

# Satellite Article

## Flexural deformities in foals

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### Introduction

Flexural deformities are a common condition of growing horses in which a joint is held in an abnormally flexed position. They affect soft tissue structures and occur in the sagittal plane, as opposed to angular limb deformities which primarily affect osseous structures and occur in the frontal plane, although periarticular soft tissue laxity can also cause angular limb deformities in newborn foals (Hunt 1998). Tendon laxities also occur commonly in newborn foals (for further information see box on *Flexor tendon laxity* later in this article).

Flexural deformities are often referred to as '**contracted tendons**', which implies a defect in the tendon itself and is **incorrect in view of the proposed pathogenesis** (see below); this term should consequently be avoided. Tendon contractions can occur as a result of scarring secondary to tendon injury; this is sometimes seen in mature horses but is rare in foals (Fackelman 1984).

### Classification

Flexural deformities can be classified as **congenital** (present at or soon after birth) or **acquired** (develops after birth during the growth period) or by the joint affected.

**Congenital** flexural deformities most commonly affect the metacarpophalangeal joint or the carpus. Rarely, the tarsus, metatarsophalangeal joint, and distal and proximal interphalangeal joint are affected. **Acquired** flexural deformities affect the distal interphalangeal joint and metacarpophalangeal joints most frequently, with the metatarsophalangeal and proximal interphalangeal joints less frequently affected. Acquired flexural deformities can be further subdivided into those which affect foals between ages 6 weeks and 6 months, yearlings between 9 and 18 months, and mature horses (Munroe and Marr 1989).

### Congenital flexural deformities

#### *Incidence*

Crowe and Swerczek (1985) reported that 20% of 608 fetuses and newborn foals submitted for necropsy had 'miscellaneous limb contractions'. The incidence in the general population of live foals is unquantified, although it is said to be increasing (Knight *et al.* 1985; Bramlage 1987).

#### *Aetiopathogenesis*

Reported causes of congenital flexural deformities include **intrauterine malpositioning** (Rooney 1966), **teratogenic effects** from ingestion of locoweed (McIlwraith and James 1982) and hybrid Sudan grass (Pritchard and Voss 1967) by the mare during gestation, a **dominant gene mutation** in a stallion (Hutt 1968), **goitre** (McLaughlin and Doige 1981), a **neuromuscular disorder** (Mayhew 1984) and an **influenza outbreak** (Fessler 1977). However, the precise cause of the majority of flexural deformities is unknown.

#### *Diagnosis and clinical signs of congenital flexural deformities*

Congenital flexural deformities may cause dystocia if severe enough (Juzwiak *et al.* 1990), or the owner may report that the foal is unable to stand. Some flexural deformities may be overlooked in recumbent foals if the joints can be straightened manually, but should become apparent once the foal is assisted to stand. Joints should be manipulated to ascertain whether manual straightening is possible. The fetlock is the most common congenitally affected joint (Fackelman 1980), followed by the carpus.

#### *Congenital deformities of the metacarpal/tarsophalangeal joint (Fig 1)*

Mild cases of fetlock flexural deformities usually resolve

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**Fig 1: A congenital deformity of the metatarsophalangeal joint.**



**Fig 2: A congenital flexural deformity of the carpus.**

spontaneously in 4 or 5 days with limited exercise. Cases which do not respond to conservative therapy can be splinted (see Kidd 2002 in this issue) and/or treated medically. Severely affected cases may require surgical intervention. One author reports that, if there are no osseous changes to the joint or the joint can be manually straightened, the prognosis is good for all degrees of severity of metacarpo/tarsophalangeal flexural deformities (Wagner von Matthiessen 1994).

#### *Congenital flexural deformities of the carpus (Fig 2)*

Carpal flexural deformities are usually bilateral and, in mild cases, the foal can stand but cannot completely straighten the carpi. Mild cases are usually self-correcting in 4 or 5 days with limited exercise but, if the deformity does not resolve spontaneously, other treatments may be instituted (see below). More severely



**Fig 3: A foal wearing splints for bilateral carpal flexural deformities. The foal required assistance to stand.**

affected cases may be unable to stand and the common digital extensor tendon may rupture secondary to the flexural deformity (see box below). If the limbs can be straightened manually (which may require sedation), the chance of resolution using splints or casts is good but the prognosis is guarded if the carpus cannot be straightened manually (Wagner *et al.* 1982; Wagner von Matthiessen 1994). If the deformity is very severe, surgical transection of the *flexor carpi ulnaris* and *ulnaris lateralis* should be considered, but **radiographs should be taken first to rule out abnormally formed carpal bones which decrease the prognosis to poor** (Wagner von Matthiessen 1994, see 'Treatment' section below).

#### **Rupture of the common digital extensor tendon**

This is a common congenital condition of foals which can be confused with carpal flexural deformities because the foal buckles forwards at the carpus. The tendons may rupture secondary to a carpal flexural deformity or due to fatigue while attempting to keep up with an exercising mare. The condition can be distinguished from carpal flexural deformities by the soft, fluctuant swelling in the tendon sheath on the dorsolateral aspect of the carpus. Often, the free ends of the tendon can be palpated within the sheath. The condition responds well to rest and some authors recommend immobilisation in splints or casts for 2 to 3 weeks. The prognosis is good.

#### *Congenital flexural deformities of the proximal interphalangeal joint*

Congenital pastern joint flexural deformities are rarely reported and often involve both osseous and soft tissue abnormalities (Wagner von Matthiessen 1994). Cases may show subluxation of the pastern joint or ankylosis of this joint in more severe cases. Subluxations may be treated using a dynamic compression plate to achieve arthrodesis of the joint.



**Fig 4:** Flexor tendon laxity occurs commonly in newborn foals, but is usually self-correcting with limited exercise.



**Fig 5a:** A stage I distal interphalangeal joint flexural deformity in which the dorsal hoof wall is vertical.



**Fig 5b:** A stage II distal interphalangeal joint flexural deformity in which the dorsal hoof wall has passed vertical.

## Radiography

**Radiography is not required for the diagnosis of flexural deformities, which can be made on the basis of clinical signs.** However, radiography is useful to identify abnormalities which may alter the prognosis for correction of the deformity. Radiographs of **carpal flexural deformities** which cannot be manually straightened may reveal **incomplete ossification** or **wedging of the carpal bones** which decreases the prognosis for correction. The same applies for the less common tarsal flexural deformities.

## Treatment of congenital flexural deformities

### Exercise

Congenital flexural deformities are best treated with moderate exercise. In many cases, the foal is unwilling to stand and walk



**Fig 6a:** An acquired metacarpophalangeal flexural deformity. The foot appears normal and is in contact with the ground but the pastern assumes a more upright position.



**Fig 6b:** A more severe fetlock flexural deformity in which the fetlock is knuckling forward.

and needs regular encouragement to do so (**Fig 3**), especially if a cast or a splint is applied. The treatments discussed below can then be used singly or in combination.

### Analgesics

**Both the primary cause and the effects of treatment of flexural deformities may be painful.** Nonsteroidal anti-inflammatory drugs (NSAIDs) have a role in the treatment of flexural deformities but should be **used judiciously** in young animals due to the potential side effects of gastroduodenal ulceration and nephrotoxicity. One author (JAK) routinely uses flunixin meglumine at a dose of 1.1 mg/kg bwt i.v. once daily and concurrent treatment with ranitidine (6.6 mg/kg bwt *per os* t.i.d. or 1.5 mg/kg bwt i.v. t.i.d.) as a gastric protectant. Other suitable analgesics include phenylbutazone, and other gastric protectants include sucralfate and cimetidine. If there is any concern about the foal's systemic health, consider biochemical monitoring of total protein, BUN and creatinine as a minimum data base.

### Tetracycline

Intravenous oxytetracycline has been used in the treatment of flexural deformities in foals. Although the method of action has not been completely elucidated, it is thought to act by chelation of calcium and inhibition of muscle contraction. Madison *et al.* (1992) examined the effect of a single 44 mg/kg bwt dose of i.v. oxytetracycline in normal foals and foals with flexural deformities. Treatment with oxytetracycline resulted in a significant decrease in the metacarpophalangeal joint angle as measured radiographically in both affected and unaffected foals. **Joints returned to their pretreatment angles by 4 days after treatment.** Oxytetracycline therapy had no significant effect on the distal interphalangeal joint angle and is **unlikely to be of use for flexural deformities of the distal interphalangeal joint.** No alterations in renal biochemical parameters were detected after a single dose. A dose of 3 g s.i.d. i.v. for 2 or 3 days has been recommended (Lokai 1992).

### Splints/casts

Splints and casts are useful in cases of metacarpo/tarsophalangeal, carpal and some hock flexural deformities. The proximal interphalangeal joint is difficult to immobilise. A method for splinting congenital flexural deformities is described in the related case report in this issue (Kidd 2002).

**Splints have the advantage over casts that they can be removed regularly and easily reapplied and can be applied with or without sedation.** Casts cannot be reset regularly (unless they have been bivalved) and do not allow inspection of the flexural deformity and the skin under the cast. **Because the forced extension of the affected limb in a cast or a splint is painful and often requires analgesia, one author (JAK) does not use casts, preferring instead to use splints which can be placed on the leg for 12 h and then left off for 12 h.**

**Splints** can be made from a variety of materials including PVC pipe, wood and fibreglass. Splints should be placed over sufficient padding that the skin is protected from excoriation, but little enough that the splints do not shift out of position. The splints will often rotate around the leg and it is often quite difficult to keep them in the desired position. Strong tape or duct tape can be used, as can splints that have been made to the contour of the limb as described in the case report (Kidd 2002).

Some foals require sedation to apply the splints to allow maximal extension of the affected joint and to allow the splints to be placed on the limb properly. Alpha-2 agonists, such as xylazine or detomidine, are useful for this purpose and have the added benefit of providing analgesia.

### Surgery

Surgery should be considered as the initial treatment for severe cases of flexural deformities and for those cases which do not respond to conservative or medical treatment. In general, congenital flexural deformities which require surgical treatment have a worse prognosis than acquired flexural deformities treated surgically (Stashak 1987), and the owners should be informed accordingly.

### Deformities of the carpus

Transection of the *flexor carpi ulnaris* and *ulnaris lateralis* has reportedly been used successfully for mild cases of carpal flexural deformities (Gerring 1989), but the literature is lacking in clinical studies; most reports are unreferenced or quote unpublished data and the surgery is, therefore, of questionable efficacy. The surgery is performed under general anaesthesia and a vertical incision is made over the lateral aspect of the accessory carpal bone and the tendons identified deep to the fascia. If these tendons are the structures preventing extension of the carpus, once they have been transacted surgically the limb can be straightened manually. After surgery, box confinement is recommended for a few days and then access to a small paddock or yard is allowed. The limb is kept bandaged until the sutures are removed.

### Flexor tendon laxity

Flexor tendon laxity (**Fig 4**), the 'opposite' of flexural deformities, occurs commonly in newborn foals. The hindlimbs are affected most frequently but the condition can affect all 4 limbs. It is commonly seen in premature or dysmature foals and is usually self-correcting with limited exercise. Foals with flexor tendon laxity bear little weight on their toes and most of their weight on their heels. Severely affected foals walk on their plantar and palmar pasterns and can excoriate their skin. In these cases, plantar heel extensions of a light material such as thin aluminium can give support to the flexor tendons. Heel extensions are probably better glued onto the foot to avoid having to nail into small feet with thin walls. Protective bandages may be needed to protect the skin but should not be substantial enough to overly support the tendons.

## Acquired flexural deformities

### Aetiopathogenesis

Acquired flexural deformities are often quoted as part of the developmental orthopaedic disease (DOD) complex which also includes angular limb deformities, osteochondrosis, phytitis and cervical vertebral malarticulation/malformations and was first described by Bramlage (1987). Congenital flexural deformities are not included in the DOD complex.

The aetiopathogenesis of acquired flexural deformities is probably multifactorial and complex, but several theories have been proposed for their occurrence. The 2 main theories are a) **a mismatch in bone and tendon/ligament growth** and b) **contraction of the musculotendinous unit in response to pain**.

### Rapid bone growth

Rapid bone growth and, especially, growth spurts, have been proposed as a cause for flexural deformities by leading to a mismatch in the tendon/bone length. The rate of bone growth is determined by genetics and nutrition, and overfeeding of foals can occur either by heavily lactating mares or excessive supplementation with concentrates (Owen 1975) or improved nutrition following a low plane of nutrition (Wagner *et al.* 1982). It has been proposed that the bones grow at a faster rate than the associated tendons and ligaments and lead to a flexural deformity of the joint flexed by the musculotendinous unit in question (Owen 1975; Metcalf *et al.* 1982).

In the forelimb, the inferior check ligament (accessory ligament of the deep digital flexor tendon) originates on the palmar carpus and inserts on the deep digital flexor tendon at the junction of the top and middle thirds of the metacarpus. The superior check ligament (accessory ligament of the superficial digital flexor tendon) originates from the caudomedial distal radius and inserts into the dorsal superficial digital flexor tendon at the distal radius and carpus.

If this pathogenesis is correct, one would expect that flexural deformities would occur at the same time that regional bone growth was occurring; in other words, distal interphalangeal flexural deformities which occur between ages 1 and 6 months would be the result of a growth spurt distal to the midmetacarpal region during this time. However,

bone growth in this region is essentially complete by age 2 months. Maximal growth of the distal radial growth plate occurs at approximately age 1 year (Auer 1992), so flexural deformities of the metacarpo/tarsophalangeal joint would be expected to have occurred by this age.

Longitudinal bone growth is insufficient at any age to create a relative shortening of the flexor tendons; therefore, while rapid bone growth may contribute to orthopaedic pain, it cannot contribute to flexural deformities. Other arguments against this hypothesis are that many acquired flexural deformities have an acute onset of 24 to 48 h and, while muscle could respond in this period, lengthening of bone would take longer than this. Also, affected foals with distal interphalangeal joint flexural deformities often do not show disproportionate growth in the affected limb but appear to be growing normally.

### Pain

Any painful condition, such as subsolar abscessation, laminitis (Adams 1976), bruised feet due to exercise on a hard surface (McGladdery 1992), decreased weightbearing due to a fracture in the contralateral limb (Turner 1986), phytitis (Fessler 1977), septic arthritis, or osteochondrosis, **can cause pain and contraction of the muscular portion of the musculotendinous unit**, as the tendons and check ligaments have a limited ability to contract. Phytitis is commonly observed in foals with flexural deformities and may be an inciting painful stimulus. Although pain may be an instigating factor in acute onset flexural deformities, more permanent states of flexion of the musculotendinous unit may occur with contracture of the flexor aspect of the joint capsule and maintain the deformity. This underscores the need for early diagnosis and treatment. Support for the secondary and permanent states of flexion are supported by foals who have flexural deformities which cannot be straightened manually even under general anaesthetic (Wyn-Jones *et al.* 1985).

### Diagnosis and clinical signs of acquired flexural deformities

The onset of acquired deformities can be divided into 2 periods of the foal's growth (**Table 1**). Early diagnosis is important to increase the chance of complete resolution but diagnosis may be made difficult by the foal being out at pasture with the mare, especially for DIPJ deformities which may be hidden by the grass. Regular examination on a hard level surface will aid early detection of acquired deformities. The metacarpophalangeal and distal interphalangeal joints are most commonly affected.

### Acquired flexural deformities of the distal interphalangeal joint (DIPJ)

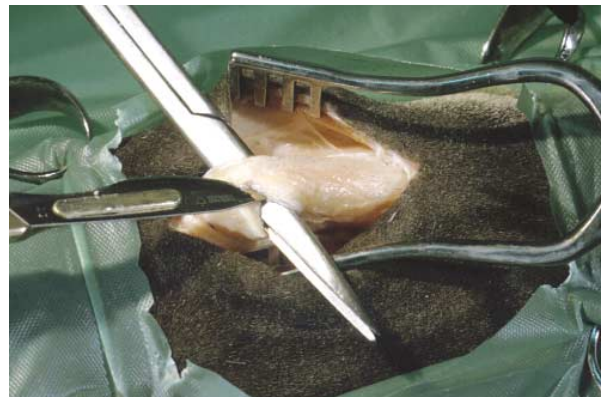
Flexural deformities of the DIPJ occur primarily in foals between ages 1 and 6 months and almost always affect the forelimbs. The condition is usually bilateral, although one limb may be more severely affected. Deformities of the distal interphalangeal joint involve the deep digital flexor tendon because this tendon

**TABLE 1: Age of onset of common flexural deformities**

Congenital	
Birth up to one month	Carpus
	Metacarpophalangeal joint
Less commonly	Metatarsophalangeal joint
	Proximal interphalangeal joint
Rarely	Tarsus
	Radiohumeral joint
Acquired	
1–6 months	Distal interphalangeal joint
Less commonly	Metatarsophalangeal joint
	Proximal interphalangeal joint
10–18 months	Metacarpophalangeal joint



**Fig 7a:** The procedure for inferior check ligament desmotomy (illustrated on a cadaver). A 5 cm vertical skin incision is made, centred at the junction of the proximal and middle thirds of the metacarpus, over the dorsal border of the deep digital flexor tendon. The underlying fascia and paratenon are incised to expose the flexor tendons. In the case of a medial approach, the neurovascular bundle is retracted palmarly.



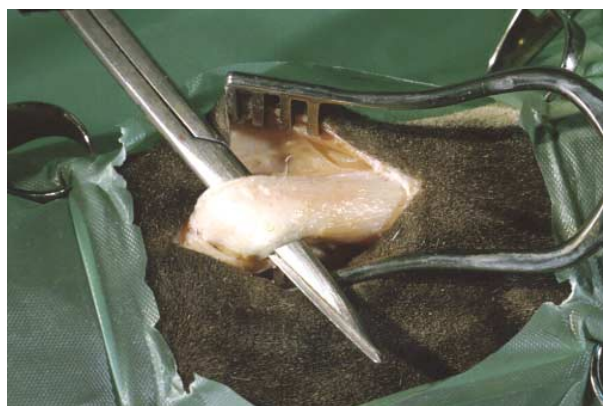
**Fig 7d:** The inferior check ligament is then elevated again and sectioned transversely using a scalpel. It is not necessary to remove a section of the ligament.



**Fig 7b:** The plane of cleavage between the deep digital flexor tendon and inferior check ligament is identified by palpation and the 2 structures are separated by a combination of sharp and blunt dissection. The division between the 2 is most easily appreciated proximally and becomes progressively harder to distinguish in the midmetacarpus.



**Fig 7e:** If the distal interphalangeal joint is extended, a gap of about 1 cm usually appears between the severed ends of the ligament. The paratenon, fascia, subcutaneous tissues and skin are then closed and a padded bandage placed around the limb distal to the carpus. The bandage is changed every 2 or 3 days and the skin sutures removed after 10 days.



**Fig 7c:** The inferior check ligament is isolated and elevated from the incision by passing a pair of curved forceps beneath it, checking that no part of the neurovascular bundle from the opposite side of the limb has been included in the tissues elevated. It is prudent to double-check the identity of the inferior check ligament at this stage by allowing it to fall back into its normal position and positively identifying the superficial digital flexor tendon, deep digital flexor tendon, inferior check ligament and suspensory ligament.

inserts on the solar surface of the third phalanx and is responsible for flexion of the DIP joint. Initially, the dorsal hoof wall assumes a more vertical angle and the heels may not contact the ground if the condition has occurred acutely. With the foot in this conformation, 2 sequelae result. First, the heels overgrow due to the lack of ground contact and the foot appears 'boxy' as the heels approach the length of the toe. Secondly, the toe is under greater stress and wear which can widen the white line and lead to flaring of the distal hoof wall. These changes in the conformation of the foot are a result of the deformity and not a cause. The deformities are divided into **stages I and II**; stage I deformities (**Fig 5a**) have a more upright dorsal hoof wall but the wall has not passed vertical and in stage II deformities (**Fig 5b**) the dorsal hoof wall is beyond the vertical. Stage II deformities have a worse prognosis for resolution than stage I (McIlwraith and Fessler 1978) and the prognosis declines with the length of time prior to treatment (Wagner *et al.* 1982). **This classification scheme is useful for description of the deformity and formulation of a prognosis, but does not always dictate the preferred method of treatment.**



**Fig 8a:** Procedure for superior check ligament desmotomy (illustrated on a cadaver). A 10 cm vertical skin incision is made, centred at the level of the chestnut, between the cephalic vein caudally and the caudomedial border of the distal radius.



**Fig 8b:** A branch of the cephalic vein penetrates the antebrachial fascia through a small foramen close to the caudomedial border of the distal radius at the level of the chestnut. This is identified, double-ligated and sectioned, allowing caudal retraction of the cephalic vein.



**Fig 8c:** The antebrachial fascia is incised vertically approximately 1 cm caudal to the radius, revealing the flexor carpi radialis muscle which is retracted caudally. This exposes the superior check ligament running from the caudal radius caudodistally across the surgical field. The ligament is progressively sectioned with scissors working from the proximal margin distally. At its distal extremity, the ligament becomes thinner and is intimately associated with the proximal extremity of the carpal sheath. Inadvertent penetration of the sheath while sectioning the ligament does not seem to have serious sequelae. The antebrachial fascia, subcutaneous tissue and skin are sutured routinely and a padded bandage applied from the fetlock to proximal antebrachium.

### *Acquired flexural deformities of the metacarpol/tarsophalangeal joint (MCPJ/MTPJ)*

These flexural deformities can be congenital but are also an acquired flexural deformity in animals from ages 10 to 18 months; some authors believe these deformities result from rapid growth of the distal radial growth plate at this time (Fackelman 1980), although this aetiopathogenesis is less favoured currently. Both front and hindlimbs can be affected, but acquired metatarsophalangeal joint flexural deformities are much less common than those affecting the metacarpophalangeal joint (Wagner von Matthiessen 1993). Acquired metacarpophalangeal flexural deformities have also recently been described in mature horses secondary to desmitis of the accessory ligament of the deep digital flexor tendon (ALDDFT) or inferior check ligament (McDiarmid 1999).

The foot usually appears normal and is in contact with the ground but the pastern assumes a more upright position (Fig 6a) and, if left untreated, the fetlock knuckles forward (Fig 6b). Deformities of the metacarpol/tarsophalangeal joint can involve one or both of the deep and superficial digital flexor tendons as these structures both support the palmar/plantar surface of the fetlock joint. **The choice of surgical treatment**, should surgery be required, is affected by which tendon is considered to be involved in the deformity. Identification of the primarily affected tendon is not always straightforward.

Cases detected and treated early carry a good prognosis for correction with conservative treatment (McIlwraith 1982; Wagner von Matthiessen 1993). Flecker (1986) reported that mild cases requiring corrective shoeing only had a good prognosis for resolution. Cases requiring surgical intervention carried a guarded (Flecker and Wagner 1986) to poor (Wagner *et al.* 1982) prognosis.

### *Acquired flexural deformities of the proximal interphalangeal joint*

This condition is seen primarily in the hindlimbs of rapidly growing weanlings (Wagner von Matthiessen 1993) and is usually bilateral. It occurs in horses with a straight hindlimb conformation in the same time period as acquired flexural deformities of the distal interphalangeal joint. The biomechanics of the condition are thought to be a shortening of the deep digital flexor musculotendinous unit and a concurrent laxity in the superficial digital flexor tendon (Shiroma 1989), which inserts adjacent to the pastern joint. A dorsal subluxation of the joint is usually present and is accompanied by an audible click as the horse walks. Radiographs may show osteoarthritis of the pastern joint in longstanding cases. Affected horses may respond to exercise restriction and analgesics but in cases unresponsive to medical treatment, surgical transection of the distal check ligament (if present) and the tendon of the medial head of the deep digital flexor tendon at the level of the chestnut (Shiroma 1989) may be performed. Three horses treated surgically with this

method had resolution of the subluxation and the one horse for whom follow-up information was available was sound after 10 months (Shiroma 1989).

## Radiography

As for congenital flexural deformities, radiography is not required for the diagnosis of acquired flexural deformities, which can be made on the basis of clinical signs. However, radiography is useful to identify abnormalities which may alter the prognosis for correction of the deformity. Secondary radiographic changes can occur as a consequence of the deformity and in relation to the distal interphalangeal joint, including modelling of the dorsodistal aspect of the third phalanx, rotation of the third phalanx in the hoof capsule, or osteoarthritis of the distal interphalangeal joint, and worsen the prognosis (Wagner *et al.* 1982). Metacarpo/tarsophalangeal joint flexural deformities may show evidence of osteoarthritis in the metacarpo/tarsophalangeal or proximal interphalangeal joints secondary to chronic subluxation (Blackwell 1980).

## Treatment of acquired flexural deformities

Early recognition and treatment of flexural deformities improves the prognosis, especially if pain-mediated flexion is involved, as these will become worse with time if left untreated. If an underlying cause of the pain can be identified, it should be addressed. Medical or conservative treatment is indicated as initial treatment for all but severely affected cases. If conservative treatment does not improve the flexural deformity, surgery should be considered. Surgery should be considered as the initial treatment for severely affected cases.

## Nutrition

Nutrition, along with genetics, controls the growth rate of the foal, and overfeeding of foals can occur via either heavily lactating mares or excessive supplementation with concentrates. Alterations in the growth rate may also occur when the nutritional plane increases suddenly, especially after a period of relative deficiency such as after weaning or after a change of ownership. **When a flexural deformity occurs in an unweaned foal**, the energy content of the diet should be reduced either by early weaning of the foal or a decrease in the mare's concentrate ration. The mineral balance of the ration for both mare and foal should also be balanced with respect primarily to calcium and phosphorus, as research has shown that calcium/phosphorus imbalances are also implicated in developmental diseases (Knight *et al.* 1985).

Older foals and yearlings should have the concentrate portion of the diet reduced to a minimum and be fed hay and a balanced mineral supplement. Presumably, if contraction of the musculotendinous unit in response to pain is the more accepted aetiopathogenesis for acquired flexural deformities, then nutritional alteration is actually aimed at the underlying

cause of the pain. **Prevention should be aimed at monitoring growth and weight and adjustment of the diet accordingly.**

## Exercise

**Opinions differ on the role of exercise in the treatment of flexural deformities.** We consider that, if the deformity is secondary to a painful condition, then exercise limitation and analgesics would be of benefit. Uncontrolled exercise may exacerbate the painful stimuli and deleterious loading of the contralateral limb.

Distal interphalangeal joint flexural deformities, in which the foal is bearing weight on the toe, generally benefit from controlled exercise on a firm surface, combined with analgesics, to allow stretching of the deep digital flexor musculotendinous unit. Turnout in a small yard that is sufficiently small to prevent uncontrolled exercise is probably a reasonable choice.

More aggressive physical therapy may be required in cases of flexural deformities of the fetlock. Enforced handwalking, walking up and down slopes, and 'hopping' by holding up the unaffected leg and necessitating weightbearing on the affected limb have all been described (Fackelman 1980). Again, the use of analgesics may be useful.

## Analgesics

See '*Treatment of congenital flexural deformities*'.

## Corrective trimming

In cases of distal interphalangeal flexural deformities, the heel overgrows and gives the typical 'boxy' conformation to the foot. The excessive heel may prevent proper realignment of the hoof-pastern axis if the heel contacts the ground and in these cases should be rasped back gradually. If the heel has been lifted off the ground by the deformity, then the heels should not be rasped as this will only apply greater forces and leverage to the dorsal laminae and the distodorsal third phalanx.

## Corrective shoeing

### *Distal interphalangeal flexural deformities*

Used in conjunction with corrective trimming and exercise, toe extensions can be used to increase the strain in the deep digital flexor tendon. **Toe extensions which are glued to the foot** have the advantages of not restricting foot expansion or growth, avoiding nailing into small feet with thin walls and can be made from lightweight materials such as plastic. Toe extensions should not be overlong (approximately 2 cm) and acrylic filler can be applied between the extension and the dorsal hoof wall to decrease the chance of the shoe being pulled off and also to distribute the pull on the dorsal laminae over a greater area. Shoeing can also help to protect the toe region from excessive forces and

wear whether or not an extension is included in the shoe. **If pain is a factor in causing these flexural deformities, then adjunctive use of analgesics seems to be indicated.**

**Conversely**, some advocate the application of a wedge under the heel to reduce the strain on the deep digital flexor tendon (Curtis 1992), although there is little evidence of its efficacy as a treatment. This treatment seems reasonable in the acute stage if the aetiopathogenesis of pain is accepted. A heel wedge can also help to decrease the pull of the deep digital flexor tendon on the third phalanx and dorsal laminae.

### *Metacarpophalangeal flexural deformities*

Flexural deformities of the fetlock joint can involve the superficial digital flexor tendon, the deep digital flexor tendon, or both. Raising the heels by the use of a wedge shoe has been suggested to decrease the strain in the deep digital flexor tendon while increasing the strain on the superficial digital flexor tendon and the palmar joint structures (Fackelman 1980; Metcalf *et al.* 1982), but the degree of involvement of the deep digital flexor tendon in metacarpophalangeal flexural deformities and the effect of a raised heel on the superficial digital flexor tendon are largely unknown. Conversely, Owen (1975) recommended lowering the heel to invoke the reverse myotactic reflex. A shoe constructed with 2 vertical metal bars to which a thick rubber sheet is attached over the dorsal fetlock has been described to maintain a normal position of the joint (Auer 1992).

### **Surgery**

Surgery should be considered for severe cases of flexural deformities and those which do not respond to conservative or medical treatment. Corrective trimming can be carried out while the animal is anaesthetised. Postoperatively, the medical treatments described above can be used to optimise the surgical results, although the prognosis must remain guarded in cases requiring surgical intervention.

### **Deformities of the distal interphalangeal joint**

**An inferior check ligament desmotomy (Figs 7a–e)** is used for surgical treatment of flexural deformities of the DIPJ. Surgery is usually performed under general anaesthesia with the horse in lateral or dorsal recumbency. If both limbs require surgery, they can both be approached with the horse in dorsal recumbency, the horse can be turned halfway through surgery from left to right lateral recumbency, or one limb can be approached medially and one laterally with the horse in lateral recumbency. The advantages of a lateral approach are avoidance of the major vasculature, which is medial at this level, and the more lateral position of the ligament. The major advantage to the medial approach is cosmetic. Corrective trimming of the feet and application of protective acrylic to the toes can be performed at this time. Corrective trimming should be aimed at restoring a normal hoof pastern axis (Curtis 1992).

**An ultrasound-guided inferior check ligament desmotomy** has been described in standing horses (White 1995). The author reported that the surgery was more successful in restoring a normal hoof conformation in the younger horses treated (median age 6 months) compared to the older group of horses (median age 12 months). The age at the time of surgery did not affect the cosmetic outcome.

**There is often an immediate improvement in the deformity** which continues for a few days postoperatively (Fackelman 1980). The surgical sites are kept bandaged until the skin sutures are removed and limited exercise is encouraged. In horses intended for showing or pleasure riding, the prognosis for athletic function is good; in one study, 86% of horses treated before age 1 year were subsequently used for their intended purpose (Wagner *et al.* 1985b). Cases treated after age 1 year had a lower success rate of 78%. Fackelman (1980) reported a good prognosis for surgical correction of mild cases but a poor response of severely affected cases to an inferior check ligament desmotomy. Stick *et al.* (1992) reported that Standardbred foals treated for DIPJ flexural deformities could reach their athletic potential, but that the prognosis was better if the foal was treated at a younger age. No foals treated after age 8 months had a favourable outcome.

**A deep digital flexor tenotomy** can be used in cases of *stage II* DIPJ flexural deformities which do not respond to an inferior check ligament desmotomy (Fackelman 1980) or are considered to be very severe (Fackelman *et al.* 1983). This surgery can be performed after an inferior check ligament desmotomy has been attempted when suitable improvement has not occurred. The technique can be performed at the level of the midmetacarpus or the palmar pastern, although a DDF tenotomy performed at the pastern level may produce a more cosmetic result. This procedure is often a salvage procedure and the prognosis for return to function is usually guarded.

### **Deformities of the metacarpo/tarsophalangeal joint**

Because both the superficial and deep digital flexor tendons cross the palmar/plantar surface of the fetlock, either or both can contribute to flexural deformities of this joint. Distinguishing which tendon is primarily affected through palpation is not necessarily straightforward. Both inferior and superior check ligament desmotomies have been used for flexural deformities of this joint and can be used together for the treatment of severe deformities.

**Inferior check ligament desmotomy**; for details, see above. Blackwell (1980) reported relief of clinical signs in 14/14 cases treated surgically in which the deep digital flexor tendon or suspensory ligament was diagnosed as the main cause of the flexural deformity.

**Superior check ligament desmotomy (Figs 8a–c)** can be performed for fetlock flexural deformities in which the superficial digital flexor tendon is contributing to the deformity. The surgery is performed under general anaesthesia using a medial approach to the affected limb at the level of the chestnut. Correction of the flexural deformity will be gradual

and is often incomplete (Fackelman 1980). **A shoe with an elevated heel may be used postoperatively** to load the superficial digital flexor tendon preferentially. Horses with chronic or severe flexural deformities respond poorly to any treatment, including surgery (Fackelman 1980; Wagner *et al.* 1985a) and superior check ligament desmotomies do not carry as good a prognosis as inferior check ligament desmotomies.

Prior to use of check ligament desmotomies for treatment of flexural deformities, desmotomies of the deep or superficial digital flexor tendons were the surgical procedure of choice but athletic performance was compromised due to scarring and adhesion formation (Fackelman 1980). **A suspensory ligament desmotomy** is a salvage procedure for cases which are unresponsive to check ligament desmotomies and removes a large element of support for the fetlock joint. Subluxation of the proximal interphalangeal joint and weakness of the resulting fibrous scar are common postoperative occurrences (Fackelman 1980).

## Conclusions

**Prevention of flexural deformities includes monitoring of a foal's weight and growth and adjustment of the diet to achieve this. Once flexural deformities have occurred, early recognition and treatment, whether medical or surgical, is important for the best prognosis.**

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