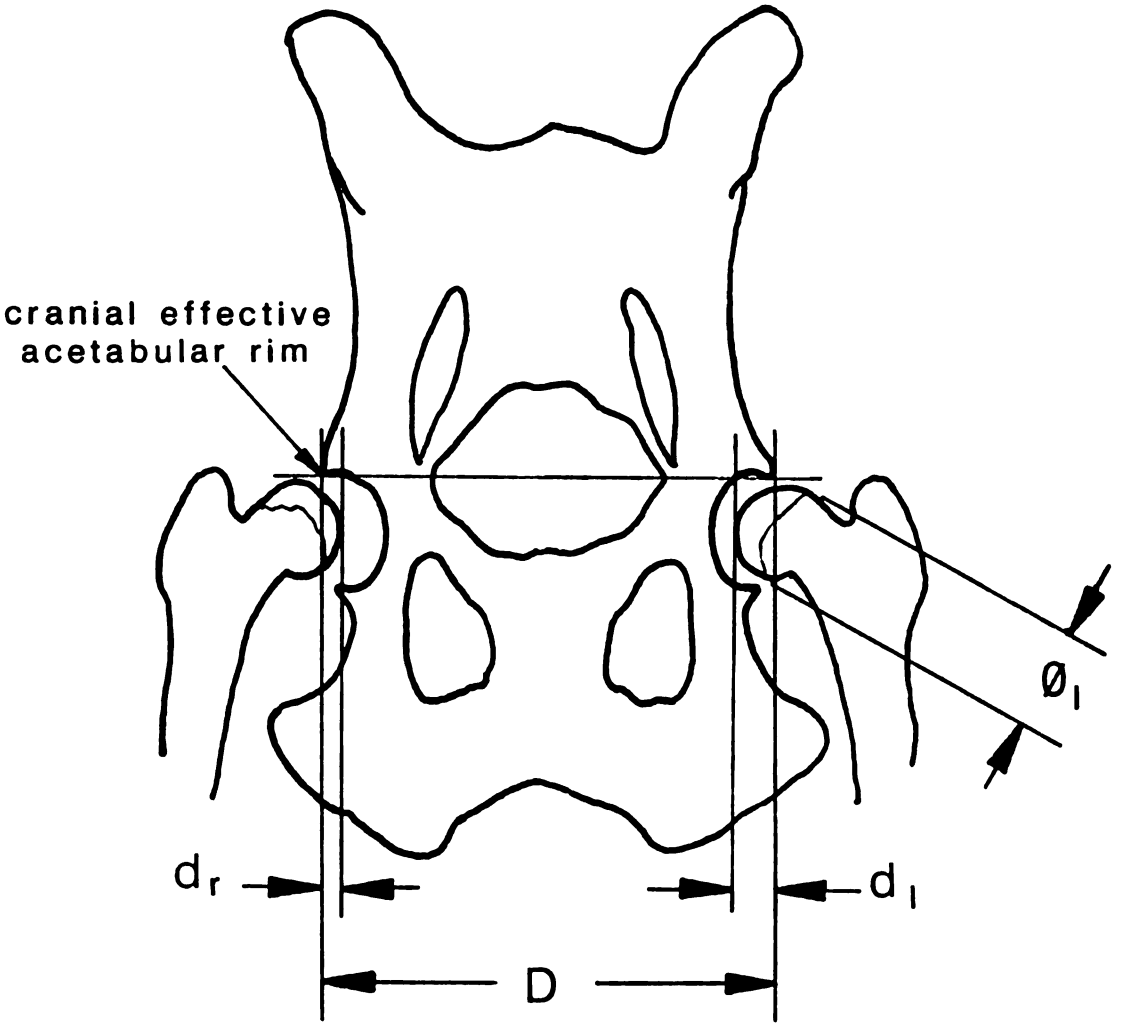


Figure 5. Measurement Diagram

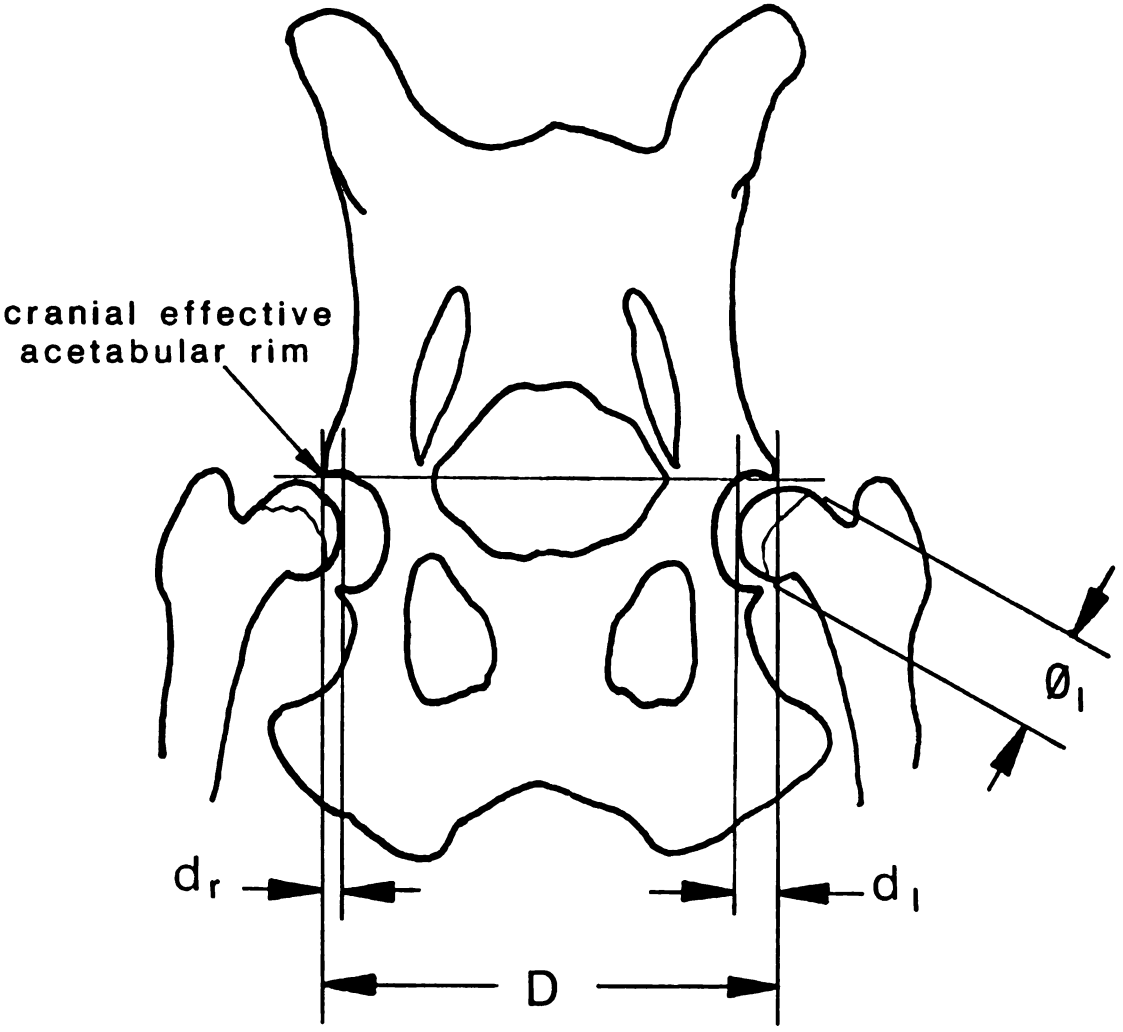


Figure 5. Measurement Diagram

labeled D. To obtain the overlap distances for the left and right femoral heads, a line was drawn connecting the cranial effective acetabular rims. Perpendicular to this line, two lines were drawn; one tangent to the femoral head and the other touching the lateral portion of the cranial effective acetabular rim, and parallel to the first. The distances between these two sets of parallel lines, are the overlap distances, $d_l$ and $d_r$, from the left and right sides, respectively. Low overlap distances indicate the femoral head is subluxated from the acetabulum. To allow comparison of overlap distances between different size dogs, the overlap distances of both joints were divided by the hip width D. This yielded ratios of overlap distances $d_l$ and $d_r$ to the hip width D and were called the overlap ratios $\delta_l = d_l/D$ and $\delta_r = d_r/D$, where the subscripts indicate the left and right sides. All measurements and ratios were made and recorded from both the OFA radiograph and the radiograph taken under load. Overlap ratios were compared between the OFA films and those radiographs taken during loading to investigate any correlation between the two methods.

Although great care was taken to apply a consistant 10 N/cm load, this was not always achieved. To account for the minor inconsistancies in loading, the normalized loads applied to the left and right hips were divided by their respective overlap ratios. These calculations were made for each hip and the results were called laxity indexes, $\mu_l = L D/d_l\emptyset_l$ and the subscripts again indicate the side.

Another more conventional method of quantifying the response of the joint to load was the calculation of the joint stiffness. This stiffness is defined as $K_l = P_l/(1-\delta_l)$ where K is the stiffness, P is

the normalized load and $\delta$ is the overlap ratio and the subscript indicates the left or right side.

Reproducibility of results was checked by retesting animals periodically at approximately one-week intervals. The data for each animal were compared for each of the tests performed.

It was assumed that the load applied to the hip joint was transmitted through the joint ligaments and not the surrounding muscle mass, as long as the animal was sufficiently anesthetized. To check this assumption, animals scheduled for euthanasia were first anesthetized, tested using the established test protocol, euthanized and immediately retested. The measurements obtained from the anesthetized and the euthanized states were recorded and compared.

For the animals tested and later euthanized, a gross post mortem examination was made of the joint ligaments and any observations were recorded. Both joints of each animal were removed intact, fixed, and stained following the standard hematoxylin-eosin procedure and then observed. The observations were then compared with the results of the laxity test.

## IV. RESULTS AND DISCUSSION

A total of 44 animals were included in initial testing to establish experimental protocol and reliability of the instrumentation. The canines ranged in age from 6 to 72 months and were mainly Siberian Husky-Labrador Retriever mixes with the exception of five chocolate Labrador Retrievers, four Siberian Huskies and one black Labrador Retriever. These animals were part of a hypertension study at Michigan State University, and they were chosen solely because of their availability. Twenty nine of the canines were females, 16 were males.

The overlap ratios obtained from the OFA radiographs are presented in Table 1. Also shown in Table 1 are the normalized loads applied to both the right and left hip joints and the overlap ratios obtained from the films taken at those loads. In cases where the normalized load varies more than $\pm$ .1 N/cm, the variation is due to the difference in femoral head diameters. That is, in those dogs, the left femoral head diameter was slightly different from the right femoral head diameter. Although the same force was applied to each joint, the joint with the smaller femoral head diameter experienced a larger normalized load. The overlap ratios range from .04 to .18 for the OFA radiographs and range from .03 to .17 for the radiographs taken under load. The significance of the overlap ratio can be seen in Figure 6. Here the left hip, (which appears on the right side of the radiograph) has an overlap ratio of only .06. This means that the overlap distance represents only 6% of the total hip span D. As the overlap ratio approaches zero, the femoral

TABLE 1. Overlap Ratios and Normalized Loads

| Case | Sex | Age(mo.) | Overlap ratios OFA $\delta_r$ | OFA $\delta_l$ | Loaded $\delta_r$ | Loaded $\delta_l$ | Normalized loads (N/cm) $P_r$ | $P_l$ |
|---|---|---|---|---|---|---|---|---|
| 16 | F | 6 | .13 | .14 | .11 | .13 | 10.5 | 9.9 |
| 17 | F | 6 | .16 | .17 | .13 | .17 | 10.6 | 10.6 |
| 18 | F | 6 | .16 | .15 | .15 | .13 | 10.0 | 10.0 |
| 19 | F | 6 | .08 | .17 | .08 | .15 | 10.5 | 10.5 |
| 20 | F | 6 | .14 | .18 | .14 | .17 | 10.4 | 10.4 |
| 21 | F | 6 | .15 | .16 | .15 | .15 | 9.4 | 9.4 |
| 22 | F | 6 | .08 | .13 | .06 | .07 | 11.1 | 11.1 |
| 23 | F | 6 | .06 | .07 | .06 | .06 | 12.0 | 11.2 |
| 24 | F | 6 | .13 | .12 | .10 | .10 | 9.9 | 9.9 |
| 25 | F | 6 | .14 | .14 | .11 | .10 | 10.1 | 10.6 |
| 30 | M | 7 | .13 | .10 | .07 | .09 | 10.0 | 10.0 |
| 31 | M | 7 | .13 | .13 | .09 | .12 | 9.9 | 9.9 |
| 32 | M | 7 | .12 | .12 | .12 | .11 | 10.0 | 10.0 |
| 33 | M | 7 | .09 | .11 | .12 | .10 | 10.5 | 10.0 |
| 34 | M | 7 | .12 | .14 | .13 | .14 | 10.0 | 9.5 |
| 26 | F | 8 | .09 | .04 | .08 | .03 | 9.1 | 9.5 |
| 27 | M | 8 | .09 | .09 | .08 | .07 | 9.5 | 9.5 |
| 28 | F | 8 | .11 | .09 | .10 | .08 | 9.5 | 9.5 |
| 29 | M | 8 | .04 | .07 | .04 | .07 | 9.2 | 10.0 |
| 6 | F | 12 | .13 | .13 | .09 | .07 | 10.6 | 10.6 |
| 10a | M | 12 | .15 | .15 | .12 | .15 | 9.1 | 9.1 |
| 38 | F | 12 | .08 | .13 | .05 | .12 | 10.0 | 10.0 |
| 40 | M | 12 | .16 | .12 | .14 | .13 | 9.1 | 9.1 |
| 39 | F | 14 | .07 | .07 | .04 | .04 | 9.5 | 9.5 |
| 42 | F | 14 | .13 | .13 | .12 | .12 | 10.0 | 10.5 |
| 1a | F | 15 | .12 | .12 | .04 | .05 | 10.6 | 10.1 |
| 11 | F | 15 | .13 | .15 | .12 | .14 | 10.0 | 9.5 |
| 10b | M | 15 | .13 | .14 | .13 | .12 | 10.0 | 10.0 |
| 36 | F | 15 | .08 | .10 | .04 | .05 | 9.5 | 9.5 |
| 41 | F | 15 | .10 | .10 | .04 | .12 | 10.2 | 10.7 |
| 1b | F | 19 | .14 | .12 | .15 | .05 | 10.0 | 10.0 |
| 7a | M | 22 | .11 | .14 | .10 | .14 | 10.1 | 10.1 |
| 12 | M | 22 | .13 | .05 | .14 | .05 | 9.2 | 9.2 |
| 13 | M | 22 | .13 | .06 | .13 | .06 | 9.5 | 9.1 |
| 14 | F | 22 | .14 | .14 | .12 | .13 | 10.0 | 10.0 |

TABLE 1. (cont'd.)

| Case | Sex | Age(mo.) | Overlap ratios OFA $\delta_r$ | OFA $\delta_l$ | Loaded $\delta_r$ | Loaded $\delta_l$ | Normalized loads (N/cm) $P_r$ | $P_l$ |
|---|---|---|---|---|---|---|---|---|
| 7b | M | 25 | .13 | .14 | .13 | .14 | 10.6 | 10.6 |
| 9 | F | 28 | .12 | .13 | .12 | .12 | 10.0 | 10.0 |
| 3a | F | 30 | .07 | .13 | .04 | .12 | 11.7 | 11.7 |
| 4 | M | 30 | .13 | .13 | .13 | .12 | 10.0 | 10.0 |
| 5a | F | 30 | .12 | .13 | .06 | .13 | 10.0 | 10.5 |
| 8a | F | 30 | .12 | .15 | .12 | .14 | 10.0 | 10.0 |
| 5b | F | 30 | .11 | .13 | .10 | .13 | 10.0 | 10.0 |
| 8b | F | 30 | .13 | .13 | .13 | .13 | 10.0 | 10.0 |
| 8c | F | 30 | .13 | .14 | .12 | .13 | 10.0 | 9.5 |
| 3b | F | 31 | .07 | .12 | .05 | .12 | 10.5 | 10.5 |
| 2 | M | 35 | .13 | .07 | .09 | .06 | 8.1 | 8.1 |
| 35a | M | 55 | .13 | .14 | .14 | .14 | 10.0 | 10.0 |
| 35b | M | 56 | .14 | .14 | .13 | .14 | 9.5 | 9.5 |
| 37 | F | 56 | .13 | .14 | .13 | .12 | 10.2 | 10.2 |
| 43a | F | 72 | .15 | .15 | .15 | .15 | 9.5 | 9.5 |
| 43b | F | 72 | .15 | .15 | .15 | .16 | 9.5 | 9.5 |
| 44a | F | 81 | .14 | .15 | .11 | .15 | 9.9 | 9.9 |
| 44b | F | 81 | .14 | .15 | .10 | .16 | 9.9 | 9.5 |

head translates laterally and approaches dislocation. Conversely, as the overlap ratio gets larger, the femoral head assumes the more desired intimate fit with the acetabulum. The left hip joint of case 10, shown in Figure 7, exhibits the physical significance of a high overlap ratio. On the right side (left in the picture) the overlap ratio is .13. Although this is not the tightest fit possible, it does represent a desirable joint configuration.

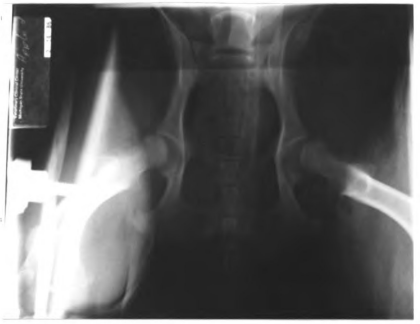

Figure 6. Load Radiograph - Case 2

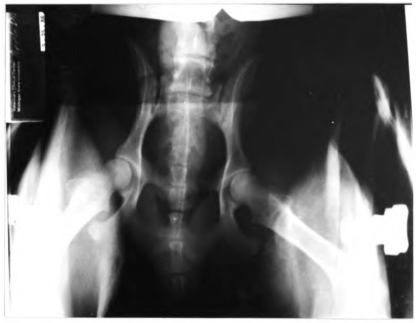

Figure 7. Load Radiograph - Case 10

As mentioned earlier, to compare cases with variations in the applied normalized loads, these normalized loads were divided by the displacement ratios to yield the laxity index. The indexes are presented in Table 2 and enable quick comparison between dogs. The indexes also offer a simple means of classifying the laxity in each joint. The higher the laxity index, the greater the laxity and vice versa. For instance, case 39 has a very high laxity index and is severely lax. Case 21, with laxity indexes of 64 for both hip joints, has normal, tight joints.

Early in this research effort, pathological tissue changes were noticed in a severely dysplastic 5 month old Samoyed. The animal was radiographed and found to have a shallow acetabulum, boney changes at the cranial effective acetabular rims as well as being severely subluxated on the left side almost to the extent of dislocation. After

TABLE 2. Laxity Indexes

| Case | Sex | Age(mo.) | Laxity index $\mu_r$ | Laxity index $\mu_l$ |
|---|---|---|---|---|
| 16 | F | 6 | 95 | 75 |
| 17 | F | 6 | 82 | 63 |
| 18 | F | 6 | 65 | 77 |
| 19 | F | 6 | 138 | 69 |
| 20 | F | 6 | 77 | 61 |
| 21 | F | 6 | 64 | 64 |
| 22 | F | 6 | 184 | 154 |
| 23 | F | 6 | 194 | 182 |
| 24 | F | 6 | 98 | 98 |
| 25 | F | 6 | 94 | 112 |
| 30 | M | 7 | 137 | 106 |
| 31 | M | 7 | 114 | 83 |
| 32 | M | 7 | 82 | 90 |
| 33 | M | 7 | 84 | 97 |
| 34 | M | 7 | 86 | 69 |
| 26 | F | 8 | 121 | 294 |
| 27 | M | 8 | 113 | 129 |
| 28 | F | 8 | 97 | 124 |
| 29 | M | 8 | 229 | 141 |
| 6 | F | 12 | 114 | 147 |
| 10a | M | 12 | 78 | 85 |
| 38 | F | 12 | 190 | 64 |
| 40 | M | 12 | 65 | 70 |
| 39 | F | 14 | 234 | 234 |
| 42 | F | 14 | 84 | 88 |
| 1a | F | 15 | 254 | 194 |
| 11 | F | 15 | 86 | 70 |
| 10b | M | 15 | 78 | 85 |
| 36 | F | 15 | 238 | 191 |
| 41 | F | 15 | 254 | 89 |
| 1b | F | 19 | 69 | 192 |
| 7a | M | 22 | 97 | 71 |
| 12 | M | 22 | 66 | 198 |
| 13 | M | 22 | 76 | 146 |
| 14 | F | 22 | 86 | 78 |

TABLE 2. (cont'd.)

| Case | Sex | Age(mo.) | Laxity index $\mu_T$ | $\mu_l$ |
|---|---|---|---|---|
| 7b | M | 25 | 80 | 75 |
| 9 | F | 28 | 85 | 85 |
| 3a | F | 30 | 263 | 96 |
| 4 | M | 30 | 76 | 83 |
| 5a | F | 30 | 167 | 81 |
| 5b | F | 30 | 100 | 77 |
| 8a | F | 30 | 85 | 72 |
| 8b | F | 30 | 77 | 77 |
| 8c | F | 30 | 85 | 74 |
| 3b | F | 31 | 192 | 87 |
| 2 | M | 35 | 92 | 138 |
| 35a | M | 55 | 70 | 70 |
| 35b | M | 56 | 72 | 67 |
| 37 | F | 56 | 80 | 86 |
| 43a | F | 72 | 64 | 64 |
| 43b | F | 72 | 65 | 60 |
| 44a | F | 81 | 87 | 64 |
| 44b | F | 81 | 96 | 57 |

this animal was euthanized, a post mortem examination revealed very thick joint capsules. The ligament of the femoral head was missing from the left hip joint, the same joint that radiographically appeared severely subluxated. Unfortunately, the laxity measuring instrument was not operational at the time this animal was radiographed and no laxity index was available for comparative purposes.

In Table 3 the laxity indexes are compared with the gross anatomical observations made at the post mortem examination for those animals which were euthanized. With the exception of case 12, joints having a laxity index of 90 or greater consistently showed thickening of the joint capsule due to edema. The joint capsule thicknesses were not recorded. Typical normal capsule thickness was approximately .5 - 1 mm whereas the thick capsules were in the order of 5 - 6 mm. According to Hickman, a stretched and thickened joint capsule and the absence of a ligament of the femoral head are common pathological findings of dysplastic dogs (23). Cases 26, 27, 28 and 29 (Table 3) had thickened articular capsules. Also observed at post mortem was the absence of the femoral head ligament and the transacetabular ligament on the left side in case 26. Joints having laxity indexes less than 90, consistently showed normal joint capsules. The only exception to this was the left hip of case 12 which appeared normal but had a laxity index of 198. There is no explanation for this exception.

The radiographic diagnoses made from the OFA radiographs by the clinician at testing were compared with the films taken under load. Animals which appeared normal under load also appeared normal in the OFA

TABLE 3. Post Mortem Observations and Laxity Indexes

| Case | Age (mo.) | RIGHT $\mu_r$ | RIGHT Post Mortem Observation | LEFT $\mu_l$ | LEFT Post Mortem Observation |
|---|---|---|---|---|---|
| 4 | 30 | 76 | NC | 83 | NC |
| 10 | 15 | 78 | NC | 85 | NC |
| 12 | 22 | 66 | NC | 198 | NC |
| 26 | 8 | 121 | TC | 294 | TC, no TAL no FHL |
| 27 | 8 | 113 | TC | 129 | TC |
| 28 | 8 | 97 | TC | 124 | TC |
| 29 | 8 | 229 | TC | 141 | TC |
| 35 | 55 | 72 | NC | 67 | NC |
| 43b | 72 | 64 | NC | 64 | NC |
| 44b | 81 | 87 | NC | 64 | NC |

TC Thick capsule

NC Normal capsule

TAL Transacetabular ligament

FHL Femoral head ligament

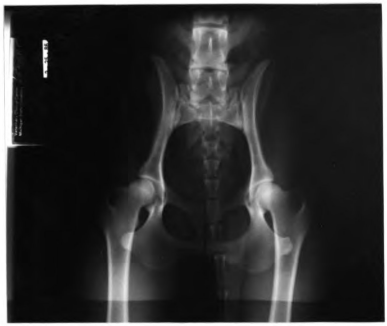

Figure 8. OFA Radiograph - Case 11

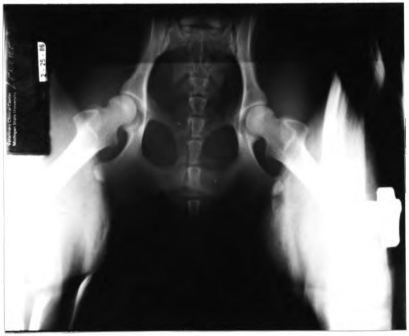

Figure 9. Load Radiograph - Case 11

radiographs (Figures 8 and 9). Both hips of case 11 exhibited normal development and lacked any secondary joint degeneration as shown in

Figure 8. The femoral head was normal, there was no apparent flattening of the acetabulum and no osteoarthritic spurring of the cranial effective acetabulum rim. Figure 9 shows the same animal with the normalized load applied to the hip. The femoral heads remained well within the acetabulum, thus, this animal exhibited no significant laxity. Both the OFA radiograph and the radiographs taken while the hips were loaded correlated well. Similarly, those animals which radiographically appeared lax or exhibited dysplastic changes, also exhibited laxity when load was applied to the hips. An example of this is case 3. Figure 10 is the OFA radiograph and showed osteoarthritic spurs on the cranial effective acetabular rim, shallowing of the

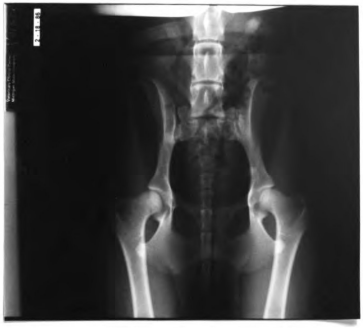

Figure 10. OFA Radiograph - Case 3

acetabulum and some minor flattening of the femoral head. These traits are all considered radiographic signs of hip dysplasia. The laxity test shows the animal being lax in the right joint (Figure 11).

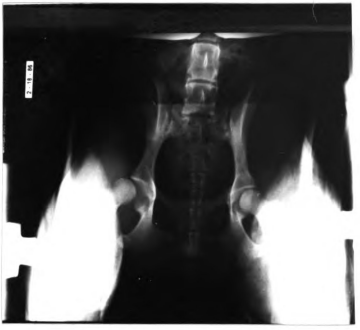

Figure 11. Load Radiograph - Case 3

In some cases, the OFA radiographs did not correlate with the radiographs taken under load. These animals were diagnosed as having normal, tight hips in the OFA radiograph; but under load, the femoral heads subluxated from the acetabula, often severely so. An example of this is case 1a, which is shown in Figures 12 and 13. In Figure 12 there are no apparent signs of secondary degenerative joint changes. Figure 13, however, shows the same animal after the normalized load was applied. Clearly, the animal is lax, having a right and left laxity index of 254 and 194, respectively. This animal was diagnosed from the OFA radiographs as normal, yet was shown to be lax. Similar observations were made by Bardens while using the wedge technique (8).

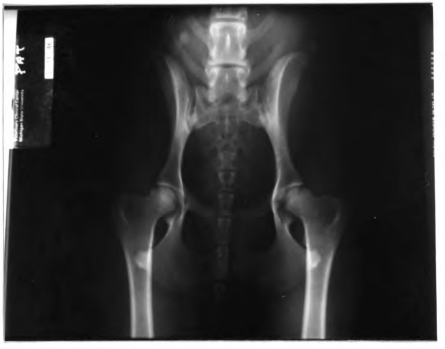

Figure 12. OFA Radiograph - Case 1

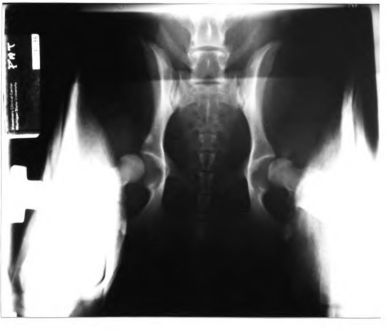

Figure 13. Load Radiograph - Case 1

Table 4 summarizes the results of several tests performed on two animals to investigate reproducibility of results. Case 8 was tested three times at one week intervals and the overlap distances under the same load did not vary more than 1 mm or 9%. Case 35 was retested after a three week interval and overlap distances did not change for the left hip, and varied only 1 mm or 7% for the right. The overlap measurements did not vary significantly between trials, and the tests were assumed to be reproducible.

Animals were anesthetized and tested and then euthanized and retested to investigate the role muscles play in stabilizing the hip joint while the animal is anesthetized. The overlap distances did not vary more than 1 mm between the two states and it was concluded that the plane of anesthesia used was sufficient to eliminate active muscle response.

TABLE 4. Result Reproducibility Test

| Case | Date of test | Age (mo.) | Load (lbs.) | Overlap Distance (mm) $d_r$ | $d_l$ |
|---|---|---|---|---|---|
| 8a | 2/21/86 | 30 | 4.5 | 11 | 13 |
| 8b | 2/28/86 | 30 | 4.5 | 12 | 12 |
| 8c | 3/05/86 | 30 | 4.5 | 11 | 12 |
| 35a | 5/21/86 | 55 | 4.7 | 14 | 14 |
| 35b | 6/18/86 | 56 | 4.7 | 13 | 14 |

The stiffness of each joint is presented in Table 5. Unlike the scale of the laxity indexes which ranged from 57 to 294, the stiffness scale is more compressed, ranging from 8.6 N/cm to 12.8 N/cm. The stiffness may be used as a measure of the connective tissue mechanical

Table 5. Joint Stiffnesses

| Case | Sex | Age (mo.) | Stiffness (N/cm) $K_r$ | $K_l$ |
|---|---|---|---|---|
| 16 | F | 6 | 11.8 | 11.4 |
| 17 | F | 6 | 12.2 | 12.8 |
| 18 | F | 6 | 11.8 | 11.5 |
| 19 | F | 6 | 11.4 | 12.4 |
| 20 | F | 6 | 12.1 | 12.5 |
| 21 | F | 6 | 11.0 | 11.0 |
| 22 | F | 6 | 11.8 | 11.9 |
| 23 | F | 6 | 12.8 | 11.9 |
| 24 | F | 6 | 11.0 | 11.0 |
| 25 | F | 6 | 11.2 | 11.8 |
| 30 | M | 7 | 11.0 | 11.0 |
| 31 | M | 7 | 10.9 | 11.3 |
| 32 | M | 7 | 11.4 | 11.2 |
| 33 | M | 7 | 11.1 | 11.2 |
| 34 | M | 7 | 11.9 | 11.2 |
| 26 | F | 8 | 9.9 | 9.8 |
| 27 | M | 8 | 10.3 | 10.2 |
| 28 | F | 8 | 10.6 | 10.3 |
| 29 | M | 8 | 9.6 | 10.8 |
| 6 | F | 12 | 11.6 | 11.4 |
| 10a | M | 12 | 11.0 | 10.9 |
| 38 | F | 12 | 10.5 | 11.4 |
| 40 | M | 12 | 10.6 | 10.5 |
| 39 | F | 14 | 9.9 | 9.9 |
| 42 | F | 14 | 11.4 | 11.4 |
| 1a | F | 15 | 11.0 | 10.6 |
| 11 | F | 15 | 11.4 | 11.0 |
| 10b | M | 15 | 11.5 | 11.4 |
| 36 | F | 15 | 9.9 | 10.0 |
| 41 | F | 15 | 10.6 | 12.2 |
| 1b | F | 19 | 11.8 | 10.5 |
| 7a | M | 22 | 11.2 | 11.7 |
| 12 | M | 22 | 10.8 | 9.8 |
| 13 | M | 22 | 10.9 | 9.7 |
| 14 | F | 22 | 11.4 | 11.5 |
| 7b | M | 25 | 12.2 | 12.3 |
| 9 | F | 28 | 11.4 | 11.4 |

Table 5. (cont'd.)

| Case | Sex | Age (mo.) | Stiffness (N/cm) $K_r$ | $K_l$ |
|---|---|---|---|---|
| 3a | F | 30 | 12.2 | 13.3 |
| 4 | M | 30 | 11.5 | 11.4 |
| 5a | F | 30 | 10.6 | 12.1 |
| 5b | F | 30 | 11.1 | 11.5 |
| 8a | F | 30 | 11.4 | 11.6 |
| 8b | F | 30 | 11.5 | 11.5 |
| 8c | F | 30 | 11.4 | 10.9 |
| 3b | F | 31 | 11.1 | 11.9 |
| 2 | M | 35 | 8.9 | 8.6 |
| 35a | M | 55 | 11.6 | 11.6 |
| 35b | M | 56 | 10.9 | 11.0 |
| 37 | F | 56 | 11.7 | 11.6 |
| 43a | F | 72 | 11.2 | 11.2 |
| 43b | F | 72 | 11.2 | 11.3 |
| 44a | F | 81 | 11.1 | 11.6 |
| 44b | F | 81 | 11.0 | 11.3 |

properties. Joints with high stiffness have low laxity indexes and high overlap ratios.

Another important aspect of this research effort was to quantitatively describe the changes which occurred in joint laxity with aging. To investigate this, four animals, cases 1, 3, 7 and 10, were tested twice with intervals of 4, 9, 3 and 3 months, respectively. As can be seen from Table 2, the left hip joint, for case 1, saw no appreciable change in laxity over the 4 month period between 15 and 19 months of age. The right hip joint, however, became substantially less lax over this time period, showing a laxity index decrease from 254 to 69. Case 10 was tested at 12 months and 15 months of age and the laxity ratios in both hips remained constant. Case 7 was tested at 22 and 25 months of age, and the laxity index decreased in the right hip and increased slightly in the left. Case 3, however, had a moderate decrease in laxity on the left side and a major decrease on the right side, going from 263 to 192 when tested at 30 months and 31 months of age. The ages at which the animals were tested were not comparable in these cases and no conclusions could be drawn concerning joint changes during maturation or aging from this small sample. A new group of research puppies will be tested at 3 month intervals from age 3 months to 18 months and for any additional animals 18 months and older, the testing will take place at 6 month intervals.

In conclusion, the coxofemoral joint laxity in dogs was quantitatively measured. The laxity measurements, reported in terms of laxity indexes and joint stiffnesses, offer an objective means of comparing joint laxity regardless of dog breed, size, or age. The

significance of these measurements, with respect to the development of hip dysplasia, has yet to be determined.

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