PHYSEAL FRACTURES

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By far the most common physeal fracture in foals are the distal physeal Salter Harris type II fractures of the distal metacarpus/metatarsus and the proximal physis of the olecranon. Other physeal fractures are far less common and may involve the distal femoral physis, the proximal femoral physis, and the distal and proximal radial physes.

Distal physeal fractures of the third metacarpal/metatarsal bones

Distal physeal fractures of MCIII/MTIII are common injuries in suckling and weanling foals. These are almost always Salter-Harris type II fractures. They usually heal relatively quickly with minimal surgical management. Foals of less than 6 weeks can be treated with cast coaptation alone for 2 to 3 weeks followed by 2 to 3 additional weeks in a splinted bandage. In older foals or fractures with marked instability, the fracture can be repaired with compression screws through the metaphyseal component. A transphyseal bridge can be added for additional stability. Such minimal internal fixation is adequate if combined with external coaptation in foals because physeal injuries heal quite rapidly. It is advisable not to cross the growth plate with any implants in animals intended for athletic function.

In young foals, the implants should be removed as early as possible in order to minimize the risk of a shortened bone (3 to 4 weeks in a neonate). In an older foal with negligible growth remaining, the implants can be removed at 2 to 3 months. Many foals will show evidence of musculotendinous laxity following cast removal. This may be characterized by marked hyperextension of the metacarpo/metatarsophalangeal joint and elevation of the toe. Such foals should be managed with minimal external support, careful trimming of the toe and orthopaedic shoes with long heel extensions to keep the sole of the foot flat on the ground. Return to exercise should be carefully monitored.

Proximal apophyseal fractures of the olecranon

The most common configuration in horses younger than 1-year of age is the type 1b fracture, where the caudal one third to one half of the physis is disrupted, propagating the fracture into the metaphysis, and finally exiting into the proximal aspect of the trochlear notch near the anconeal process. Less commonly, the fracture breaks out the cranial cortex of the olecranon proximal to the anconeal process and remains nonarticular (also type 1b). Physeal separations (type 1a) are most common in neonates.

Open reduction and internal fixation using the tension-band principle, provide stable fixation of nearly all fracture configurations with minimal complications and a favorable prognosis. In foals, distal screws should not engage the caudal cortex of the radius. If the body of the ulna is transfixed to the radius in foals younger than 12 months, growth of the proximal radial
epiphysis forces the anconeal process into the humeral condylar notch, and subluxation of the cubital joint develops, resulting in elbow dysplasia.

The proximal location of type 1 fractures challenges the fixation, because the small size of the proximal fragment limits the number of screws available for purchase. With a type 1b fracture, the plate is contoured over the dorsal aspect of the apophysis to allow purchase with three short screws. The prognosis following plate fixation of type 1b ulnar fractures is good and 81% of horses in one study were reported to be sound.

Tension-band wire fixation has been also used to manage olecranon fractures in horses that weigh 250 kg or less. Fractures in foals and weanlings are repaired with at least two or three 1.2-mm diameter wires, whereas four to six 1.5-mm diameter wires are used in older horses. The advantages of tension-band wiring compared to plate fixation are less-expensive equipment, less risk of screws entering the joint space or engaging the caudal cortex of the radius, less risk of fracture of the apophysis because screw holes in the proximal fragment are not necessary for fixation, and, with more distal fractures, less tissue dissection required.

**Proximal and distal physeal fractures of the radius**

Fractures involving the proximal physis are candidates for internal fixation, with special considerations. Salter-Harris types I and II fractures of the proximal physis are accompanied by ulnar fractures. Plate fixation of the ulnar fracture, with screws engaging both cortices of the radius where possible, combined with a laterally applied narrow plate with the most proximal screw in the radial epiphysis, is recommended.

Salter-Harris type I distal physeal fractures occur in neonates and are treated by transphyseal bridging across the medial aspect of the physis. A T-plate or screws and wires can be used. The implants are removed when the fractures are healed at approximately 4 to 6 weeks.

**Proximal physeal fractures of the tibia**

The Salter-Harris type II proximal physeal fracture of the tibia is most common and a lateral triangular metaphyseal fragment is invariably attached to the epiphysis. These fractures can be adequately repaired with a medial plate and a tension band to stabilize the tibial crest. The prognosis after repair of proximal physeal fractures is favorable, but the smaller the foal, the better the prognosis.

**Proximal and distal physeal fractures of the femur**

Proximal head and neck fractures are seen almost exclusively in foals and have a guarded prognosis with either surgical or conservative management. The favored surgical technique involves placement of at least two screws in lag fashion.

Distal femoral fractures are most commonly encountered in older weanlings and yearlings and are nearly always of the Salter-Harris type II. Most have the metaphyseal fragment positioned caudally. These fractures require surgical repair with a lateral plate (LCP or DCS). The prognosis for these fractures is guarded because of the high risk of complications.
Reference

DIAGNOSIS AND TREATMENT OF ANGULAR LIMB DEFORMITIES IN FOALS

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Introduction

Angular limb deformities (ALD) are an important problem in foals and young horses. The prevalence of ALD that require intervention in thoroughbred horses has been reported to be as high as 4.7 % (Wohlfender, Barrelet et al. 2009). Deformities of the carpus, fetlock and tarsus lead to abnormal stress on the affected limbs and can limit athletic performance. Although many foals have less than ideal conformation at birth, the majority of angular deformities resolve spontaneously and relatively few foals require surgical management. Treatment of ALD has varied over the years: surgical correction of ALD, popular many years ago, was followed by a period when conservative treatment was preferred. There has been a resurgence in the popularity of surgical treatment of ALD in foals, which is reflected by the large number of recent publications in journals and books. However, some studies have questioned the benefit of some surgical procedures. Angular limb deformities can occur in one or in two planes, mainly in young foals. There is either a valgus deformity (a lateral deviation from the normal axis of the limb distal to the location of the deformity) or a varus deformity (a medial deviation of the limb distal to the location of the deformity). Most animals with ALD also have some degree of rotational deformity initially, which may simply be of postural origin. Angular limb deformities may be congenital or acquired.


**Diagnosis**

A thorough examination is paramount in foals with ALD. Foals should be observed standing at rest and at a walk to establish whether the angulation displayed is consistent or variable. The affected limbs should be palpated to evaluate possible ligamentous laxity and the presence of pain and swelling. It is crucial that the limbs be assessed in a dorsopalmar/dorsoplantar plane rather than simply assessing limb conformation by looking at the patient from the front and back.

Radiographs should be taken to evaluate the bones, especially the cuboidal bones, and to quantify the angulation of the limb. To achieve this, geometric assessment is carried out by drawing lines that bisect the long bones immediately above and below the joint.

**Aetiology**

**Angular limb deformities caused by ligamentous laxity**

Angular limb deformities attributable to ligamentous laxity are typically present and most pronounced at birth. The majority of ALD attributable to ligamentous laxity resolve spontaneously over the first few days of life. The foal should be confined to a box stall for several days. Mechanical support in the form of a stiff bandage or a splint may be indicated in some cases.

**Angular limb deformities caused by cuboidal bone hypoplasia**

In foals, underdeveloped cuboidal bones are due to inadequate ossification, and affected animals are skeletally immature. The cuboidal bones are essentially cartilaginous. Foals with cuboidal bone hypoplasia generally have a mild to moderately severe angular limb deformity. Deviation in the front limbs usually occurs in a medial-to-lateral direction but, in the rear limbs, the angulation typically occurs in a dorsoplantar direction. Affected foals should be
confined to a box stall for 14-21 days. If the foal is very active, a splint or tube cast should be used to provide mechanical support.

**Angular limb deformities caused by disparity in long bone growth**

Angular limb deformities caused by a disparity between medial and lateral long bone growth may be present a birth or the deformity may be an acquired developmental condition. Uterine positioning is believed to be the major cause of congenital deformities. The most common deformity is a lateral (valgus) deformity originating in the distal radius. There is a constant angulation and no swelling or laxity of the involved joint. Radiographic evaluation confirms normally-shaped cuboidal bones, and the geometric pivot point is located proximal to the joint.

In the majority of foals with ALD caused by a growth disparity of the long bones, spontaneous correction of the deformity occurs. The challenge is to identify the foals that need more aggressive intervention. Exercise restriction and corrective hoof care are routinely used to reduce biomechanical stress on the concave side of the angled limb and to ensure that the compressive physiologic limit of the physis is not exceeded. Severely affected foals or foals failing to respond to conservative treatment are candidates for surgical treatment. Follow-up examination of the foal to determine whether the deformity is improving, static or becoming worse is very important when contemplating surgical intervention.

**Management of angular limb deformities**

In the past, surgical treatment was elected in very young and immature foals, but in recent years a more conservative approach has been used for correction of ALD. The exception to this is very severe angular limb deformities. Deformities of the fetlock must be addressed before 4-6 weeks of age, whereas deformities of the carpus/tarsus can be handled with a “wait and see” approach because of the longer period of growth in the distal radial/tibial physis.

**Stall rest**

Foals that have straight limbs but incomplete ossification or dysmaturity on radiographs should not be left out on pasture. These animals should be housed in a stall for about two weeks, after which time the limbs can be re-evaluated radiographically.

**Splints**

If the deformity can be corrected manually, it is important to apply splints over the carpi and tarsi. The limbs are first covered with a light bandage, and the splint is applied over the bandage.

**Manipulation of the hoof**

Manipulation of the hoof should be encouraged. Application of “Dallmer” shoes with an extension to one side or to the back is better than rasping and unbalancing the hoof because excessive manipulation of the hoof wall is avoided.
Surgical techniques

Generally, the available techniques can be divided into growth acceleration and growth retardation techniques. These techniques are used when disproportional growth between the medial and lateral aspect of the bone is verified radiographically, or in foals that have a residual deformity after ossification has progressed to a stage where splints are no longer indicated.

**Growth acceleration = Periosteal stripping**

The growth acceleration technique, through stripping and loosening of the periosteum from the underlying bone, was developed over 30 years ago for the correction of angular limb deformities in foals (Auer and Martens 1980). This procedure stimulates growth through either a vascular effect or a periosteal-release effect. There have been numerous reports detailing its use, but after 20 years, this technique has come under fire (Slone 2002) (Read, Read et al. 2002). Many foals that undergo periosteal stripping are exercise restricted and receive corrective hoof care. This raises the question of whether the observed correction is in fact due to exercise restriction, hoof care or the actual periosteal stripping.

The operation is carried out on the shorter side of the leg. It is supposed to facilitate unilateral acceleration of the growth and therefore correction of the deformity. After the periosteum is elevated from the underlying bone, the distal and proximal ends are separated from the attachment to the bone by approximately 5 mm. Within about 2 months, the periosteal gaps will again be bridged, at which point the effect of the manipulation ceases.

The advantages of periosteal transection include: overcorrection cannot occur, it is a simple ambulatory technique, no implants are inserted and therefore postoperative infection is minimal. If required, the procedure can be repeated after 2 months, provided that the bone is still growing.

**Growth retardation**

With this technique, growth on the longer side of the leg is temporarily slowed down by applying implants over it. That allows the shorter side to catch up. Once both sides are even, the implants must be removed because overcorrection will occur otherwise. Growth retardation can be achieved either with staples, with screws only, with screws and cerclage.
wire or with smaller screws and a small bone plate. The implants cause compression of the growth plate and growth will be slowed down.

**Staples**

Staples are still used in some clinics. An incision slightly larger than the length of the staple is centred over the growth plate of the deviated bone. The periosteum is surgically approached and the staple is inserted using the appropriate instruments. The staple should not be hammered in too forcefully because this could cause injury to the periosteum and possible permanent and premature closure of the physis. It can also make removal of the staple difficult.

**Screw and wire**

The screw and wire technique is the most frequently used technique. A stab incision is made over the middle of the epiphysis and another proximal to the physis. The subcutaneous tissue between the two incisions is undermined. One screw is inserted through each of the stab incisions, but not completely tightened. A loop of cerclage wire is passed from the proximal incision down to the distal incision and hooked over the screw head. The wire is crossed and then twisted on top of the proximal screw. The wire is tightened as much as possible without breaking it. Once this is achieved, a second wire loop is applied over the first one. The screws are then tightened and the stab incisions closed with two skin sutures each.

**Screws and plate**

A similar technique is carried out when screws and a small bone plate are used. The only difference is that the distal screw must be inserted first through the distal-most plate hole and the second screw through the most proximal plate hole. A plate that can be manually bent should be used.

**Single transphyseal screw (STS)**

This technique has become extremely popular in recent years because of its simplicity, effectiveness and excellent cosmetic results. An STS is the method of choice for the distal cannon bone, but there is some concern when used in the distal radius and tibia.

Implant removal is carried out using short-term intravenous anaesthesia. With the screw and wire technique, the proximal screw can always be palpated and a stab incision is made over it to partially remove the screw. The distance between the proximal and distal screw is determined on the preoperative radiograph and a second stab incision carried out at that distance from the proximal screw. Once the screw has been identified by probing with a hypodermic needle, it is removed as well. The wire is then identified at the proximal incision and pulled out with a sharp jerk. The stab incisions are subsequently closed with simple interrupted sutures.
Varus deviation in both fetlock joints

Single transphyseal screw

Correction after 4 weeks

Right leg

Left leg

Valgus deviation in both carpal joints

Radiographic pictures

Growth retardation with plates

Growth retardation with plates

Perfect correction after 3 months

Combination of growth acceleration and retardation techniques

In animals with severe deformities (axis deviation greater than 15°), growth retardation and growth acceleration techniques are carried out simultaneously with the aim of straightening the limbs as quickly as possible. Obviously the implants must be removed once the limbs are straight. This combination may also be carried out in older animals, where growth is not as rapid as in very young animals.

Surgical complications

Surgical complications include implant failure such as breakage of the cerclage wire, which is particularly problematic when only one wire has been applied. Irritation of the surrounding
tissue by the wire ends and penetration of the joint or the growth plate with one of the implants are other complications. It is therefore important to take radiographs immediately after application of the implants. Should penetration of a joint be detected on radiographs or during surgery, the implant should be removed immediately and redirected or shortened. In doing so, major damage is avoided and the animal can mature normally. Obviously, aseptic technique is mandatory with any of the techniques that manipulate growth.

**General remarks**

It is very important to limit surgical correction of ALD to animals that are still growing. None of these techniques will be successful after growth has stopped. This is especially important in miniature horses or Shetland ponies because often, owners as well as veterinarians wait too long to carry out surgical correction. These animals may have atavism in the form of persistent complete ulna and tibia, which may lead to further complications such as degenerative joint disease. In the absence of growth potential, these surgical techniques are ineffective and should be replaced by corrective osteotomies or ostectomies.
TREATMENT OF SUBCHONDRAL CYSTIC LESIONS

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Introduction:

Subchondral cystic lesions (SCLs) are defined as radiolucent areas of bone, which usually contain fibrous connective tissue and serous synovial-like fluid and sometimes cartilage- and bone like structures. Depending on their stage of development, SCLs may be well demarcated from the surrounding tissue by a sclerotic rim (Fürst et al. 2007).

Subchondral cystic lesions can be treated conservatively with stall rest, controlled exercise and systemic or intra-articular medications (Wallis et al. 2008), which include hyaluronan, corticosteroids, polysulphated glycosaminoglycans and benzopyrones (Jackson et al. 2008). Methylprednisolone acetate has been used successfully for the treatment of UBCs in humans (Scaglìetti et al. 1982), and corticosteroids have been investigated for treatment of SCLs in horses (Vandekeybus et al. 1999). In a recent study, injection of corticosteroids into the fibrous tissue lining of SCLs in the medial femoral condyle via arthroscopy had a success rate of 67%, regardless of the age of the horse (Wallis et al. 2008). When conservative treatment fails, surgical curettage of the cyst is recommended (Bertone et al. 1986; Kold et al. 1986; Jackson et al. 2000; Deiss et al. 2000; Story and Bramlage 2004; Smith et al. 2005). Surgical management includes debridement of the cystic contents, forage and microfracturing, after which the lesion may be filled with cancellous bone grafts (Jackson et al. 2000), chondrocytes and insulin-like growth factor (IGF I) (Nixon 2002), tricalcium phosphate granules (Fürst et al. 1997), chondrocytes or mesenchymal stem cells in fibrin glue (Fortier and Nixon 2005), mesenchymal stem cells without fibrin glue (Kraus and Kircher-Head 2006), multiple osteochondral autografts in the form of a mosaic plasty (Bodo et al. 2004; Janicek et al. 2010) or with PTH (Jackson et al. 2011). The results of these treatment
modalities vary and the reported success rates range from 64 to 82% in young horses (White et al. 1986; Howard et al. 1995). After surgical debridement the prognosis in older horses with an SCL in the medial femoral condyle, is worse than in younger horses (Smith et al. 2005). Moreover, there is a risk of injury to the medial meniscus after curettage of large SCLs in the medial femoral condyle (Hendrix et al. 2010). The technique of forage or osteostixis is no longer used because this transsclerotic drilling technique may lead to expansion of the lesions (Howard et al. 2005). For more than eight years, our clinic has used parathyroid hormone (PTH$_{1-34}$) cross-linked with hydrogel via a fibrin base to fill surgically curetted SCLs. Parathyroid hormone is secreted by the parathyroid gland and has been thoroughly researched. It is normally involved in bone resorption, provided that it is constantly secreted in high concentrations. However, when PTH was administered systemically, intermittently and at lower dosages, it induced bone formation, mainly in cancellous bone. The exact mechanism involved in the anabolic effects of PTH is not clear, although the main function of the hormone is thought to be extension of the lifespan of osteoblasts and osteoclasts. Treatment of our patients was based on the hypothesis that PTH$_{1-34}$ induces new bone formation and accelerates bone healing in surgically debrided SCLs by down-regulating cytokines and inflammatory mediators. It was expected that PTH$_{1-34}$ bound to the structured hydrogel, would support early filling of the SCL with new bone, decrease bone inflammation and thus, be a considerable improvement of the current management regimes. We have treated over fifty horses with SCLs in different locations bones. There was a rapid improvement in most horses and, compared with curettage alone, bone healing seemed better (Fürst et al. 2007, Jackson et al. 2011). In-vitro studies have shown that cell cultures of fibrous tissue harvested from SCLs produce local inflammatory mediators, including PGE$_2$, IL-1 and IL-6, and neutral matrix metalloproteinases (NMPs) (von Rechenberg et al. 2000; von Rechenberg et al. 2001). These mediators are capable of recruiting and activating osteoclasts in laboratory animals and humans (Jilka et al. 1992; Manolagas et al. 1993) and are thought to be responsible for the osteolysis observed in equine SCLs (von Rechenberg et al. 2000). This could explain, at least partially, the beneficial effect of intraleseional corticosteroid administration in UBCs and SCLs (Scaglìetti et al. 1982, Wallis et al. 2008) because PGE2 synthesis is inhibited by corticosteroids (von Rechenberg et al. 2000). However, the time required for healing after intraleseional injection of corticosteroids may be prolonged and complete remission not achieved (Fürst et al. 1997) because corticosteroids also inhibit matrix molecule production, such as collagen. For these reasons, surgical curettage of the cystic lesions with complete removal of the cellular lining of the cavity is considered by the authors to be the treatment of choice for SCLs. Residual cystic wall tissue is considered a contributing factor in recurrence of the cystic lesions because it may secrete cyst fluid or inhibit revascularization and new bone formation (Kold et al. 1986). In most patients, thorough curettage of the cyst and removal of its contents are achieved via an articular or an extraarticular transosseous approach, depending on the location of the cyst. Arthroscopy minimises the risk of postoperative complications (Trotter and McIlwraith, 1996) and therefore is the approach of choice for debridement of many bone cysts. The extraarticular approach is used in all SCLs without joint communication or small joints with insufficient space for arthroscopic instrumentation (Fürst et al. 2007). This approach is technically more difficult, but has the advantage of causing less damage to the
articular cartilage covering the cyst (Fürst et al. 2007). The disadvantage of this approach is that complete removal of the cyst contents cannot be verified.

At the moment we are investigating the possibility of treating SBC with recombinant bone morphogenetic proteins (BMPs). The BMPs are potent cell signaling proteins that involve in many aspects of embryonic development because of their osteoinductive effect. Their principal function is to induce transformation of undifferentiated mesenchymal cells into chondroblasts and osteoblasts. BMPs have been used with success in other disorder of poor bone formation. Among the BMPs family, BMP-2 has been shown to have the ability to improve or accelerate fracture healing in humans and animals (Perrier, Lu et al. 2008).

The ideal material for filling the cyst is still not defined, because there are still 20 % of subchondral cysts, which are curetted and filled, which do not heal.
MRI OF TENDON INJURY AND HEALING IN HORSES

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MRI has numerous advantages over other modalities for imaging of tendons. Mainly it has high intrinsic contrast and resolution, particularly for soft tissues, resulting in good anatomic separation between different tissues. In addition it can give information on the biochemical nature of tissues which may be useful in determining the stage of healing in an injured tendon.

Ligament and tendon margins and symmetry are best evaluated on sequences that produce MR images with high anatomical detail, like proton density and 3D spoiled gradient echo sequences. T2-weighted and STIR images on the other hand have high fluid contrast making them useful for looking at fluid in tendon lesions. A protocol that combines information from T1-, T2-weighted and fat suppressed images is needed for a complete evaluation. In general, fluid is hypointense on T1-weighted and hyperintense on T2-weighted and PD images. Increased protein or cellular content in fluid can lead to increased signal intensity of fluid on T1-weighted images. Immature granulation tissue has high signal intensity on T2- and T1-weighted images, whereas mature fibrotic tissue has low signal intensity on T2-weighted images but often remains hyperintense in T1-weighted images.

Normal tendons emit zero signal on T1- and T2-weighted images. Normal ligaments produce more signal variation than tendons and vary from light gray to black because ligaments are often composed of different fiber bundles with varying orientations which makes them more susceptible to magic angle effects than tendons. Mild signal increase may be seen in normal areas of cartilaginous tissue in tendons, such as the dorsal aspect of the superficial digital flexor tendon (SDFT) in the fetlock region and the dorsal aspect of the deep digital flexor tendon (DDFT) at the level of the middle phalanx and navicular bone.

In general increased signal intensity in tendons and ligaments indicates tissue damage. Tendon damage is seen as focal or diffuse, marginal or central, intratendonous signal increase on both T1- and T2-weighted sequences, variably accompanied by swelling in the acute stage of injury. Mild damage may be detected by the presence of peritendinous signal increase on T2-weighted images. More severe damage is characterized by either focal or diffuse intratendonous increase in signal intensity. This may be accompanied by enlargement and shape changes. A partial transverse tear may result in thinning, elongation or a wavy or partially interrupted contour of a ligament or tendon. With complete transverse tears, the contour is interrupted by a visible hyperintense defect and stumps may be present at the ligament or tendon ends. In the acute and subacute stages of tendon or ligament injury, signal increase is present in both T1- and T2-weighted images. At later stages of healing, T2 signal progressively returns to normal but T1 signal hyperintensity may persist, sometimes indefinitely.
MR signal intensity varies between different echo sequences with duration and severity of a tendon lesion. Acute lesions generally have higher T2 signal intensity due to the presence of fluid and increased cellularity, whereas fibrous scar tissue in chronic lesions produces a more intermediate to low T2 signal intensity. It may therefore be possible to use T1-to-T2 signal differences to estimate the age or stage of healing of a tendon lesion (Schramme et al. 2010). In the chronic stages of healing by fibroplasia, signal intensity in core lesions generally decreases in T2 and STIR images but can remain high in T1-weighted sequences. Focal T1 hyperintensity has been identified in DDFT lesions of more than 12 months’ duration, in spite of histological evidence of healing with mature scar tissue. In addition, the presence of T2 and STIR signal hyperintensity in lesions is not only related to the age but also to the severity of injury. Signal hyperintensity in T2 or STIR images was found in areas of more severe fiber disruption and pseudocyst formation in both equine DDFT lesions (Busoni et al. 2005) and human Achilles tendonopathy (Karjaleinen et al. 1996). No T1-to-T2 signal differences were observed between acute and chronic core lesions in the DDFT in a different study (Blunden et al. 2009), although the only T2 sequences used in this study were T2* gradient echos. In the same study, increased signal in STIR images was found to be useful to determine whether necrosis or fibroplasia was the dominant histological feature of a lesion. STIR signal hyperintensity in fat suppressed images only occurred in tendon lesions with fascicular necrosis but not in lesions with core fibrosis and/or fibrocartilaginous metaplasia, regardless of the age of the lesion.

In conclusion, a focal area of abnormally high T1 (and possibly T2*) signal in an injured tendon that is not accompanied by high signal on the corresponding T2 and STIR images, is likely to reflect an area of chronic fibroplasia without any active inflammation or necrosis. However, the overall MRI appearance of an injured tendon may never become completely normal again because of the persistence of signal hyperintensity on some sequences. Based on MR imaging therefore, it may not be possible to establish when a tendon or ligament injury has healed adequately for resumption of sporting activities safely.

References

BIOLOGICAL MEDICINE FOR THE TREATMENT OF TENDONS AND LIGAMENT INJURIES

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Introduction

Over-strain and traumatic tendon and ligament injuries are common in the horse and, for the most part, heal (repair) naturally by the formation of scar tissue. Regenerative medicine offers the prospect of restoring normal structure and function to an injured organ and thereby resulting in a successful restoration of activity without the risk of re-injury. Regenerative medicine traditionally utilises one or more of three different components: a scaffold, an anabolic stimulus (eg growth factor), and a cell source.

1) SCAFFOLDS

Scaffolds can have a variety of potential beneficial effects – they can be used to carry or attract cells, help align reparative tissue by their structure, and protect the cells immediately after implantation and before new matrix has been synthesised. However their influence can be both positive and negative.

ACell Vet™ is an intralesional treatment for tendinopathy/desmopathy using acellular tissue components derived from porcine urinary bladder submucosa. This preparation has been suggested to deliver appropriate growth factors to the injured tissue as well as attracting mesenchymal stem cells. It is injected in liquid form (as a reconstituted powder). However, it has been associated with significant inflammatory reaction after injection and it is recommended that horses are pre-medicated with anti-inflammatories and cold is applied locally after treatment.

2) GROWTH FACTORS

Insulin like growth factor–1 (IGF-1) stimulates extracellular tendon matrix synthesis and is also a potent mitogen. In collagenase-induced models of tendinitis, initial swelling was decreased following intralesional injections if IGF-1 compared with controls, although no differences were found at later time points and there was no difference between the quantities of type I and type III collagen synthesized.

TGF-β has been considered as another appropriate growth factor treatment although clinical experience has been limited. Treated horses showed significant enlargement of the tendon and, although re-injury rates were similar to conservatively managed horses, these re-injuries were all on contralateral, untreated, limbs.
Most recently, platelet-rich plasma/concentrate has gained popularity in the treatment of tendon and ligament injuries. PRP contains high levels of those growth factors sequestered in platelets – most notably platelet-derived growth factor (PDGF) and transforming growth factor beta (TGF-beta1) but also Vascular Endothelial Growth Factor (VEGF) which promotes neovascularisation. It is not known as to whether these growth factors are optimal for tendon and ligament healing but they have been demonstrated to have anabolic effects in vitro on both tendon explants from the superficial digital flexor tendon and suspensory ligament explants and desmocytes and are therefore logical factors to consider. However, because of platelets’ close association with the normal inflammatory process, it could be postulated that PRP would promote an exaggerated fibrotic reaction rather than regeneration. Studies in experimental mechanically created tendon lesions in the horse equine models have demonstrated improved organisation and increase strength after PRP treatment although the stiffness of the repairing tissue was also increased. Given this concern, we have hypothesised that PRP may be most suited to the treatment of suspensory ligament lesions where exaggerated fibrosis is potentially less of a concern functionally than for the superficial digital flexor tendon. Recent publications have suggested positive effects in small clinical series of both suspensory ligament and superficial digital flexor tendon injuries.

3) MÉSÉNCHYMAL STEM CELL (MSC) THERAPY

MSCs have been considered an ideal source of cells for regenerative medicine because they can be demonstrated, in horses as in other species, to differentiate into different cell lines and synthesise new matrix (usually chondrogenesis, adipogenesis and osteogenesis).

Equine digital flexor tendon strain injuries provide many of the elements required for tendon tissue engineering – the lesion manifests within the central core of the tissue thus providing a natural enclosure for implantation and, by the time of stem cell implantation, is filled with granulation tissue which acts in the role of a scaffold. It has the added advantage of being highly vascularised and therefore capable of nutritional support of the implanted progenitor cells. The cytokine and mechanical environment, which are potentially important drives for differentiation, is provided by the intra-tendinous location of the cells and the suspension of MSCs in bone marrow supernatant which we have shown to have significant anabolic effects on cultures of equine ligament-derived cells.

For clinical and equine experimental studies, bone marrow recovered from the sternum is used to prepare a pure MSC preparation that is implanted into the damaged tendon (10-50x10^6 cells, depending on the extent of the lesion) under ultrasound guidance. The cells are suspended in citrated bone marrow supernatant for implantation so that no ‘foreign’ material is implanted and to gain potential beneficial effects of the rich mix of growth factors present in the supernatant. After implantation, the limb is bandaged and the horses undergo a week of box rest followed by a controlled exercise programme for up to 48 weeks.

In an experimental study of 12 horses with naturally occurring SDFT injury 10 million autologous bone marrow derived MSCs were implanted into the damaged SDFT of the treated group while saline was injected into the control group. After 6 months, MSC-treated
tendons exhibited normalisation of their mechanical, morphological and compositional parameters towards that of uninjured tendons.

Analysis of clinical outcome for 113 National Hunt and flat racehorses treated with MSCs and followed up for 2 years after treatment, was found to halve the re-injury rate compared to previously published data for horses treated conventionally and with either superior check ligament desmotomy, bar firing, or IGF-1.

References

NEW TREATMENT OPTION IN THE THERAPY OF TENDON INJURIES

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Summary: A new therapy as a treatment for tendon injuries is discussed. The special production process prepares a soft tissue adapted Hyaluronic acid (STABHA) with the name TendoPlus. The regeneration of tendon lesions is improved due to a suspected trigger function on stem cells and a positive effect to fibrin polymer formation. Alternative therapeutic options in tendon repair (Surgery-shockwaves-injections with PEP-ACP-and others) are presented and their results are compared with the TendoPlus treatment. Different cases are shown and the results by sonographic examination confirm the positive effect of this therapy.

A hyaluronic acid developed specifically for application in soft tissue has been on the market of human medicine for some time. The preparation, which was developed here in Switzerland, promised a large range of uses also for the horse. Thus, it was no surprise that first attempts for administration in the horse quickly followed. The surprisingly positive results led to the intensification of treatment attempts and to further development of an equine preparation, i.e. the well-known TendoPlus.

Due to the special production process, the affinity of TendoPlus for soft tissue has been increased so that the effect on tendons and ligaments was optimised. The active substance of TendoPlus is not conventional hyaluronic acid but was especially developed for soft tissue, with the protected name STABHA (Soft Tissue Adapied Biocompatible Hyaluronic Acid). STABHA is produced in a patent-protected production process and has an extremely high degree of purity.

This purity profile is essential for the effect in soft tissue, since only this can achieve biocompatibility with the soft tissue and thereby, a long retention time in the tissue. This long retention time is a precondition for the positive effect on wound healing. The exogenous administration of hyaluronic acid into the freshly traumatised tissue leads to a faster formation of the fibrin matrix with stronger cross-linking, resulting in a more stable fibrin matrix.

The production process, among other things, uses special procedures that effect targeted elimination of nucleic acids and endotoxins. This is a further crucial difference to the production process of conventional hyaluronic acid, since not only the impurities but the macromolecules themselves are precipitated there. By this inverted production reaction, i.e. the precipitation of impurities, a clearly higher degree of purity is obtained. This in turn leads then to substantially higher receptor binding and prevents the degradation by the body's own reactions.

The basis for the development of TendoPlus was the work of a team around Prof. Rinaudo of the University in Grenoble. This team has been working for years, actually even for decades, on the biochemical behaviour of the most diverse aminoglycan macromolecules.

Among other things, a direct chemical reaction of hyaluronic acid (HA) with fibrin could be demonstrated, leading to a cross-linking and thus a stabilisation of this structure beyond the physiological extent (LeBoeuf et al. 1986, LeBoeuf et al. 1987, Weigel et al. 1988). This reaction, in connection with results of other teams (Weiss et al., 1995) who could demonstrate an elevation of the body's own HA concentration in fresh soft tissue traumas, led to the rationale for developing TendoPlus.

At our hospital, we have performed all new forms of therapy for the treatment of tendon injuries, and over the course of the time, we were able to gain a lot of experience with the effectiveness of the various procedures. Aside from the surgical approaches such as tendon splitting, use of carbon fibre or PDS ligaments and physical procedures such as therapeutic ultrasound,
laser or shock waves, conservative therapies with various injections have proven themselves above all others.

Already in 2001, I had the privilege to present my work on tendon therapy with the so-called Müller-Wohlfahrt-Therapy (a combination of homeopathic preparations with heparin, Medivitan and a calf blood derivative) in comparison to shock wave therapy here in Geneva at the CEMS. Ever since then, we have taken up the newly developed therapy forms for tendon damage again and again and have compared their effectiveness with our experiences.

Particularly the development of stem cell therapy with cultured stem cells from autologous bone marrow has shown a great many successful courses of therapy. Also the use of stem cells cultured from fatty tissue was intensively tested.

Nearly in parallel, various systems for obtaining growth factors from the blood of patients were introduced to the market. Aside from the Orthokin system, the company Arthrex presented a practice-friendly system with which the growth factors could be isolated. A system of the most recent generation promises an up to 10 times higher concentration of growth factors.

Completely new and not yet published is the use of trigger substances for the development of stem cells, which are in part administered but are also in part made available by the body itself.

This opens a new possibility for therapy, because crucial for the healing process is not just the availability of the substances necessary for healing but also the possibility to provide the body with the correct information to produce the right cell once more. Not inelastic scar tissue but elastic connective tissue with the ability to withstand stress prevents the feared recurrence.

Even if no scientific studies have been finalised yet, the work of Marguerite Rinaudo still shows that the special hyaluronic acid (STABHA) contained in TendoPlus fulfills a trigger function in the healing process and thus, explains its convincing effect. We work on the development of further trigger substances under the leadership of Professor Augustinus Bader of the University of Leipzig or the Bionethos Innovation Institute, respectively, in order to further optimise the healing process. Aside from the trigger function, the soft tissue adapted hyaluronic acid also possesses improved properties for a matrix formation that supports the healing process.
TENDON AND TENDON SHEATH WOUNDS

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1. Introduction

As a rule, all tendon sheath injuries should undergo the same treatment. Typical tendon sheath injuries include puncture wounds, such as those caused by pitch forks and nails, kick injuries and tears. Evaluation of the severity of the injury must take into account the location of the wound and its proximity to vital structures including tendon sheaths. Thus, the prognosis for rupture of the tendon sheath of an extensor muscle is better than that for injury to the sheaths of the digital flexor tendons or tarsal flexor tendons.

2. Examination

Injuries in the immediate region of a tendon sheath must be thoroughly examined to determine whether the tendon sheath is in fact affected. This is extremely important for treatment as well as for a prognosis.

In some cases, for example large abrasions over the carpus, determination of tendon sheath injury can be done with the naked eye. However, this is not possible for the majority of injuries, especially puncture and bite wounds, and special diagnostic techniques are required to ascertain whether the tendon sheath is involved.
Diagnostic Imaging

An open synovial structure can sometimes be detected by the presence of gas shadows within the synovial structure on radiographs. The flow of contrast medium out of the synovial space following injection can also be documented via radiography.

Synoviocentesis

**Synoviocentesis is the most important diagnostic aid.** Centesis of the affected synovial space must always be done in an area distant to the injury. The area is prepared aseptically for surgery, and sterile gloves and a sterile needle are used. In cases where there is localised wound infection, centesis of neighbouring synovial structures bears the risk of iatrogenic infection, and in these cases, centesis is contraindicated. Assessment of synovial fluid provides important information. A sample is collected into an EDTA tube for macroscopic and microscopic evaluation and for culture. Changes in the parameters listed below occur before the onset of clinical signs such as lameness or pyrexia. It should be remembered that synovial fluid may leak out of torn synovial structures making it difficult or impossible to collect a sample.

**Flow of turbid synovial fluid from a perforating tendon sheath injury**

**Turbid synovial fluid**

**Evaluation of synovial fluid**

Macroscopic evaluation provides many indications of whether the synovial space has been affected. Normal synovial fluid is clear, whereas turbid synovial fluid is abnormal and may indicate involvement of the synovial space.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal synovial fluid</th>
<th>Infected synovial fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Macroscopic appearance</td>
<td>Clear</td>
<td>Turbid</td>
</tr>
<tr>
<td>Viscosity</td>
<td>Viscous</td>
<td>Watery</td>
</tr>
<tr>
<td>Cell count</td>
<td>&lt;1000/µl</td>
<td>&gt;30 000/µl</td>
</tr>
<tr>
<td>Differential cell count</td>
<td>&gt;90 % mononuclear cells (Macrophages, lymphocytes)</td>
<td>&gt;90 % neutrophils, some with degenerative changes</td>
</tr>
<tr>
<td>Protein</td>
<td>&lt;25 g/l</td>
<td>&gt;35 g/l</td>
</tr>
<tr>
<td>pH</td>
<td>&gt;7</td>
<td>&lt;7</td>
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</table>
When synovial fluid cannot be aspirated, the synovial structure must be filled with sterile Ringer’s solution after attempted centesis. Sufficient pressure must be produced and the limb flexed and extended to break down fresh adhesions. When the synovial space retains the injected solution with no fluid leakage, it is probably safe to conclude that the structure has not been affected.

Puncture wound: How deep is this injury? The injury may be superficial...or it may be very deep!

This question can only be answered after special diagnostic testing, usually during surgical treatment!

3. Procedure for ruptured synovial structures

Surgical wound debridement

The importance of surgical wound debridement cannot be overemphasized. All necrotic tissue must be carefully removed, even if that means enlarging the wound. This entails removal of all contaminated tissue from the level of the skin to the depth of the lesion. Good drainage must be provided, more so than with other wounds.

A small puncture wound that is barely visible Surgical revision of the puncture wound
Lavage of the synovial structure

General considerations

Open synovial structures must be generously (3 – 6 litres) lavaged with sterile solution to remove foreign bodies and bacteria as well as inflammatory mediators and enzymes such as collagenase, caseinase, lysozyme, elastase and gelatinase. Irritating solutions including iodine, chlorhexidine or DMSO must not be used. Instead, sterile Ringer’s solution or lactated Ringer’s solution is recommended. Non-irritating antibiotics (amikacin, gentamicin, nebacetin or cefquinome) can also be added to the lavage solution. The concentration of antibiotics must not exceed the daily dose for the patient. Lavage should be carried out through an opening opposite the injury so that bacteria and foreign bodies can be flushed out through the wound opening. It is critical that all parts of the synovial space undergo lavage whenever possible. For example, in patients with injuries to the digital flexor tendon sheath, all blind sacs must undergo synoviocentesis and lavage. Depending on the clinical signs and results of synovial fluid evaluation, lavage should be repeated two days later to ensure removal of residual foreign bodies, bacteria and inflammatory products. The nature of the injury dictates whether lavage is carried out using a hypodermic needle or adjunctive arthroscopy or via incisions into the joint. In patients with fresh wounds (1 day old), lavage can be done with a hypodermic needle. Wounds that are 2 or more days old should be assessed via arthroscopy. Established infection may benefit from open drainage. Lavage is usually done with the patient under general anaesthesia, and the cell count after effective lavage should be less than 10,000 cells/µl.

Arthroscopic lavage

In old injuries or established infection of a synovial structure, there may be clots of fibrin and purulent material with compartmentalisation of the joint from adhesion formation. Cartilage lesions may also be present. In these cases, lavage using a hypodermic needle is not adequate to remove the tissue debris, and lavage of the synovial structure via arthroscopy is required (McIlwraith et al. 2005; Wright et al. 2003). The degree of invasiveness of arthroscopy is relatively minimal. The entire joint including the cartilage, synovial membrane and contents can be evaluated using this method, and visual control of the lavage process is facilitated. Arthroscopy is very informative in patients with suspected joint infections. It also allows collection of biopsy samples of the synovial membrane, evaluation of early cartilage lesions and removal of cartilage or bone fragments and diseased synovial membrane.

Fibrin clumps and necrotic debris must be removed from the synovial space with small forceps during lavage. The use of a shaver is a distinct advantage for removal of synovial villi. Great care must be taken to protect the joint cartilage during removal of tissue debris. The cartilage and all other joint structures should be carefully assessed, and curettage carried out if required. Foreign bodies such as hair, grass and other organic debris are found in 20 to 40% of synovial structure injuries; these must be removed via arthroscopy.
4. Adjunctive treatment

Treatment of infection must be started as early as possible to prevent irreversible joint damage, which occurs as early as 48 to 72 hours after infection has been established. Administration of systemic antibiotics using a wide-spectrum antimicrobial agent such as marbofloxacin (Marboxyl®) or cefquinome (Cobactan®) or a combination of drugs such as penicillin and gentamicin or sulfonamide/trimethoprim (Rota-TS®) (Widmer et al. 2009) is carried out independent of the duration of infection. Administration of local antibiotics into the synovial space reduces fibrin formation, the loss of joint cartilage and the severity of synovialitis.

Regional retrograde perfusion of intravenous antibiotics

A tourniquet is applied in the sedated and standing patient, and gentamicin or amikacin is injected distal to it into the superficial venous circulation, for example the medial or lateral digital vein in the region of the proximal sesamoids (Ceppi 1999). A dose of 1g gentamicin or 500 mg amikacin in 60 ml isotonic electrolyte solution is used, and the injection is carried out slowly over approximately 1 minute. The tourniquet blocks the superficial venous circulation, which causes an increase in the diffusion pressure in the tissues. This leads to a high concentration and good distribution of antibiotics in the bone, joint or tendon sheath. The tourniquet is kept in place for 30 minutes after which time it is removed allowing the antibiotics to enter the systemic circulation. This treatment is done daily for 3 to 5 days.
Regional intravenous antibiotic administration

NSAID should be administered to provide patient comfort and to interrupt the cascade of inflammatory mediators so that fewer toxic products are produced in the synovial space. Pain management also facilitates better weight bearing, which is critical in the prevention of laminitis in the contralateral limb (Widmer et al. 2009).

A bandage must be applied in all patients to protect the wound from contamination. A splint can be used in the first few days to limit movement in the affected limb and promote wound healing. Bandage changes must be done a minimum of every 2 to 3 days at the beginning so that wound secretion does not interfere with healing. Bandage changes must follow sterile procedure so that the synovial space does not become infected secondarily. The interval for bandage changes can be extended once there is a reduction in the amount of wound secretion.

The patient should be housed in a box stall. Healing times may be lengthy and box stall confinement should be extended beyond the resolution of clinical signs to allow complete healing of the joint cartilage.

5. Prognosis:

Because of the enormous physiological demands on the locomotor system, the prognosis for full recovery after injuries involving synovial structures is guarded in horses. The prognosis depends on the age of the lesion and whether adequate treatment can be implemented. The timing of the treatment is of essence; the earlier that treatment is started, the better the chances of healing and the smaller the risk of permanent damage to the injured tendon.
DISORDERS OF THE ESOPHAGUS

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Esophageal diseases are among the less commonly seen disorders in equine veterinary practice around the world. The clinical signs and diagnosis of esophageal disorders and their therapy are specific in horses. Clinical presentation of esophageal obstructive disorders is typically manifested by feed and saliva appearing at the nostrils and mouth, hypersalivation and flapping of the lip. Odynophagia (painful swallowing) and repeated extension of the head and neck could also be present. Affected horses are usually inappetent. Coughing may occur because of feed and water aspiration. Recurrent signs of esophageal obstruction may indicate a persistent narrowing of the esophagus due to some other anatomic pathology or esophageal dysfunction.

Diagnosis of esophageal diseases is based on physical examination including passing a nasogastric tube, endoscopy and radiography. Additional information can be obtained via clinical pathology, ultrasound and manometric evaluation. Palpation of the laryngeal and cervical region could reveal subcutaneous emphysema or masses. Detailed oral examination can exclude oral causes of dysphagia such as cleft palate or dental diseases. Insertion of a nasogastric tube could confirm impassibility of the esophagus and exact location of an obstruction. Basic clinical pathology in horses with esophageal disease includes packed cell volume (PCV), total plasma protein (TPP) and plasma electrolyte concentrations to determine the horse’s metabolic and hydration status. Dehydration, neutrophilia, hypochloremia, hyponatremia and hypokalemia are reported to be the most consistent abnormalities accompanying esophageal disorders.

Endoscopic examination of the upper airways can reveal the presence of feed and saliva in the nasal cavities. If an empyema of the guttural pouches is the reason of regurgitation purulent discharge could be seen at the orifice of diseased guttural pouch. Endoscopy of the trachea could reveal the feed aspiration. Esophagoscopy is an important and essential part of the examination of a horse with suspect esophageal disorder. Esophageal obstruction, mucosal lesions, esophageal strictures, diverticula and other disorders can be diagnosed with this method.

Radiographic examination can visualise the cervical and thoracic esophageal region in the adult horse. It is an important diagnostic procedure in esophageal disorders other than simple obstruction. Esophagoscopy and esophageal radiographic contrast studies are necessary before surgery to precisely locate and determine the extent of the condition requiring surgical treatment. Native radiographic study will confirm the presence of radio-opaque foreign bodies and esophageal and periesophageal gas. For a complete examination, contrast oesophagography may be used. Negative contrast study can be performed by air insufflation.
through a nasogastric tube into the esophagus. Esophageal diverticula and fistula can be visualised by this method. Positive contrast studies are conducted using barium sulphate or iodinated solutions. Esophageal strictures and diverticula are best evaluated by obtaining a double-contrast oesophagogram. In cases with suspected esophageal perforation, iodine contrast instead of barium should be used. Thoracic radiography is also indicated in cases suffering from aspiration.

Esophageal disorders are connected with some complications. Common respiratory complications include aspiration pneumonia, mediastinitis or pleuritis. Esophageal mucosal ulcerations are also frequent following esophageal obstructions. Circumferencial mucosal lesions can result in esophageal strictures. Perforation of the esophagus as well as other pathologies (mural abscesses, diverticula etc.) could also follow after esophageal obstruction. Laryngeal hemiplegia and Horner’s syndrome can occur especially after esophageal surgery.

The lecture includes general discussion about esophageal disorders as well as the description of an esophageal obstruction, esophageal rupture, esophagitis, esophageal strictures, esophageal diverticula, esophageal congenital disorders, megaesophagus and esophageal neoplasia. Further information is available in a paper of Bezdekova (2012).

References and further reading


COLIC EXAMINATION

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The majority of colic cases respond quickly and completely with medical treatment such as analgesics and laxatives if appropriate. However, approximately 10% of colic cases need surgical intervention if the life of the horse is to be saved. The important decisions to be made when dealing with an acute colic case are:

1. Medical vs. surgical management.
2. Surgical management vs. euthanasia.

Fortunately, the vast majority of colic cases are trivial in nature and do respond well to analgesia. The initial assessment of a colic case should include:

**Degree of pain**

This is an important indicator of the need for surgery. Horses that show severe pain always have a surgical lesion. It may be impossible to examine the violently painful horse but the level of pain indicates the need for surgery.

**Heart rate**

An elevated heart rate (>60 beats/min) indicates dehydration and/or endotoxaemia. This can only arise from ischaemic damage to intestine so surgery is necessary. Small elevations in heart rate (40-60 bpm) may be associated with spasms of pain, but these are transient with period of normal heart rate between.

**Intestinal sounds**

If these are present, it is usually a good indicator. Endotoxaemia, distension and peritonitis will all lead to depressed gut motility. Flatulent colic cases and impactions require normal motility for gas or ingesta to progress along the intestine.

**Rectal examination**

This is an important part of any colic examination. The following abnormalities (amongst others) may be palpated: distension of large or small intestine, impaction of colon, displacement of large colon, taught taenial bands. This information is vital in deciding the nature of the horse’s underlying disease. It is sometimes possible to make an absolute diagnosis by rectal examination e.g. nephrosplenic entrapment.
Nasogastric intubation

This is both therapeutic and diagnostic. Relief of gastric distension is analgesic, but a distended stomach is also indicative of small intestinal obstruction. More than 2L of gastric reflux is abnormal.

Additional tests that may be possible in a clinic or with laboratory assistance include:

**Packed cell volume (PCV)**

This is another indicator of cardiovascular status. PCV will rise if a horse is becoming dehydrated as a result of endotoxaemia. Recent studies have indicated that even small elevations in PCV are associated with significant decrease in prognosis.

**Paracentesis abdominis**

A sample of peritoneal fluid can be very helpful in horses with colic pain but no other signs of a surgical condition. Normal fluid is clear and straw-coloured with protein content of <20g/l. Early ischaemia leads to red blood cells entering the fluid to give it an orange appearance, which gradually darkens with time. A normal peritoneal fluid sample indicates the absence of ischaemic bowel, hence the clinician is at liberty to investigate the horse in a more leisurely fashion.

No one clinical parameter is a sensitive indicator of the need for surgery. The clinician must gather all the information possible, and use it to work out the most likely cause of the animal’s colic pain. Key questions are:

- Is this horse’s level of pain consistent with a surgical lesion?
- Does the animal have evidence of endotoxaemia?
- Is there evidence of ischaemic bowel?
- Does the horse have displaced intestine that can only be corrected surgically?

If the answer to any of these questions is “Yes,” then surgery is indicated.

Medical management of acute colic relies upon analgesic drugs, with the use of intestinal lubricants and gentle exercise in some cases. Time is also important. Most colic case will resolve in less than two hours. The analgesics indicated for use in the colicing horse are:

- Non-steroidal anti-inflammatory drugs (phenylbutazone, flunixin, Buscopan compositum)
- “Spasmolytics” (Buscopan)
- Opiates (butorphanol)
- Alpha-2 antagonists (detomidine, romifidine).

One consideration when choosing analgesia is its duration of action. The use of long-lasting analgesics, e.g. flunixin, is only indicated if the cause of the colic has been determined. Often, this is not possible at a first visit so short-acting analgesics should be used. Buscopan
or butorphanol or detomidine/butorphanol are safe, effective first-line analgesics. Failure to respond to analgesia or an unexpectedly short duration of pain relief may indicate the presence of a surgical lesion.

Horses that show signs indicating the need for surgery should be referred at the earliest opportunity. This will maximise the probability of a successful long-term outcome.

Sometimes euthanasia of the colicing horse is necessary. The three most commonly encountered situations where this is appropriate are:

1. A horse showing consistent or worsening signs indicating the need for surgery, where surgery is declined by the owner.
2. A horse that is showing such severe pain, that is non-responsive to analgesia, that transport to a surgical facility would be dangerous.
3. A horse showing signs consistent with gastric rupture:
   a. Heart rate >90bpm
   b. Sweating profusely
   c. Purple membranes
   d. Reluctance to move and boarded abdomen.

Horses fulfilling one of these 3 criteria meet the BEVA guidelines for humane destruction under All Risks Mortality Insurance i.e. “The insured horse...manifests an illness or disease that is so severe as to warrant immediate destruction to relieve incurable and excessive pain and that no other options of treatment are available to that horse at that time.”
Stomach disorders in a horse include mainly ulcerative disease. Equine gastric ulcer syndrome (EGUS) is characterized by ulceration of the distal esophagus, stomach (proximal squamous or nonglandular mucosal part, distal glandular part) and proximal duodenum. EGUS represents a significant clinical and economic problem in horses due to its high prevalence, nonspecific clinical picture and negative effect on the horse’s performance.

An increase in aggressive factors including acid content, decreased pH and decrease in protective mucosal factors have been confirmed as causes for gastric ulceration in horses. Equine gastric ulcer syndrome seems to be extremely prevalent in the equine population. EGUS was first described as a serious highly prevalent disease of foals in 1964. Later, frequent occurrence of the syndrome in adult horses was also found. According to various authors, prevalence of the disease varies from 55% to 100%.

The ethiology of equine gastric syndrome is multifactorial and many risk factors have been studied including exercise, stress, stomach microbial infection, age, sex, breed, non-steroidal drug administration, horse diet and feeding management. Various results were obtained.

Clinical signs are non-specific in equine gastric ulceration. Acute and recurrent colic, diarrhoea (in foals), poor hair coat, poor appetite, weight loss, attitude changes, depression and poor performance are described in horses which suffered from gastric ulceration. Partial anorexia (poor general appetite) is one of the nonspecific clinical sign which accompany the syndrome of gastric ulceration in horses. Poor performance is frequently mentioned as a common sign of gastric ulceration, but only one study showed a direct association. Evaluation of a larger number of horses is needed to prove that poor performance is indeed a sign of equine gastric ulceration.

Ante mortem diagnosis of equine gastric ulcer syndrome is based on history, clinical signs, gastroscopic examination and treatment response.

Gastroduodenal ulceration can lead to pyloric or duodenal stenosis which is the most common cause of delayed gastric emptying syndrome (gastric outflow obstruction syndrome) in horses. Duodenal and gastric ulceration can be complicated by perforation leading to fatal septic peritonitis (especially in foals). The prevalence of duodenal perforation in adult horses is not well documented, but it appears to be low.

Current therapy for equine gastric ulcer syndrome tries to provide pain relief, ulcer healing and prevention of secondary complications. Medical treatment is based on blocking gastric acid secretion and encouraging the subsequent increase in gastric pH. This higher pH creates
a suitable environment for ulcer healing. Surgical treatment is an option for delayed gastric emptying following EGUS. Prevention of recurrence is focused on dietary management. Treating pain originating from stomach ulceration in the horse is difficult. NSAIDs (especially repeated administration) should be avoided due to their role in EGUS pathogenesis. Ulcer healing is promoted by antacid therapy. Proton pump inhibitors (omeprazole) offer a better option than H$_2$ antagonists (ranitidine, cimetidine) in stomach ulceration treatment. They can be used once daily and their antiulcer effectiveness is long acting. Omeprazole (4 mg/kg of body weight orally every 24 hours) inhibits gastric secretion in horses and it was proven to be effective in EGUS treatment and prevention. It is difficult to recommend the duration of pharmacologic treatment. Gastric ulcerations (similarly to skin wounds) in horses are individual. Endoscopic examination is recommended after two weeks of omeprazole treatment to evaluate ulcer healing.

Coating and binding agents such as sucralfate and bismuth subsalicylate can promote ulcer healing in the equine stomach. Sucralfate is the hydroxyl aluminium salt of sucrose octasulfate and in the stomach environment is converted to a sticky mass covering the mucosal lesions. Sucralfate is widely used in neonate foals where antacid therapy can lead to increased risk of nosocomial infections. The use of synthetic prostaglandin 1 (PGE 1) analog and somatostatin analog were studied in connection with EGUS. Prostaglandins would be most indicated in horses with NSAIDs administration or under stressful conditions. The cost limits their use in horses and it is contraindicated in pregnant and nursing mares because of its effect on the female reproductive tract.

Even though the role of the recently found Helicobacter-like sp. in EGUS has not been confirmed yet there are some studies focusing on the influence of antibiotics and probiotic treatment on gastric ulcer healing in horses. Probiotic preparations containing Lactobacillus sp. given to horses improved healing of spontaneously occurring nonglandular gastric ulceration. Moreover the severity of nonglandular ulceration decreased with antibiotic treatment (trimethoprim sulphadimidine administration).

The recurrence of gastric ulceration in horses is common, therefore preventive strategies for environmental, nutritional and dietary management have been developed and their effect is discussed. As is mentioned above, exercise, dietary management, stall conditions and diet are all considered as risk factors for EGUS. The protective role of alfalfa hay and the risk from a high starch diet are proven. Numerous dietary supplements have been developed for equine gastric ulcer supportive treatment but the majority of them lack scientific confirmation of their efficiency. Feeding dietary oils (corn oil) is a popular management practice for horses suffering from gastric ulceration. Some results confirmed their effect while the others did not. Dietary oils are at least the very good option for energy supplementation in horses instead of starch which is contraindicated in horses suffering from EGUS.

Despite the widespread use of antiulcer treatment, the prevalence of EGUS remains high. This could be probably due to the cost of antiulcer medicaments which leads to shorter
courses of treatment than is recommended, the administration of subtherapeutical doses or substitution of other ineffective medications or feed supplements.

**Further readings:**


CONSERVATIVE TREATMENT OF COLIC

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The horse is a hindgut fermenter with a highly specialised caecum and colon. The normal function of this organ is to ferment fibrous ingesta by microbial action producing volatile fatty acids, an essential source of energy, and large volumes of gas. The large colon is relatively mobile which allows displacement and entrapment within the abdominal cavity. This presentation will use computer graphics to illustrate some of the more commonly encountered displacements of the large intestine and discuss their surgical management.

As our understanding of large intestinal disease has increased so management of large intestinal displacements has become more conservative. Many displacements cause only a partial obstruction of the large intestine and, with patience and appropriate medication, these can resolve without the need for surgery. However, some obstructions of the large intestine do require immediate surgery if the horse is to have a reasonable prognosis for long-term survival. Indications for immediate surgery include:

- **Gross distension of the abdomen.** This indicates that a complete obstruction has occurred and this is unlikely to resolve spontaneously. Delayed surgery will allow gas distension to increase, thereby increasing the risks associated with general anaesthesia.
- **Severe pain, unresponsive to analgesia.** This is often the earliest indicator of ischaemic damage to intestine. Referring a horse for surgery at this stage maximises the probability of a successful outcome.
- **Signs of cardiovascular compromise.** Elevations in heart rate, packed cell volume and congestion of mucous membranes are indicators of ischaemic change to intestine. The earlier surgery is performed, the better the horse’s long-term prognosis.
- **Intestinal wall oedema palpable on rectal examination.** This is another indicator of ischaemic change, indicating the need for immediate surgery.

Early surgery of horses with complete or strangulating obstructions of the large intestine carries a good long-term prognosis. If in doubt, the horse’s best interests are served by early referral.

**Diseases of the large colon**

1. **Tympanic colic.** This type of colic arises through temporary obstruction of the large intestine or over-production of gas. Gas distension rapidly develops causing stretching of the intestine and its mesentery. However, so long as the underlying cause is transient in nature, colic of this type usually respond well to medical treatment. Common causes of tympanic colic include dietary change e.g. turnout onto lush pasture in Spring, over-feeding
concentrates. Some tympanic colic cases are likely to be due to transient obstruction of large intestine which corrects itself due to normal gut motility.

2. Pelvic flexure impaction. This is one of the more commonly diagnosed types of large intestinal colic in general practice. The pelvic flexure is a hairpin bend in large colon where the intestine undergoes a marked reduction in diameter. Impaction of ingesta at this site causes partial obstruction of the large intestine although gas can usually bypass the obstruction. This type of colic can be readily palpated by a veterinary surgeon on rectal examination and usually responds well to medical treatment with intestinal lubricants and softening of gut contents. Such impaction can cause low grade colic signs for several days whilst the impaction softens and intestinal motility pushes the food material along the intestinal tract. The most widely recognised cause of pelvic flexure impaction is change in management, especially the transition from an all-grass diet, to a diet high in dry fibre and readily available carbohydrate. This type of colic usually responds well to medical treatment and carries a good prognosis.

3. Left dorsal displacement/nephrosplenic entrapment. The nephrosplenic ligament is a short ligament joining the spleen to the left kidney (a pic or diagram would be good). Occasionally the large intestine migrates between the spleen and the body wall and becomes caught over the nephrosplenic ligament. Larger horses are at greater risk of this condition than smaller breeds. Constriction of the large intestine as it passes over the nephrosplenic ligament causes obstruction of the intestine. In most cases this is a simple obstruction (i.e. no damage to blood supply of the gut), but occasionally the obstruction is strangulating.

The treatment of this condition has altered over the past fifteen years. Once it was considered that surgical management was necessary in most cases in order to correct the displacement. Experience has proved that many cases of nephrosplenic entrapment will resolve spontaneously given time. Certain medical treatments and light exercise under veterinary supervision can also be useful. The underlying cause of this type of large intestinal colic is unknown.

[Image of Colon draped over the nephrosplenic ligament]

[Image of Spleen]
4. Right dorsal displacement. The large colon is highly mobile within the abdominal cavity. Abnormal migration of the colon so that it comes to lie between the caecum and the right body wall, often with a degree of volvulus (see below), is termed “right dorsal displacement.” Displacement of the colon in this fashion causes obstruction of the large intestine and gas distension. Specific causes of right dorsal displacement are unknown but it is more common in larger breeds of horse and is associated with dietary change. Although some cases of right dorsal displacement may respond to conservative management, the majority are only diagnosed at exploratory surgery and do require surgical correction. The prognosis for this type of colic is good.

5. Volvulus (torsion) of the large intestine. The mobility of the large intestine and the fact that it is only anchored to the dorsal abdominal wall at one point near its origin, allow this organ to rotate about its long axis. Rotation of 180° is probably physiological. Rotation of 270° causes simple obstruction of the large intestine but 360° of volvulus or greater causes strangulation of blood supply and death of intestinal tissue.
In its most severe form this is one of the most painful and rapidly progressive types of colic. A large intestinal volvulus of $360^\circ$ will cause rapid abdominal distension due to tympany of the gut. Endotoxeamia due to ischaemic damage to the intestinal mucosa will lead to rapid onset of dehydration and endotoxaemic shock. Horses can die from this type of colic within a few hours. If surgical treatment is to stand any chance of success it must be carried out within 1-2 hours of the onset of colic signs. Prognosis following a volvulus of less than $360^\circ$ is good. The prognosis for volvulus that is strangulating is often poor, even with surgical treatment.

The causes of large intestinal volvulus are poorly understood. However, post-parturient mares, large breeds of horse and animals with recent dietary change are believed to be at greater risk.

**6. Sand impaction colic.** This type of colic arises when sand accumulates in the large intestine of horses. It tends to occur in horses kept on poor quality pasture that have to graze close to the roots of the grass. Inadvertent sand ingestion occurs during grazing and the large intestine is unable to expel the unwanted sand. The result is impactions of sand, most commonly at pelvic flexure and in right dorsal colon. A milder form of sand colic can also occur due to the abrasive effect of sand on the large intestine. This form of sand colic can be managed medically with pain relief and anti-inflammatory drugs. Sand impactions usually require surgical evacuation. The prognosis following sand impaction colic is usually good, so long as exposure to sandy pasture and poor quality grazing is minimised. This type of colic is particularly common in dry sandy regions of the world; far less common in the UK and rest of Europe.
Endotoxaemia and Colitis in Adult Horses

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Endotoxin (lipopolysaccharide, LPS) is a structural component of the cell membrane of gram negative bacteria and is formed from 3 subcomponents: the inner lipid A (which mediates pathophysiologic effects), the core oligosaccharide and the outer polysaccharide. Endotoxin is liberated when gram negative bacteria die or rapidly multiply. As mammals have evolved to carry countless quantities of gram negative bacteria and endotoxin within their gastrointestinal (GI) tracts, effective defences against this potent “enemy from within” have also developed primarily in the form of a highly effective and exclusive GI mucosal barrier preventing passage of endotoxin into the systemic circulation. The majority of endotoxaemic cases seen in equine practice have acute GI disease associated with compromise of this barrier although gram negative infections at other sites such as pleuropneumonia or metritis may also be associated with endotoxaemia.

The innate immune response is highly sensitive for the detection of endotoxin should it escape from the gastrointestinal lumen and this results in an inflammatory cascade intended to limit the pathogenicity of perceived bacterial invasion. However, given the almost limitless quantities of endotoxin within the equine GI tract, massive systemic absorption may occur leading to excessive stimulation of mechanisms intended to prevent harm, which may then become exaggerated and injurious as a “systemic inflammatory response syndrome” (SIRS) develops.

Colitis in horses may vary from the mild and innocuous with soft faeces but no detectable systemic illness to a peracutely life-threatening endotoxaemic crisis with severe compromise of large areas of colonic mucosa. Specific aetiologic diagnosis of colitis in horses is frequently problematic although the most commonly recognised causes of endotoxaemic colitis include antimicrobial drugs, Clostridium difficile, Clostridium perfringens, Salmonella sp., starch overload and Neorickettsia risticii.

Diagnostic Methods

The diagnostic approach in a colitis case firstly involves evaluation of key characteristics that determine treatment and prognosis; and secondly, efforts to determine aetiology (e.g. faecal analysis). The most important pathologic effects of endotoxaemia and SIRS are cardiovascular and are readily detected by clinical examination (tachycardia, hypotension (poor pulse quality), dark (or pale) membranes, slow capillary refill time, cold extremities, poor urine production) and examination of blood samples (polycythaemia, leucopaenia, hyperlactaemia, azotaemia, high urine specific gravity). Additionally serum proteins and electrolytes (calcium, magnesium, potassium) are important to measure as significant losses
may also occur in colitis. Further to collection of these data for their initial prognostic and diagnostic importance, they are also useful to monitor effectiveness of treatment and guide appropriate changes in the therapeutic plan. Changes in these measurements over the course of 4-12 hours of initial therapy is a better predictor of final outcome and it follows that almost all endotoxaemic colitis cases warrant, at least, initial attempted treatment (depending on financial constraints).

**Treatment**

Endotoxaemia is a medical emergency and requires prompt and aggressive therapy. There are 3 fundamental arms of treatment: to counter the effects of circulating endotoxin; to neutralise circulating endotoxin; and to reduce the ongoing entry of endotoxin into the circulation.

**Counter effects of circulating endotoxin**

Many clinical effects of endotoxin are mediated by prostaglandins and therefore NSAIDs play a useful role in moderating the clinical signs of endotoxaemia. Flunixin (0.25 mg/kg q 6 hours IV to 1.1 mg/kg q 12 hours IV) remains very popular although COX2-specific agents such as firocoxib (0.1 mg/kg q 12-24 hours IV) might be used with possible benefits to mucosal recovery. Further products that might interfere with the inflammatory cascade initiated by endotoxin include pentoxyfylline (10 mg/kg q 12h PO) or DMSO (0.02 - 1 g/kg as 10% solution IV). Recent work describing the effects of ethyl lactate infusion (150 mg/kg IV) raises the prospect of this product being clinically useful also.

Plasma expansion is vital to counteract the hypotensive effects of endotoxaemia along with fluid and electrolyte losses from diarrhoea. Hypertonic (7.2%) saline (2-4 mL/kg IV bolus) can be remarkably effective in rapidly improving circulatory status and should be immediately followed by at least 5 times the volume of isotonic fluids (preferably Hartmann’s/lactated Ringers). Protein losses are commonly seen as a result of mucosal damage and colloid replacement is important to hold fluids in the intravascular compartment. Plasma transfusion is, in this author’s view, clinically the most effective colloid although access to multiple donors is useful (e.g. 5-20 L plasma required for adult horse). Synthetic colloids such as hetastarch (10 mL/kg IV) or pentastarch (10-15 mL/kg IV) might be more readily available although their clinical benefit may be less.

Endotoxin has strong procoagulant effects which are sometimes clinically evident as venous thrombosis and increased bleeding following consumption of platelets and coagulant proteins. Use of plasma and also low-molecular-weight heparins (dalteparin 50 IU/kg or enoxaparin 0.4 mg/kg q 24 hours SC) may be useful to prevent such problems.

**Neutralising circulating endotoxin**

“Hyperimmune plasma” containing anti-endotoxin antibodies against an *E. coli* mutant (J5) or *Salmonella typhimurium* and may bind and encourage removal of circulating endotoxin (e.g. 1-2 L per 500 kg). The lipid A segment of endotoxin may also be attracted and bound by Polymixin B (2000-6000 IU/kg q 12 hours IV) or exogenous lipid infusion.
Reducing ongoing entry of endotoxin into the circulation

Specific treatment of any suspected aetiologic agents (e.g. metronidazole therapy for *C. difficile* colitis) is clearly key in limiting further entry of endotoxin. Additionally various binding agents might be used for their possible effects on enteric endotoxin and other bacterial toxins such as di-tri-octahedral smectite (Biosponge) (3 g/kg followed by 1 g/kg q 6-8 hours PO) and even mineral oil (e.g. 1 L per 100 kg by nasogastric tube). Misoprostol, a prostaglandin E agonist (5 microg/kg q 12 h PO), might also be used for its possible beneficial effects on the mucosal barrier. Lidocaine infusion (1.3 mg/kg loading dose followed by 3 mg/kg/hour infusion) may have a double benefit of reducing enteric inflammation and providing analgesia.
Acute abdominal pain is common clinical problem in foals. Foals are less tolerant to pain than adult horses and they may behave colicky even in non-intestinal disorders. It makes colic evaluation in the foal very difficult. Simple meconium retention or enterocolitis can be accompanied by violent colicky behaviour in neonate and they do not require surgical approach. The decision of need for referral or surgery is based on several factors including the definitive or possible diagnosis, practitioner facilities (knowledge, time, equipment) and the cost of planned surgical treatment and/or intensive care.

Clinical signs of colic in a foal are diverse. The abdominal cavity can be distended, the foal can show diarrhoea (even in strangulating lesions), feces can contain blood, the foal can show straining (tenesmus). Foals can be depressed and anorectic, bruxism can be presented (together with hypersalivation in gastric ulcers), rolling and kicking and keeping dorsal recumbency can be seen. Tenesmus can be the clinical signs in intestinal (dorsal flexed back) and urinary disorders (ventrally flexed back).

The colic diagnostics in a foal is based on the history, examination results and the treatment response. Further diagnostic in a foal is usually applied after the pain therapy. The pain treatment in the foal differs from adult. The first choice in meconium impaction (being the commonest reason for colic in neonate) is butylscopolamine. Nonsteroidal anti-inflammatory drugs should be avoided in colicky foal, because of possible adverse effect. If needed metamizol and ketoprofen are used. Pain killers commonly used in neonates are short acting opioids (butorphanol). The influence of peristaltic is minimal. The further colic treatment depends on underlying cause.

In colicky foal, the history must be obtained and important data are the term and date (hour) of delivery, colostrum intake, meconium passing, the time of colic onset, degree of pain, urination, diarrhoea presence and previous treatment.

The physical examination in a colicky foal differs slightly from adults. Respiratory rate should be measured from distance if possible. Heart rate is easily increasing under the stress conditions including clinical examination itself. The rectal temperature can help to differentiate septic or inflammatory problems. In a foal, there is a need of detailed umbilicus, joints, ribs and scrotal region palpation. Rectal examination is replaced by gentle digital palpation and abdominal cavity palpation. Mucous membranes examination is important. Together with capillary refill time, pulse quality and extremities temperature it helps to evaluate regional blood perfusion and possible need of infusion therapy. Auscultation of gut sounds is also important.
The diagnostic imagining is very useful tool in colicky foal and partly replaces complete rectal examination in adults. Ultrasonography and radiography can reach the final diagnosis and can be very helpful in the decision of surgical treatment (small intestinal volvulus, intussusception, peritonitis, uroperitoneum). Blood work can be also very useful in colicky foal. The basic hematology, biochemical panel, arterial blood gas analysis, IgG level and peritoneal fluid analysis are performed.

The basic differential diagnosis of colic in a foal is:

**Congenital disorders:**
- Hernias (umbilical, diaphragmatic, scrotal, inguinal)
- Atrezia ani, coli, recti
- Ileocaecal agangliosis

**GIT obstructions:**
- Meconium impaction
- Intestinal volvulus
- Intussusception
- Colon impaction
- Large colon displacement
- Pyloric and duodenal stenosis
- Ascarid impaction

**Other causes:**
- Gastric and duodenal ulceration
- Enteritis
- Enterocolitis
- Uroperitoneum
- Gastric or intestinal rupture
- Peritonitis
- Abdominal abscess

Further reading:


http://www.vet.ohio-state.edu/assets/courses/vm70016/foalscolic.pdf

ROLE OF PARASITES IN EQUINE COLIC

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1. Large strongyles

- *Strongylus vulgaris* has a migratory lifecycle. L3’s travel through caecal and colonic mucosa to the cranial mesenteric artery. Damage to the arterial intema leads to thrombus formation and occlusion of arteries.
- In the 1980’s, the widespread use of ivermectin did lead to a massive reduction in prevalence of *S. vulgaris*. Presently, this parasite represents less than 2% of the equine strongyle burden.
- In spite of the successful control of this parasite, there has probably been little change in the incidence of colic!

2. Small strongyles

The cyathostomins are a large and varied family of parasites with more than thirty different sub-species. These can be differentiated morphologically, but only with difficulty. As the epidemiology of these different species only varies slightly they can conveniently be grouped together as “cyathostomins.” The term “mixed strongyle infection” is used when no attempt is made to differentiate between large and small strongyles (e.g. faecal strongyle egg counts).

- Resident in the lumen or mucosa of the caecum and large colon.
- Larval stages can become encysted in the mucosa and undergo hypobiosis.
- Widespread benzimidazole (BZ) resistance amongst this group of parasites.
- Mass emergence of encysted stages can cause severe disruption of the colonic mucosa leading to the clinical condition of larval cyathostominosis (Mair et al. 1990, *Vet Record* 126, 479-481).
- Risk factors for larval cyathostomosis investigated by Reid et al. (1995, *EVJ* 27, 127-130). Winter time, recent use of anthelmintics and age <6yrs are all risk factors for this disease.

Cyathostomin burdens have also been associated with colic. Uhlinger, in the USA, conducted a cohort intervention study over five years (*EVJ*, 22, 251-254). The study looked at five horse “farms” and measured the incidence of colic on each farm during each year of the study. Anthelmintic prophylaxis varied on each farm during this five year period (e.g. no treatment; regular BZ use; regular ivermectin use).
3. Tapeworms

*Anoplocephala perfoliata* is the most important tapeworm of the horse. This parasite has been the subject of much speculation and a little research (including my own). The parasite is unusual because of its predilection for the ileo-caecal junction. Many case reports implicate it in the aetiology of intestinal accidents at this site, but is the relationship circumstantial or causal?

- The pathological changes associated with tapeworm attachment have been shown to be proportional to the number of parasites present (Fogarty *et al.*, 1994, *Vet Record* **134**, 515-518).
- The same study showed no seasonal effect on parasite numbers.
- A case-control study of over 100 spasmodic colic cases and matched control indicated an increased risk of this type of colic in the presence of tapeworms (Proudman, French and Trees *Equine veterinary Journal* **30**, 194-199.).

A serological assay for tapeworm-specific antibody has been developed. This assay has been used, along with conventional coprological assays, to investigate tapeworms as a risk factor for. An association between tapeworm infection and two different types of colic has been demonstrated. Spasmodic colic (mild colics, readily responsive to medical treatment) and ileal impaction colic have been shown to be associated with tapeworm infection. Furthermore, a dose-response relationship was evident when the tapeworm antibody ELISA was used to evaluate infection intensity. This means that the risk of spasmodic colic increases with the level of tapeworm infection.

![Graph demonstrating the dose-response relationship between tapeworm infection and spasmodic colic.](image-url)
The take-home message from these studies is that every case of tapeworm-associated colic is potentially preventable. Tapeworm control is an important means of preventing colic.

4. Parascaris equorum

This parasite is often present in large numbers in young horses (up to the age of 4 years). The large size of the mature adult, which resides in small intestine, can cause intra-lumenal obstruction. This has been associated with recent anthelmintic use. Prevention of ascarid-associated intestinal disease is dependent upon minimising the number of parasites present in young animals. This can be done by treating last year’s young stock to minimise environmental contamination, or by regular treatment of foals and young animals to prevent the accumulation of large numbers of worms.

A worrying recent development is the finding of ivermectin resistance in this parasite in The Netherlands and USA.

5. Oxyuris equi

This colon-dwelling parasite has been associated with small colon impaction. The parasite is susceptible to all anthelmintics used for equine treatment.

6. Mechanisms of intestinal disturbance by helminths

1. **Mechanical obstruction** (eg. *Parascaris equorum, Anolpocephala perfoliata*).


3. **Inflammation or the effects of inflammatory mediators.** Mason et al. (1996, *Parasitology* 113, 173-182) demonstrated that in an experimental *N. brasiliensis* model the parasite induces a change in neuronal regulation from cholinergic to one dependent upon substance-P, a pro-inflammatory neuropeptide. Could this be a mechanism by which *A. perfoliata* or encysted cyathostomes cause disease?

4. **Vascular occlusion causing ischaemia or infarction.** The mesenteric arteritis caused by *S. vulgaris* falls into this category. The migratory stages of large or small strongyles may also affect the small blood vessels of the caecal and colonic bowel wall. Sellers et al. (1982, *Am. J. Vet. Res.*, 43, 390-396) showed a 65% decrease in colonic blood flow in ponies experimentally infected with *S. vulgaris.*
INFLAMMATORY BOWEL DISEASE IN THE HORSE

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Inflammatory bowel disease (IBD) represents a poorly characterised group of conditions that may present with colic, weight loss, diarrhoea, oedema and lethargy. IBD has generally been categorised according to the predominant inflammatory cell type in the intestinal wall: eosinophilic, lymphocytic-plasmacytic, granulomatous and mixed. Intestinal infiltration by eosinophils may be further subdivided into diffuse eosinophilic enteritis (DEE) characterised by abnormal eosinophilic infiltrates found diffusely within the wall of small and/or large bowel; focal/multifocal eosinophilic enteritis (FEE) characterised by focal nodules or circumferential mural bands of eosinophilic inflammation affecting the small or large bowel; and multisystemic eosinophilic epitheliotropic disease (MEED) involving abnormal eosinophilic infiltrates in intestine and other organs. Of 75 horses diagnosed with IBD at Liphook Equine Hospital (1999–2007), 36% had mixed lymphocytic-plasmacytic/eosinophilic enteritis (LP/EE); 31% had DEE; 19% had FEE; and 15% had lymphocytic-plasmacytic enteritis (LPE).

Generally cases of eosinophilic enteritis (DEE or FEE) in the literature have presented with abdominal pain rather than weight loss, whereas horses with LPE have presented with weight loss, diarrhoea and colic. In the case series from Liphook, there was an association between eosinophilic infiltrates and colic (especially acute colic) (Figure 1a) and between lymphocytic-plasmacytic infiltrates and diarrhoea and weight loss (Figure 1b). Nevertheless colic was still the predominant clinical sign in all IBD subtypes and several horses with eosinophilic infiltrates were affected by weight loss and diarrhoea.

Fig. 1. Association between IBD subtypes and history of a) colic; and b) weight loss and diarrhoea (n=75).
Diagnosis

The only serum biochemical markers found to be abnormal in more than 50% of cases tested were hypoalbuminaemia (<26 g/L) which was found in 22/39 (56%) horses and increased serum amyloid A (>4 mg/L) which was found in 10/14 (71%) horses. Increased serum alkaline phosphatase was seen in only 6/24 (25%) horses in which it was measured. An oral glucose absorption test (OGAT) indicated total or partial malabsorption (peak <85% above baseline glucose) in 9/15 (60%) cases involving the small intestine. The results of OGAT did not correlate with serum albumin or degree of intestinal inflammation. An increased total nucleated peritoneal fluid cell count (>5 x 10^9/L) was found in only 3/39 (8%) horses although peritoneal protein concentration (>20 g/L) was increased in 10/39 (26%) horses. Peritoneal inflammation appeared to be associated especially with eosinophilic infiltrates.

Intestinal thickening (>4mm) was identified ultrasonographically in 11/20 (55%) cases. Definitive diagnosis of IBD necessitates biopsy. Minimally invasive techniques include duodenal mucosal and rectal mucosal biopsy although success of both techniques depends upon disease extending into the proximal and distal extremes of the intestinal tract respectively. Biopsy size and collection artefact may also present problems especially with duodenal biopsy. The diagnostic usefulness of full thickness biopsies is likely to be greater and the cases selected for the Liphook study had all been subject to this procedure. Visible inflammation and palpable thickening were only identified in a minority of cases at surgery demonstrating the importance of histopathological examination in the diagnosis of IBD.

Knowledge of normal cell distributions is a prerequisite to histopathological diagnosis and classification of IBD. Quantification of leucocyte populations in normal equine intestine has been published although appears to be rarely referred to. Given the difficulties in distinguishing pathological infiltration from normal variation it is possible that eosinophilic, lymphocytic and plasmacytic cellular infiltrates are over-interpreted and further quantitative and objective criteria for diagnosis are required.

<table>
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Table 1. Absolute numbers of leucocytes per mm^2 of lamina propria in 14 normal horses (adapted from Packer et al 2005, J Comp Pathol. 132:90-5).

Treatment

The most common treatment for equine IBD is prednisolone administered orally (1-2 mg/kg sid). Lack of response to initial therapy often leads to an increasing dose of prednisolone or a change to oral or parenteral dexamethasone (0.05-0.1 mg/kg q 24-48 h). The treatment period is rarely less than 3 months and can be as long as 2 years (6-9 months most frequent).
Prognosis

Thirty four (61%) of 56 horses for which follow-up data was available were alive at 12 months. Prognosis was favourable for horses with DEE and LP/EE (70% survival) but guarded for LPE and FEE. Previous reports of horses with FEE have documented better survival rates when the intestine was decompressed but not resected. The low survival rate for FEE in the Liphook cases (3/8, 38%) might be associated with the fact that all cases underwent intestinal resection. In previous reports LPE has been associated with a hopeless prognosis in contrast to the Liphook study which found that 6/11 (55%) of LPE cases were still alive 12 months after diagnosis. Of the horses in all groups which survived, 25% (9/36) continued to suffer at least one episode of colic and 6% (2/36) had other problems such as failure to gain weight. Horses with moderate or marked villous atrophy were less likely to survive than those with mild or no villous atrophy (p = 0.03). Villous atrophy was more likely to be present in the FEE (8/12) and LPE (5/11) groups than in the DEE (6/23) and LP/EE (7/27) groups.
DISORDERS OF THE SMALL COLON AND RECTUM

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The small (descending) colon in the horse may be affected by simple obstructions, vascular and strangulating lesions.

1. Simple obstructions

Meconium retention

Meconium retention, the most common colic in newborn foals, is described elsewhere.

Diffuse small colon impaction

Diffuse obstructive lesions of the small colon have been associated with certain predispositions: breed (pony, American miniature horse), female gender, and higher age. Possible aetiology includes colon dysfunction and submucosal oedema (possibly *Salmonella* infection), decreased water intake, poor dentition with inadequate mastication, poor quality roughage and lack of exercise. Horses with small-colon impaction have mild colic with a scant amount of faeces. Findings on rectal examination include a tubular small colon filled with firm faeces. Small-colon impaction may be treated with medical therapy (oral rehydration therapy, intravenous fluid, enemas, NSAID); in some cases surgery may be necessary. The prognosis is usually favourable.

Focal intraluminal obstructions

Focal obstructions of the small colon are caused by faecaliths, phytoconglobates, enteroliths and ingested foreign bodies. Faecaliths are hardened masses of faecal material resulting from poor mastication or digestion of hay. They have often been reported in foals and young horses, especially in American miniature horses and ponies. Similar obstructive masses are phytobezoars and trichobezoars. Phytoconglobates are concretions of matted plant residues formed into balls. Enteroliths are common in specific geographic areas. Foreign body obstructions of the small colon are uncommon, being caused by ingestion of ropes, hay nets, rubber fencing etc. The diagnosis of focal obstruction can be made by transrectal palpation, in ponies and miniature horses with the aid of diagnostic imaging. Only a few cases respond to medical treatment and an exploratory celiotomy is usually necessary.

2. Vascular and strangulating lesions

Strangulation-obstruction of the small colon is an uncommon condition. The literature includes reports of cases in which small colon was entrapped by ovarian pedicle, teratoma, mesenteric rent, strangulating lipomas. Other less common lesions are volvulus, herniation
and intussusception. The clinical signs are associated with an acute obstruction of the small colon and lead to acute faecal stasis, abdominal distension and colic. Absence of faeces in the rectum and large colon tympany are typical findings; rectal palpation is often painful. Treatment is surgical.

**Mural lesions**

Mural lesions are represented by oedema of the terminal small colon and submucosal haematomas. Submucosal oedema may develop at the junction of the small colon and rectum, causing mild colic with protracted course. The caudal part of the oedema may be reached rectally by fingertips. Conservative treatment is successful in some cases but others require surgical treatment. Similar obstruction may be caused by intraluminal submucosal haematoma. Treatment of choice is surgery which usually requires resection of the affected segment.

**Lesions associated with parturition**

Mares in the second stage of parturition may incur intestinal and mesenteric contusion in the small colon area without disruption of continuity or with a separation of the mesentery from the intestine leading to ischemic necrosis of the bowel. Conservative treatment is valueless and operative treatment is very difficult.

**The equine rectum can be affected by rectal tears, rectal prolapse, perirectal abscesses and neoplasia.**

1. **Rectal tears**

Rupture of the rectum occurs most commonly as a complication of rectal examination, mating accident, enema, dystocia or external trauma. Spontaneous tears were also described. Rectal tears can be divided into four grades: Grade I – only mucosa and submucosa are torn; Grade II – only muscular layer is disrupted causing the mucosa and submucosa to prolapse through the muscle defect; Grade III – involves all layers except the serosa (grade IIIa) or mesorectum (grade IIIb); Grade IV – involves all layers.

Clinical signs depend on the severity of the tear. Horses with grade I and II tears may show mild colic, horses with grade III and IV tears rapidly develop colic, fever and depression. As the treatment and prognosis are dependent on the type of rectal tear, an accurate classification should be made. It may be enhanced by endoscopy.

Grade I tears may be successfully managed medically (antibiotics, NSAID, tetanus antitoxin and laxative diet). Horses with a grade II tear are treated similarly but antimicrobial therapy is unnecessary. Grade III and IV tears require emergency management to prevent further damage to the rectum (rectal packing with a rectal tampon or repeated manual evacuation of the rectum). The techniques described to surgically treat rectal tears include primary closure through the rectal lumen or via celiotomy, colostomy or insertion of a temporary rectal liner.
The prognosis is guarded. Grade IV tears usually result in peritoneal contamination justifying euthanasia.

2. Rectal prolapse

Causes of rectal prolapse are straining from constipation, diarrhoea, neoplasia, dystocia, urethral obstruction or colic. In type I only the rectal mucosa and submucosa project through the anus. Type II represents a complete eversion of all or a portion of rectal ampulla. In type III a variable amount of small colon intussuscepts into the rectum in addition to a type II prolapse. In type IV the peritoneal rectum and variable length of the small colon prolapse through the anus. The presentation of a prolapse is a mucosal mass (types I, II, III) or tube (type IV) protruding beyond the anus. Colic and peritonitis develop with type III and IV. Treatment depends on the type of prolapse and health of the exposed tissue. If the prolapsed mucosa or ampulla recti cannot be simply reduced, submucosal resection or amputation is necessary. The prognosis is favourable with prolapse types I and II, guarded in types III and IV.

3. Perirectal abscess

Possible causes are rectal puncture or tear, inflammation or gluteal abscess. The most common signs are low-grade abdominal pain, reduced faecal production, tensemus and fever. The abscess can usually be palpated as a firm mass inside the rectum. Ultrasonography is helpful. Depending on the location, the abscess can be drained into the anus, rectum or vagina and post-operatively flushed.

4. Perirectal neoplasia

The most common neoplasms of the perineal region and anus are squamous cell carcinomas and melanomas. Treatments include surgical excision, cryosurgery, laser surgery, radiation therapy, immunotherapy, chemotherapy or a combination of these methods.
Liver failure in the horse results in recognisable clinical signs due to compromise of its normal diverse functions. However, many cases of liver disease are subclinical or result in very mild and vague clinical signs but nevertheless are deserving of further investigation rather than simply waiting for the case to deteriorate (and have a worse prognosis) before intervening.

Biochemical substances measured in the blood of suspected hepatopathy cases can be subdivided into those reflecting damage to liver cells and those reflecting impaired liver function. Increased serum concentrations of several intracellular enzymes may reflect hepatic injury including alanine aminotransferase (ALT), alkaline phosphatase (AP), arginase, aspartate aminotransferase (AST), gamma glutamyltranferase (GGT), glutamate dehydrogenase (GLDH), iditol dehydrogenase (IDH) and lactate dehydrogenase (LDH). Increased serum concentrations of liver enzymes arise primarily from damaged liver cells which may or may not recover and therefore enzyme increases are not always predictive of severity and prognosis of the hepatopathy. Serum concentrations of other biochemical substances have been reported to reflect the capability of the liver to perform its normal functions. These are endogenous and exogenous substances which accumulate in the blood as a result of failure of extractive and processing functions normally performed by the liver and other substances whose serum concentrations are normally maintained by hepatic biosynthesis. They include various amino acids, ammonia, bile acids, bilirubin, fibrinogen, globulins, glucose and urea. Additional functional biochemical tests include blood clotting times, half-life of plasma bromosulphthalene (BSP), indocyanine green (ICG) and radionucleides (eg. $^{99m}$Tc-mebrofenin). These functional analytes might be more closely associated with the remaining mass of functional liver cells and therefore better reflect remaining hepatic function in comparison to estimation of serum liver-derived enzymes. Abnormalities of these functional parameters might therefore be more useful in the differentiation of hepatic failure from cases of adequately compensated hepatic disease and consequently have both diagnostic and prognostic value.

The liver is easily imaged using ultrasound immediately below the ventral margin of the lung. Most liver is situated on the right and is typically imageable via at least 5 intercostal spaces (ICSs) somewhere between the 6th and 15th ICS as a relatively hypoechoic triangular structure superficial to the bright highly echogenic are of the colonic mucosa-ingesta interface. A smaller area of liver is imageable from the left side via 2 to 4 ICSs immediately caudal to the heart and cranial to the spleen and stomach somewhere between 6th and 9th ICSs. When imaged from the left side the liver should not be confused with the caudomedially adjacent...
and overlapping spleen which has similar ultrasonographic architecture but is significantly more hyperechoic than the liver and has fewer blood vessels.

Although occasional abnormal features are identified ultrasonographically, the primary usefulness of hepatic ultrasonography is to guide liver biopsy and a site is usually chosen based on thickness of imaged hepatic tissue, absence of large blood vessels and, occasionally, focal presence of abnormal hepatic tissue. If liver disease is suspected on the basis of preliminary non-invasive tests then liver biopsy remains the ‘gold-standard’ technique by which to establish diagnosis, prognosis and appropriate therapy. The widespread availability of diagnostic ultrasound makes the ongoing use of unguided biopsy techniques questionable.

The main theoretical adverse effect of liver biopsy in the horse is haemorrhage although in the author’s experience this is extremely rare, especially when the technique is performed under ultrasonographic guidance. The requirement for pre-biopsy coagulation assessment is questionable and is not performed by this author. Nevertheless when collecting a liver biopsy from a horse with known coagulopathy then pre-treatment with 2-10 L of freshly sedimented plasma or whole blood might be wise.

A biopsy scoring system was developed to attribute a prognostically useful broad comparative index of histopathologic severity (table 1). Biopsy scores from 73 cases showed a strong and statistically significant association with survival and survival times. Essentially, horses with scores 0-2 have a good prognosis; horses with scores 8-14 have a poor prognosis; and horses with scores 3-7 have a guarded prognosis and deserve reasonably aggressive therapy and close further monitoring.

<table>
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<td>2</td>
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<tr>
<td>Biliary hyperplasia</td>
<td>-</td>
<td>-</td>
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*Table 1. Biopsy scoring system. Minimum score = 0, maximum score = 14*

(From Durham et al 2003. EVJ 35, 534.)

Specific therapy can only be applied to cases with a specific diagnosis, which is invariably as a result of liver biopsy. It is common for the biopsy to fail to identify the precise aetiology of liver disease, although in such cases logical therapy can nevertheless be applied based upon histopathologic patterns. For example marked lympho-plasmacytic infiltrates might indicate prednisolone, biliary neutrophilic infiltrates may indicate antimicrobials, fibrosis may indicate prednisolone, pentoxyfylline or vitamin E, haemosiderosis may indicate phlebotomy etc…..

Dietary management of hepatic insufficiency is a delicate balance between provision of adequate nutrients to limit catabolism but at the same time preventing excess nutrient supply that may precipitate encephalopathy. Free access to fresh grass or grass hay is advisable.
Provision of supplementary feed divided into at least 4 to 6 daily meals also appears sensible. Excessive dietary protein should be avoided but low protein diets may be equally undesirable as they result in catabolism.

Micronutrients with a particular capacity for hepatic insult include iron, copper, manganese and vitamin A and therefore supplements should be free from these substances. Zinc supplementation may be helpful as well as the fat soluble vitamins D, E and K.