

Developmental Orthopedic Disorders in Foals and Yearlings

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INTRODUCTION

Orthopedic problems in foals can be congenital or acquired. Congenital problems are present at birth and are usually obvious. Congenital angular and flexural deformities sometimes lead to dystocia and in some cases necessitate fetotomy or Caesarean section to relieve dystocia.

Many foals are born with some degree of flexural or angular limb problem. The most common appearance is a degree of flexor laxity and carpal and/or tarsal valgus deformity. Fortunately, these deformities usually self-correct with exercise and age and should be considered normal findings in a newborn foal. Likewise, congenital flexural deformity of both the metacarpal/tarsal phalangeal joint, distal interphalangeal joint, and carpus may be present. Mild forms of these conditions will self-correct with age and exercise, but in my experience moderate to severe congenital flexural deformity usually requires some type of intervention.

Orthopedic evaluation of a foal can be difficult, and foals will often look different inside and outside of a stall. For example, a foal with even a mild lameness may be nonweight-bearing when loose in a stall but look much less lame when walked with the mare. Also, it is difficult to accurately assess flexural and angular deformities with a foal standing in a stall. For this reason, I usually perform palpation of the limbs with the foal restrained in a stall and evaluate conformation and gait with the foal outside of the stall.

Flexor Laxity

Many foals are born with flexor laxity. It is usually most obvious in the hind limbs with the fetlock dropping close to the ground and occasionally the toe will lift off the ground when the foal is weight-bearing. In general, this laxity self-corrects over a few days as muscle tone improves with exercise.

In some circumstances, laxity is severe enough to prevent the foal from getting up to nurse or is such that the palmar/plantar aspect of the fetlock develops abrasions. In these cases, lowering the heels with a rasp or the use of glue-on shoes with extended heels or the use of acrylic, such as Equilox®, is helpful in providing heel support. Exercise should be encouraged but limited to prevent fatigue. The use of bandages (except to protect the soft tissues) and splints is generally contraindicated because they encourage further laxity. The use of casts is discouraged. Most foals improve significantly once normal loading of the flexor tendons occurs.

Flexural Deformity (Contracted Tendons)

Flexural deformity can be congenital or acquired. The congenital form may involve the carpus, fetlock, and/or coffin joints. Acquired flexural deformity of the coffin joint typically occurs in four- to eight-month-old horses. Flexural deformity of the fetlock joint usually occurs in eight- to fourteen-month-old horses. Acquired flexural deformity of the carpus is usually associated with a severe lameness in that limb.

In neonates with congenital flexural deformity, exercise, splints, casts, and oxytetracycline have been used to help achieve tendon laxity. In mild cases of flexural laxity, corrective hoof trimming and exercise are often curative. Polyvinyl chloride (PVC) splints placed over a padded bandage are used

to place the limb in a weight-bearing position. It is important to reset the splints daily and to use well-padded bandages to prevent rub sores, which easily occur in foals. I often will place the splints in the morning and remove them at night, repeating this procedure daily.

Casting will cause flexor tendon laxity. Casts should be left on no longer than ten days and early removal may be necessary if a cast rub is suspected. Commercially available splints (Dynasplint®) can be custom fitted and depend upon dynamic loading of the limbs to correct flexural deformities.

The use of the antimicrobial oxytetracycline to induce tendon relaxation has gained acceptance. It is thought that the oxytetracycline binds calcium and causes muscle relaxation and effective tendon lengthening. Three grams of oxytetracycline is given intravenously. Since this is such a large dose, the author usually dilutes the drug in one liter of saline and gives it over a 15-minute period.

In the United States, oxytetracycline is not approved for use in horses, so it is important to inform the client of this and of the potential side effects, which include diarrhea, renal failure, and acute death. I have not witnessed these side effects in my use of this drug. In foals less than two weeks of age, the effects are often dramatic and occasionally flexor laxity may occur. If the desired effect is not seen, a second or third dose is given on successive days. Splinting is often used in concert with oxytetracycline treatment. Oxytetracycline has proven to be an effective medical treatment in the management of flexural deformities in foals less than two weeks of age. In older foals (greater than 30 days), oxytetracycline treatment is often not effective, probably due to the increased muscular development in these foals.

Acquired flexural deformity of the coffin joint is usually seen in foals between four and eight months and is associated with large, fast-growing foals on a high plane of nutrition. Nonsurgical methods of correction include extended toe shoes, phenylbutazone, and exercise, and are chosen for mild cases. For cases that are moderate to severe in nature or unresponsive to medical treatment, inferior check ligament desmotomy and extended toe shoes are recommended. Correction after inferior check ligament desmotomy is usually dramatic and immediate. Corrective shoes usually are necessary for four to eight weeks postoperatively.

Acquired flexural deformity of the fetlock joint is usually seen between eight and fourteen months of age and is also associated with large, fast-growing foals on a high plane of nutrition. In general, nonsurgical methods are only helpful in mild cases and consist of raising the heel with a wedge pad, phenylbutazone, and exercise. For cases that are moderate to severe in nature or unresponsive to medical treatment, superior check ligament desmotomy and elevation of the heels with wedge pads is recommended. Horses that are beyond vertical cannot load their tendons adequately for conservative management to work, and surgical treatment is recommended. In severe cases, a distal check ligament desmotomy and splinting are often combined with the proximal check ligament desmotomy to achieve maximize tendon/muscle unit lengthening. If excessive feeding has been a contributing factor for the deformity, nutritional adjustment should be made.

Angular Deformities

Most foals are born with some degree of tarsal and carpal valgus deformity, so this is generally considered normal. As the foal matures, many angular deformities completely improve. Moderate to severe (>10 degrees) valgus deformities that are still present at six weeks are considered abnormal and require evaluation. Causes of valgus deformity include ligamentous joint laxity, physal dysplasia, and cuboidal bone abnormalities. Ligamentous joint laxity can be assessed by manipulation of the foal's limb. Radiographs are required to diagnose physal dysplasia or cuboidal bone problems.

Dorsopalmar radiographs reveal the location of the angular deformity by the center of the angle formed by lines drawn down the center of the metacarpus and radius. If the lines meet in the physis or

epiphysis, the abnormality originates from the physis. If the lines meet in the carpus, the abnormality springs from the cuboidal bones. Fortunately, most of the bony causes for angular limb deformities are in the physis, which is correctable surgically. Inflammation of the physis (physisitis) can be a contributing cause of angular deformities by causing disproportionate growth at the growth plate. Causes of physisitis include mineral imbalances, overfeeding, and poor conformation or hoof care.

Periosteal elevation and transection has been accepted as an effective means of correcting angular limb deformities in growing horses. The surgery is performed on the “short” side of the bone (in the case of a valgus deformity on the lateral side) and the transection performed two centimeters proximal to the physis. Growth acceleration occurs on the operated side, and overcorrection is not possible. Surgical correction for fetlock deformities should be performed before eight weeks of age and for carpal deformities by four months of age. The earlier the surgery is performed, the more rapid the correction; however, case selection is important because some foals will self-correct.

In cases of severe angular deformity or in older foals and yearlings, physal retardation with screws and wires or transphyseal screws is necessary to slow the growth on the “long” side of the bone. In our practice, physal retardation of the fetlock is exclusively accomplished with transphyseal screws and that of the carpus with screws and wires up through approximately 14 months of age; after 14 months, it seems to be safe to use transphyseal screws with limited risks of overcorrection of the carpus in yearlings after removal.

Environmental or nutritional factors must also be addressed in the overall treatment of angular limb deformities, especially if a herd problem exists.

Osteochondrosis

Osteochondrosis is a form of developmental orthopedic disease that occurs commonly in all breeds of horses. Clinical signs include mild to moderate lameness and joint effusion. Some osteochondrosis lesions can be clinically silent but become important sources of lameness when the horse is placed in work or even later in its career after years of normal activity.

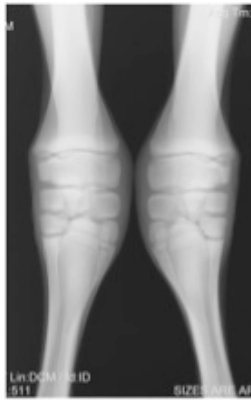
The cause of osteochondrosis is multifactorial and can be related to trauma, environmental, nutritional, and genetic factors. For these reasons, it can be difficult to give a specific cause of the abnormality. In situations where a high percentage of horses are affected, it may be possible to determine a specific cause of osteochondrosis. Essentially, there is an abnormality in the normal process of endochondral ossification or the transformation from cartilage to bone. In general terms, the incidence of osteochondrosis can be reduced by allowing normal pasture activity and by providing a well-balanced diet that includes trace minerals.

Some breed associations have made efforts to reduce the incidence of osteochondrosis in their respective breeds by avoiding mating of affected horses. In an individual horse, some clinical cases of osteochondrosis will heal over time. Fragments that are separated from the parent bone are usually most reliably treated with arthroscopic removal. In most circumstances, removal of the abnormal cartilage and bone is curative and allows the horse to resume normal athletic activity.

Take-Home Message

Management of orthopedic disorders in foal can be challenging and rewarding. In my experience, a combination of early recognition and intervention coupled with a patient attitude to allow the foal to improve is best in the management of angular and flexural deformities. Other forms of developmental orthopedic disease including osteochondrosis can cause joint effusion and lameness and can be reliably treated with arthroscopic surgery and debridement.

DEVELOPMENTAL ORTHOPEDIC DISEASE



Eight-week-old Standardbred foal before and after placement of a transphyseal bridge and correction of its carpal valgus angular deformities.

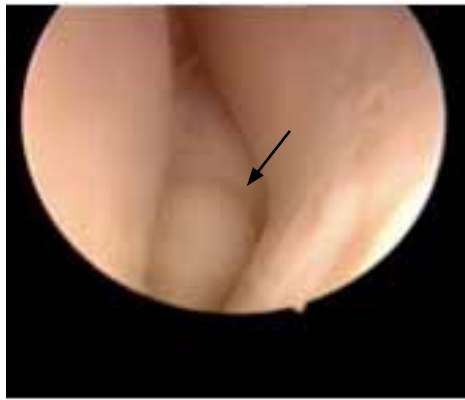


Two-month-old Thoroughbred foal with left fore fetlock varus deformity (toeing in). Radiograph taken after transphyseal bridge was placed for correction.

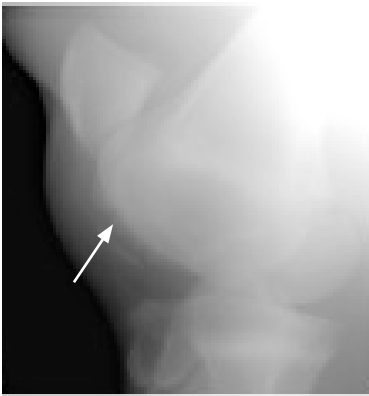


DEVELOPMENTAL ORTHOPEDIC DISEASE

Preoperative and postoperative radiographs and two days postoperative photograph of a four-month-old Arabian foal with moderate flexural deformity of the distal interphalangeal joint. Treated with inferior check ligament desmotomy and therapeutic trimming.



Preoperative and arthroscopic view of an osteochondrosis lesion of the fetlock in a Thoroughbred yearling.



Preoperative and arthroscopic view of an osteochondrosis lesion of the stifle in a Thoroughbred yearling.

DEVELOPMENTAL ORTHOPEDIC DISEASE



Preoperative and arthroscopic view of an osteochondrosis lesion of the tarsus in a Thoroughbred yearling.