Juvenile orthopedic diseases affect the musculoskeletal system of immature animals, and most of these diseases can be traced to pathologic events (eg, diseases, toxins, inappropriate nutrition, trauma) occurring in this period.

This 2-part series addresses the most common pathologic conditions affecting juvenile dogs and cats, including:

- **Congenital and neonatal orthopedic diseases:** Defined, for these articles, as diseases that occur in the prenatal period or within the first 3 to 4 weeks of life
- **Pediatric bone, cartilage, and joint diseases:** Diseases that occur in the skeletally immature dog.

Part 1 of this series presented an overview of musculoskeletal development and pediatric bone diseases (diseases that occur after 1 month of age and before skeletal maturity), which generally have a good prognosis. This article discusses congenital and neonatal orthopedic diseases as well as pediatric cartilage and joint diseases.

### CONGENITAL & NEONATAL ORTHOPEDIC DISEASES

Neonatal diseases are apparent at birth or within the first 3 to 4 weeks of life. While these diseases are often congenital and inherited, a direct cause for each disease has not yet been determined, and other causes, such as in utero factors, may play a role.

Such diseases can be categorized according to the tissue affected. For example, dysostoses refer to diseases of individual bones or a defect in mesenchymal bone formation, whereas osteochondrodysplasia refers to defects of endochondral or intramembranous ossification.1 Part 1 of this series—*Musculoskeletal Development & Pediatric Bone Diseases* (May/June 2016)—presents a basic overview of the steps of ossification.

A thorough history is crucial and useful in obtaining a diagnosis; it should include information regarding:

- Age at which the disease became apparent
- Whether other littermates or relatives have similar presentations
- Breeding environment and general care of the dam and puppies (eg, raised in “puppy mill”)
- Exposure to medications or radiation
- Maternal health.

### Dysostoses

**Overview**

A *dysostosis* is a defect in the development of a bone or part of a bone.1,2 In contrast, a *synostosis* is a defect in the development of 2 or more adjacent bones, leading to fusion. Dysostoses result from 1 or more failures in proper development of mesenchymal bone, transformation of the mesenchymal bone model into cartilage, or conversion of cartilage into bone. Such defects can occur in the axial or appendicular skeleton.2,3

**Axial dysostoses** include hemivertebra, block vertebra, butterfly vertebra, transitional vertebra, spina bifida, facet aplasia, and dens malformation;4 the most commonly encountered axial dysostoses are summarized in Table 1. Consequence of such malformations can lead to varying degrees of spinal cord or nerve root compression and, thus, varying degrees of subsequent neurologic dysfunction.4,5

Furthermore, any of these conditions can be seen in the absence of clinical signs, and their presence alone does not necessarily warrant intervention. For example, hemivertebra often results in characteristic spinal angulation at the site of the defect, as seen by kyphosis, lordosis, or scoliosis.4

Any breed can be affected by such conditions (Table 1) and, while few direct cause-and-effect relationships have been determined, many of these conditions are likely inherited.5
**Appendicular dysostoses** include amelia, hemimelia, dimelia, ectrodactyly, polydactyly, and syndactyly; the most commonly encountered conditions are summarized in Table 2. Hemimelia and dimelia can be seen in any appendicular location.²³

- **Terminal hemimelia** refers to a defect in which all or some of the bones distal to a certain point are missing.
- **Intercalary hemimelia** refers to a condition in which the bones proximal and distal to the missing bone or bones are present.
- Subdivisions of both intercalary and terminal hemimelia include *transverse*, or complete absence of

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**TABLE 1. Axial Dysostoses⁴⁵**

<table>
<thead>
<tr>
<th>AXIAL DYSOSTOSIS</th>
<th>DEFECT</th>
<th>GROSS APPEARANCE</th>
<th>CLINICAL AFFECT</th>
<th>BREED PREDISPOSITION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemivertebra</td>
<td>Failure of sclerotome formation</td>
<td>Wedge-shaped, incomplete</td>
<td>Spinal malangulation (kyphosis, lordosis, scoliosis) at level of the defect</td>
<td>* Bulldogs (English and French), pugs, and Boston terriers, Thoracic hemivertebra is inherited in German shorthair pointers</td>
</tr>
<tr>
<td>Block vertebra</td>
<td>Failure of vertebral segmentation, lack of adjoining disk space</td>
<td>Fusion of adjacent vertebra</td>
<td>Varies</td>
<td>None recognized</td>
</tr>
<tr>
<td>Butterfly vertebra</td>
<td>Midline cleft through the body</td>
<td>“Butterfly” appearance</td>
<td>Varies</td>
<td>None recognized</td>
</tr>
<tr>
<td>Spina bifida</td>
<td>Failure in fusion of dorsal laminae; may be present along with herniation of meninges and/or spinal cord</td>
<td>Open bone defect in dorsal spinal column with (spina bifida aperta) or without (spina bifida occulta) soft tissue herniation</td>
<td>Varies</td>
<td>* Manx cats, Bulldogs (English and French), pugs, and Boston terriers</td>
</tr>
</tbody>
</table>

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**TABLE 2. Appendicular Dysostoses¹³**

<table>
<thead>
<tr>
<th>APPENDICULAR DYSOSTOSIS</th>
<th>DEFECT</th>
<th>TREATMENT</th>
<th>PROGNOSIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amelia</td>
<td>Absence of 1 or more limbs; frequently have concurrent life-threatening conditions</td>
<td>Physical rehabilitation, prostheses and/or carts</td>
<td>Generally poor if more than 1 limb affected; however, uncommon successes have occurred</td>
</tr>
<tr>
<td>Hemimelia</td>
<td>Complete or partial absence of 1 or more bones</td>
<td>Varies according to location and type of hemimelia</td>
<td>Varies according to location and type of hemimelia</td>
</tr>
<tr>
<td>Dimelia</td>
<td>Duplication of entire, or part of limb</td>
<td>Conservative treatment or amputation of nonfunctional portion of limb</td>
<td>Generally good</td>
</tr>
<tr>
<td>Ectrodactyly</td>
<td>Digital cleft between metacarpal bones</td>
<td>Conservative (splinting) or surgical (reconstruction or amputation)</td>
<td>Dependent on degree of deformity and progression</td>
</tr>
<tr>
<td>Polydactyly</td>
<td>1 or more extra digits</td>
<td>Only in case of trauma or infection of extra digits; may include amputation of diseased digits</td>
<td>Generally good</td>
</tr>
<tr>
<td>Syndactyly</td>
<td>Lack of differentiation between 2 or more digits</td>
<td>Weight-bearing digits can be surgically separated for symptomatic cases</td>
<td>Generally good</td>
</tr>
</tbody>
</table>
bones along the width of the limb, and longitudinal, or absence of bones along the long axis (medial or lateral) of the bone (Figures 1 and 2).

**Signalment**
Most reports of hemimelia appear to lack a hereditary linkage, but there are exceptions, including Chihuahuas, Siamese cats, and domestic shorthair cats. Ectrodactyly is autosomal dominant in cats, and polydactyly is autosomal dominant in cats and most dogs. The exception is seen in the Saint Bernard and collie, in which preaxial (medial) polydactylism is likely an autosomal recessive trait.

**Diagnosis**
Many dysostoses can be diagnosed on physical examination, but the extent of abnormality may be better characterized by orthogonal radiography. In general, most dysostoses are radiographically apparent. In the case of axial dysostoses, advanced imaging (magnetic resonance imaging [MRI]) is indicated in the presence of neurologic dysfunction to further characterize the degree and source of spinal cord compression.

**Treatment**
Treatment is generally aimed at conditions in which clinical effects degrade patient quality of life and can be divided into medical and surgical interventions.
- Medical intervention may include physical rehabilitation to alleviate pain, build muscle, and regain function.
- Splinting may be indicated when bone and limb deformities or associated soft tissue laxities or contractures are present, as can be seen with ectrodactyly or hemimelia.
- Other medical therapies are generally aimed at alleviation of clinical signs and may include pain-modulating medications and nonsteroidal anti-inflammatory drugs (NSAIDs).
- The objectives of surgery can either be palliative or reconstructive: Palliative surgery includes total or partial amputation of severely deranged limbs or digits that interfere with ambulation or are a source

![Figure 1](image1.png)
**Figure 1.** An 11-week-old, male beagle presented for evaluation of left forelimb lameness. Diagnosis is ectrodactyly of the left manus with absence of the second and third carpal bones. Lateral (A) and craniocaudal (B) views of the left distal antebrachium; note that only 3 digits are present on the left manus. The base of the presumed fifth metacarpal is located more laterally and caudally, and this bone is articulating with the presumed ulnar and fourth carpal bone. Courtesy University of California–Davis Veterinary Medical Teaching Hospital

![Figure 2](image2.png)
**Figure 2.** A 4-month-old, female domestic shorthair cat presented for evaluation of bilateral thoracic limb angular deformity. Diagnosis is bilateral radial agenesis or hemimelia. Lateral (A, C) and craniocaudal (B, D) views of the left and right forelimbs; findings are similar in both limbs, with the radius absent and the ulna abnormally shaped. On the left, the radial head appears to be incorporated into the ulna (arrow, A). On the right, a hypoplastic radial head is present (arrow, C). There is carpal varus, and only 1 carpal bone is present in the proximal row. Courtesy University of California–Davis Veterinary Medical Teaching Hospital
of discomfort due to misuse or self-mutilation. Reconstruction is aimed at limb salvage through realignment and, frequently, arthrodesis. These surgeries include amputation, arthrodesis, or reconstruction, when possible.

• Neutering is advised in conditions with known inheritance.

Prognosis
The prognosis for axial dysostoses is highly variable and depends on the degree of malformation, amount of neurologic compression, and degree and progression of secondary changes to both bone and soft tissue. See Table 2 for appendicular cases.

Osteochondrodysplasias

Overview
Osteochondrodysplasias are a group of cartilage and bone disorders that occur due to defective endochondral or intramembranous ossification (Table 3). The resultant defects manifest as slowed growth and small stature when compared with animals of the same breed and age. Such animals, often termed dwarfs, can exhibit proportionate or disproportionate growth. The latter term refers to patients in which the limbs or trunk are relatively short or long. Interestingly, some forms of osteochondrodysplasia can be intentional, which occurs with selective breeding of certain breeds to create a specific phenotypic appearance (eg, miniature dachshund). Strictly speaking, dwarfism refers to the condition by which unintentional defects occur.

Signalment
Numerous breeds have been reported with this condition; several are listed in Table 3.

Diagnosis

History (including familial) and physical examination (identifying concurrent congenital defects) in conjunction with radiography are generally sufficient for diagnosis of osteochondrodysplasia.

The following is the minimum database for an animal with dwarfism:

- Nutritional history
- Complete blood count
- Serum biochemical profile
- Urinalysis
- Fecal analysis (parasites)
- Radiography

<table>
<thead>
<tr>
<th>BREED</th>
<th>TRAIT</th>
<th>MODE OF INHERITANCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Akita</td>
<td>Achondrogenesis</td>
<td>Unknown</td>
</tr>
<tr>
<td>Alaskan malamute</td>
<td>Chondrodysplasia</td>
<td>Simple autosomal recessive</td>
</tr>
<tr>
<td>Beagle</td>
<td>Chondrodysplasia punctata</td>
<td>Unknown</td>
</tr>
<tr>
<td></td>
<td>Multiple epiphyseal dysplasia</td>
<td>Simple autosomal recessive</td>
</tr>
<tr>
<td></td>
<td>Osteogenesis imperfecta</td>
<td></td>
</tr>
<tr>
<td>Bulldog</td>
<td>Osteochondrodysplasia</td>
<td>Unknown</td>
</tr>
<tr>
<td>Bull terrier</td>
<td>Osteochondrodysplasia</td>
<td>Unknown</td>
</tr>
<tr>
<td>Cocker spaniel</td>
<td>Hypochondrodysplasia</td>
<td>Unknown</td>
</tr>
<tr>
<td>Collie</td>
<td>Osteogenesis imperfecta</td>
<td></td>
</tr>
<tr>
<td>Dunker</td>
<td>Multiple epiphyseal dysplasia</td>
<td>Unknown</td>
</tr>
<tr>
<td>Great Pyrenees</td>
<td>Chondrodysplasia</td>
<td>Simple autosomal recessive</td>
</tr>
<tr>
<td>Hygenhund</td>
<td>Multiple epiphyseal dysplasia</td>
<td>Unknown</td>
</tr>
<tr>
<td>Irish setter</td>
<td>Hypochondrodysplasia</td>
<td>Simple autosomal recessive</td>
</tr>
<tr>
<td>Labrador retriever</td>
<td>Oculoskeletal dysplasia</td>
<td>Simple autosomal recessive</td>
</tr>
<tr>
<td>Miniature poodle</td>
<td>Achondrodysplasia</td>
<td>Simple autosomal recessive</td>
</tr>
<tr>
<td></td>
<td>Multiple epiphyseal dysplasia</td>
<td>Unknown</td>
</tr>
<tr>
<td>Mixed-breed dog</td>
<td>Mucopolysaccharidosis VII*</td>
<td>Simple autosomal recessive</td>
</tr>
<tr>
<td>Norwegian elkhound</td>
<td>Chondrodysplasia</td>
<td>Simple autosomal recessive</td>
</tr>
<tr>
<td>Plott</td>
<td>Mucopolysaccharidosis I*</td>
<td>Simple autosomal recessive</td>
</tr>
<tr>
<td>Pointer</td>
<td>Enchondrodystrophy</td>
<td>Homozygous recessive</td>
</tr>
<tr>
<td>Samoyed</td>
<td>Oculoskeletal dysplasia without hematologic abnormalities</td>
<td>Simple autosomal recessive</td>
</tr>
<tr>
<td></td>
<td>Oculoskeletal dysplasia with hematologic abnormalities</td>
<td>Unknown</td>
</tr>
<tr>
<td>Scottish deerhound</td>
<td>Pseudoachondrodysplasia</td>
<td>Simple autosomal recessive</td>
</tr>
<tr>
<td>Scottish terrier</td>
<td>Achondrodysplasia</td>
<td>Unknown</td>
</tr>
<tr>
<td>Shiba Inu</td>
<td>Idiopathic multifocal osteopathy</td>
<td>Unknown</td>
</tr>
<tr>
<td>Domestic shorthair cat</td>
<td>Mucopolysaccharidosis I*</td>
<td>Unknown</td>
</tr>
<tr>
<td>Siamese cat</td>
<td>Mucopolysaccharidosis VI*</td>
<td>Simple autosomal recessive</td>
</tr>
</tbody>
</table>

* Genetic test available

Table 3. Canine & Feline Osteochondrodysplasia

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In patients in which an endocrinopathy is suspected, include measurement of:
- Serum thyroxine
- Thyroid-stimulating hormone
- Growth hormone (stimulation test)
- Insulin-like growth factor-1 serum concentrations.

Radiographic evaluation should include the lumbar spine and radius/ulna, with assessment for abnormal growth plate morphology, delayed epiphyseal growth center development, and reduced length of the axial/appendicular skeleton.3

Biopsy of growth plates (eg, rib, postmortem) can confirm the diagnosis (Figure 3).1,3

Treatment
Treatment is generally aimed at ameliorating clinical signs. As in other conditions discussed in this article, the heritable nature of this condition warrants neutering the affected animals or known carriers.

A general rule
of thumb is that nutritional diseases and osteochondrodysplasias most often result in disproportionate growth, whereas metabolic, endocrine, and polysystemic diseases result in proportionate growth.3

Prognosis
Prognosis varies according to the type and severity of osteochondrodysplasia.

Swimmer Syndrome
Overview
Swimmer syndrome—also known as flat-pup syndrome, turtle-pup, splay leg, myofibrillar hypoplasia, and twisted legs—is a developmental abnormality generally apparent within the first few weeks of life.3,7,8

The syndrome is characterized by hindlimb paraparesis or tetraparesis, with paraparesis being the most common form of presentation.3 Affected animals move with a “swimming” motion of the limbs, often in a sternal position due to their inability to adduct their limbs to rise.3,7,8

Animals with thoracic limb involvement commonly have concurrent thoracic abnormalities, including pectus excavatum, sterna concave, or dorsoventral flattening of the chest.3,7,8 These concurrent abnormalities can lead to respiratory compromise.

The cause of this condition is unknown, although poor traction in the environment has been implicated.3 It is thought to be a rare syndrome; the prevalence in a population of small-breed dogs in Thailand is around 2%.7

Signalment
This condition, which can occur in any dog or cat, becomes apparent around 1 to 3 weeks of age, when walking is normally initiated. Certain breeds appear to be predisposed,7,8 including dachshunds, Yorkshire terriers, Pekingese, basset hounds, and French and English bulldogs. It has also been reported in a Devon rex cat.8

Diagnosis
Physical examination alone is the most common means of diagnosis. As mentioned earlier, affected animals often display signs within 1 to 3 weeks of birth and the condition is characterized by sternal recumbency with lateral splaying of the hindlimbs and, occasionally, the forelimbs.3,7,8

Animals are unable to walk and instead move their affected limbs in a paddling motion.3 After some time, chronic positioning in sternal recumbency results in dorsoventral compression of the chest with lateral widening, which, without intervention, may become permanent.3,7

Other conditions, such as encephalomeningitis, canine distemper, toxoplasmosis, neosporosis, myopathy, and spina bifida, should be considered.7
**Treatment**

Treatment generally involves intense physical therapy, including splinting, bandaging (eg, hobbles), and muscle strengthening exercises. With aggressive treatment and environmental changes, a response is often noticed within the first 3 to 4 weeks.

Extreme care must be exercised when bandages and/or splints are placed on growing animals because these devices must be changed frequently to avoid complications. Owners must also be properly educated on the care of such devices, with instructions to monitor for soiling, movement/slipping, swelling, or exposed digits.

For animals with pectus excavatum, surgical correction of the deformity is recommended, if they have respiratory compromise or severe deformity. The reader is referred to the references for more specific details on the management of pectus excavatum because this is beyond the scope of this article.

**Prognosis**

Prognosis can be good for patients whose condition is detected and treated early with therapy and supportive care. For chronic or untreated cases, prognosis is guarded. Humane euthanasia is often chosen when owners are unable to meet the high demands of nursing care and physical rehabilitation exercises or when severe joint/limb rigidity has developed.

**PEDIATRIC CARTILAGE DISEASES**

**Osteochondrosis**

**Overview**

Osteochondrosis (OC)—also known as osteochondritis dissecans and osteochondrosis dissecans (OCD)—is a complex, multifactorial condition. Discussion of the etiopathogenesis is beyond the scope of this article; however, the interested reader is referred to Ytrehus et al for a thorough review.

OC is a common disorder of developing cartilage in humans and domestic animals. The exact cause has been theorized, but one common unifying theme has not been identified. It is generally accepted that the disease results from an aberration in endochondral ossification, the process whereby cartilage is gradually converted into bone.

Ytrehus et al have proposed classifying articular OC on the basis of disease stage. In this system:

- **Osteochondrosis latens** refers to the presence of an early, microscopic lesion
- **Osteochondrosis manifesta** refers to subclinical lesions that are macroscopically and radiologically apparent

**Table 4.**

**Reported & Accepted Clinical Manifestations of Osteochondrosis Dissecans in Dogs**

- Caudomedial aspect of the humeral head
- Medial aspect of the humeral condyle
- Lateral or medial femoral condyle
- Medial or lateral trochlear ridge of the talus
- **Osteochondrosis dissecans** refers to patients in which attached or loose cartilage flaps are present, typically resulting in clinical signs (Table 4). Lesions can occur in both the physeis and epiphysis; however, for this discussion we will focus on OCD of the epiphysis (articular lesions).

**Signalment**

Animals are typically 4 to 8 months of age, with males more commonly affected than females. Large- and giant-breed dogs are most commonly affected, but the condition can occur in small breeds and cats.

**Diagnosis**

Affected animals are often not clinical until a cartilage flap develops. OC should be considered in any young, large-breed dog with lameness and a swollen, painful joint. The contralateral joint should be evaluated regardless of clinical signs because OC is most typically bilateral. Orthogonal radiographs of the affected joint are often diagnostic. Defects in the subchondral bone, flattening of the normal contour and, in some cases, sclerotic margins are typically noted (Figures 4 to 6, page 30). Mineralization of the cartilage flap may also be noted.

A positive contrast arthrogram can be helpful in identifying lesions when the cartilage flap has not mineralized. Computed tomography (CT) and MRI can be helpful in detecting early lesions and in imaging more challenging joints (eg, tarsus and elbow).

Finally, arthroscopy can be used for direct visualization of the lesion and, in some cases, allow treatment at the same time.

**Treatment**

Treatment options vary according to the joint affected and the lesion size. Medical management—maintaining a lean body condition, physical rehabilitation, regular controlled activity, and NSAIDs—is an option in most cases.

- Shoulder OCD is very amenable to fragment removal with curettage of subchondral bone
(abrasion arthroplasty). The specific techniques and goals of abrasion arthroplasty are beyond the scope of this paper.

- Stifle OCD can also be treated successfully with surgical resurfacing methods (eg, Osteochondral Autograft Transfer System [OATS] and SynACART [Arthrex Vet Systems, arthrexvetsystems.com]) (Figure 7).
- Elbow and tarsal OCD are more challenging: Treatment of elbow OCD centers on an abrasion arthroplasty of the humeral lesion. Tarsal OCD, in cases of small fragments, may be amenable to...
fragment retrieval but, in cases of larger fragments, pantarsal arthrodesis may be advocated.

All dogs should be neutered given that the reported heritability of all forms of osteochondrosis ranges from 10% to 45%.10

**Prognosis**

When contemplating the prognosis for OC, it is prudent to consider that not all joints are equal. A joint that is relatively nonconstrained, such as the shoulder, has a greater tolerance for abnormalities in cartilage pathology, whereas a more highly constrained joint, such as the tarsus, has little tolerance and, thus, a worse prognosis.

Medical management of shoulder OC can be successful unless the cartilage flap dislodges, becomes mineralized, and is trapped in a joint capsule recess or near the biceps tendon, where it may cause irritation and inflammation. After removal, the prognosis appears to be excellent, with minimal to no development of osteoarthritic changes.13

Due to cartilage loss, joint incongruity, and secondary osteoarthritis, stifel OC is traditionally given a fair to poor prognosis; however, treatment with joint resurfacing techniques is promising and appears to provide a more favorable prognosis.15

Treatment for OC of the elbow and hock will often result in improved lameness. However, osteoarthritis will develop/progress and lameness is usually evident after exercise. In severe cases of tarsal OC, the prognosis for maintaining a functional joint is poor.12

**Retained Cartilage Cores**

**Overview**

Retained cartilage cores (RCC)—also known as retained endochondral cartilage cores—represent a failure of endochondral ossification that most commonly affects the distal ulnar physis. These lesions are characterized:

- Histologically, by the retention of hypertrophic chondrocytes
- Grossly, as cones of physial cartilage that project from the distal ulnar physis proximally into the distal metaphysis.3,12,16

Several etiopathogeneses of RCC exist, with no single theory universally accepted. Some postulate that RCC may result from a chondrocyte abnormality, which prevents progression of endochondral ossification,6,8 while others suggest that RCC is a physial manifestation of osteochondrosis.3,12,16 Given that RCC occurs primarily in large, fast-growing dogs, nutritional contributions have also been suspected.12

**Signalment**

Large- and giant-breed dogs are most commonly affected. An average age of 5 months was noted in one retrospective study.16

**Diagnosis**

In some cases, RCC causes retarded growth of the distal ulnar physis, which can result in shortening, external torsion, and procurvatum of the radius. Clinically this is observed as a torsion-angulation deformity of the distal antebrachium.3,12,16 If RCC is bilateral, so should be the resulting antebrachial deformities.

Radiographically, RCC is characterized by a radiolucent core of cartilage extending from the distal ulnar physis proximally to the metaphysis, ranging in length from 2 to 6 cm.3,16 This core may be surrounded by a zone of sclerosis (Figure 8).3 Although the size of the core can be impressive, it is important to note that there is no known correlation between size of the lesion, histology, and severity of angular deformity.3

![Figure 8](image) A 10-week-old, male Great Dane evaluated after an injury to his right thoracic limb. An incidental finding is presence of bilateral retained cartilage cores (RCC) in the distal ulna. Lateral (A) and craniocaudal (B) images of the distal right forelimb; the area outlined in the box in B is enlarged to highlight the presence of the RCC (arrow). Courtesy Dr. Dan Bucy, University of California–Davis Veterinary Medical Teaching Hospital
**Treatment**

Dogs without angular limb deformation do not require treatment, but these dogs must be monitored with weekly to every-other-week physical examination for the development of an angular deformity. Early intervention, in many cases a distal ulnar physeal ostectomy (Figure 9), is the key to a successful outcome. Those with more severe angular deformities may require corrective osteotomy of the radius and, in some cases, multiple surgical procedures are necessary.

In all cases, owners should be questioned about the dog's diet and environment, and a balanced diet appropriate for age and size should be prescribed.

**Prognosis**

Ultimately, prognosis depends on the severity and presence of any angular deformity. Animals identified and treated early can have a favorable prognosis. Those with a severe deformity have a more guarded prognosis. For all patients, owners should be warned that additional surgical procedures may be necessary.

**PEDIATRIC JOINT DISEASES**

**Puppy Carpal Laxity**

**Overview**

Puppy carpal laxity—also known as carpal instability—is a syndrome in young dogs characterized by carpal hyperextension (angle of extension greater than 190°) and carpal hypoextension (angle of extension less than 180°). Hyperextension conditions are also known as dropped carpus; hypoextension conditions are also known as carpal flexural deformity, bucked carpus, and carpal flexion syndrome.

The cause is not known but may be related to unbalanced growth, poor muscle tone, or weakness between the flexor and extensor muscle groups. Improper exercise, poor footing (e.g., slippery surfaces), inappropriate nutrition, and genetics have also been implicated.

**Signalment**

Age of presentation is usually 6 to 16 weeks; animals may be unilaterally or bilaterally affected. While all breeds can be affected, large breeds tend to be affected more often than small breeds.

**Diagnosis**

Diagnosis is usually based on clinical signs, orthopedic examination, and radiographs of suspected dogs. The orthopedic examination is characterized by carpal laxity and, in cases of carpal hypoextension, a palmograde stance (Figure 10). In cases of carpal hypoextension, the dogs stand with the carpus sitting more cranial than usual (hence the term...
**IN SUMMARY**

This series outlines some of the more common juvenile orthopedic diseases encountered in small animals. As noted previously in Part 1 of this series, normal musculoskeletal development is key because, as discussed in this article, deviation from normal can result in many orthopedic diseases.

**Prognosis**

The prognosis is good to excellent for most patients with mild to moderate severity.