# Two Orthopedic Conditions And Treatments In Dogs

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As more and more people have moved into urban and suburban areas, there has been an increase in the amount of bone and joint surgery on small animals. Many of these cases are fractures but other conditions such as osteochondritis dissecans and Legg-Perthes' disease are increasing in incidence.[5] The following discussion deals with these two conditions as cases submitted to the Iowa State University Veterinary Clinic, small animal orthopedic section.

### *Legg-Perthes' Disease*

Avascular necrosis of the femoral head (Legg-Perthes' disease) is a condition primarily seen in dogs between the ages of four to eleven months. The most common breeds affected are Wire-Haired and Manchester Terriers, Miniature and Toy Poodles, Miniature Schnauzers, Pugs, and Pekingese.

### *Pathogenesis and Anatomy*

The blood supply to the femoral head varies with the age of the dog. In a very young dog, the primary blood supply is from the epiphyseal arteries which also supply the joint capsule. The epiphyseal cartilage, which closes at seven to eight months of age in these small breeds, acts as a barrier to blood coming from the metaphysis of the femur and only a small amount of blood comes from the artery in the round ligament. As the dog matures, the epiphyseal cartilage disappears and no longer blocks the flow of blood from the shaft of the femur.[5] The articular cartilage is nourished by synovial fluid and is therefore independent of blood supply to the bone. It follows that any interference to this delicate blood supply to the femoral head in young dogs could lead to ischemia and necrosis of the bone between the articular cartilage and the epiphyseal plate. (Figures 1 & 2)

### *Etiology*

Many causes have been suggested for this decrease in circulation to the femoral head. For a long time trauma to the joint capsule was incriminated since the condition was seen more often in very active puppies.[5] Inflammation due to infections or rise in synovial fluid pressure could cause a decrease in blood supply to the femoral head. Hormonal influences have recently been incriminated. It has been found that these miniature breeds usually mature earlier than larger breeds of dogs.

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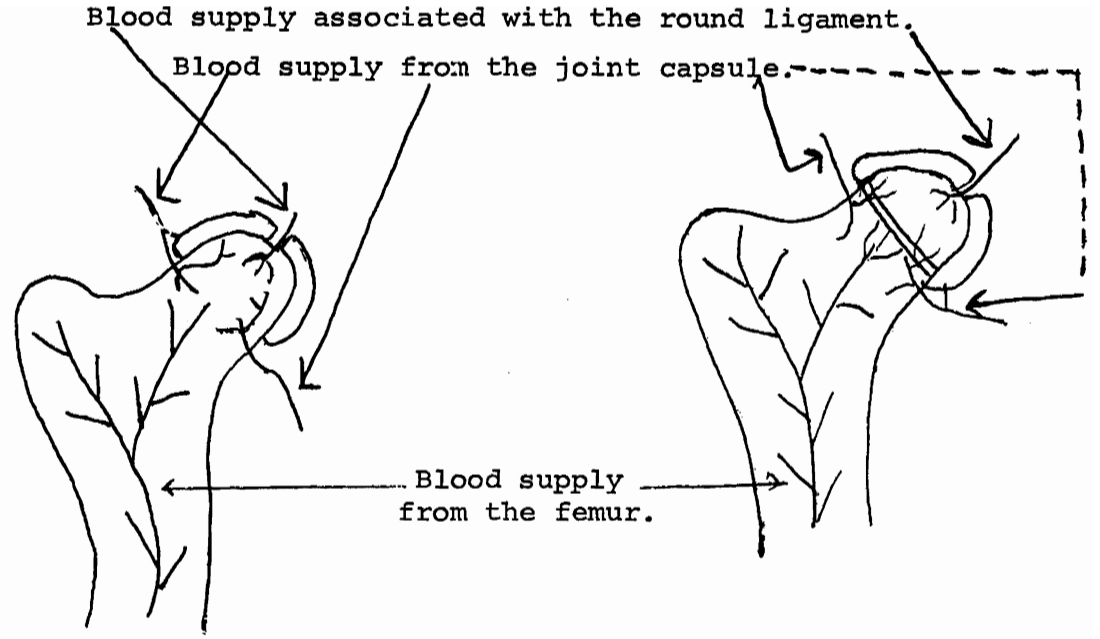


FIG. 1

FIG. 2

Along with the increase in sex hormones there is a decrease in growth hormones which in turn causes premature closure of the epiphyseal plate and excessive endosteal bone formation. This interferes with the blood supply and could be the causative factor. Many other conditions such as toxemias, allergies, or genetic factors could be involved but as of now, no definite etiology has been proven.

### *Case History*

On August 19, 1972, Spot, a male terrier, was admitted to the ISU Veterinary Clinic. The primary complaint was a lameness in the right hind leg. The animal would put no weight on the leg and upon deep palpation over the greater trochanter, pain was elicited. The dog was one year old when first admitted but the lameness was first noticed six months earlier and had been getting progressively worse. A radiograph of the right coxofemoral joint was taken and a diagnosis of Legg-Perthes' disease made. (Figure 3)

Surgery was performed eleven days later on August 30, 1972. The Brown approach was used. This consisted of surgically preparing an area large enough to make an incision 1½ inches proximal and 1½ inches distal over the greater trochanter of the femur. When the skin was incised, the attachment of the tensor fascia lata to the biceps femoris was cut and the biceps reflected posteriorly. At this point, one should be able to see the superficial gluteal muscle and the sciatic nerve, which runs along the posterior border of the femur. Being careful not to cut this nerve, the superficial, middle, and deep gluteals were isolated near their attachment to the greater trochanter of the femur and their tendinous attachments cut. After reflecting these muscles the joint capsule was exposed and subsequently cut around its attachment to the

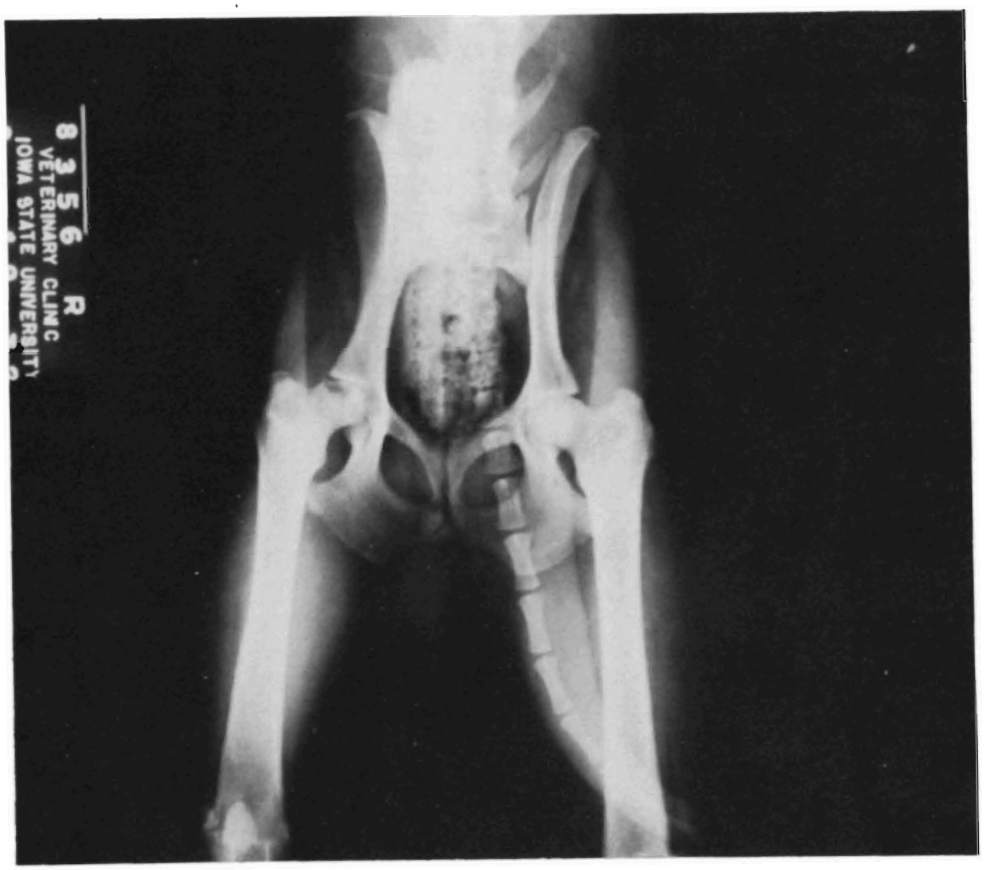


**FIG. 3. Legg-Perthes' Disease. Note necrosis in right femoral head.**

femoral head. By cutting the round ligament with a curved scissors, the femoral head can be luxated dorsally. The head was removed with a wire saw starting at the base of the neck of the femur. The edges of the bone were filed where needed to prevent trauma to tissues. The joint capsule was closed with 2–0 chromic catgut. Each gluteal muscle was sutured separately using 32 gauge stainless steel in a horizontal mattress pattern. The tensor fascia lata and biceps femoris were reunited using simple interrupted 00 catgut sutures. The skin was brought into apposition by subcuticular sutures of 00 catgut and stainless steel sutures were used to close the skin incision.

### *Discussion*

In a typical clinical case of Legg-Perthes', the condition is characterized by sudden occurrence, lameness with pain upon deep palpation over the greater trochanter, and the breed and age of dog. The condition is best diagnosed by radiograph and best treated by femoral head ostectomy. Medically, one can relieve the pain with aspirin but this is usually unsuccessful. In this case, the femoral head was submitted to the pathology department and necrosis of the femoral head confirmed. The dog was using the leg on September 5, 1972, just before it was released.

### *Osteochondritis Dissecans*

This condition was first diagnosed in 1956 but not until 1966 was a surgical approach perfected for this condition.[1] As was previously stated the occurrence of this condition has been increasing in the last ten years. This condition is seen primarily in larger breeds of dogs between the ages of one to twelve months. Of 43 cases of this condition at the small animal clinic of the College of Veterinary Medi-


**FIG. 4. Osteochondritis, dissecans. Note characteristic dishing of the posterior aspect of the humeral head.**

cine in Helsinki, most were in German shepherds, Labradors, German pointers, and Rottweilers. 41 of the 43 cases were between the ages of four to fourteen months.[3] Another study indicates that the occurrence is twice as great in males as it is in females.

### *Pathogenesis and Etiology*

This condition results from the delayed calcification of the articular cartilage on the posterior aspect of the humeral head. If untreated, this will progress until cleavage of the cartilage occurs.[3] Joint fluid now comes in contact with the subchondral tissue and causes pain. The loose piece of cartilage eventually becomes calcified on its underside and thickens. When this free particle comes in contact with surrounding tissue, it will also elicite pain. If it is not removed, the free particle will fragment and the particles now known as "joint mice" will gravitate to the posterior aspect of the joint.[1]

The cause for this delayed ossification of articular cartilage has not yet been agreed upon. Hormone imbalances causing an upset in enchondral ossification at this area has been somewhat ruled out due to studies performed on hormone treated dogs.[3] Infections, interference to blood supply, and allergies all have been incriminated but trauma seems to be the most likely etiology. Repeated trauma by the posterior rim of the glenoid cavity with delayed calcification of the cartilage in this area as a predisposing factor is at this time the most logical cause of the condition.[1]

### *Case History*

Trampus, a six month old Golden Retriever, was admitted to ISU Vet Clinic on May 1, 1973, with a history of shifting

lameness of the forelimbs. When the legs were extended or flexed to a 90° angle at the shoulder joint, pain was elicited. Radiographs (figure 4) showed the characteristic dishing of the posterior aspect of the articular surface of the humeral head. On May 4th, the dog was prepared for surgery and induced with Na Thiamyl (Surital.) Halothane was used to maintain surgical anesthesia. The humerus in this procedure is approached from its posterior aspect. The dog was placed in lateral recumbency and the leg draped in such a manner as to permit manipulation during surgery. The skin was incised from the middle of the scapular spine to the middle of the humerus. The incision is made curving slightly caudally on the lateral side of the leg. Care should be taken to prevent severance of branches of the cephalic vein which should be at the distal end of the incision. Two bellies of the deltoideus muscle and the omotransversarius can be seen attaching to the scapular spine. Next, the belly of the deltoideus that attaches to the humerus is elevated and retracted posteriorly out of the surgical field. The distal two centimeters of the omotransversarius muscle is separated from the acromion process and retracted. The teres minor tendon is then visible and is transected. By retracting the incised parts of the teres minor muscle, the joint capsule is exposed and incised leaving sufficient segments in which to place sutures during closure. Both shoulder joints had flaps of cartilage barely attached to the articular surface. These flaps were removed and the depression curetted to get rid of necrotic tissue and smooth the edges. The joints were flushed with saline and suction applied to remove pieces of loose cartilage. The joint capsules were closed with simple interrupted sutures of chromic 2–0 catgut with care being taken not to go completely thru the capsule. The teres minor muscles were not sutured but the fascia and tissue were replaced with simple interrupted sutures of 00 chromic catgut. The skin was brought together with a subcuticular stitch of 00 catgut and closed with 32 gauge stainless steel wire sutures in a simple interrupted pattern. The dog was placed on 750 mg. chloramphenicol orally bid for five days post-operatively and restricted with cage rest. The owners were informed to restrict exercise for six weeks post-operatively and on May 17, 1973, the sutures were removed. At this time, the dog was using the forelimbs somewhat and the incisions were healed.[4]

### *Discussion*

In treating a case of Osteochondritis dissecans, one must consider both signs and radiographic lesions. Certain criteria are used to decide upon surgical or medical treatment.

1. If the dog is showing no pain and is not lame, the dog can be treated medically. This consists of restricted exercise for approximately three months. At ISU we feel that steroids or phenylbutazone is contraindicated. These drugs tend to make the dog feel better and be more active than it should. Also, they tend to slow the healing process.

2. If the animal shows lameness and/or pain for three months duration or longer, surgery is indicated.

3. If the radiographs show any loose cartilage in the joint cavity, it should be removed.

Since all the cases which were submitted to the clinic at ISU were of the advanced state, surgical correction has been the primary mode of treatment, the results of which have been good.

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