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Acquired Flexural Deformities

By David G. Wilson, DVM

Acquired flexural deformities of the coffin and fetlock joints are an important problem in foals and young horses, respectively. Deformities at the coffin joint can lead to clubfoot and caudal heel syndrome, while deformities at the fetlock joint can lead to degenerative joint disease. This issue of *Large Animal Veterinary Rounds* discusses the etiology and treatment of flexural deformities.

Acquired flexural deformities of the coffin and fetlock joint are well-recognized developmental orthopedic conditions in foals and juvenile horses. Early descriptions termed the condition “contracted tendons”; however, this is a misnomer, since tendons have little capacity to contract. In the truest sense, muscle contraction leads to flexion and persistent flexion leads to a state of contracture. Abnormal flexion at the coffin joint generally occurs in foals <4 months of age, whereas involvement at the fetlock joint typically develops in juvenile horses between 10 and 18 months of age. The condition is frequently bilateral.

Etiology

When first described, acquired flexural deformities were attributed to an imbalance of growth between the long bones of the limb and the soft-tissue structures, specifically, the digital flexor tendons and accessory ligaments of the deep digital flexor (DDF) and superficial digital flexor muscles. Early support for the “imbalance of growth” theory was based on knowledge of the periods of growth in the metacarpus and radius. While growth may play a role, the imbalance theory fails to explain the sudden onset of the condition at a time when long bone growth has essentially stopped. It is known that growth of the metacarpus is minimal after 90 days of age; yet, flexural deformities at the coffin joint develop after that time.

Previously, animals with unilateral involvement were believed to have developed flexural deformity secondary to an injury, resulting in a period of reduced weight bearing on the affected limb. Proponents of this pathogenesis believed that during periods of reduced weight-bearing, the soft-tissue structures on the flexor surface of the limb become shortened because they are not repeatedly stretched during the stance phase. Firth dispelled this theory by clearly demonstrating that failure to bear weight alone does not result in the development of a flexural deformity.¹

Acquired flexural deformities have often been associated with high-energy diets and foals pushed for development in preparation for autumn futurities were thought to be particularly at risk. In humans, limb pain associated growth is referred to as “growing pains.” Most clinicians now believe pain is likely the precipitating factor in the development of acquired flexural deformities in horses. A natural reaction to limb pain is “stenting” or persistent contraction of the muscles in the limb. The stronger digital flexor muscles overcome and fatigue the extensors, resulting in abnormal flexion at the coffin or fetlock joints. The capacity of the coffin and fetlock joints to resist flexion is determined not only by the opposition of the digital extensor muscles, but also by the simple lever arm mechanics of the individual joints. In foals, mechanical resistance to flexion of the coffin joint is initially less than that of the fetlock joint. As the hoof grows – effectively lengthening the lever arm of the coffin joint – the fetlock becomes easier to flex than the coffin joint. These changes in



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Figure 1: Flexural deformity of the coffin joint results in elevation of the heel (clubfoot)



lever arm mechanics as the horse matures offer a rational explanation for the age predisposition of these deformities relative to the joint involved. Finally, if abnormal flexion is sustained, the supporting structures on the flexor side of the limb become shortened, preventing a return to a normal joint angle, even when the pain is gone and the muscles have relaxed. In these cases, surgical manipulation offers the best prognosis for a return to normal posture.

Acquired flexural deformities at the coffin joint

A flexural deformity at the coffin joint is commonly referred to as a “clubfoot” and it typically develops in foals <4 months old. In this case, an important factor in the development of flexural deformities is the deep digital flexor muscle where direct action is exerted on the flexor surface of the distal phalanx. With inappropriate contraction, the coffin joint is pulled into flexion, resulting in the elevation of the heel from the ground and the transfer of weight-bearing to the toe (Figure 1). In the short-term, if abnormal muscle contraction ceases, flexion at the coffin joint resolves and allows normal hoof-to-ground contact. In most cases, abnormal muscle contraction persists long enough for the toe to wear down and results in the heel returning to ground contact with the coffin joint abnormally flexed (Figure 2). At this point, corrective hoof care, in the form of lowering the heels, fails to re-establish a normal joint angle because the deformity has reached the point of contracture. Specifically, the inferior check ligament tendon unit is anatomically shortened due to the lack of mechanical stimulus for the ligament to elongate during the period of muscle contraction. The toe of the hoof tends to grow slower than the heel and, if left untreated, horses with this condition are relegated to a lifetime of corrective shoeing. Each time the horse is trimmed or shod, the farrier must

Figure 2: When abnormal muscle contraction persists, the toe wears down, the heel returns to ground contact, and the coffin joint is abnormally flexed



remove more heel than toe in an attempt to establish a normal hoof angle. During the immediate post-trimming or shoeing period, the strain on the deep digital flexor (DDF) tendon is elevated and more pressure is placed on the navicular area, potentially predisposing the horse to the development of “caudal heel syndrome.”

Classification

Flexural deformities at the coffin joint are classified based on the severity of the angle between the dorsal hoof wall and the ground surface. In mild-to-moderate deformities (Stage I), the dorsal hoof wall has not gone beyond a line perpendicular to the ground surface (Figures 1 and 2). In horses with severe deformities (Stage II), the dorsal hoof wall has gone beyond the perpendicular (Figure 3).² Horses with severe deformities are unlikely to have athletic careers, although, with appropriate treatment, mild and moderately affected animals can lead normal lives.

Figure 3: In severe flexural deformity (Stage II), the dorsal hoof wall is held in a position beyond perpendicular



Flexural deformities at the coffin joint are easily diagnosed based on the clubfoot appearance. Affected foals are rarely foot sore and radiographic assessment, although useful for the farrier, rarely demonstrates any abnormality other than flexion at the coffin joint. Unlike foals, an older horse with a clubfoot may present with lameness and a hoof-tester examination will consistently reveal sensitivity over the toe. If a farrier has repeatedly tried to correct the condition, the horse may also be heel sore. In these cases, a palmar digital nerve block will improve, but not resolve, the lameness. Radiographs typically reveal pedal osteitis and varied radiographic changes in the navicular bone.

Treatment

If presented for evaluation and treatment early in the development of the condition (stance still up on the toes, as in Figure 1), an aggressive nonsurgical approach may be successful. It is imperative that the pain responsible for the muscle contraction be lessened. Administration of non-steroidal anti-inflammatory drugs (NSAIDs) is indicated, as is the immediate balancing of the diet with energy restriction. Mechanically, placement of shoes with extended toes may be advantageous; however, while the flexural deformity at the coffin joint may be resolved, it is possible that a deformity could develop at the fetlock. Ulcer prophylaxis is recommended in foals receiving NSAIDs.

Oxytetracycline is commonly used for the treatment of congenital flexural deformities. In normal foals, oxytetracycline has a transient effect on the fetlock joint angle and, until recently, the mechanism of action was attributed to a possible effect on intracellular calcium. Recent investigations confirming the presence of myofibroblasts in both the inferior check ligament and the DDF tendon provide support for the concept that chelation of calcium may produce some relaxation (lengthening) of the ligament and tendon.³ Interstitial collagenase [matrix metalloproteinase-1 (MMP-1)] is involved in collagen remodeling. Investigators have confirmed that oxytetracycline inhibits tractional structuring of collagen fibrils by myofibroblasts through an MMP-1-mediated mechanism,⁴ possibly making developing ligaments and tendons more susceptible to elongation during weight-bearing. This inhibition of normal collagen organization may provide an explanation for the benefits of oxytetracycline in foals with congenital flexural deformities. Some clinicians use oxytetracycline for nonsurgical treatment of acquired flexural deformities at the coffin joint; however, published reports on the efficacy of this treatment are lacking. The author has not found oxytetracycline useful in the treatment of acquired flexural deformities at either the fetlock or coffin joints.

Flexural deformities at the coffin joint that have progressed to the point of having the heels in contact with the ground (ie, the classic clubfoot; Figure 2) require surgical management. Ideally, surgery should not be performed until the pain that precipitated the condition has resolved. Excel-

lent results can be expected with inferior check ligament desmotomy in foals with uncomplicated mild-to-moderate deformities.⁵ Surgical management of the older clubfooted horse (Stage I) can be especially rewarding if treatment is undertaken before lameness is present. The prognosis for a lame horse with chronic clubfoot is uncertain; nevertheless, the author has been impressed with the positive response to check ligament desmotomy in some of these horses. Following desmotomy, weight-bearing on the foot returns to a normal angle. However, although the contracted foot will expand after surgery, it is unlikely to regain its normal size. Horses with severe deformities can have athletic careers with DDF tenotomy, but the prognosis is guarded.⁶

Inferior check ligament desmotomy

The inferior check ligament can be approached from the medial or lateral aspect of the limb. The author prefers the medial approach for 3 reasons.

- The medial common digital artery is in full view and can be avoided by choice rather than by chance, as in the case of the lateral approach.
- The check ligament is easier to define on the medial side.
- The post-surgical blemish that develops in some cases is less noticeable on the inside of the leg.

The surgical landmarks for the skin incision are: the head of metacarpal (MC) 2, proximally; the medial common digital vein, dorsally; and the deep digital flexor tendon, palmarly. A 6-cm skin incision is made, beginning at the level of the head of MC2 and extending distally. Hemorrhage is controlled and scissors are used to dissect through the remaining subcutaneous tissue to expose the subcarpal fascia. A scalpel is used to make a stab incision through the subcarpal fascia immediately palmar to the medial common digital vein. The incision in the subcarpal fascia is extended to the proximal and distal limits of the skin incision. The subcarpal fascia is retracted and blunt dissection is used to separate the inferior check ligament from the suspensory ligament. The neurovascular bundle, consisting of the medial palmar nerve and medial common digital artery, are identified and reflected in a palmar direction to expose the tissue plane between the check ligament and the DDF tendon. The check ligament is separated from the tendon, isolated, and elevated using curved scissors. The coffin joint is extended and the check ligament is sharply transected. The subcarpal fascia and subcutaneous tissue layers are closed separately, using an absorbable suture in a simple continuous pattern. The skin is apposed using simple interrupted sutures.

Aftercare includes a pressure bandage for a minimum of 14 days. Bandaging the limb until 30 days postoperatively may enhance the cosmetic appearance. Exercise is restricted to handwalking until the sutures are removed 10 days after surgery. Corrective trimming and shoeing (if required) should be performed immediately after recovery from

Figure 4: Mild fetlock flexural deformity with abnormal upright pasterns



anesthesia. A normal coffin joint angle must be attained no later than a week after surgery. Further corrective hoof care is rarely required beyond 3 to 4 months. Nutritional management to limit energy intake is indicated for a period of 2 to 3 months.

Deep digital flexor tenotomy

Tenotomy of the DDF tendon can be performed at the level of the mid-metacarpus or distal to the fetlock. Some surgeons prefer the mid-metacarpal approach because it can be performed in the standing horse. Unfortunately, horses undergoing mid-metacarpal tenotomy experience a higher level of discomfort during the postoperative period due to the stretching and tearing of peritendinous tissues that must take place to allow the joint to return to a normal angle. The author prefers tenotomy distal to the fetlock joint. A 6-cm palmar midline skin incision is carried through the palmar retinaculum into the digital tendon sheath. The tendon is elevated from the incision and transected. Closure is routine and bandage support is required for 10 days after surgery.

Postoperative management should include corrective trimming or shoeing to establish a normal coffin joint angle within the first 2 weeks after surgery. Ongoing corrective hoof care may be needed for a period of up to 6 months. While transection of the DDF tendons in normal horses results in the toe tipping up, this does not occur in horses with severe flexural deformities at the coffin joint. NSAIDs are indicated in the immediate postoperative period.

Flexural deformities at the fetlock

Acquired flexural deformities at the fetlock generally develop between 10 and 18 months of age. Flexion

Figure 5: Moderate fetlock flexural deformity: angle becomes almost normal in motion



at the fetlock joint is affected by the superficial flexor muscle; however, contraction of the DDF muscle can also play a role through its lifting action as it passes palmar to the proximal sesamoid bones. Individually, or together, contraction of the digital flexors results in varying degrees of abnormal flexion at the fetlock. Persistent muscle contraction eventually results in contracture with likely involvement of the accessory ligaments of both superficial and DDF muscles, the suspensory ligament, and the joint capsule of the fetlock.

Classification

Flexural deformities at the fetlock have been classified based on severity.⁷

- Mildly affected horses have abnormal upright pasterns (Figure 4).
- Moderately affected horses exhibit dorsal flexion while standing, but the fetlock angle increases toward normal when the horse is in motion (Figure 5).
- The fetlock joint is flexed at all times in severely affected horses (Figure 6).

Horses with flexural deformities at the fetlock are predisposed to degenerative joint disease that reduces their prognosis.

Flexural deformities at the fetlock are readily apparent on physical examination and the degree of severity is assessed with the horse at rest and in motion. Concomitant involvement at the coffin joint is rare and can be ruled-out by the observation of a normal hoof and a normal hoof angle (coffin joint angle). Interestingly, the deformity is often worsened by well-intentioned corrective trimming that involves lowering the heels. Radiographic evaluation should

Figure 6: Severe fetlock flexural deformity: joint is always flexed



be performed to look for evidence of degenerative changes in the fetlock joint.

Treatment

Early intervention is directed toward relieving the pain that is responsible for the deformity. Mild and moderately affected horses may also benefit from energy restriction and corrective trimming and shoeing in the form of heel elevation and toe extension. Elevating the heels reduces the contribution of the DDF muscle to fetlock support. This places more strain on the superficial flexor tendon check ligament unit and the suspensory apparatus and possibly promotes adaptive remodeling. An extension of the toe delays breakover and further stretches the flexor support structures. If this non-surgical management results in a positive response, usually, the hoof angle can be gradually returned to normal beginning 4 to 6 months after the onset of the condition. Horses that fail to respond within 6 to 8 weeks are candidates for surgical treatment. The prognosis for mild and moderately affected horses is good in the absence of degenerative joint disease. The prognosis for severely affected horses is poor and treatment is directed toward salvage.

Surgical management options for management of mild and moderate flexural deformities at the fetlock include: inferior check ligament desmotomy, superior check ligament desmotomy, or both. Although many clinicians recommend nonsurgical management for mildly affected horses, the author believes these horses are the best candidates for surgery and can be returned to normal with an inferior check ligament desmotomy. Moderately affected horses are best managed by performing both an inferior and a superior check ligament desmotomy. In the immediate postoperative

period, the heels should be elevated by approximately 5° and an extended toe shoe should be placed. A gradual return to a normal hoof angle can be started 3 months after surgery. Horses with severe deformities rarely respond to check ligament desmotomies. In these cases, a superficial digital flexor tenotomy, a suspensory ligament desmotomy, a tenotomy of the DDF tendon and, possibly, surgical manipulation of the joint capsule can be employed. The goal of treatment is salvage, eg, for use as a brood mare; however, failure to resolve the deformity is common.

Superior check ligament desmotomy

Superior check ligament desmotomy is technically more demanding than desmotomy of the inferior check ligament. The ligament is approached from the medial aspect of the limb just proximal to the carpus. Early descriptions involved transecting the ligament immediately adjacent to the caudal surface of the radius.⁸ This approach was consistently complicated by the indistinct nature of the ligament at this location and the vessels of the supracarpal rete. Bramlage modified the original technique by moving the incision caudally, allowing the ligament to be more easily isolated through the tendon sheath of the flexor carpi radialis muscle.⁹

Summary

Acquired flexural deformities develop in foals and juvenile horses secondary to persistent painful conditions of the limbs. Foals <4 months of age develop deformities at the coffin joint, while the deformity occurs at the fetlock in older animals. A good-to-excellent outcome is achieved with inferior check ligament desmotomy in mild to moderately severe clubfooted foals. Inferior and superior check ligament desmotomy can achieve satisfactory results in mild to moderately severe deformities at the fetlock in juvenile animals. The prognosis for severe deformities at the coffin joint is guarded and the prognosis for severe deformities at the fetlock is grave.

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Dr. David G. Wilson is a large animal surgeon in the Department of Large Animal Clinical Sciences at the Western College of Veterinary Medicine. Dr. Wilson's research interests include developmental orthopedic diseases in horses and biomechanics of orthopedic fixation.

Abstracts of Interest

Glycogen branching enzyme deficiency in Quarter horse foals.

VALBERG SJ, WARD TL, RUSH B, KINDE H, HIRARAGI H, NAHEY D, FYFE J, MICKELSON JR. ST PAUL, MINNESOTA
Seven related Quarter Horse foals that died by 7 weeks of age were examined for glycogen branching enzyme (GBE) deficiency. Clinical signs varied from stillbirth, transient flexural limb deformities, seizures, and respiratory or cardiac failure to persistent recumbency. Leukopenia (5 of 5 foals) as well as high serum creatine kinase (CK; 5 of 5), aspartate transaminase (AST; 4 of 4), and gamma glutamyl transferase (GGT; 5 of 5) activities were present in most foals, and intermittent hypoglycemia was present in 2 foals. Gross postmortem lesions were minor, except for pulmonary edema in 2 foals. Muscle, heart, or liver samples from the foals contained abnormal periodic acid Schiff's (PAS)-positive globular or crystalline intracellular inclusions in amounts proportional to the foal's age at death. Accumulation of an unbranched polysaccharide in tissues was suggested by a shift in the iodine absorption spectra of polysaccharide isolated from the liver and muscle of affected foals. Skeletal muscle total polysaccharide concentrations were reduced by 30%, but liver and cardiac muscle glycogen concentrations were normal. Several glycolytic enzyme activities were normal, whereas GBE activity was virtually absent in cardiac and skeletal muscle, as well as in liver and peripheral blood cells of affected foals. GBE activities in peripheral blood cells of dams of affected foals and several of their half-siblings or full siblings were approximately 50% of controls. GBE protein in liver determined by Western blot was markedly reduced to absent in affected foals, and in a half-sibling of an affected foal, it was approximately one-half the amount of normal controls. Pedigree analysis also supported an autosomal recessive mode of inheritance. The affected foals have at least 2,600 half-siblings. Consequently, GBE deficiency may be a common cause of neonatal mortality in Quarter Horses that is obscured by the variety of clinical signs that resemble other equine neonatal diseases.

J Vet Intern Med 2001;15(6):572-80.

Acquired flexural deformity of the metacarpophalangeal joint in five horses associated with tendonous damage in the palmar metacarpus

MCDIARMID A. ROSLIN, MIDLOTHIAN.

Five cases of acquired flexural deformity of the metacarpophalangeal joint (MCPJ) in older horses and ponies were studied. The mean age of affected horses was 14-8 years. Four deformities developed following desmitis of the accessory ligament of the deep digital flexor tendon (ALDDFT) and superficial digital flexor tendon (SDFT) and one following tendonitis of the SDFT alone. All cases were markedly lame and demonstrated variable degrees of flexural deformity. Ultrasonographic examination was performed on all cases, which revealed extensive adhesion formation between the ALDDFT and SDFT and reduced cross-sectional area of the deep digital flexor tendon in each case. A variety of treatments was unsuccessfully employed to treat this condition. The prognosis for acquired flexural deformity of the MCPJ in old horses following tendonous damage in the palmar metacarpus is likely to be poor.

Vet Rec 1999;144(17):475-8.

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