A COMPARISON OF THE WEIGHTS OF THE PELVIC MUSCLES OF NORMAL AND DYSPLASTIC DOGS

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INTRODUCTION

The description "congenital dysplasia of the hip joint" has been adopted by the veterinary profession to depict a condition in dogs characterized by a failure of the proper development of the hip joint as a whole. It can be shown radiologically as varying degrees of failure of proper fit of the femoral head to the acetabulum. Clinically, it is characterized by an abnormal gait and at necropsy by the reaction of soft and bony tissue to constant microtrauma implicit in abnormal mobility of the femoral head (Snavely, 1959).

Reduced to its etymological translation, dysplasia of the hip simply means "bad form of the hip." Records of a similar disorder in man date from Hippocrates. The first description of the malformation in the dog, however, was not made until 1935 by Schnelle.

During the past three decades there has been much semantic discussion of the propriety of the name given to the condition seen in the dog. Congenital does not seem to be applicable since the joint of the newborn puppy has been shown to be normal in appearance, both grossly and histologically.

Many other terms have been applied to the condition; these include congenital dislocation, congenital subluxation, congenital luxation and acetabular dysplasia.

Hip dysplasia is not Legg-Calves-Perthes disease (asceptic necrosis) or epiphysiolysis, two other conditions affecting the hip joint. These conditions occur at different ages and in different types of dogs than hip dysplasia and are the result

of interrupted vascular supply to the area of the femoral head (Wamberg, 1961).

This maldevelopment of the joint seems to be inherited and affects most of the large breeds of dogs with an incidence of 50 per cent or more (Riser, 1963). Since this involves the working, hound and sporting groups of dogs, which are still used in capacities other than pets and whose functioning is disabled by hip dysplasia, the importance of establishing the etiology of the condition is evident.

The hip joint is so structured that muscle pull is necessary to prevent luxation when the joint bears weight. Riser (1963) determined that the pelvic muscle mass of dysplastic dogs is less than that of normal animals.

Since one factor in holding the joint in congruity is the muscle mass of the area, this study was performed to determine whether the lack of development of the pelvic muscle mass of dysplastic dogs is general or whether it involves one of the action groups (flexors, extensors, adductors, abductors) or whether it involves only individual muscles. With the establishment of the exact muscular components involved, further study could be directed toward these muscles.

REVIEW OF LITERATURE

Clinical symptoms are present in only twenty to twenty-five per cent of all radiologically diagnosed dysplastic dogs (Wamberg, 1961). The first symptoms to appear in puppies are; clumsiness, swaying of the rear quarters, a slight limp in one or both hind limbs and an insufficient drive with the rear quarters. With increasing age further symptoms appear such as lameness following exercise, difficulty in climbing up and down stairs, a shorter hind limb stride, pain with pressure over the hips, sitting with the hind legs extended forward or to the side rather than under the pelvis on its haunches, incoordinated gait and a desire to sit (Konde, 1954; Wamberg, 1961 and Mostosky, 1962).

In man, hip dysplasia can be initially diagnosed by the Ortolani click, which is produced by a manual luxation of the joint resulting in a clicking noise. During this procedure, one can feel the femoral head slip in and out of the acetabulum (Andren and von Rosen, 1958). There is some debate as to whether this can be done in dogs. Henricson et al., (1965) claim to be able to perform this manipulation in dogs under anesthesia at the earliest age of two weeks. Bardens and Hardwick (1968) were very enthusiastic about this as a diagnostic aid in pups at four weeks of age.

Even with the clinical signs, which are not seen with a great amount of frequency, and palpation, which at best can only be positive for the more extreme cases of dysplasia, the condition must be diagnosed radiologically for certain confirmation. Although

the radiograph is the most accurate diagnostic tool of the veterinarian, the picture must be taken with extreme caution and with standard positioning. Lawson (1963), Riser (1962), Riser and Rhodes (1966) and Whittington et al., (1961) considered the accurancy of positioning and gave directions for the correct procedure. Age of radiographic diagnosis varies with the severity of the condition and can be made at four weeks (Maksic and Small, 1962), although most suggest six months or older (Wittington, et al., 1961).

There have been two systems worked out to measure the acetabular depth on radiographs and thus determine the degree of shallowness that is present. It is the shallowness or the degree of development of the acetabulum which is diagnostic for hip dysplasia (Rhodes and Jenny, 1960, and Norberg, as cited by Olsson 1961b). On the basis of the different degrees of severity of the pathological changes, Schnelle (1954) set up a grading system of one to four, representing a range of a slightly poor fit between the femoral head and the acetabulum to a very severe displacement of the head of the femur from the acetabulum.

Since the variations of hip dysplasia affect at least forty breeds of dogs, it is essential to determine its etiology. The mode of inheritance was at one time considered to be recessive (Schnelle, 1954), but more recently workers in the field indicate this is a misconception. Work done by Faber, Hooff and Verschuer as quoted by Schales (1956) demonstrated that in man hip dysplasia is transmitted as a dominant characteristic with irregular penetrance. After studying the Swedish Army dogs, Henricson and

Olsson (1959) postulated an incomplete penetrance of possible monogenic characters, rather than a simple recessive inheritance. They further indicate that the incompleteness may be a result of external environment or the effect of the genetic environment. Schales (1962) did a thorough geneological study of dogs and concluded that in fact canine hip dysplasia is inherited as a dominant characteristic with irregular manifestation. Hutt (1967) theorized a polygenic trait caused by cumulative action of an undetermined number of genes. A common feature of this type of character is a gradation between extremes of the expression of a trait, which is characteristic for hip dysplasia. He also stated that the degree of expression can be influenced by the environment. Kamen and Gossling (1967) also proposed that the genetic connotation is complex and multifactorial. Hein (1963) considered the manifestation of dysplasia as a phenotype to be influenced by the presence or absence of an epistatic gene or environmental factors or both together, thus explaining incomplete penetrance. She also theorized that inheritance is multifactorial or polygenic due to the wide variation in severity of the defect. Bonfors et al., (1964) described the inheritance as autosomal since there is an equal sex distribution in dogs. He indicated the penetrance was according to the formula $\frac{n}{N}$ (the number of parental combinations with at least one defective divided by the total number of litters comprising at least one defective) which equals sixty percent; or in other words forty percent of the German Shepherd breed carrying the genetic background for hip dysplasia are phenotypically normal.

Further evidences, other than paper geneological studies, that hip dysplasia is inherited are actual reductions of the condition by planned breeding. Thus, hip dysplasia was eliminated from a kennel of Samoyeds (McClave, 1957) and the Swedish Army has also been able to reduce the incidence by almost one half in their kennel by selective breeding (Henricson and Olsson, 1959).

Establishment that hip dysplasia is inherited is not generally contested, but much controversy does arise in determining what prime etiological factor is genetically carried. Basically there are three different theories of primary cause:

a. A congenital malformation of the bones of the joint which causes dislocation.

b. Weakness and laxity of the joint capsule and ligaments due to a hormonal imbalance and therefore failure to keep the joint in congruity.

c. Lack of muscle mass for some unknown reason, possibly disuse atrophy or a physiological atrophy, which results in a failure to hold the joint in place.

Badgley (1949) considered the condition a response to a timing concept malfunction. He proposed a dynamic concept of a changing structure with an inherited alteration in the proper timing of growth or overstimulation leading to faulty development. Although growth is potentiated intrinsically, extrinsic factors may alter the intrinsic design.

Hart, as quoted by Innes (1957), is the major proponent of the theory of a primary defect in the development of the acetabulum, which he considers as the predisposing cause of hip dysplasia.

Schnelle (1959) and Schales (1959) also considered the lesion to be primarily the result of a faulty development of the acetabulum. Paatsama et al., (1966) demonstrated histological and histochemical changes occurring first in the acetabular cartilage and the epiphysis of the femoral head of dysplastic dogs. Paatsama and Rissanen (1965) found these changes occurring as early as three and one half months of age, before the condition was radiologically seen.

Although Henricsson and Olsson (1959) considered an acetabular lesion primary, Henricsson et al., (1966) reversed this opinion and stated there is no evidence of a primary underdevelopment of the acetabulum. Some experimental work by Smith (1958) indicated that aplasia of the acetabulum is a result of dislocation rather than the cause. This theory is based on the fact of experimental dislocation leading to an acetabular dysplasia (Smith, 1963 and Smith et al., 1958). Langenskiold and Laurent (1966) also found that experimental dislocation is followed by dysplasia. Caffy et al., (1956) studied the normal variation of the acetabular angles of infants and concluded that the acetabular predislocation theory was not valid. Andren and von Rosen (1958) also considered the hypoplasia of the acetabulum to be a result of hip dislocation rather than the cause.

Chapple and Davidson (1941) and Hofmeyer (1963) emphasized the fact that the conversion of the cartilagenous acetabulum and femoral head to bone depends on induced pressure of the two structures on each other. Therefore, if the femoral head is not

closely invested in the acetabular socket the needed stimulus for ossification is diminished. They concluded that hip dysplasia is a result of a relaxation or looseness of the soft tissues associated with the joint, which are incapable of keeping the bones in tight congruity. Forsyth and Paschall (1963) believed that hip dysplasia is only one component of an inherited, generalized condition involving variation in strength and tone of the joint capsule and supporting structures, which are, in turn, susceptible to forces applied by weight bearing, muscle pull and an alteration in the positioning of the bony elements composing the joint in question.

According to Henricson et al., (1966) the main feature of hip dysplasia is a varying degree of laxity of the hip joint permitting subluxation early in life, therefore giving rise to skeletal changes. This same theory of relaxation of the joint capsule was also proposed by Howorth (1947). The primary cause of dysplasia is considered by Langenskiold et al., (1962) to be a flaccidity of the capsule and ligaments of the hip joint, the stretched capsule therefore allowing displacement and deformities of the acetabular socket and femoral head. This dislocation is only a part of an increased pelvic instability. Somerville (1961) also considered the pathological changes secondary to a primary relaxation of the capsule, predicated on maternal hormone influence.

Andren (1960) stated that the etiology of hip dysplasia is primarily due to maternal hormones which cause a relaxation of the capsule and ligaments. Therefore the inherited characteristic is an increased reaction to maternal hormones and not the

dislocation or dysplasia. In 1962 he further stated that hip dysplasia was only one sign of a general pelvic instability and that the effect of maternal hormones is specific for all the tissues of the pelvis. This concept is based on work done by Andren (1961) and Andren and Borglin (1961a,b) where they found, by urine analysis, a disordered estrogen metabolism of newborn children. They also administered large doses of estrogen and caused a widened symphysis pubis in experimental animals. They have also induced abnormal 17-B estrogen excretion (which is diagnostic for dysplastic children) in normal children by injecting exogenous 17-B estrogen. They conclude that there is some abnormal enzymatic malfunction in the metabolism of estrogen.

Zaffaroni (1958) reported that an overdose of estrogenic hormones in cattle and sheep causes elasticity of the ligaments which is followed by luxation of the femoral head from the acetabulum. Wilkenson (1963) produced hip dislocation in rabbits by inducing joint laxity with hormones, and also considers breech malposition as a contributing factor by stretching the weakened capsule. Mansson and Norberg as quoted by Riser (1964) treated newborn puppies with relaxin and estrogen or treated the dam with relaxin and thus induced dislocation of the hips in the puppies. Pierce et al., (1964) were able to induce joint laxity and eventual dislocation of the coxofemoral articulation by prolonged treatment with estradiol. Pierce and Bridges (1966) found a lowered capacity to metabolize biologically active estradiol in animals with inherited hip dysplasia. They

considered dysplasia to be secondary to the joint laxity produced by hyperestrogenism due to an inherited malfunction of the enzyme activity involved in estrogen metabolism.

Riser (1963) considered the condition as the result of a biomechanical imbalance and thus the joint demands a certain amount of development of the ligaments and muscles of the area in order to have the needed integrity. He believed the prime reason for the malformation of the joint was a lack of development of the pelvic muscle mass. Riser and Shirer (1967) established a difference in muscle mass between dysplastic and normal dogs and found that they could differ by as much as fifty percent. Brookes and Wardle (1962), working with human material, found that unbalanced muscle action alters the shape of bone.

There are others who consider that there is a basic muscle weakness, but the definition of an exact muscle or group of muscles that is responsible varies. Bardens and Hardwick (1968) found the pectineus muscle especially atrophic and suggested a close look at all the adductor muscles with special attention given to the muscles innervated by the obturator nerve. Bado (1961) suggested an insufficiency of the adductor muscles while Mau (1961) believed there is a reduced efficiency of the abductor muscles. Riser and Shirer (1964) also suggested the abductor muscles and rotator muscles are most responsible for holding the joint stable.

Working with normal neonate dogs, Fox (1963a,b) observed an initial muscle flaccidity at birth, which changed to an increasing dominance of flexor tone and then to a period of extensor dominance

with a balanced tone as the animal matured. Salter et al., (1963) found that constant extension will lead to dysplasia in pigs while constant flexion will not. Fox (1964) observed a longer period of extensor dominance in neonate dogs that proved to be dysplastic with maturity.

As a companion of a lowered muscle mass Riser et al., (1964) suggested that in those large breeds predisposed to dysplasia, rapid growth and weight gain during the critical first four and one half months may injure the soft supporting tissue. Pierce and Bridges (1966) agreed that the increased weight and rate of growth may alter the performance of the weakened articulation, thereby enhancing abnormal development.

MATERIALS AND METHODS

The basis for this research was a litter of purebred Labrador Retrievers which had been radiographically diagnosed to have grade four hip dysplasia. These animals had been originally donated to the College of Veterinary Medicine of Michigan State University. The litter was comprised of two females and three males whose weights ranged from 25.0 to 17.1 kg (55.0 to 37.5 pounds). Age at the time of dissection ranged from seven to nine months.

With these animals as a reference point and proceeding on the assumption of Riser (1963) that there is a critical weight of 25 to 30 pounds, such that dogs above this weight will be prone to dysplasia while dogs below this weight will most likely be normal, a second group was approximately matched by weight. Thus, dogs were sought with normal hips whose weight and body conformation closely approximated the dysplastic group. Five adult mongrel dogs were obtained from the Departments of Physiology and Pharmacology. There were two females and three males in this group also and their weights ranged from 25.0 to 20.4 kg (55.0 to 45.0 pounds).

As a matter of interest and to evaluate a proposal of Riser (1963) that those dogs below the critical weight should have a relatively higher pelvic muscle mass than the dysplastics, five additional mongrel dogs were acquired from the Michigan State Veterinary Clinic. These were two females and three males weighing from 9.1 to 7.7 kg (20 to 18 pounds) and whose age

ranged from approximately six months to adulthood. Table 1 summarizes these groups.

All fifteen dogs had been euthanatized the day of dissection or one day previously in which case the cadaver was stored in a cooler. At the time of dissection each animal was skinned at the pelvis and pelvic limb and any excess fat was trimmed from the area. The sixteen muscles dissected were as follows:

Quadriceps femoris
Biceps femoris
Adductor magnis et brevis with adductor longus
Semimembranosus
Middle gluteal
Semitendinosus
Gracilis
Sartorius
Iliopsoas
Superficial gluteal
Internal obturator
External obturator
Deep gluteal
Pectineus
Quadratus femoris
Gemelli

Determination of which muscles to weigh was made on the basis of a similar project completed by Riser (1963). According to Miller et al., (1965) who have listed the muscles of the region and their actions at the hip joint, five action groups of muscles were composed. Two groups of flexors were made since only one head of the quadriceps actually is considered to act on the hip. The groups and their muscles are as follows:

1. Adduction
   a. adductor magnus et brevis with adductor longus
   b. gracilis
   c. sartorius
   d. pectineus

2. Abduction
   a. biceps femoris
   b. internal obturator
   c. external obturator
   d. deep gluteal
   e. quadratus femoris
   f. gemelli

3. Extension
   a. biceps femoris
   b. semimembranosus
   c. middle gluteal
   d. gracilis
   e. semitendinosus
   f. superficial gluteal
   g. deep gluteal
   h. quadratus femoris

4. Flexion without the quadriceps femoris
   a. adductor magnus et brevis with adductor longus
   b. sartorius
   c. iliopsoas

5. Flexion with the quadriceps femoris
   a. adductor magnus et brevis with adductor longus
   b. sartorius
   c. iliopsoas
   d. quadriceps femoris

The dissection was carried out on both hind limbs and each muscle was individually weighed to the nearest tenth of a gram. In order to calculate the total pelvic muscle mass the muscles of both limbs were added together and divided by total body weight.

After the dissection was completed the ossa coxae and the femurs were disarticulated and boiled to remove any adherring tissue. The denuded bones were then thoroughly examined to determine the condition of the hips, especially the mongrel groups since there had been no radiographs taken to determine normalcy of these two groups of dogs.

Table 1. Animals used in the study.

Group	Animal	Sex	Age	Weight (kg.)
Normal 50 pound	1	male	adult	25.0
Normal 50 pound	2	female	adult	23.0
Normal 50 pound	3	male	adult	22.7
Normal 50 pound	4	male	adult	22.0
Normal 50 pound	5	female	adult	20.4
Normal 20 pound	1	male	adult	9.1
Normal 20 pound	2	female	adult	8.6
Normal 20 pound	3	female	6 months +	8.2
Normal 20 pound	4	male	6 months +	8.1
Normal 20 pound	5	male	6 months +	7.7
Dysplastic	1	male	9 months	25.0
Dysplastic	2	male	7 months	21.6
Dysplastic	3	female	7 months	19.3
Dysplastic	4	male	7 months	19.1
Dysplastic	5	female	7 months	17.1

RESULTS AND DISCUSSION

Statistical Analysis

An analysis of variance was computed to determine whether there was a significant difference in the relative weights of the pelvic muscles of three groups of dogs - normal 50 pound dogs; normal 20 pound dogs; and dysplastic dogs. Analysis was made on the total weight of the pelvic muscles; the total weight of each action group (flexors, extensors, abductors and adductors) and the weight of each individual muscle comprising the pelvic mass. The raw data used in the statistical analysis is shown in Appendix 1. The F-test (Guenther, 1964) was used to determine the existence of any significant difference among the three groups of dogs.

Since the F-test only indicates the existence of a significant difference, further calculations had to be made. The Tukey method for multiple comparison (Guenther, 1964) was used to find where the significant difference among the three groups — normal 50 pound, normal 20 pound and dysplastic — might be. For that data shwoing no significant F-statistic at the 0.05 level of rejection of the hypothesis of equal means, the power of the F-test was computed. The results of these tests are incorporated in Tables 3 and 4.

Pelvic Muscle Mass

The differences among the pelvic muscle mass of the normal 50 pound, normal 20 pound and dysplastic groups is presented in Graph 1. The Tukey analysis shows there is a significant difference

Table 2. **Results of statistical analysis and calculation for relative underdevelopment in dysplastic dogs for pelvic muscle mass and action groups.**

Action	Group	Frequency	Mean g.	F-Statistic	Sig. prob. of F	Power of F	T-test	Rel. underdevel.
Pelvic mass	N_{50}	5	10.16	12.96	.001		$N_{50} > N_{20}$	30.3
	N_{20}	5	7.70				$N_{50} > D$	
	D	5	7.08				$N_{20} = D$	
Extension	N_{50}	5	2.75	17.36	$< .0005$		$N_{50} > N_{20}$	37.1
	N_{20}	5	2.03				$N_{50} > D$	
	D	5	1.73				$N_{20} = D$	
Flexion with quadriceps	N_{50}	5	2.16	8.39	.005		$N_{50} > N_{20}$	23.4
	N_{20}	5	1.67				$N_{50} > D$	
	D	5	1.65				$N_{20} = D$	
Adduction	N_{50}	5	1.78	14.29	.001		$N_{50} > N_{20}$	30.3
	N_{20}	5	.96				$N_{50} > D$	
	D	5	.89				$N_{20} = D$	
Flexion without quadriceps	N_{50}	5	1.16	9.97	.003		$N_{50} > N_{20}$	30.0
	N_{20}	5	.97				$N_{50} > D$	
	D	5	.83				$N_{20} = D$	
Abduction	N_{50}	5	1.13	13.51	.001		$N_{50} > N_{20}$	33.3
	N_{20}	5	.81				$N_{50} > D$	
	D	5	.76				$N_{20} = D$	

Normal 50 pound dogs= N_{50}, Normal 20 pound dogs= N_{20}, Dysplastic dogs= D

Table 3. **Results of statistical analysis and calculation for relative underdevelopment in dysplastic dogs for each pelvic muscle.**

Muscle	Group	Frequency	Mean g.	F-Statistic	Sig. prob. of F	Power of F	T-test	Rel. underdevel.
Quadriceps	N_{50}	5	.99	8.47	.005		$N_{50} > N_{20}$ $N_{50} > D$ $N_{20} = D$	16.7
	N_{20}	5	.70					
	D	5	.83					
Biceps	N_{50}	5	.93	15.24	.001		$N_{50} > N_{20}$ $N_{50} > D$ $N_{20} = D$	41.4
	N_{20}	5	.62					
	D	5	.54					
Adductors	N_{50}	5	.70	12.22	.001		$N_{50} > N_{20}$ $N_{50} > D$ $N_{20} = D$	31.0
	N_{20}	5	.55					
	D	5	.49					
Semi-membranosus	N_{50}	5	.57	13.23	.001		$N_{50} = N_{20}$ $N_{50} > D$ $N_{20} = D$	38.1
	N_{20}	5	.44					
	D	5	.35					
Middle Gluteal	N_{50}	5	.45	27.16	$< .0005$		$N_{50} = N_{20}$ $N_{50} > D$ $N_{20} = D$	45.5
	N_{20}	5	.36					
	D	5	.24					
Gracilis	N_{50}	5	.30	19.27	$< .0005$		$N_{50} > N_{20}$ $N_{50} > D$ $N_{20} = D$	37.7
	N_{20}	5	.19					
	D	5	.19					
Semi-tendinosus	N_{50}	5	.32	4.17	.042		$N_{50} = N_{20}$ $N_{50} = D$ $N_{20} = D$	21.6
	N_{20}	5	.27					
	D	5	.25					
Sartorius	N_{50}	5	.23	7.28	.009		$N_{50} > N_{20}$ $N_{50} > D$ $N_{20} = D$	20.2
	N_{20}	5	.18					
	D	5	.18					

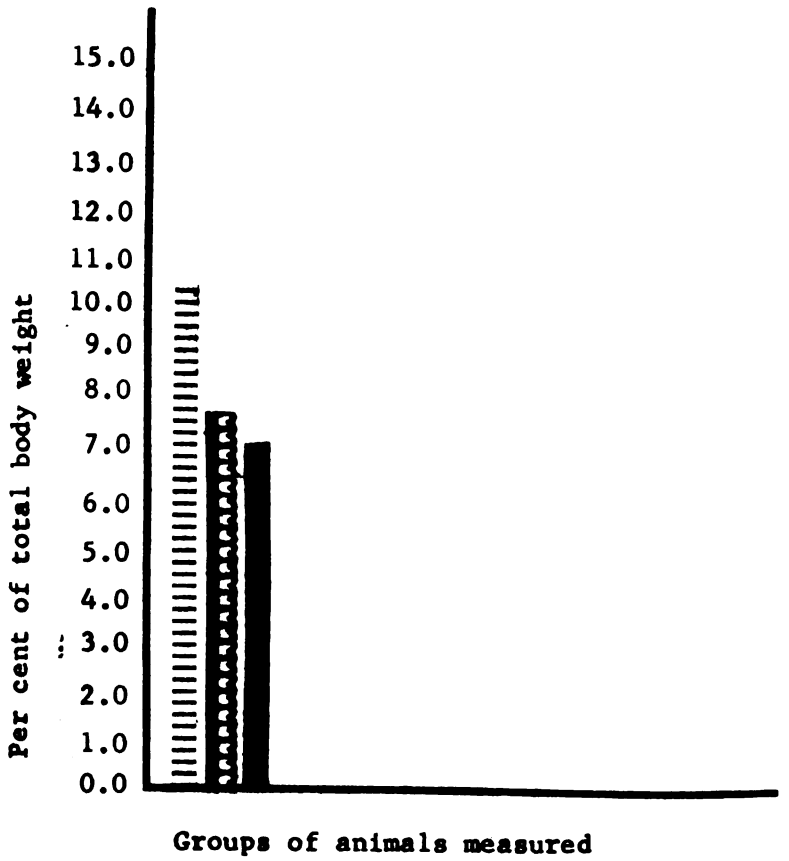
Normal 50 pound dogs= N_{50}, Normal 20 pound dogs= N_{20}, Dysplastic dogs= D

Table 3. (contd.) **Results of statistical analysis and calculation for relative underdevelopment in dysplastic dogs for each pelvic muscle.**

Muscle	Group	Frequency	Mean g.	F-Statistic	Sig. prob. of F	Power of F	T-test	Rel. underdevel.
Iliopsoas	N_{50}	5	.23	3.65	.058	.53		
	N_{20}	5	.24					
	D	5	.16					31.7
Superficial Gluteal	N_{50}	5	.10	13.07	.001		$N_{50} > N_{20}$	
	N_{20}	5	.07				$N_{50} > D$	
	D	5	.08				$N_{20} = D$	40.8
Internal Obturator	N_{50}	5	.06	8.00	.006		$N_{50} > N_{20}$	
	N_{20}	5	.05				$N_{50} > D$	
	D	5	.05				$N_{20} = D$	23.7
External Obturator	N_{50}	5	.05	3.66	.057	.50		
	N_{20}	5	.05					12.3*
	D	5	.06					
Deep Gluteal	N_{50}	5	.05	1.07	.373	<.30		
	N_{20}	5	.05					8.5*
	D	5	.06					
Pectineus	N_{50}	5	.05	1.59	.245	.30		
	N_{20}	5	.04					21.5
	D	5	.04					
Quadratus	N_{50}	5	.03	1.55	.250	.30		
	N_{20}	5	.03					12.3
	D	5	.03					
Gemelli	N_{50}	5	.01	6.23	.011		$D > N_{50}$	
	N_{20}	5	.01				$D = N_{20}$	
	D	5	.01				N_{50} N_{20}	37.8*

* overdevelopment rather than underdevelopment
Normal 50 pound dogs= N_{50}, Normal 20 pound dogs= N_{20}, Dysplastic dogs= D

Graph 1. Total pelvic muscle mass relative to body weight



Normal 50 pound dogs

Normal 20 pound dogs

Dysplastic dogs

between the pelvic muscle mass of the normal 50 pound and the dysplastic group (see Table 4). This corroborates the work of Riser and Shirer (1967) and Riser (1963) that there is a lack of muscle mass of the pelvic area in dogs with hip dysplasia.

Although a lowered pelvic muscle mass in the dysplastic animals was shown, further attempts at comparison of values found in this study with those of Riser and Shirer (1967) proved futile. The mean values of the pelvic muscle mass of the dissected dogs in this experiment are lower than those reported by the above workers. The pelvic muscle mass relative to the body weight of each animal used is shown in Appendix 3. Table 4 gives the group means.

Since there was interest in Riser's theory of similitude (1963), the differences between the normal 20 pound and dysplastic groups and those between the normal 20 pound and normal 50 pound groups were observed. No significant difference was reported between the means of the normal 20 pound and dysplastic groups. Although there was a significant difference between the normal 20 pound and the normal 50 pound groups, the value for the pelvic muscle mass of the normal 20 pound group was much higher than that reported by Riser and does not agree with his theory that a dog of 50 pounds and 24 inches high at the shoulder will have a pelvic muscle mass three times that of a dog 20 pounds and 12 inches at the shoulder.

Results of this study show a definite decrease in the total muscle mass of the pelvic area of dogs with hip dysplasia. There does not seem to be a direct causal relationship between lack of muscle mass of the area and a biomechanical imbalance.

Action Groups of Muscles

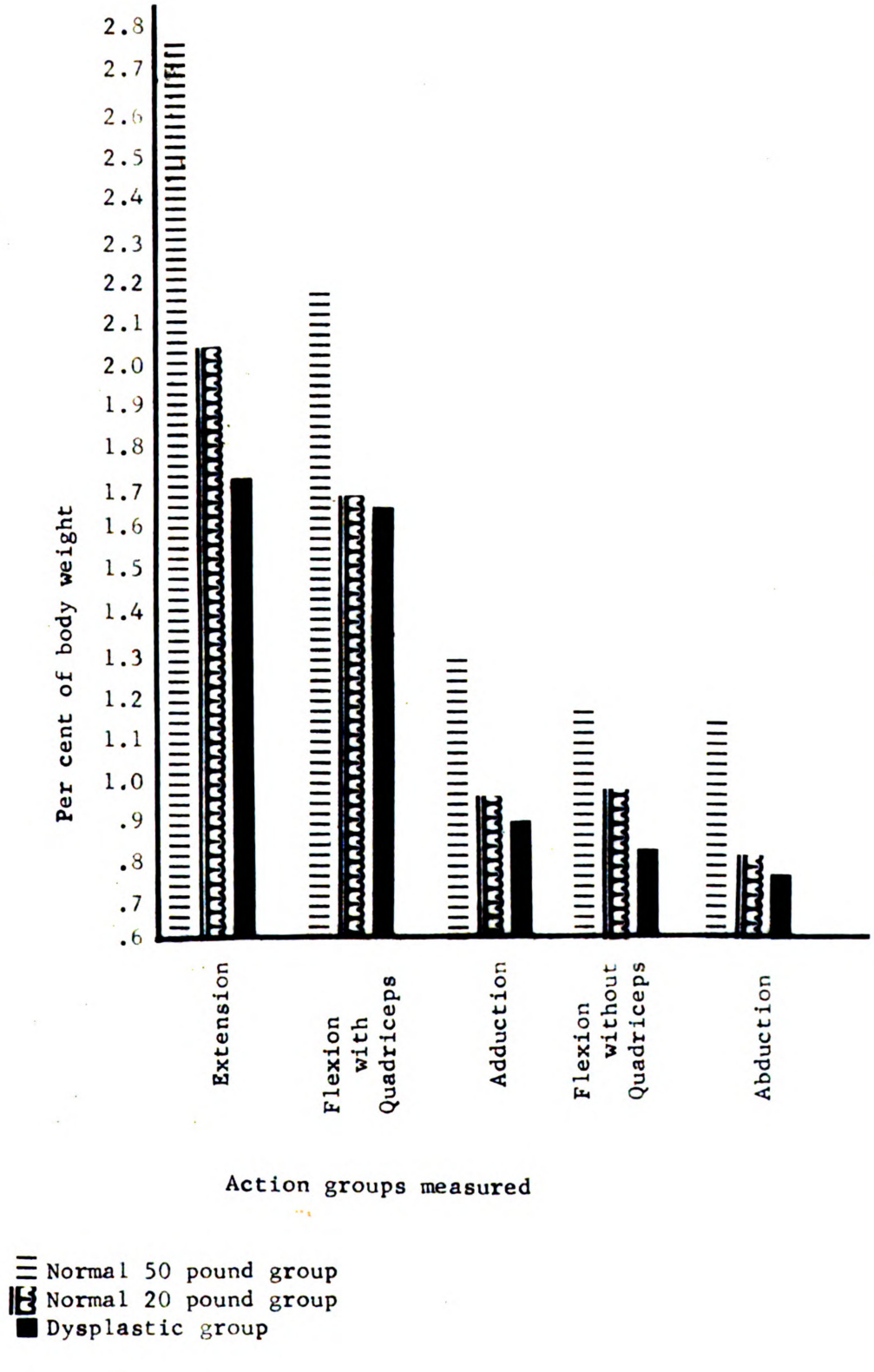
The differences in the weights of the action groups of muscles of the three groups of dissected dogs is shown in Graph 2. The data shown by this graph indicated that there is a lack of development as the muscles are grouped according to their action on the hip joint.

An attempt was made to determine which one of these action groups was most affected by the underdevelopment. This calculation was done by taking the difference between the means of the normal 50 pound group and the dysplastic group and dividing the difference by the mean of the normal 50 pound group i.e. $\frac{N_{50} - \text{Dysplastic}}{N_{50}}$. It was not possible to satisfy the requirements for a statistical test for the significance of the above calculation, therefore only trends are reported.

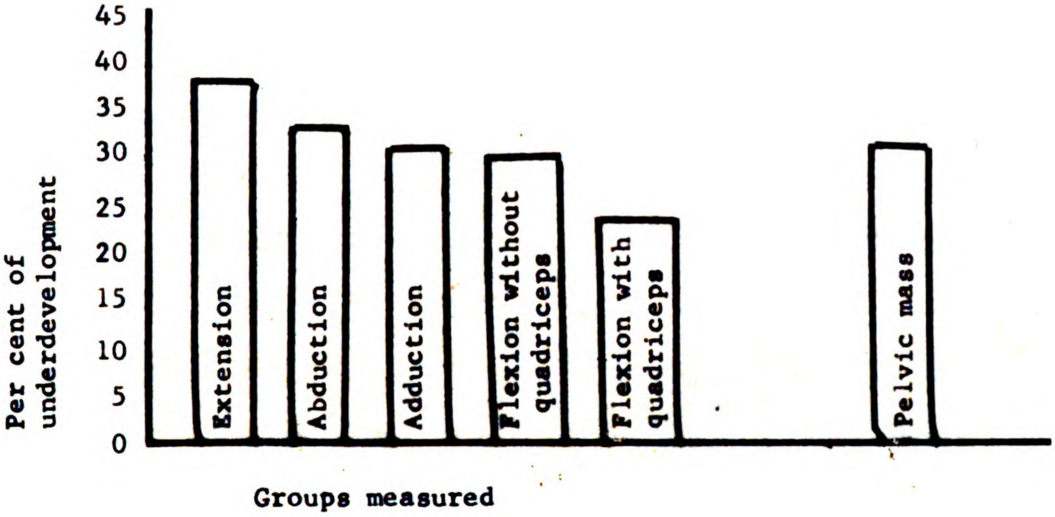
The extensor and abductor groups seem to be most affected (see Graph 5). This work strengthens the suggestions by Riser and Shirer (1964) and Mau (1961) that the abductors may be unduly affected in the dysplastic animals. This study also indicated that the extensors, which in the normal animal contributes most to the pelvic mass, are the most underdeveloped in the dysplastic animal and may be an area for further concern.

Since there was some question about the action of the quadriceps femoris, other than its rectus femoris head, two groups of flexors were constructed - one containing the quadriceps and one without. Graph 5 demonstrates what contribution the quadriceps makes to the flexor group. Graph 4 indicates a lack of development in this muscle. From both of these graphs it can be inferred that this

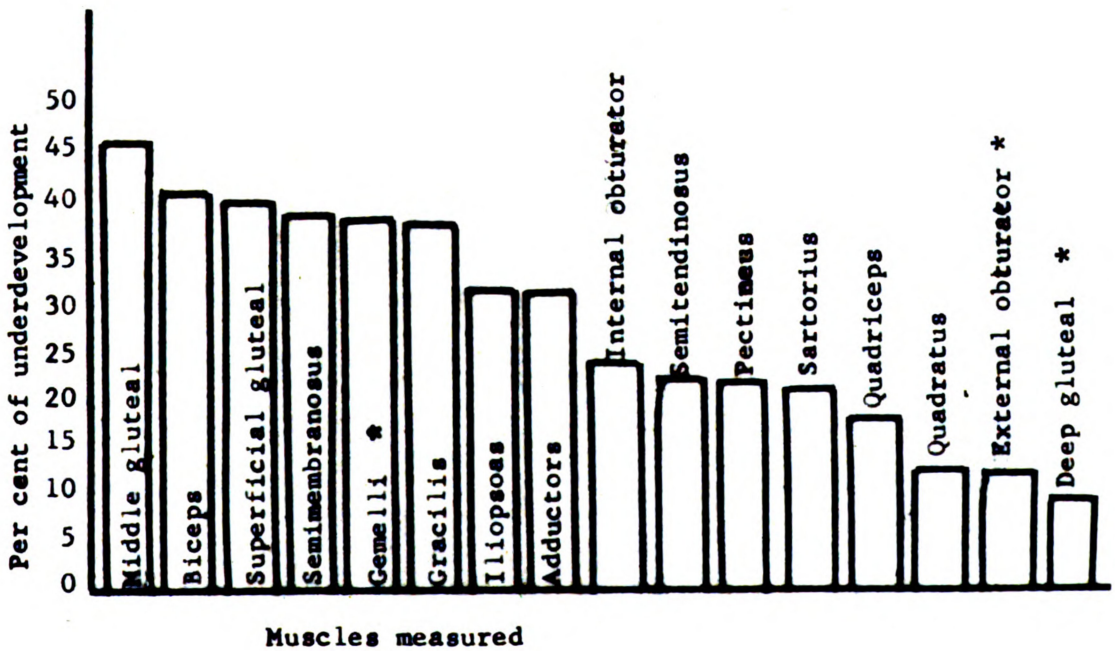
Graph 2. Action groups of muscles relative to body weight.



Graph 4. Amount of underdevelopment in the total pelvic mass and action groups of dysplastic dogs relative to the normal 50 pound dogs.



Graph 5. Amount of underdevelopment in the individual pelvic muscles of dysplastic dogs relative to normal 50 pound dogs.



*-overdevelopment rather than underdevelopment

muscle as a whole is not greatly affected and does not have a great affect on the act of flexion at the hip.

Individual Muscles of the Pelvic Mass

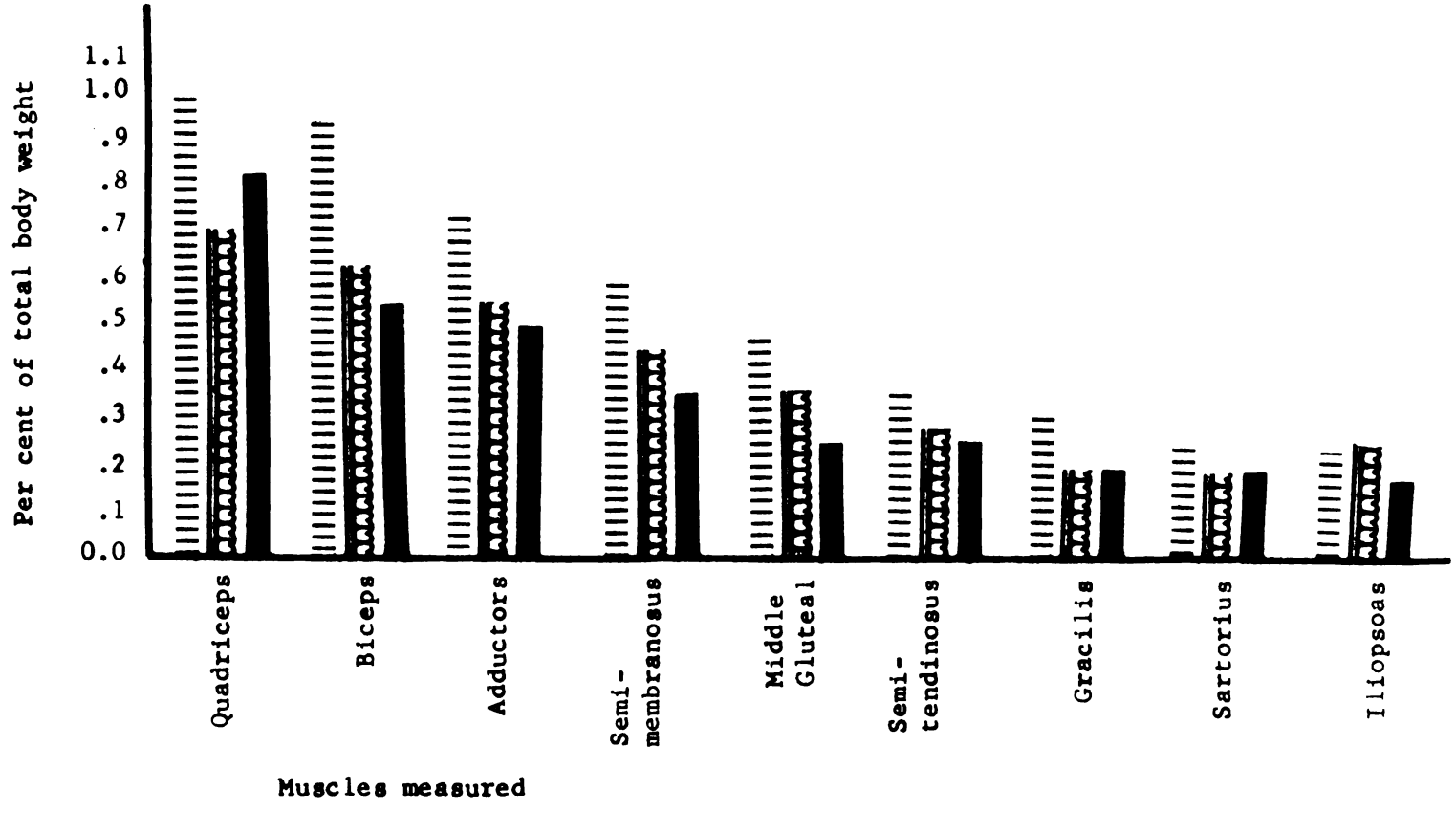
A general trend of atrophy throughout all of the muscles of the pelvic area of dogs with hip dysplasia is shown in Graph 3. With the exception of five muscles, iliopsoas, external obturator, deep gluteal, pectineus and quadratus femoris, the statistical data in Table 3 demonstrates a significant difference between the normal 50 pound and the dysplastic groups.

The calculation using the formula $\frac{\text{Mean } N_{50} - \text{Mean Dys.}}{\text{Mean } N_{50}}$, used to determine the lack of development of the action groups, was also used to find the lack of development occurring in each individual muscle. Since this data cannot be analysized statistically, the following is a report of the trend shown.

The progression demonstrated in Graph 4 indicates an extreme lack of development in the middle gluteal, biceps femoris, superficial gluteal, semimembranosus and gracilis muscles. These muscles are involved in the actions of extension or abduction - the muscle groups which were shown to be affected most by the dysplastic condition. Those three muscles showing the least decrease due to the dysplasia - deep gluteal, external obturator, and quadratus femoris - have also shown no significant difference from the normal.

In further examining the data of those muscles which showed no significant difference, the iliopsoas and the external obturator muscles were extremely close to meeting the 0.05 level of rejection. It is of interest to note that even though a significant difference was not shown in the external obturator and the deep gluteal muscles,

Graph 3. Individual pelvic muscles relative to total body weight



Graph 3. (contd.) Individual pelvic muscles relative to total body weight

there was an increase in mass rather than a decrease. Likewise, there was a significant increase in the gemelli as demonstrated in Table 3. Thus it is possible that these smaller muscles are actually increasing in mass as a compensatory reaction to the general underdevelopment occurring in the dogs with dysplasia. These three muscles also are included in the abductor action group, although the general condition of this group is one of a more extreme lack of development.

An observation made during the dissection of the dysplastic dogs was the total lack of the samll capsularis coxae muscle. Even when present in the normal dogs the muscle is only measurable at the level of .1 grams at most, but its absence may be an indication that one aspect of the dysplastic condition is in the muscle tissue itself and not only the result of the general disorder.

Although Spurrell (1967) admits there is an underdeveloped condition of the muscles of the pelvic area which this study confirms, he suggests it is a result of the dysplasia which he considers may have some sort of neural cause. Fitzgerald (1961) also suggests a neural involvement resulting in the atrophy of the muscles. With the possibility of some sort of neural cause, Miller et al., (1965) was checked for the innervation of all the muscles of the pelvic area. Special attention was given to those muscles and action groups which seem to be particularly affected in accordance with the trend indicated in Graph 4. There is no one lumbar level that goes to each of these specified muscles or groups. Since there were five muscles that were not shown to be

significantly different, the innervations of these were also checked and again there is no involvement at one single level. The three muscles of increased development have no common level of innervation.

Collateral Observations

During dissection special attention was taken to determine the condition of the ligamentum teres or round ligament and the condition of the joint capsule. In nine of the dysplastic legs the round ligament was ruptured and the one that was found intact was present in the 17.1 kg female of seven months of age. Riser and Shirer (1966) consider the round ligament to be an aid in the stabilization of the hip joint during the first few months of life. They do agree with Olsson (1961a) that the absence of the round ligament is the result of the dysplasia rather than the cause.

The joint capsules of the dysplastics were much thickened and looser than those of either of the groups of normal dogs. This observation is in agreement with those of Snavely (1959), Howorth (1965) and Massie and Howorth (1951). When the dysplastic capsules were incised the joint fluid found was viscous, yellow and was in considerable excess of that found in the normal groups, which was colorless and the consistency was more watery and thinner. This observation has also been made by Snavely (1959).

Thus Schnelle (1965) has said that hip dysplasia is part of a generalized fault or disease of development involving the joint and its supporting structures. The muscular underdevelopment seen in this study seems to be only one step in the sequence of

maldevelopment of this area.

SUMMARY AND CONCLUSIONS

Dissection of sixteen muscles comprising the mass of the pelvic area was performed on three groups of dogs, with a frequency of five in each group. These groups were normal 50 pound mongrels, normal 20 pound mongrels and dysplastic purebred Labrador Retrievers of 45 pounds. An analysis of variance was calculated to determine whether there was a significant difference between the pelvic muscle mass of the normal and dysplastic dogs.

With a confirmation of a significant difference in pelvic mass between the dysplastic and the normal 50 pound dog further observation and calculation was made to determine whether there was a significant difference between the action groups of muscles and whether there was a significant difference between the individual muscles of the area in the two groups of dogs.

The data showed that there was a definite underdeveloped condition and difference between the action groups of dysplastic dogs compared to those dogs of approximately the same weight with normal hips. There was a trend implying that there may be an exaggerated lack of development of the extensor and abductor groups of muscles.

There was also shown a difference between the individual muscles of the dysplastic compared to the 50 pound normal. There was also a trend of exaggerated atrophy being shown in the biceps femoris, middle gluteal, semimembranosus, superficial gluteal and gracilis muscles. These muscles also help make up the already mentioned underdeveloped extensor and abductor action groups. There was also an observation of the total lack of the capsularis

coxae muscle in the dysplastic dogs. The question of whether the underdevelopment is due to disuse, neural involvement or a hormone disorder is point for further study.

It was also seen that the ligamentum teres was ruptured in nine of the ten dysplastic limbs examined. Upon examination of the joint capsule as a whole it was seen that there was a thickening or hypertrophy of the connective tissue and that the capsules of the dysplastic animals were stretched and a looser enclosure of the joint was seen in these animals as compared to the two normal groups.

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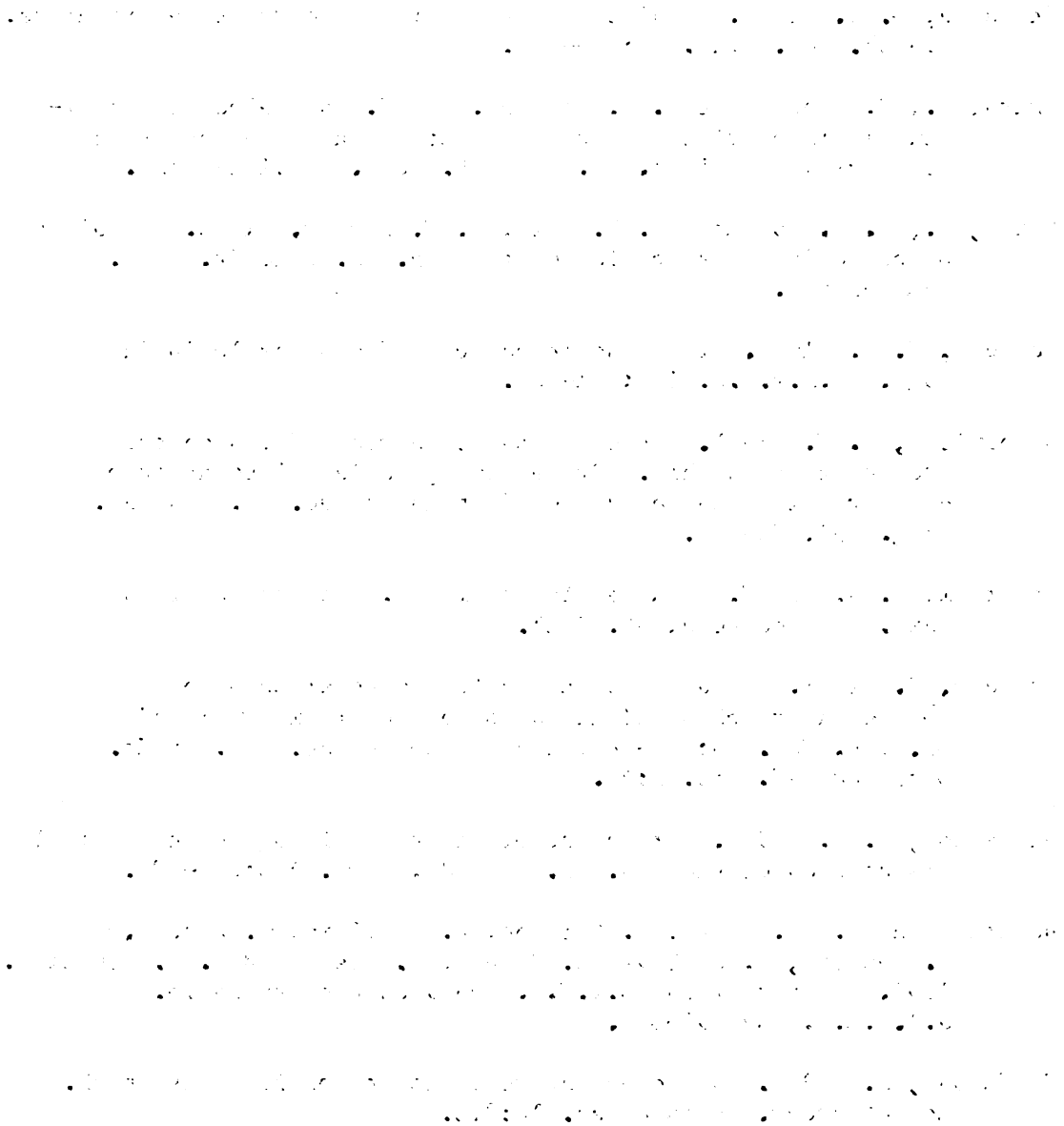

Appendix 1. Raw weights of each pelvic muscle of the 50 pound group.*

	25.0 kg.		23.0 kg.		22.7 kg.		22.0 kg.		20.4 kg.	
	rt.	lf.	rt.	lf.	rt.	lf.	rt.	lf.	rt.	lf.
Quadriceps	275.5	273.7	169.8	165.8	223.8	225.8	227.9	218.3	235.3	231.1
av.	274.6		166.8		224.5		223.1		233.2	
Biceps	237.6	243.1	177.2	181.0	209.0	210.8	199.8	203.8	214.6	219.7
av.	240.4		179.1		209.9		201.8		217.2	
Adductors	183.4	176.4	130.8	134.3	161.8	142.8	157.1	152.8	167.3	174.3
av.	179.9		132.6		154.7		155.0		170.8	
Semi-membranosus	145.3	155.9	109.0	93.2	123.6	126.8	142.3	134.3	126.7	130.6
av.	150.6		101.1		125.2		138.3		128.4	
Middle Gluteal	127.6	117.0	87.1	92.0	93.9	97.6	106.2	106.2	97.5	90.4
av.	119.6		89.6		95.7		106.2		94.0	
Semi-tendinosus	86.1	89.8	58.0	53.1	69.0	71.6	73.5	71.0	83.8	79.5
av.	85.5		59.7		70.3		72.3		81.7	
Gracilis	85.8	85.6	58.1	61.2	56.2	60.6	70.2	63.8	72.2	71.0
av.	85.7		59.7		58.4		67.0		71.6	
Sartorius	58.4	53.8	41.7	39.6	49.0	52.9	59.8	56.5	53.5	53.2
av.	56.1		40.7		51.0		57.7		53.4	
Iliopsoas	80.9	81.7	39.0	40.5	40.5	46.6	48.6	47.7	47.9	49.1
av.	81.3		39.8		43.6		48.2		48.5	
Superficial Gluteal	25.9	24.5	16.2	17.3	21.0	20.1	25.0	23.0	25.4	24.2
av.	25.2		16.8		20.6		24.0		24.8	
Internal Obturator	15.1	15.2	13.2	12.4	13.7	17.0	14.9	12.4	16.0	12.0
av.	15.2		12.8		15.4		13.7		14.0	
External Obturator	13.7	14.8	8.4	10.0	10.7	14.4	11.3	11.0	12.9	10.9
av.	14.3		9.2		12.6		11.2		11.9	
Deep Gluteal	15.1	12.4	10.0	9.0	14.9	14.1	10.7	11.0	10.4	9.3
av.	13.6		9.5		14.5		10.9		9.9	
Pectineus	10.7	9.7	9.2	7.9	11.5	12.2	9.7	8.3	13.0	12.4
av.	10.2		8.1		11.9		9.0		12.7	
Quadratus	7.5	7.5	6.0	5.9	5.6	6.8	6.6	7.2	7.0	5.6
av.	7.5		6.0		6.2		6.9		6.3	
Gemelli	2.1	2.5	1.3	1.0	2.0	2.1	1.2	1.4	2.8	2.7
av.	2.3		1.2		2.1		1.3		2.8	

* recorded to the nearest .1 g.
the group is arranged left to right in a progression of decreasing weight.
av.=average weight of both legs, rt.= right leg, lf.= left leg

Appendix 1. (contd.) Raw weights of each pelvic muscle of the 20 pound group.*

	9.1 kg.		8.6 kg.		8.2 kg.		8.1 kg.		7.7 kg.	
	rt.	lf.	rt.	lf.	rt.	lf.	rt.	lf.	rt.	lf.
Quadriceps	53.1	52.9	70.0	69.6	65.7	65.9	54.9	53.5	48.8	50.0
av.	53.0		68.8		65.8		54.2		49.4	
Biceps	39.8	35.8	67.4	68.6	63.1	62.5	50.3	50.7	39.3	36.7
av.	37.7		68.0		62.8		50.5		38.0	
Adductors	38.2	39.8	49.4	52.2	49.6	50.4	43.5	43.6	43.4	43.0
av.	39.0		50.7		49.9		43.6		43.2	
Semi-membranosus	30.6	28.2	40.7	40.7	43.7	43.0	32.7	32.2	35.7	36.4
av.	29.4		40.7		43.4		32.5		36.1	
Middle Gluteal	25.2	24.3	31.6	32.5	33.9	31.9	25.9	26.4	35.2	34.4
av.	24.8		32.1		32.9		26.2		34.8	
Semi-tendinosus	22.5	21.5	23.7	25.7	23.4	23.3	18.3	18.2	23.0	21.9
av.	22.0		24.8		23.4		18.3		22.5	
Gracilis	13.2	15.5	16.7	16.6	17.8	18.1	13.2	13.2	16.1	15.6
av.	14.9		16.7		17.5		13.2		15.9	
Sartorius	15.7	17.8	16.7	16.1	15.7	14.9	15.0	15.5	13.3	13.4
av.	16.8		16.4		15.3		15.3		13.4	
Iliopsoas	14.4	14.3	17.4	17.1	18.5	17.2	24.8	24.3	23.5	22.9
av.	14.3		17.3		17.9		24.6		23.2	
Superficial Gluteal	6.3	6.0	6.4	6.7	6.2	6.9	5.2	5.7	5.9	5.7
av.	6.2		6.6		6.6		5.5		5.8	
Internal Obturator	5.8	4.7	4.2	4.6	4.0	4.2	3.9	3.9	4.6	4.5
av.	5.3		4.1		4.1		3.9		4.6	
External Obturator	4.1	3.9	3.9	4.4	3.9	4.1	3.3	3.5	4.1	4.3
av.	4.0		4.2		4.0		3.4		4.2	
Deep Gluteal	3.3	3.6	4.4	4.1	4.2	4.0	4.5	4.6	4.0	3.5
av.	3.5		4.3		4.1		4.4		3.8	
Pectineus	2.8	2.9	3.3	3.7	3.7	3.5	2.6	3.1	2.5	2.9
av.	2.9		3.5		3.6		2.9		2.7	
Quadratus	2.2	2.5	2.7	2.8	2.9	2.9	3.1	3.3	3.0	3.0
av.	2.4		2.8		2.9		3.2		3.0	
Gemelli	.8	.8	.8	.8	.8	1.1	.7	.9	1.0	1.0
av.	.8		.8		1.0		.8		1.0	

* recorded to the nearest .1 g.
the group is arranged left to right in a progression of decreasing weight
av.=average weight of both legs, rt.= right leg, lf.= left leg

Appendix 1. (contd.) Raw weights of each muscle of the dysplastic group.*

	25.0 kg.	21.6 kg.	19.3 kg.	19.1 kg.	17.1 kg.
	rt. lf.	rt. lf.	rt. lf.	rt. lf.	rt. lf.
Quadriceps	193.5 207.8	162.7 201.2	161.3 142.3	176.2 171.0	123.0 132.0
av.	197.7	182.0	151.8	173.6	137.5
Biceps	146.2 152.9	110.7 130.7	94.8 78.7	109.6 110.5	80.0 104.5
av.	149.2	120.7	86.8	110.2	92.3
Adductors	126.5 137.6	100.2 103.8	80.1 80.3	104.1 93.3	71.8 97.2
av.	132.1	102.0	80.2	98.7	84.5
Semi-membranosus	95.8 103.1	68.1 86.5	60.6 52.1	69.7 64.5	51.5 72.7
av.	99.5	77.8	56.4	67.1	62.1
Middle Gluteal	65.7 67.8	40.7 56.7	53.7 38.1	52.3 49.3	34.4 40.8
av.	66.8	48.7	45.9	50.8	37.6
Semi-tendinosus	65.5 69.1	47.3 50.2	43.0 39.6	60.0 54.0	43.0 47.3
av.	67.3	48.8	41.3	57.0	45.2
Gracilis	58.5 59.0	36.8 36.6	32.0 28.9	34.8 35.6	30.5 36.4
av.	58.8	36.7	32.5	35.2	33.5
Sartorius	45.6 49.4	37.9 43.0	32.6 30.1	36.5 37.7	29.5 32.8
av.	47.5	40.5	31.4	37.1	31.7
Iliopsoas	40.0 41.8	30.2 30.4	28.0 24.5	35.0 35.3	24.0 29.5
av.	40.9	30.3	26.8	35.2	26.8
Superficial Gluteal	11.5 18.9	8.9 14.0	15.5 13.2	11.6 11.0	7.0 8.5
av.	15.2	11.5	14.4	11.3	7.9
Internal Obturator	13.2 12.3	9.7 11.2	9.3 8.0	10.5 11.3	6.8 6.3
av.	12.8	10.5	8.7	10.9	6.6
External Obturator	13.8 12.9	12.8 12.0	12.0 11.0	13.9 14.5	9.3 9.0
av.	13.4	12.4	11.5	14.2	9.2
Deep Gluteal	12.5 14.9	11.3 14.9	11.1 10.9	8.7 15.4	10.0 9.5
av.	13.1	13.1	11.0	12.2	9.8
Pectineus	14.9 13.5	6.0 6.0	5.6 5.7	6.1 5.8	5.5 6.8
av.	14.2	6.0	5.7	6.0	6.2
Quadratus	8.0 8.2	7.9 5.3	4.8 4.8	7.2 6.5	9.5 5.0
av.	8.1	6.6	4.8	6.9	7.3
Gemelli	3.2 3.4	3.6 2.9	2.7 3.0	3.0 2.0	2.5 2.1
av.	3.3	3.2	2.9	2.5	2.3

* recorded to the nearest .1g.
the group is arranged left to right in a progression of decreasing weight
av.= average weight of both legs, rt.= right leg, lf.=left leg

Appendix 2. Total pelvic muscle mass and action groups for each animal relative to its body weight*

	Normal 50 pound dogs **					Normal 20 pound dogs **					Dysplastic dogs **				
	25.0	23.0	22.7	22.0	20.4	9.1	8.6	8.2	7.7	8.1	25.0	21.6	19.3	19.1	17.1
Pelvic mass	10.9	8.1	9.8	10.4	11.6	6.1	8.4	8.7	7.4	7.8	7.0	7.6	6.3	7.2	7.6
Extension	2.9	2.3	2.7	2.9	3.1	1.6	2.3	2.4	1.9	2.1	1.7	1.8	1.5	1.7	1.9
Flexion with quadriceps	2.2	1.7	2.1	2.9	3.1	1.6	2.3	2.4	1.9	2.1	1.7	1.8	1.5	1.7	1.9
Adduction	1.3	1.0	1.2	1.3	1.5	.8	1.0	1.1	1.0	.9	.9	.9	.8	.9	1.0
Flexion without quadriceps	1.2	.9	1.1	1.3	1.3	.8	1.0	1.0	1.0	1.0	.8	.9	.7	.8	.9
Abduction	1.1	.9	1.1	1.2	1.3	.6	1.0	1.0	.7	.8	.7	.8	.7	.8	.8

* per cent of body weight

** each group is arranged left to right in a progression of decreasing weight (kg.)

Appendix 3. Individual muscles for each animal relative to its body weight*

	Normal 50 pound dogs				**	Normal 20 pound dogs				**	Dysplastic dogs				**
	25.0	23.0	22.7	22.0	20.4	9.1	8.6	8.2	7.7	3.1	25.0	21.6	19.3	18.	17.1
Quadriceps	1.01	.73	.99	1.10	1.14	.58	.80	.80	.64	.67	.80	.91	.79	.84	.79
Biceps	.92	.78	.92	.96	1.06	.41	.79	.77	.49	.62	.54	.57	.45	.56	.60
Adductors	.70	.57	.68	.72	.84	.43	.58	.61	.56	.54	.49	.52	.42	.47	.53
Semi-membranosus	.63	.44	.55	.60	.63	.32	.47	.53	.42	.45	.36	.35	.29	.36	.40
Middle Gluteal	.48	.39	.42	.48	.46	.27	.37	.40	.34	.43	.22	.27	.24	.23	.27
Gracilis	.30	.26	.26	.34	.35	.16	.19	.21	.21	.16	.20	.18	.16	.17	.24
Semi-tendinosus	.33	.24	.31	.34	.40	.24	.29	.29	.24	.28	.26	.30	.21	.23	.27
Sartorius	.26	.18	.22	.22	.26	.18	.19	.19	.17	.19	.19	.19	.16	.19	.19
Iliopsoas	.22	.17	.19	.33	.24	.16	.20	.22	.30	.30	.16	.18	.14	.14	.16
Superficial Gluteal	.11	.07	.09	.10	.12	.07	.08	.08	.07	.07	.05	.06	.07	.05	.06
Internal Obturator	.062	.055	.067	.061	.069	.058	.048	.050	.060	.048	.039	.057	.045	.049	.051
External Obturator	.050	.040	.055	.057	.058	.044	.049	.049	.055	.042	.054	.074	.059	.057	.054
Deep Gluteal	.049	.041	.064	.054	.048	.038	.050	.050	.059	.046	.057	.063	.056	.060	.043
Pectineus	.040	.035	.052	.041	.062	.031	.040	.043	.035	.035	.036	.031	.039	.037	.050
Quadratus	.031	.026	.027	.030	.030	.026	.032	.035	.039	.039	.042	.030	.024	.030	.032
Gemelli	.006	.006	.009	.009	.013	.009	.009	.012	.012	.009	.013	.013	.015	.014	.013

* per cent of body weight

** each group is arranged left to right in a progression of decreasing weight (kg.)