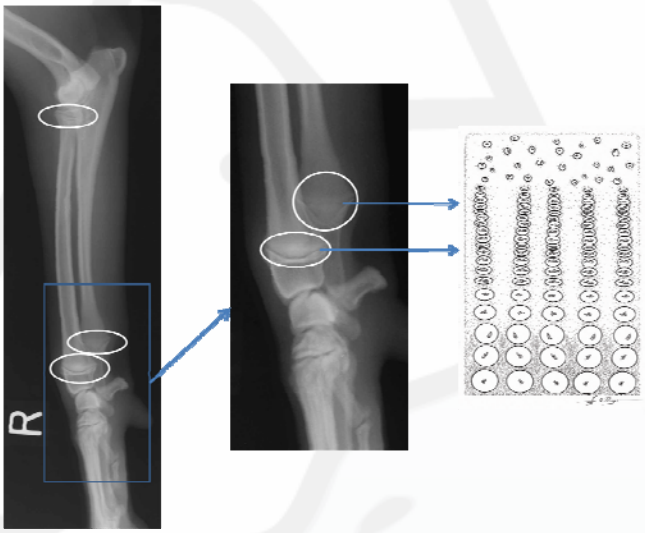
Orthopedic Problems in the Immature Dog

Dirsko J.F. von Pfeil, Dr.med.vet. DVM; Diplomate American and European College of Veterinary Surgeons;
Adjunct Professor, Small Animal Surgery, Michigan State University

To better understand growth related diseases of young animals, it is important to have some basic knowledge of the normal growth process. Bone forms from a cartilage model at the level of the growth plate. Growth plates consist of several specific cells that allow bone to form. The growth plates are located in long bones such as the femur (thigh bone) or tibia (shin bone) a few millimeters away from the adjacent joints, and are seen as a dark line on radiographs (x-rays). In the picture below, showing a normal immature dog's front leg, they are outlined with white circles. The bone on the left is called the radius, the one on the right is called the ulna. The picture on the right depicts the normal cellular arrangement of cells within the growth plate.



The growth plate allows growth of bone to occur until the animal is mature. Most of the growth happens between 3-9 month of age. Once the animal is mature, the growth plate closes. At this point, the growth plate cannot be seen anymore on radiographs (x-rays). In a normal sized dog, most growth plates are closed at approximately 1 year of age. However, in very large or giant breed dogs, they may remain open until 18-20 month of age.

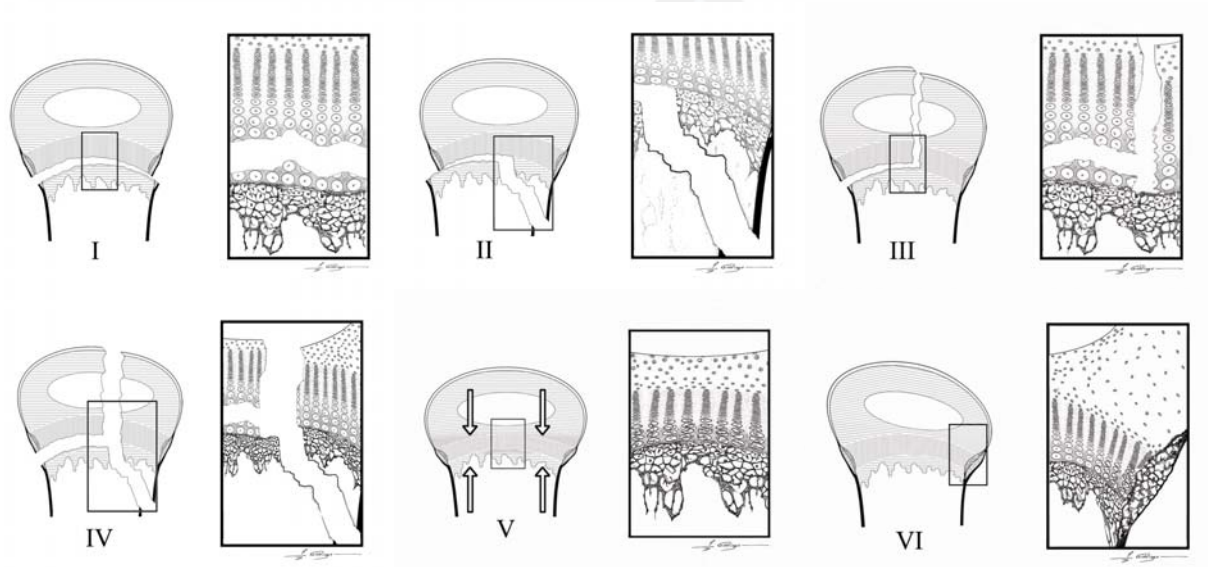
Synchronized growth of all bones is necessary for normal development when a dog or cat transforms from a small puppy or kitten into an adult. Several factors may interfere with normal growth, and a few of these are discussed in the following paragraphs. Early correction of each specific condition is warranted to avert permanent damage. Dogs and cats that are affected with a potentially heritable problem should not be considered for breeding purposes.

More specific discussions for each disease and case will be discussed with you during your appointment with the board certified surgeon at VSOA.

Fractures of the Growth Plate

Fractures of the growth plate are usually the result of trauma and vary in severity. Obviously, the cells that form the growth plate are not able to function normally after they have been damaged. Clinically affected animals show pain, are not weight bearing, or show an initial period of pain, followed by slow development of angulation of the distal part of the leg. The amount of damage can be anticipated depending on what is seen on radiographs (x-rays). The classic description of growth plate fractures was established by two pediatric surgeons, Drs. Salter and Harris, in 1963. Today this classification has been slightly modified and includes

types I-VI. These types are drawn below. On the left is what you would see on a radiograph, on the right the damage to the cells of the growth plate is depicted.

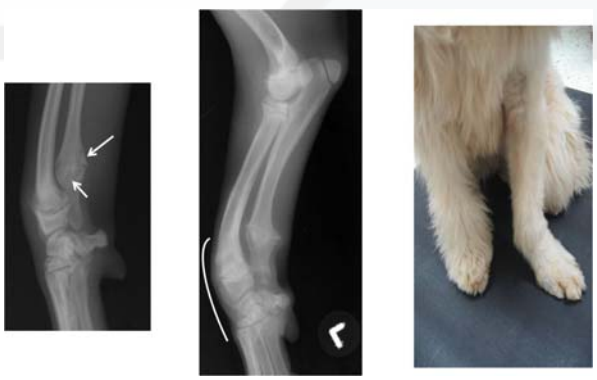


Modified classification system of Salter-Harris. The numbers I-VI indicate the type of fracture.

In general, with increasing type, the prognosis for continued, uncomplicated growth decreases. However, it has been recently shown that severe damage can also occur in dogs with type I fractures. It is therefore of uttermost importance to understand that independent of growth plate fracture type, any animal that has suffered from a growth plate injury may develop an angular limb deformity, may need corrective surgery in the future, or may have other orthopedic problems down the road. The repair for growth plate fractures is usually performed using small pins, which have to be removed in most cases a few weeks after surgical stabilization of the fracture.

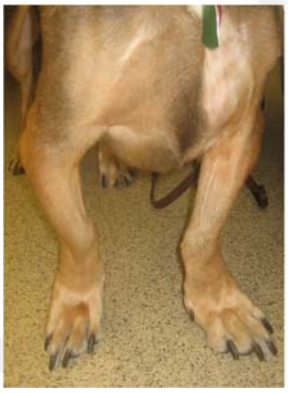
Traumatic Damage to a Growth Plate of the Radius or Ulna

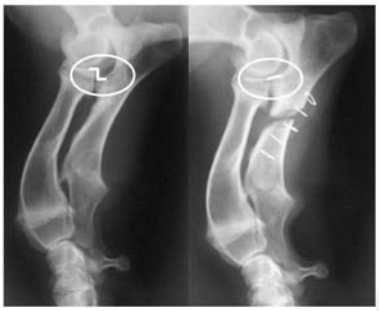
In the lower forearm there are two bones. The radius and the ulna. Both of them have to grow at the same rate as to prevent orthopedic problems. Most frequently clinical signs are observed when the distal (lower) growth plate of the ulna is damaged (see picture below this paragraph on the left side. The damage is outlined by white arrows). This growth plate is responsible for 90-100% of growth in length of the ulna, and must equal the combined growth of the two growth plates of the radius. Damage to this growth plate limits growth of the ulna. Since the radius and ulna are linked together at the ends, the radius becomes deformed as it continues to grow. The radius is not allowed to grow in length, but starts growing in a bow (see white line in the picture below in the middle). The wrist may start bowing laterally (to outside of the leg axis), as depicted in the picture below on the right side.



In severe cases there may be a step defect developing in the elbow joint, between the top parts of the radius and the ulna (see an example in the paragraph of inherited premature closure of the ulnar growth plate)
Affected animals can compensate for the abnormal formed bones remarkably, but after a certain point, they will show gait abnormalities because the joints above or below the deformed bones start to hurt, and develop signs of arthritis. Affected animals usually show discomfort when getting up or when walking. There may be a decreased range of motion of the affected joints, there may be some grinding in the joints, or the leg may be obviously shorter than the other front leg. These signs may increase with increased activity. Depending on what stage of growth the animal is in at the time of diagnosis, different surgical procedures may be performed. It may be necessary to perform two or more surgeries in certain cases.
If a growth plate of the radius is affected, the radius cannot grow at the same speed as the ulna. In these cases, the ulna "pushes" the elbow upwards, and another step defect in the elbow joint develops. This time, the ulna appears to be riding "higher" than the radius.
Affected animal may show similar signs, except that the wrist may start turning to the inside of the leg. Depending on what stage of growth the animal is in at the time of diagnosis, different surgical procedures may be performed. It may be necessary to perform two or more surgeries in certain cases.
The specifics of this surgery vary with each case and will be discussed with you during your appointment with the board certified surgeon at VSOA.

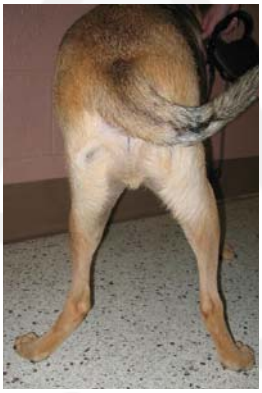
Inherited Premature Closure of the Distal Ulnar Physis
This condition is heritable in Sky Terriers, but is also seen in Welsh corgis, basset hounds, and other chondrodystrophic breeds. Affected animals present with forelimb lameness between 3 and 5 months of age. Radiographic and physical exam findings included 1) laterally deviated wrist, 2) elbow swelling, 3) circumduction of the elbows when walking, and 4) decreased range of motion of the elbow joint. Treatment consists of corrective osteotomy. The specifics of this surgery vary with each case and will be discussed with you during your appointment with the board certified surgeon at VSOA.
A typical case and radiographs are shown below. Note the step defect in the elbow joint (marked in white in the left radiograph), and how it resolved after corrective surgery (see white flat line in the elbow joint on the right radiograph)





Damage to a Growth Plate of the Tibia or Fibula

Similar to the front leg, there are also two bones forming the lower hind leg (shin bones). These are the tibia and the fibula. The tibia is a much stronger and thicker bone than the fibula. Each of these bones have two growth plates. If one of these growth plates is damaged, there will be slowed growth at the affected bone. If the fibula is affected, the tibia will grow faster. This can lead to outside bowing of the hock, as indicated in the picture below.



Depending on the stage of growth the dog is in at the time of injury, surgical correction may be indicated. This condition is overall uncommon. A premature closure of the distal tibial growth plate without any history of trauma can occur as well. It is mainly seen in the Dachshund in which it may be a heritable condition. In this breed the term "Pes varus" is used. Corrective surgery consists of adjusting the angle of the leg, and stabilizing the site of surgery using a bone plate or an external fixator. The specifics of this surgery vary with each case and will be discussed with you during your appointment with the board certified surgeon at VSOA.

Nutrition

Deficiencies in vitamin D, trace elements, or excessive calcium, vitamin C, and high-energy intakes have been discussed as reasons for developmental growth abnormalities. Calcium and energy are two of the most important factors. Commercially prepared large-breed-puppy diets, that meet the nutritional guidelines of the Association of American Feed Control Officials (AAFCO), and are judged to be 'complete and balanced', should be fed until a growing dog reaches approximately 80 percent of its mature size. Recommended levels of key nutrients in a diet for large breed puppies to prevent orthopedic disease are summarized in table 1 (next page).

Recommended Levels of Key Nutrients in a Diet for Large Breed Puppies		
Protein	29-34 % DMB	6-8 % as fed canned
Fat	11-16 % DMB	2-4 % as fed canned
Mean Energy	3.4-4.1 % DMB	13.4-15.9 KJ/g DMB
Calcium	0.8-1.4 % DMB	-
Phosphorous	0.7-1.2 % DMB	-
Fiber	2.4-5.6 % DMB	1 % as fed canned

Table 1: Recommended ranges of key nutrients in a diet for large breed puppies.
KJ/g = Kilojoule per gram; DMB = dry matter basis.

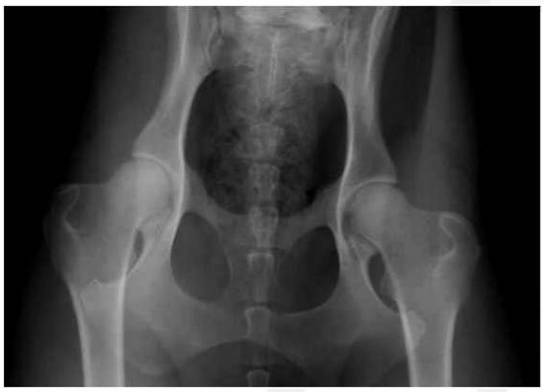
Too much **calcium** may lead to problems during the bone forming process, and possibly lead to the development of osteochondrosis.
Very high levels of **phosphorus**, for example from "Meat only diets" decreases calcium absorption in the gut. The result is poorly calcified soft bones, which are predisposed to pathological fractures. It is therefore recommended to maintain a dietary calcium –phosphorus ratio between 1.1:1 and 2:1
Vitamin D and its metabolites are important in the regulatory mechanism of calcium metabolism and thus skeletal development in the dog and cat. Vitamin D deficiency will lead to inadequate mineralization of bone. The growth plate fails to calcify. The resultant disease, rickets, is a very rare disease that is characterized by deformable bones, lameness, pain, angular limb deformation, and pathologic fractures.
Foods too high in dietary **energy** (fat or proteins) have been shown to lead to weaker growth plates and bone, predisposing the animals to mechanical injuries, such as fractures or osteochondrosis. Excessive dietary protein and fat also increased total body weight of dogs, which could lead to obesity as well as secondary changes in joints and prolonged epiphyseal plate maturation. Feeding commercial dry diets with an energy density below 4.0 kcal/g can help minimize the negative effects of high calorie intake.[10] Restricted total dietary intake has been associated with decreased development of hip dysplasia and arthritis in several joints, as well as with increased life span and delayed onset of chronic disease.
Although rare, **copper** deficiency can impair the metabolism of bone quality.
Zinc is an important trace element for physiological growth. Zinc deficiencies can lead to impaired growth and skeletal development as well as to skin problems and decreased immune function.

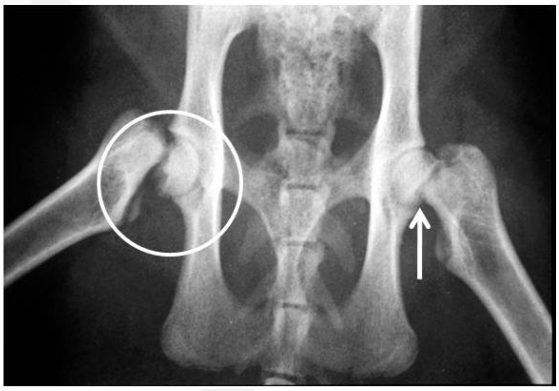
Panosteitis
Panosteitis is an inflammation of the growing bone. Clinical signs are spontaneously occurring lameness that usually occurs in large breed dogs. German shepherds seem to be particularly predisposed to this condition. The cause for panosteitis is unknown, but genetic, viral, or nutritional causes have been discussed. Affected dogs are usually between 5 - 14 month of age. The disease has been reported in dogs as young as 2 months and can also occur in young mature dogs. The lameness tends to occur very suddenly, usually without a history of trauma or excessive exercise. In most cases one or the other front leg is affected first and then it appears that the lameness is shifting from leg to leg.

There are often periods of improvement and worsening of the symptoms in a cyclic manner. This makes evaluation of treatment difficult since many dogs will spontaneously recover with or without treatment and then relapse. If pressure is applied over the long bones, pain can usually be elicited. Radiographs (x-rays) usually reveal a typical "cloud-like" pattern. The X-ray signs do not always match the clinical signs. In most cases, the worst pain lasts between one and two months but may persist in a cyclic nature for up to a year. Analgesics, i.e. a FDA approved non-steroidal anti-inflammatory for dogs can be be helpful. In severe cases, corticosteroids may provide relief. As diet may play a role in the etiology of this disease, it may be preferable to feed the animal an adult dog food, instead of puppy food. This condition is self limiting, meaning that it will eventually go away, with or without treatment. Pain control can go a long way towards helping your pet feel more comfortable and should be used, though.

Slipped capital femoral epiphysiolysis

The skeletal changes associated with prepubertal spay or neuter have been associated with an increased risk in developing slipped capital femoral epiphysiolysis (SCFE) in cats and rarely in dogs. On the picture below you can see on the left side a normal hip of a dog, on the left a hip of a cat, affected from SCFE. The white circle shows a complete loosening of the head of the femur, the white arrow indicates a slipped head.

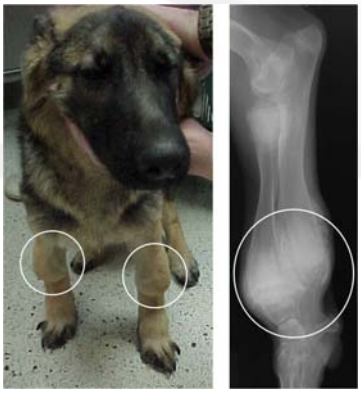




It is therefore a hormonally based disease. Animals are lame in the affected rear leg for a variable amount of time and variable severity. A recent study that evaluated the disease in cats, affected animals were 5-24 months old, 85 percent were male, 23 percent were Siamese, and 90 percent were obese. These cats were neutered between 4-8 months of age. Depending on clinical signs, conservative treatment or surgery may be indicated. The specifics of this surgery vary with each case and will be discussed with you during your appointment with the board certified surgeon at VSOA.

Hypertrophic Osteodystrophy

Hypertrophic osteodystrophy (HOD) is a condition that is seen in growing large- or giant breed dogs with open epiphyseal plates. Affected animals present with intermittent lameness, fever, and extremely painful, swollen wrists or ankles, and radiographs (x-rays) show specific signs, like a double-physeal line. Below a typical case example. The white circles indicate where the problem lies in this dog.

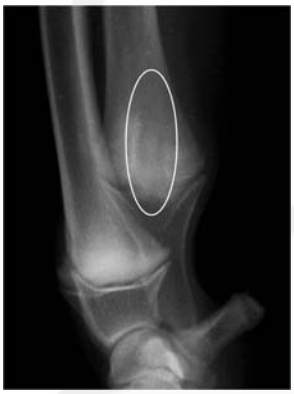


The cause of hypertrophic osteodystrophy is unknown. Nutritional (Vitamin C deficiency) or viral factors (Distemper virus) have been suggested. However, these theories remain unproven. The disease is usually self-limiting, but affected dogs can be extremely ill. Treatment consists of supportive care with fluids, antibiotic,

pain medication, non-steroidal or steroidal anti-inflammatories. Permanent bone changes and angular limb deformities, secondary to either asymmetric or asynchronous growth, or bridging of bone, and retardation of axial growth have been reported.

Osteochondrosis
This disease occurs also in young animals, and is discussed on another client information sheet.

Retained Cartilaginous Core
Retained cartilaginous cores are most commonly seen in the distal ulna of growing, large and giant breed dogs. It is a developmental disorder of abnormal bone formation, in which the processes at the growth plate is disturbed, resulting in decreased bone growth of the ulna. The etiology is uncertain, however, dietary imbalances or a form of osteochondrosis have been suggested. Depending on the severity, affected dogs present with variable degree of lameness, multiple deformities, including valgus deformity of the carpus and cranial bowing of the radius (with or without lameness). A radiolucent cartilage core in the center of the distal ulnar physis can be seen radiographically, as outlined with the white oval marks below.



Treatments include return to a complete and balanced diet, and cessation of excessive dietary supplements in growing dogs. If clinically indicated, corrective osteotomy should be performed. The specifics of this surgery vary with each case and will be discussed with you during your appointment with the board certified surgeon at VSOA. Prognosis varies according to the degree of deformity and degree of lameness at initial presentation.

Ununited Anconeal Process
This disase occurs also in young animals, and is discussed on another client information sheet (Ununited Anconeal Process).

Incomplete Ossification of the Humeral Condyle
This disase occurs also in young animals, and is discussed on another client information sheet (Elbow fractures / Incomplete ossification of the humeral condyle).

Ocular Skeletal Dysplasia

This heritable condition occurs mainly in the Labrador retriever, but has also been described for the Samoyed and German shepherd. Affected dogs present with a typical "downhill-conformation" (shorter front limbs compared to the longer hind limbs; see picture below), and various bony abnormalities.

Courtesy: Dr. Ulli Reif

Hip dysplasia as well as retarded growth of the tibia may also occur. The degree of lameness varies with severity. The eye component of this disease may present as "night blindness". Several eye disease can be the cause. Depending on the degree of orthopedic and/or ophthalmologic disease, surgical treatment may be beneficial. The specifics of this surgery vary with each case and will be discussed with you during your appointment with the board certified surgeon at VSOA. Prognosis for complete restoration of normal orthopedic function is guarded to poor and affected dogs should not be bred.

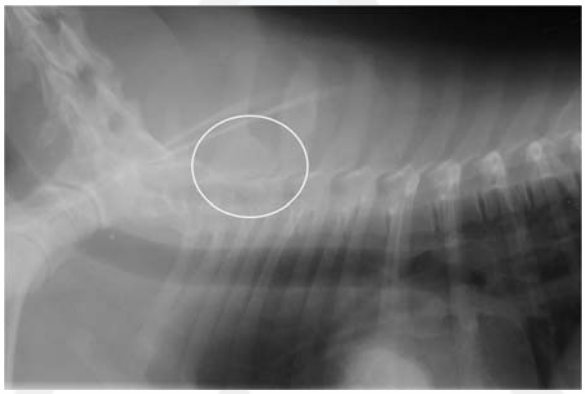
Chondrodysplasia (Dwarfism)

Chondrodysplasia, also commonly called dwarfism, has been described as a heritable disease in the great Dane, Scottish deerhound, Alaskan malamute, Norwegian elkhound, and miniature poodle. Chondrodysplasia is characterized by severely shortened limbs but with normal body length and normally sized skull, leaving the impression of a disproportionate dwarf (see picture below. This dog was a Golden Retriever with dwarfism. Note the short legs and the large head).

The disease is genetically transmitted. In the Alaskan malamute, this condition is combined with occurrence of a macrocytic hypochromic anemia. Affected animals are usually not lame. If no severe abnormalities occur, the quality of life of affected dogs may be good. However, if animals show signs of decreased bone density, bowed down backs, joint laxity, reduced diameter of the windpipe, or angular limb deformities, the prognosis is guarded.

Multiple Cartilaginous Exostosis

Multiple cartilaginous exostoses (MCE) are multiple "mini-growth plates" that can be found in random areas of bone. In the picture below there is an abnormal growth noted at one of the vertebral bodies (part of the back bone), outlined by the white oval mark.



They are identified in cats and dogs of any age as bony proliferations on the back bone, the shoulder blade, sternum, ribs, and hip bone. Frequently, the disease is an incidental finding, because animals do usually not show clinical signs. These malformations are a result of disturbed bone formation at the periphery of the growth plate with abnormal, "benign" development of cartilage and fibrous connective tissue. MCE seems to be associated with feline leukemia virus in cats. This condition is reported to occur predominantly in siamese cats.

The disease is a heritable entity in dogs, and has been identified in the vertebrae, ribs, and long bones. Interestingly, lesions are not observed to affect long bone growth. Dogs and cats may have no symptoms, unless the disease causes dysfunction of a joint or vital structure such as the wind pipe or spinal cord. Affected dogs or cats may present with muscle weakness, commonly at an age younger than one year. Depending on the severity of the lesion, progressive neurological signs may be seen. While the disease is often aggressive and carries a poor prognosis in cats, dogs usually have a good prognosis as the process ceases at maturity. However, in rare cases, transformation into malignant neoplasia at several years after initial diagnosis has been reported.

Mucopolysaccharidosis

Mucopolysaccharidosis is a rare storage disease, in which different lysosomal enzyme defects result in the inability to degrade glycosaminoglycans. Bone formation is disturbed, resulting in skeletal abnormalities. There are different types. Mucopolysaccharidosis type VI occurs in the Siamese cat. Animals present with dwarfism, a disproportionate small and broad maxilla, small ears, large paws, hip dysplasia, crepitus and hypermotility in multiple joints, as well as fusion of vertebral bodies with subsequent neurologic deficiencies. A case example is depicted in the picture below. Note the small ears and large head. This cat also had crepitus and hypermotility in several joints.

Breed-specific DNA tests have been established for the diagnosis of affected animals and carrier detection. Treatment and prognosis depend on type of MPS and the severity of affected bone. The specifics of this disease vary with each case and will be discussed with you during your appointment with the board certified surgeon at VSOA.

We hope that this information pamphlet was helpful to help you. Please do not hesitate to call or ask at your next appointment if you have any questions or concerns.

Dirsko J.F. von Pfeil, Dr.med.vet. DVM
Diplomate American College of Veterinary Surgeons
Diplomate European College of Veterinary Surgeons
Adjunct Professor: Small Animal Surgery, Michigan State University
Staff Surgeon: Veterinary Specialists of Alaska, P.C.
Telephone: (907) 274 0645
Fax: (907) 929 3320
Website: www.VSOAK.com

Contents of this handout are in part also available in following publications:

- Von Pfeil DJF, DeCamp CE. The Epiphyseal Plate: Physiology, Anatomy, and Trauma. Compend Contin Educ Vet 2009;31(7):E1-E12
- Von Pfeil DJF, DeCamp CE, Abood S. The Epiphyseal Plate: Nutritional and Hormonal Influences; Hereditary and Other Disorders. Compend Contin Educ Vet 2009;31(7):E1-E14