

The gastrointestinal system

11

Introduction including body condition scoring	11.1
Debility and poor body condition – Approach to the thin equid	11.2
Diagnostic aids for examination of the GI system	11.3
Conditions of the mouth and oesophagus	11.4
Colic – A practical approach to diagnosis and treatment	11.5
Diarrhoea in adult equids	11.6
Conditions affecting the rectum (prolapse and perforation)	11.7
Peritonitis	11.8
Liver disease	11.9
Case study – Impaction colic caused by foreign material consumption	11.10
References	11.11



11.1

Introduction including body condition scoring

The ability of the working equid to digest and absorb nutrients (and ultimately gain energy to work) depends on a well-functioning gastrointestinal (alimentary) system. It is necessary to consider all aspects of the gastrointestinal tract (GIT), the teeth, mouth, oesophagus, stomach, small and large intestines, caecum and rectum, as disruption to any part of this system can be very debilitating.

Symptoms of a problem with part or all of the GIT include the following:

- Poor body condition
- Dropping food from mouth 'quidding' (see Chapter 10 The Teeth)
- Weight loss
- Inappetance
- Colic
- Diarrhoea
- Impaction/constipation
- Rectal prolapse

Taking a 'GIT' history

Ask specific questions regarding the GIT if it is suspected that this is the system affected:

- Eating patterns/appetite altered?
- Dropping food/pain?
- Excessive salivation?
- Weight loss?
- Faecal consistency and colour – diarrhoea/constipation?
- Signs of colic?
- Other animals showing the same signs?
- Type of feed/feeding pattern changed?

Body condition scoring (BCS)

This is an important assessment to make; a lot of information can be gained about the health of the animal and the quality of the husbandry it receives from the owner. It can also be used as a monitoring tool to assess progress in poorly conditioned animals.

Body condition score is an estimation of the fat and muscle coverage on the animal's body.

Procedure

- View the animal from at least two positions in order to assess the body condition.
- Stand approximately 3 metres away from the animal, facing towards its side.
- Stand approximately 3 metres away from the animal, facing towards its tail.

Horses

A number of BCS systems are described. Carroll and Huntington (1988) described a system with six categories (0 = very poor, 1 = poor, 2 = moderate, 3 = good, 4 = fat, 5 = very fat). The paper describes the body parts to be observed and the conditions for each score. The neck, back, ribs and pelvis are the main indicators.

Henneke et al. (1983) used a system of nine categories (1 = poor, 2 = very thin, 3 = thin, 4 = moderately thin, 5 = moderate, 6 = moderately fleshy, 7 = fleshy, 8 = fat, 9 = very fat) and recommend palpating the following body parts: neck withers, back crease, tail head, ribs and behind the shoulder.

Donkeys

Vall et al. (2003) reported the use of a body condition scoring system for donkeys with four categories (1 = emaciated, 2 = thin, 3 = average, 4 = good) considering the appearance of the hindquarters, ribs and spine.

When body condition scoring donkeys, consider the following:

- Donkeys have a deceptively large belly.
- Fat pads: these are large uneven lumps which vary in position on the animal (e.g. neck, hindquarters and belly).
- Do not take these features into account when scoring body condition. Use the other descriptors to decide on a score.

Repeatability

The main consideration when choosing a BCS system is to select one that adequately describes each score so that it is repeatable between different observers and the same observer on different occasions. Thus individual animals and groups can accurately be assessed for improvements or deterioration in BCS.

Both Carroll and Huntington (1988) and Vall et al. (2003) stated that their systems were repeatable and reproducible.

BCS illustrations

For pictures illustrating BCS refer to the papers mentioned above.

11.2

Debility and poor body condition – Approach to the thin equid

Debilitated equids are commonly presented for treatment (Figure 11.2.1). Conduct a clinical examination to identify the underlying cause(s) and treat appropriately. Remember to obtain a thorough history about the animal's husbandry, nutrition, workload and any previous health problems (see Chapter 1).

ENERGY INTAKE < ENERGY OUTPUT = WEIGHT LOSS AND DEBILITY

When this imbalance is prolonged, body reserves of fat and muscle are used to provide energy, resulting in a thin, weak animal.

Causes of reduced energy intake

- Insufficient or poor quality food provided (most common) (Finkler-Schade 2007)
- Inability to take in food properly, eg. overgrown teeth, sore mouth
- Inability to digest food properly, eg. dehydration, diarrhoea, worms
- Inability to metabolise food properly, eg. liver disease

Causes of excessive energy output

- Overwork (most common) (Maranhão et al. 2006)
- Concurrent disease, pain or fever (Dobromylskyj et al. 2000, Almeida et al. 2008)
- Cold environment leading to excessive loss of body heat
- Lameness (Weishaupt et al. 2004)

Principles of management of thin equids

- 1 Identify underlying cause Often both health and management issues are involved so it is important to identify all issues and communicate these to the owner. Where possible, a blood smear should be taken to look for signs of anaemia and blood parasites (see Chapter 16).
- 2 Communicate with the owner Discuss improvements which could be made to the energy content of the animal's diet, using locally available feed, to ensure adequate quality and quantity. Be realistic about what the owner can afford. Emphasise that reducing the animal's workload is essential if it is to regain condition.

Feeding sick equids

Sick animals have higher energy requirements than healthy ones.

They may also have a reduced appetite (Dobromylskyj et al. 2000, Almeida et al. 2008) and poorer ability to digest food efficiently. Aim to give small volumes of high quality, digestible food 5–6 times per day.

Stimulate the appetite with small meals of green fodder; this acts as an appetite stimulant, improves gastrointestinal function and provides vitamins, minerals, energy and protein. Adding a cup of vegetable oil to each feed increases the energy content.

If eating is painful or difficult, feed (such as grain/cereals) can be soaked in water or cooked to make it easier to eat, and digest. Some sick equids may need to be fed by hand.

A sick animal will not eat well if it is nervous or threatened by a more dominant animal next to it. Feed sick animals separately from healthy ones to prevent bullying and ensure that they receive their share.



Figure 11.2.1 Weight loss and debility in a working equid.

Ensure that fresh water is available at all times.

Feeding foals

Foals grow at an extremely fast rate especially in the first 6 months of life; daily weight gains of over 1 kg a day are common in this period (Coleman et al. 1999). Before any milk replacer is offered (Stoneham 2005) it is absolutely essential that all foals have suckled within the first 6 hours of birth so that adequate colostrum is consumed containing a rich mix of antibodies which helps to protect the neonate from disease (Naylor 1979).

Foals are very dependent on their mother's milk during the first few weeks of life. It supplies all their needs up to 6 weeks of age (Ousey et al. 1997).

The mare's milk is the main source of nutrition until 4–5 months of age. Foals are born with a monogastric GIT and do not have the capacity to digest fibre in the first few months of life – this develops as the foal grows. Some foals will eat the mare's droppings in the first 2 months of life and this coprophagia is thought to help populate the hindgut with bacteria, provide some nutrients, provide exposure to pheromones for growth and other substances which may increase the gut immunity and maturation of the nervous system (Crowell-Davis and Houpt 1985).

Foals suckle very frequently – up to 7 times a day in the first week of life.

This frequent suckling means it is very important a foal is left with the mare (Carson and Wood-Gush 1983). This is also essential in building the mother-foal relationship which is

11 THE GASTROINTESTINAL SYSTEM

important for good welfare. Owners should be fully educated about this requirement and a mare with a foal should not be worked.

Feeding lactating mares

Lactation places a huge drain on nutrient requirements – especially for energy, water, protein, calcium and phosphorus.

Reports state lactating mares require 65% more feed than pregnant mares (Boulot et al. 1987).

This should be accounted for in the feeding of the mare and her body condition should be carefully monitored. A nutritional deficiency in lactation will not only affect the health of the mare but will also reduce the quality of the milk and, therefore, affect the health of the foal.

11.3 Diagnostic aids for the examination of the gastrointestinal system

As with any diagnostic aids, use of the following has the potential to cause harm or even death to the animal if not done correctly. Rectal examination and abdominocentesis are usually only done in cases of severe colic and so will be found under the Colic section (11.5) of this chapter.

Mouth speculum for examining the teeth and oral cavity

See Section 10.4 on teeth and dentistry.

Auscultation

Both left and right sides of the abdomen should be auscultated; about 4–5 minutes is the time required to do this properly in a suspected GIT case.

Normal gut sounds

- ‘Rumbling’, ‘bubbling’ and ‘splashing’ noises can be heard on auscultation of the abdomen.
- Ileocaecal sounds should be heard in the right paralumbar fossa (see Section 1.4 for this location, and Figure 11.3.1) at a rate of 1–3 a minute; this sounds like water flowing down a drainpipe.
- Borborygmi are small intestinal sounds in the ventral abdomen, low-pitched fluid sounds.

Abnormal gut sounds

- Increased ‘rumbling, bubbling and splashes’ indicate possible spasmodic colic.

- Absence/decreased gut sounds are a poor sign (Orsini 2011), indicating ileus associated with possible obstruction or torsion.
- A 'pinging' sound on percussion indicates gas accumulation. This is significant if there is associated abdominal distension and may indicate an obstruction or impaction.

Repeated auscultation over some hours is vital when monitoring a colic case. A progressive decline in frequency or intensity of gut sounds indicates a poor prognosis, especially if the other clinical signs are also deteriorating.



Figure 11.3.1 Auscultation of the right paralumbar fossa.

Anti-spasmodic drugs (e.g. hyoscine-N-butyl bromide/N-butylscopolammonium bromide) can be used for analgesia in various types of colic: spasmodic, tympanic and simple impaction.

Never administer anti-spasmodic drugs in cases of impaction associated with ileus (Bertone 2002).

Faecal examination

It is important to look at an equid's faeces as this can give very useful information about a case and is non-invasive.

Collect the sample of fresh faeces, either on rectal examination, if this is being carried out as a part of the clinical examination, or freshly deposited faeces straight off the ground. Do not rectal the animal simply to collect faeces, only if it is part of the clinical examination, e.g. when investigating a colic case. Otherwise wait for the animal to deposit a fresh sample.

Look for the following:

- Parasites – may be grossly visible.
- In cases of ileus and some cases of colic, faecal output will be reduced.
- In impaction colic and dehydration, the faecal balls will be very small and dry.
- In dental disease, long fibre may be visible in the faeces as this has not been adequately chewed (masticated) as a result of dental pain/pathology.
- In diarrhoea cases, faeces will be loose and watery. Check for any signs of blood or melaena.

The following tests can be carried out:

- Direct observation (see above)
- Smear (see parasitology texts or specialist laboratories)
- Faecal flotation – McMaster (see Chapter 16 Parasitology) and Baermanns test for lungworm
- Culture, e.g. bacterial, if suspected Salmonella or clostridium infection (Weese et al. 2001)

11 THE GASTROINTESTINAL SYSTEM

Nasogastric intubation

Why is this often done in a case of colic?

Passage of a nasogastric tube can be used to assess whether there is reflux of gastric material.

Remember that equids cannot vomit.

Passage of a stomach tube will release any gastric contents which have built up as a result of this inability to vomit (due to the sharp angle of the oesophagus entering the stomach and the strong oesophageal muscles). Gastric reflux is significant because it indicates an accumulation of fluid in the stomach resulting from an obstruction in the proximal jejunum/duodenum; this obstruction can be a strangulating lesion or a functional obstruction. If the stomach is not decompressed it can rupture leading to peritonitis and fatal endotoxaemia. (See Section 4.1).

Further GIT diagnostics are usually only indicated in severe colic cases – see Section 11.5 of this chapter for further tests.

Glucose absorption test

This test is indicated in suspected malabsorption cases (Mair et al. 2006). When weight loss is observed in cases where the equid is consuming sufficient food, malabsorption may be suspected. There may be concurrent diarrhoea and ventral oedema. Rule out more common problems such as parasites, blood protozoa and dental problems before conducting a glucose absorption test. The procedure is as follows:

- Fast the animal for 12 hours (Venner and Ohnesorge 2001).
- Administer 1 g/kg glucose as a 10% (or 20%) solution by stomach tube.
- Collect blood samples in oxalate fluoride tubes at 0 (pre), 30, 60, 90, 120, 150, 180 minutes post glucose administration.

Result

Between 60 and 120 minutes post administration (Mair et al. 2006), assess glucose levels:

- In normal horses, peak concentrations of glucose of over 85% above the resting level is seen.
- Partial malabsorption is seen with results between 15 and 85% above resting blood glucose levels.
- Complete malabsorption is seen with levels of glucose less than 15% of the resting blood glucose level.

Differential diagnoses

Inflammatory bowel syndrome, infiltrative bowel disease (neoplastic), infections of the intestinal tract (bacterial, parasitic, fungal), enteritis in foals (Mair et al. 2006).

Treatment

Depends on the cause and diagnosis. Nutrition is a major factor; drug therapies include corticosteroids, antibiotics and anthelmintics.

Conditions of the mouth and oesophagus

11.4

Cleft Palate

See Chapter 18.1.

Lampas

Lampas is reported as a swelling of the hard palate just behind the incisor teeth (Pringle 1871).

Cause

It is a **normal physiological adaptation** to a very fibrous diet, commonly seen in working equids fed on poor quality roughage such as straw and husks.

Treatment

Lampas requires no treatment. Explain the cause to owners and try to discourage the use of astringent, blistering agents or surgical removal as these will cause unnecessary pain, suffering and affect normal eating. Even in an equine veterinary textbook of 1871 by Pringle, it is stated that this is a natural process and 'cruel practices – such as firing – should never for a moment be thought of'. Alterations to the diet and good feeding practices from the start can help prevent, resolve or prevent enlargement of such swellings. If the swelling is large it may make placement of a mouth speculum uncomfortable, so take extra care during dental examination and rasping.

Remember, lampas is a normal physiological adaptation requiring no treatment.

Oral ulceration

This can be due to a variety of causes: trauma, nutrition, autoimmunity and neoplasia (Tell et al. 2008). A high prevalence of traumatic ulcers caused by bits and bridles has been recorded (Tell et al. 2008). Infectious causes include 'vesicular stomatitis', which is a viral disease of equids and a zoonosis and bacterial diseases such as *Pseudomonas*. Additional causes are the ingestion of brittle plant materials, thorns or toxic chemicals (often from drinking from inappropriate water containers), dental disease and oral foreign bodies. Uncommonly, ulcers can occur from kidney disease and associated uraemia, rarely associated with long-term phenylbutazone administration.

Clinical signs

Initially, small vesicles develop into large ulcers over the oral cavity and tongue, sometimes extending into the pharynx and larynx. There may be excessive salivation and inappetence due to pain. In some cases, the tongue may become hard or 'spoon-like', resulting in inability to eat.

Diagnosis

Based on clinical signs and, in the case of toxicity, a history of ingestion of inappropriate substances. Always check the oral cavity using a full mouth speculum (remember, the animal might have pain in opening its mouth) and ensure there are no foreign bodies present. Also check the teeth as ulcers can be secondary to dental disease (see Chapter 10). Caution – Vesicular

11 THE GASTROINTESTINAL SYSTEM

stomatitis is **zoonotic**, so wear gloves. Lesions appear on the tongue, gums, lips, teats, prepuce and coronary band (Letchworth et al. 1999); horses are depressed with an increased temperature. If renal disease is suspected from the general examination, biochemistry can confirm this.

Treatment

Treat the underlying cause, e.g. remove the foreign body, treat dental disease and ensure that bits and bridles are well-fitting (Tell et al. 2008). Rinsing the mouth with dilute antiseptic solution daily may be useful to prevent secondary infection (if the animal allows this). Give anti-inflammatory drugs/analgesia if the animal is in pain, and encourage eating by providing soft palatable food.

Trauma to the lips and tongue

This is common in working equids due to ill-fitting or harsh bits, injuries from wood and wire, and eating inappropriate caustic substances.

Trauma can also be caused by excessive force on the tongue during examination – so be careful.

Clinical signs

Reluctance to eat, often accompanied by blood or excessive saliva dripping from the mouth. Inspection of the bit can reveal dried blood. Often the mouth will be open with the tongue hanging out. There is sometimes swelling under the tongue (sublingual cellulitis).

Diagnosis

Based on history, clinical signs and thorough examination (Hague and Honnas 1998)

Treatment

If fresh wounds are present on the lips and tongue, debride and suture if necessary, preferably with a dissolvable material. Landmarks for the mental nerve block are found in Section 10.8. Lesions of the tongue heal well by secondary intention, often rapidly and without complications. Equids can manage well with even severe tongue defects. Nursing care is important, so give pain relief and soft, palatable foods; administer antibiotics if secondary infection is severe. As with all wounds, ensure that the animal is either vaccinated for tetanus or, if the vaccination history is unknown, give tetanus anti-toxin if available.

Oesophageal obstruction ('Choke')

Oesophageal disorders are uncommon in equids but, among these, the most common presentation is choke.

There are a variety of inflammatory or traumatic forms of choke which cause partial or full blockage of the oesophagus. The most likely scenario with working equids is choke due to eating inappropriate feed which gets lodged in the oesophagus part-way down and presents as an acute condition.

Always consider choke an emergency (Widman 2008).

Clinical signs

Pain and retching when attempting to swallow, nasal discharge often accompanied by extension of the neck when swallowing often with regurgitation of food through the nostrils. Often a swelling can be seen halfway down the neck due to the impacted food or foreign body. If choke has been present for some time there may be evidence of aspiration pneumonia (dyspnoea, increased lung sounds, pyrexia) from food passing back up the nose and into the trachea.

Diagnosis

Based on the clinical signs, confirmed with passage of a stomach tube which will not pass further when it reaches the level of the blockage



Figure 11.4.1 Discharge from the nose in a case of choke.

Be very careful when passing a nasogastric tube if choke is suspected, to avoid rupturing the oesophagus.

Treatment

Over the telephone to the owner, tell them to remove food and water.

Most cases of choke resolve spontaneously. It is important to relax the animal (sedation with e.g. xylazine or acepromazine) and provide pain relief. Oxytocin (0.11–0.22 IU/kg IV, administered once only) can help oesophageal relaxation (unless the animal is pregnant) if the obstruction is in the top third of the oesophagus.

If none of the above is effective then lavage can be performed.

Insert the stomach tube to the level of the impaction and lavage with warm water to encourage lubrication and mass breakdown. Lavage slowly and gently and keep the head as low as possible to avoid causing aspiration pneumonia (see complications of nasogastric tube insertion in Section 4.1). Placing 20–50 ml of local anaesthetic down the tube promotes oesophageal relaxation and helps with the animal's discomfort. Lavage may need to be repeated over a few hours.

Ensure that only soft food is fed for a few days after the obstruction has been cleared and provide pain relief if the obstruction was severe and required a lavage to remove it, as the animal will continue to suffer discomfort when swallowing.

After every choke case check the teeth and carefully discuss diet with the owner as this may reveal a predisposing cause that can be avoided in the future. For example, choke can be caused by feeding very dry food which swells in the oesophagus; this can be easily avoided by ensuring that soaked feed is given.

11.5

Colic – A practical approach to diagnosis and treatment

‘Colic’ is a general term meaning abdominal pain. There are a large number of known causes of colic in equids, so a thorough work-up is necessary.

Colic is a symptom, not a diagnosis!

This section covers colic due to gastrointestinal (GI) disturbances, although there are a number of other causes – see relevant sections.

Colic can be fatal and is one of the most frequent causes of mortality in horses (Nolen-Walston et al. 2007).

However, 80% of colic cases resolve spontaneously or with simple medical treatment, with a further 10% requiring intensive medical treatment (Orsini 2011). It is the remaining 10% that are fatal without surgical intervention; humane euthanasia may be considered in these cases when surgical intervention is not possible or appropriate. To determine whether a colic case is mild or serious conduct a thorough clinical examination and monitor the response to treatment closely.

Do not delay in the response to a colic case.

What might be happening internally when colic signs are observed?

- Inflammation of the stomach or intestinal walls This could be due to parasitic worm damage, sand ingestion, bacterial infection or ingested foreign bodies (e.g. plastic bags). It can lead to altered motility and diarrhoea.
- Stretching of the intestinal wall This is due to distension with gas (tympany) or food material (grain overload, or colonic impaction).
- Altered intestinal motility Peristaltic movements may be abnormally increased (spasm), decreased or absent (ileus). Changes in motility may be caused by dehydration, dietary changes, parasitic worms, toxins, or obstruction from inappropriate ingested materials. They often lead either to diarrhoea or to constipation.
- Loss of blood supply to an area of intestine (ischemia) This is caused by intestinal torsion, strangulation, intussusception or total blockage. It leads to the release of toxins into the blood, inflammation and severe pain followed by necrosis and death of the affected section of intestine.

In many cases, the underlying cause of the colic will result in a combination of the above effects on intestinal walls. The first three categories listed often recover spontaneously or with medical treatment alone. Those in the fourth category can develop from one of the other three, or may have no obvious cause. Medical treatment does not relieve the symptoms, in this last category, which progress rapidly over a few hours and are fatal without surgical intervention; euthanasia is recommended in these cases to relieve pain and suffering.

The approach to a colic case

History

The following questions are important:

- When did the colic signs start?
- Has the animal had colic before? If so, when was the last time?
- When did it last pass faeces? How much? What consistency?
- Does it have diarrhoea?
- When was the last de-worming treatment?
- When did the animal last eat? What does it eat? Has the diet changed recently?
- When did it last drink and how much?
- When did it last pass urine?
- Is the animal pregnant?
- History of teeth problems/quidding? (See Chapter 10 Teeth.)

Dehydration is a common cause of colic in working equids.

Around 100 L of fluid passes daily through the GIT of an adult horse and is partly responsible for keeping ingesta moving through the system.

Urine retention is an extremely rare cause of colic; it is more likely that colic signs are due to dehydration.

Never give diuretics in cases of colic – they will make the animal more dehydrated.

When the animal is rehydrated and given pain relief it will urinate without the need for diuretics or catheterisation (see Section 13.1).

Always examine the teeth as dental problems are a common cause of colic in working equids.

Clinical signs

It is important first to observe the animal at a distance to ascertain physical signs of colic.

- Horses show varying symptoms, some of which can be very dramatic and can potentially harm the animal or people in the vicinity. Lying down 'flat out', rolling and kicking are common. Milder signs include sweating, pawing at the ground and frequent looking back at the abdomen (Figures 11.5.1). (See Chapter 2 for further behavioural signs of abdominal pain.) Some animals may strain as though attempting to pass faeces, whilst others are just subdued with dehydration and decreased appetite. Try to perform a thorough clinical examination before giving any drugs, as these may quickly hide the clinical signs and make diagnosis more difficult.
- Donkeys often show more subtle pain symptoms which may be overlooked (Figure 11.5.2). They must be examined thoroughly in order to assess the severity of colic as behavioural signs of pain may be different from those shown by horses. Dullness and depression are signs of colic as well as subtle changes in behaviour.



Figure 11.5.1 Colic signs in horses: flank-watching (top left), stretching (top right), sitting down (bottom left) and lying flat out on the ground (bottom right).



Figure 11.5.2 Colic signs in the donkey may appear more subtle than in the horse, including sitting down and stretching out.

Clinical examination

A thorough clinical examination should take place, including the length of the GI tract that is either physically palpable (mouth, oesophagus, abdomen, rectum), or via auscultation and percussion (all four quadrants of the abdomen). Auscultation of the abdomen, and knowledge of what normal gut sounds are, will be the first step in the colic diagnosis as, even with mild/inapparent clinical colic signs, changes in gut motility can be detected. Heart rate, respiratory rate, mucous membrane colour (pale/anaemic, icteric/jaundice, purple/toxic), capillary refill time, pulse quality and hydration status should all be assessed.

Other conditions can be mistaken for colic in equids, especially if the signs are mild.

In equids exhibiting colic signs, check also for laminitis, rhabdomyolysis, tetanus or even foaling.

Other diagnostic procedures

These include the following:

- Nasogastric intubation
- Rectal examination
- Abdominocentesis (peritoneal fluid sampling)
- Blood sampling

Nasogastric intubation

This procedure should be carried out in all equids suspected of colic, if there is a suitably trained and competent veterinarian to carry out the procedure, a suitable nasogastric tube, a calm (possibly sedated) animal and a competent handler. Passing the tube will help with diagnosis of an obstruction in the pharynx or oesophagus, it will release air and gas from the stomach and, in severe colic cases, fluid accumulated in the stomach can be released or siphoned off. This procedure will ease the pain and suffering caused by a distended stomach as well as aiding in diagnosis and treatment of the case. The same technique can be used to pass fluid into the animal if required as part of the treatment regime. For details of the full technique refer to Section 4.2.

Rectal examination

Why do a rectal examination in a case of colic?

- Rectal examinations give us an insight into what is happening internally so that we can refine or confirm our diagnosis or gain a better indication of prognosis. Although frequently carried out for colic cases, rectal examination is also performed to confirm pregnancy or to assess for tumours or other abnormalities in animals with a history of chronic weight loss or diarrhoea. Rectal examination is not generally well tolerated by mules; it is most commonly used in horses; it can also be safely carried out in donkeys. Do not attempt in mares known to be heavily pregnant as it is difficult to palpate structures other than the foal.

Only the caudal third of the abdomen can be palpated, so abnormalities higher up in the digestive system (small intestine or stomach) will not be detected.

11 THE GASTROINTESTINAL SYSTEM

When is the best time to do a rectal examination? And what if it seems too dangerous?

- A rectal examination should be done as soon as possible in cases of severe colic (see Table 11.5.2 on page 271 for other external indicators of whether colic is severe) to get an early diagnosis and prognosis. It is important that the severity of the colic is determined as early as possible so that cases with a poor prognosis (gut torsion, rupture, etc.) can be euthanased quickly to prevent further suffering.
- In many cases of severe colic the animal may harm the clinician while the rectal examination is being carried out, for example if it is rolling on the ground or kicking out. An early attempt at a rectal examination in this case could also damage the sensitive rectal mucosa so calm the animal first with analgesia (NSAIDs such as flunixin meglumine) and sedate with an alpha2-agonist sedative drug (xylazine or detomidine), then attempt the examination after 10–20 minutes. Weigh up the potential harm caused by the hypotensive effects of sedation compared to the harm the animal will do to itself, or those present, if it is not sedated. Alternatively, leave out a rectal examination altogether if the potential harm is going to outweigh the benefit to the animal, and use other information you can gather (mucous membrane colour, heart rate, history, response to analgesia etc.) to make an informed decision about the prognosis.
- In cases of mild to moderate colic a rectal examination may be performed at a later stage of treatment than it can in severe cases. This would be appropriate if there is no response to fluid or analgesic therapy after several administrations, no faeces have been passed after 48 hours despite settling of clinical signs, or the colic signs are recurring.

Blood on an equine rectal glove is a bad sign. Do not use the same force as when conducting a rectal examination on a bovine animal.

What if there is blood on the glove after examination?

- The rectal wall of the horse is extremely sensitive and damage is often indicated by the presence of blood on the rectal glove. Any amount of blood is serious and the depth of the rectal tear will determine the consequences. Refer to Section 11.7 of this chapter (Rectal perforation) for guidance on what to do if a rectal tear is suspected.
- The seriousness of a rectal tear and associated peritonitis reiterates the importance of using good restraint, patience and good technique when doing a rectal examination. Peritonitis is a very painful, unnecessary and fatal consequence of poor rectal technique.

How to perform a good, safe rectal examination?

A good knowledge of the internal anatomy is essential for conducting a rectal examination. Without this knowledge a rectal examination cannot be justified.

Observe the following technique:

1. Excellent restraint and lots of lubrication are vitally important. Stand beside the animal and insert an arm slowly and gently into the rectum. If the animal is straining, do not push against it as this may damage the rectal wall.
2. Pull the faeces out slowly and observe consistency (e.g. dry and hard) or whether it contains blood, sand or mucus. Removal of faeces gives better access to the rectum.

3. Gently palpate the internal organs with a flat hand.

- a. Rectal mucosa should be smooth. Roughening or thickening of the rectal wall is abnormal.
- b. Small colon (left side) is detected by palpation of small faecal balls within the lumen and is relatively mobile. Distension or impaction of this is abnormal.
- c. Pelvic flexure is normally in the ventral part of the abdomen, left of the midline. Impaction is common due to the sharp bend in the colon at this point and the natural reduction in luminal diameter there. If impacted, it will feel distended and 'doughy' on palpation and, when gently pressed, a small indent will remain. When impacted, the pelvic flexure may be displaced from its normal position; it may be the first thing palpated on entering the rectum.
- d. Caecum This sits in the right-hand side of the abdomen, internal to the right paralumbar fossa. The ventral band is felt running top to bottom of the caecum from right to left, in a diagonal direction. Distension of the caecum with gas or ingesta changes the direction of this band, and tight bands are pathognomonic. If it is possible to palpate the bottom part of the caecum it is distended.
- e. Large colon Distension and bands (similar to the caecum) are abnormal.
- f. Small intestine This cannot usually be palpated. If it is palpable in the middle part of the abdomen (usually because it is distended with gas), it could indicate a problem higher up the tract, such as a small intestinal obstruction, strangulation or torsion. When distended it feels like a tight balloon or rubber rings.
- g. Spleen This is close to the left abdominal wall near the level of the last rib. It should feel very close to the abdominal wall and is firm to palpate with a sharp caudal edge. Always check the dorsal margin of the spleen and feel along the nephrosplenic ligament which joins to the left kidney on the dorsal aspect of the left abdominal wall because the large colon can displace and lodge here. This is known as nephrosplenic entrapment.
- h. Peritoneum This is the final structure examined. Be very gentle so as not to rupture the large intestine. It should be smooth. Nodules, roughening and fibrous tags indicate possible peritonitis.
- i. Other Occasionally other abnormalities such as tumours, uterine torsion or adhesions can be found. These are useful findings to aid diagnosis especially if associated with chronic weight loss or diarrhoea.

Abdominocentesis/paracentesis/peritoneal tap

What information can abdominal fluid provide, and when should it be sampled?

- Examination of the colour and viscosity of abdominal fluid (see Table 11.5.1 on the next page) is useful in determining the prognosis for suspected peritonitis cases and can also be used to give more information about the cause of colic. It should be done if clinical signs are severe or worsening over time to give a possible indication of whether torsion or rupture has occurred.

Sterile technique is extremely important; do not introduce infection into the abdomen.

11 THE GASTROINTESTINAL SYSTEM

1. Restrain the animal (sedate if necessary) and clip and disinfect the abdominal midline thoroughly. Find the umbilicus; the ideal place for needle insertion is 2–3 fingers' width from the umbilicus back towards the tail end of the animal at the most dependant point of the abdomen.
2. Stand next to the animal at the elbow, facing the rear. It is easier to move the arm, rather than the whole body, out of the way if the animal kicks.
3. Using a sterile 18G, 1.5-inch needle, insert it sharply and quickly through the skin and muscle and then slowly advance it into the peritoneal space. Once it is inserted move it around gently until fluid appears at the hub. Collect a sample in a sterile tube. Normal peritoneal fluid is yellow/straw coloured and clear enough to read the text of a newspaper through the vial.
4. If not successful repeat more caudally.
5. Refractometry Put a drop of peritoneal fluid onto the refractometer and read the total protein concentration (opposite scale to the USG). This is a simple and useful tool which can provide useful information (see table below).

Colour	Possible condition	Total protein (TP) (g/dL)
Yellow/straw, clear	Normal	< 2.0
Streaked with fresh blood	Hit a blood vessel in the skin or muscle on the way through the abdominal wall (blood free in abdomen will be defibrinated and will not clot)	
Yellow/straw/amber, opaque	Impaction, obstruction, peritonitis, early gut compromise	< 3.0
Red/brown, serous	Possible strangulation, early torsion. Dark blood that clots is from the spleen.	2.5–6.0
Brown/black/green-tinged	Bowel necrosis/rupture, torsion, peritonitis, severe gut compromise (turbid and cells settle out)	5.0–6.5
Green/fibrous	Intestinal contents/penetrated gut with needle (with sediment)	Variable

Table 11.5.1 Findings when examining peritoneal fluid.

Normal peritoneal fluid

- Approximately 5–10 mls in drips over 4–5 minutes
- Clear (low turbidity) pale yellow fluid, low cellularity
- Can use a refractometer to measure TP, < 2 g/dl is reported as within normal limits.
- Look at the cells under a microscope for WBC count, for adult horses < $10 \times 10^9/l$ has been reported as the maximum limit of the normal range, for foals a count > $1.5 \times 10^9/l$ should be considered abnormal (Grindem et al. 1990).

Guidelines for making informed decisions in the management of colic cases

Colic, especially if severe, is an emergency.

Stay with the animal to monitor the response to treatment, and follow up over the following days until the animal is passing faeces and behaving normally again.

Table 11.5.2 is included to help determine how severe a colic case is, the possible cause, and what actions should be taken after the initial examination. Use this as a guide only and treat each case individually based on clinical findings. It is impossible to classify every case into a table with specific guidelines for approach to management.

Clinical sign	Mild/moderate colic	Severe colic
Pain	Mild/moderate	Severe
Sweating	Absent/mild	Severe
Mucous membranes	Normal or mild congestion	Severe congestion, purple, grey, 'toxic line' at the base (gum line) of the teeth
Capillary refill time	Normal	> 3 seconds
Pulse quality	Normal	Weak
Heart rate, beats per minute (bpm)	< 70 bpm	> 70 bpm and rising
Gut sounds	Normal or increased	Decreased or absent
Faeces	Present	Absent or small dry faecal balls
Hydration	Normal or mildly dehydrated	Severely dehydrated
Gastric reflux*	None or very little	Large volume
Rectal examination*	Normal, impaction, mild tympany	Displacement, severe tympany, loops of small intestine, foreign body
Paracentesis*	Normal	Abnormal
Response to analgesia	Good response within 30 minutes to an hour. Symptoms do not recur if underlying cause is treated.	Poor/slight response after an hour. Symptoms recur quickly within hours and often appear worse than before.

Those diagnostic aids marked with an asterisk (*) are normally only done if other clinical signs are severe. If mild colic is suspected treat and reassess 1–2 hours later.

Table 11.5.2 Interpreting clinical signs in colic cases.

11 THE GASTROINTESTINAL SYSTEM

Diagnosis

Mild to moderate clinical signs with increased gut sounds

This usually indicates spasmodic colic or intestinal wall inflammation due to intestinal worm damage or diet.

Treat with NSAIDs and rehydrate. Anti-spasmodic drugs (hyoscine) are good if available although never use them if gut sounds are decreased. Give anthelmintic treatment only when the animal has recovered; giving this while the animal still has clinical signs can greatly increase the colic symptoms.

Remember, flunixin meglumine is a very potent visceral analgesic and will mask early signs of deterioration and the development of endotoxaemia.

After analgesia has been given animals should be carefully monitored for signs of further deterioration.

Mild to moderate clinical signs with decreased/absent gut sounds

This can indicate an impaction or partial obstruction. Rectal examination findings may reveal foreign objects such as plastic bags, rope or cloth in the rectum (see Figure 11.5.3 and the case study in Section 11.9 of this chapter). The pelvic flexure is a common site of impaction due to the narrow lumen at this point; observe its position and consistency noting any abnormalities (see details of rectal examination earlier).

Treatment

Impaction

- Analgesics to control the pain (see above)
- If gastric reflux is not present, and there is evidence of gut sounds, give oral fluids by nasogastric tube. 3–6 litres can be given, depending on the size of the animal, and electrolytes can be added. This can be repeated every 2–4 hours; if the impaction is large this may be necessary. Because foreign body impactions are difficult to distinguish from food impactions, laxatives such as mineral oil are often used. However, frequent administration of fluids per nasogastric tube has been shown to be as effective, if not more so. Refer to Section 6.2 on fluid therapy for information on how to make an isotonic solution suitable for nasogastric intubation.
- Food should be withheld and the treatment repeated until the animal starts to pass faeces again.
- Recent research on treatment of large colon impactions shows that oral fluid hourly is the most effective way to treat an impaction. This



Figure 11.5.3 An example of a foreign body impaction from plastic bags and rope eaten by the donkey.

provides the most rapid resolution and is cheaper, as well as avoiding the complications associated with IV parenteral fluid administration (e.g. thrombophlebitis).

In the context of working equids the administration of oral fluids, in the case of large colon impactions, is more practical, readily available and safer, so should be considered the treatment of choice.

Refer to the paper Colic – A Practical Approach to Diagnosis and Treatment by Hallowell (2008) for more information.

- Once the animal has started to pass faeces, small amounts of a soft laxative diet such as fresh green fodder or crushed concentrates soaked in large amounts of water can be offered 5–6 times daily (little and often) for 3–4 days. Ensure plenty of water is available to drink at all times; this keeps the contents of the intestine soft and reduces the likelihood of recurrence. Warn the owner of the possibility for recurrence, especially if water is withheld or the animal is allowed to eat unsuitable feeds (hard, fibrous or rubbish).

Colonic displacement

Displacement of the left colon dorsally over the nephrosplenic ligament and right dorsal displacement, where the colon rotates around the caecum, are the two most common forms. The cause of these displacements is not known but it is thought to be the result of alterations in gut contents, resulting in an accumulation of gas, or altered motor activity.

- **Nephrosplenic entrapment** The left portion of the colon becomes lodged in the space between the left kidney and the spleen, hooked in place by the underlying nephrosplenic ligament. Once trapped in this position the colon becomes occluded resulting in a partial obstruction. On rectal examination the colon can be felt in this position between the kidney and the spleen in the upper left quadrant.
- **Right dorsal displacement** The large colon rotates 180 degrees around its mesenteric attachment. This can be felt rectally as a gas-distended colon and the pelvic flexure is no longer palpable as this has been displaced more cranially in the abdomen by the rotation.

Treatment regime

- Analgesics to control the pain
- Correct fluid imbalances. Provided that gut sounds are present and there is gastric reflux, this can be administered through nasogastric intubation.
- Withhold food. In mild cases, if food is withheld, the reduction in gut fill allows the colon to shrink and possibly move back to the normal anatomical position. Gentle walking can help this movement.
- Administration of phenylephrine at 20–80 µg/kg dissolved in 500 ml of 0.9% NaCl given slowly over 30 minutes, results in splenic contraction and facilitates the movement of the colon into the normal position. It should only be used if a nephrosplenic entrapment has been definitively diagnosed without doubt. The heart should be carefully monitored for the duration of the infusion and it should be stopped immediately if any arrhythmia is detected. Do not use in cases of myocardial failure.
- In some cases, displacements will not self-correct.

11 THE GASTROINTESTINAL SYSTEM

In the context of working equids corrective colic surgery is not an option.

If a case is not responsive to analgesia or becomes endotoxaemic (i.e. develops into a more serious disease state) then euthanasia should be advised.

Severe colic signs

Treat initially with pain relief.

It is very important to stay for at least an hour and monitor the response to pain relief since these drugs should act within 30 minutes. If it is safe to do so, carry out further diagnostics (rectal examination, abdominocentesis) even if pain relief has worked, as there is still likelihood of a critical condition if severe signs are seen. Further work-up will assist in determining a prognosis. If there is no response, or the animal's condition gets worse over a few hours, the prognosis is hopeless.

Euthanasia should be carried out to alleviate suffering if the prognosis is hopeless.

If the owner does not agree to euthanasia, heavy sedation and pain relief should be given until the animal dies.

Why is colic surgery not advocated in working equids?

Surgical treatment of colic requires a large amount of specialist equipment, strict aseptic conditions, an expert surgeon and excellent facilities and personnel for general anaesthesia and recovery. Post-operative care is intensive, lengthy and the animal always requires at least 4–6 months' rest post-recovery which is normally not possible for a working equid. Colic surgery is therefore logistically impossible in mobile clinic conditions and is not attempted.

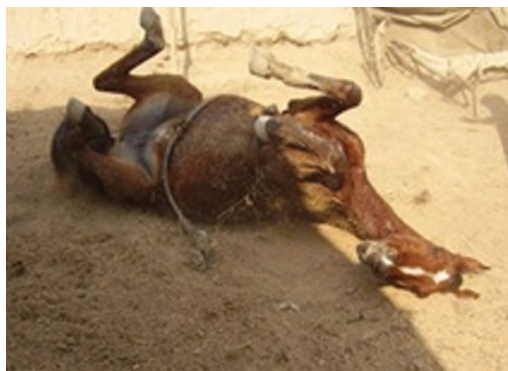


Figure 11.5.4 A horse showing severe abdominal pain.

Remember harm v. benefit for the animal.

Even with state of the art facilities, 24-hour nursing care, expert equipment and experienced equine surgeons, many colic surgeries result in prolonged suffering and eventual death of the animal, or long-term gastric complications.

Prevention of colic

The key to a healthy equine gastrointestinal system is the continuous provision of appropriate food and water.

Sometimes colic occurs for no identifiable reason. Good nutrition and feeding practices, free access to clean water, an appropriate living environment, care of dentition, some moderate daily exercise, and management of internal parasites can help prevent many of the common causes of colic.

Risk factors for colic in equids

A number of reviews have been written on the risk factors for colic in equids (see References). Listed risk factors are: age, sex, breed, type of housing, outdoor access to pasture, changes in management practices, activity level of the animal and changes in activity, water supply, control of parasites (use of de-worming products, presence of intestinal parasites particularly tapeworm, and type of de-worming programme), quality and type of food (such as concentrates, feeding of wholegrain corn, changes in feeding practices and types of feed), competence of the person who cares for the animal daily, and medical history such as previous colic episodes or administration of medical treatment (Reeves et al. 1996, Proudman and Trees 1999, Gonçalves et al. 2002, Proudman 2003). However, many reported risk factors have conflicting reports and weak evidence (Nolen-Walston et al. 2007).

Identification of risk factors is important to be able to advise owners on management that will reduce colic incidence (Scantlebury et al. 2011).

Risk factors for colic related to feeding practices are the most prevalent, especially changes in diet, low levels of forage, and high levels of carbohydrates.

Anthrax in equids presents as acute colic, with septicaemia and enteritis. Treatment can be attempted, with BID penicillin, if early in the disease course. The disease course is usually 48–96 hours, and is generally fatal. Burn and bury suspect cases that die; do not conduct a post-mortem as this is a zoonotic disease.

Diarrhoea in adult equids

11.6

See Chapter 18 for information about diarrhoea in foals.

Acute diarrhoea (acute colitis)

In most cases, acute diarrhoea results from inflammation of the large colon and caecum.

Clinical signs include depression, abdominal pain, pyrexia and dehydration, which can progress to shock and death. This progression can occur quickly, so rapid identification and correction of fluid deficits must be a first priority (Naylor and Dunkel 2009).

Cause may not always be identified.

Treatment of clinical signs should be implemented without delay. Other treatment should aim to reduce systemic and intestinal inflammation (NSAIDs) and then to promote intestinal mucosal repair (sucralfate). Response to treatment should be monitored.

Caution! Antibiotic-associated diarrhoea (AAD) has been linked to nearly all antibiotics used in equids (McGorum and Pirie 2010) – so do not use them in treatment of diarrhoea (especially in adult horses) as this may exacerbate the situation.

The macrolide group of antibiotics (erythromycin and clindamycin) has been particularly indicated as a cause of AAD (see below) and is therefore contra-indicated in equids (McGorum and Pirie 2010); see Chapter 5 Medicines. Antibiotics should be avoided but, if used prudently, should be carefully selected based on diagnostic analysis.

Fluid therapy in diarrhoea

Mucosal inflammation, often found in cases of diarrhoea, can compromise mucosal integrity and function, which in turn can affect the absorption of water and electrolytes. However, in mild to moderate cases of diarrhoea, enteral fluid therapy via a nasogastric tube is still considered an appropriate method of fluid therapy (Naylor and Dunkel 2009). In most cases involving working equids this would be the most appropriate method of rehydration because it is readily available, practical, cheap and avoids the risk of thrombophlebitis associated with venous catheterisation (often increased in diarrhoea cases). See Chapter 6 Dehydration and fluid therapy for instruction on how to deliver isotonic enteral therapy.

Salmonella

Cause *Salmonella* spp. of bacteria. Diarrhoea is due to toxin release by the bacteria resulting in increased fluid secretion and inflammation of the intestinal mucosa. Equids of all ages can be affected although younger animals seem more susceptible. This can occur in outbreaks, and stress is often indicated as an underlying factor, e.g. transportation, gastrointestinal disease, changes in feeding patterns, high temperatures and antibiotic therapy (Feary and Hassel 2006). Recovered animals may become chronic carriers, shedding bacteria into the environment and affecting other animals (see Chronic diarrhoea below).

Clinical signs Signs of depression, inappetance, pyrexia, and colic are often present a few days before the diarrhoea starts. **Profuse watery foul-smelling diarrhoea**, possibly bloody, is typical, along with increased systemic parameters and congested mucous membranes. Septicaemia and endotoxaemia are common sequelae, which could lead to complications such as laminitis. Sometimes salmonella can become a chronic condition, reappearing in times of stress and causing chronic poor body condition and failure to thrive.

Diagnosis Should be confirmed by faecal culture but this can be difficult as false negatives are possible. As the bacteria are shed intermittently, a minimum of three samples (ideally five) taken on subsequent days, are recommended to rule out the salmonella infection (Feary and Hassel 2006).

Treatment Correct the fluid deficit via nasogastric tube with oral supplementation. NSAIDs are recommended. The use of antibiotics has been shown in some cases not to reduce shedding of salmonella, and can be detrimental. Indeed, antibiotics have been shown to be associated with the cause of some cases (Feary and Hassel 2006). Antibiotics should only be proposed in severe cases of septicaemia and for immune-compromised animals with careful selection of a narrow-spectrum bactericidal agent (Feary and Hassel 2006).

Remember, salmonella is very contagious to most species, including humans. Appropriate isolation of the animal and owner advice must be given.

Clostridium perfringens

Cause *C. perfringens*

Clinical signs Acute onset depression and shock, with death often occurring before diarrhoea. Intense pain, often unresponsive to analgesia, means that euthanasia is usually indicated; most animals die within 24 to 48 hours of onset. Endotoxaemia is a common finding, and the associated systemic signs of this. Seen in foals in the first few days of life.

Diagnosis Isolation of bacteria in fresh faeces or faecal swabs

Treatment Aggressive fluid therapy can be initiated if *C. perfringens* infection is recognised in the early stages. Foals can be treated with antibiotics (e.g. penicillin and aminoglycoside, plus metronidazole PO/IV 10–15 mg/kg BID). Avoid antibiotic therapy in adult equids as this is reported to induce further diarrhoea (Feary and Hassel 2006).

Chronic diarrhoea

Parasitic enteritis

(See also Chapter 17)

Cause Usually due to the emergence of larval cyathostomes (small strongyles) which damage the walls of the caecum and colon. These cyathostome larvae hypobiose (go into arrested development) in the gut wall at times when external environmental factors are detrimental to their development/life cycle, e.g. cold winters and possibly drought periods. Large strongyles have also been implicated.

Clinical signs Diarrhoea, weight loss, parasitic larvae may be seen in faeces, severe cases may show limb oedema (due to hypoproteinaemia) and increased pulse/respiratory rates. Faecal egg counts will not reveal the true extent of the infection as the hypobiosed larvae are prepatent. Cyathostomiasis has been linked to fatal colitis and is more commonly reported in 1–6 year olds (Feary and Hassel 2006).

Diagnosis Signalment, history, risk factors, faecal and blood analysis, parasitic larvae in faeces

Treatment Due to over-use of de-worming drugs, certain anthelmintics are no longer effective, e.g. benzimidazoles (fenbendazole). Repeated dosing is needed to kill developing larvae; there are no reports of resistance to ivermectin (Feary and Hassel 2006).

Emergence of resistance of cyathostomes to anthelmintics is a major parasitic control problem and warrants careful monitoring.

Chronic salmonellosis

Clinical signs Soft faeces with intermittent bouts of diarrhoea, persistent weight loss (MacLeay et al. 1997). May have fever or poor appetite.

Diagnosis Serial faecal cultures

Treatment As for acute salmonellosis; however, it is usually unsuccessful.

11 THE GASTROINTESTINAL SYSTEM

Antibiotic Associated Diarrhoea (AAD)

Clinical signs Diarrhoea starts 2–6 days after antibiotics are first administered.

Nearly all antibiotics used in horses have been reported to cause diarrhoea; therefore, it is important to use antibiotics responsibly.

It is thought that the antibiotics kill the beneficial gastrointestinal (GI) flora which leads to an overgrowth of *Clostridium difficile* and *C. perfringens* (McGorum and Pirie 2010). Decreased roughage consumption may predispose animals.

Diagnosis By clinical signs and recent antibiotic therapy

Treatment Stop the antibiotic therapy. Give dry forage diet and supportive care. Correct the fluid deficit via nasogastric tube if possible, ideally with isotonic solution (see Chapter 6). NSAIDs may be required to ensure comfort, but use sparingly. Flunixin meglumine is a good choice for its anti-endotoxic properties. Metronidazole 15–25 mg/kg PO 6–8 hours may help in cases of acute toxic enterocolitis (McGorum and Pirie 2010) but, if improvement is not seen, stop the antibiotics.

NSAID toxicity

Cause Phenylbutazone, flunixin meglumine and ketoprofen have been implicated in causing disease. Phenylbutazone is thought to have the highest propensity to cause diarrhoea (MacAllister et al. 1993).

Clinical signs Diarrhoea, colic (Hillyer 2004), inappetance, hypoproteinaemia, and dependent oedema

Diagnosis Recent history of NSAID use and clinical signs. Drugs may have been used in the correct doses, or may have been overdosed or used for a prolonged period of time.

Treatment Withdraw the NSAID. Supportive treatment for the diarrhoea: fluids (preferably by nasogastric tube), dry forage diet. Sucralfate 22 mg/kg BID/TID may help.

GI tumours

This should be considered in older equids with chronic, non-progressive diarrhoea which does not respond to treatment.

The most common form is intestinal lymphosarcoma (Taylor et al. 2006), although mesenteric lipomas are also common in older animals.

Clinical signs Persistent weight loss, diarrhoea, colic, and oedema of the ventral abdomen and limbs as, over time, the protein-losing enteropathy leads to hypoproteinaemia. Contrary to other causes of chronic diarrhoea, the animal may or may not have a good appetite or fever.

Diagnosis Clinical signs and a lack of response to therapy. A rectal examination may reveal enlarged mesenteric lymph nodes, or the tumour itself, and a haemogram or abdominal paracentesis may show increased numbers of lymphocytes.

Treatment None available which is curative. Supportive therapy (rest, good quality feed, and pain relief if required) is indicated until the animal is too debilitated to work. These tumours

often lead to colic signs, especially if the bowel becomes obstructed or strangulated which is often the case with pedunculated lipomas.

Prognosis Poor – chronic weight loss and debility leads to death or euthanasia. If the animal cannot work and has become anorexic, these are indications that it is suffering and euthanasia should be recommended to the owner. Reported time from the start of signs of the disease to death or euthanasia is less than 2 months (Taylor et al. 2006). Make the owner aware of this very poor prognosis to reduce suffering for the animal.

Conditions affecting the rectum (prolapse and perforation)

11.7

Rectal prolapse

Cause This is quite common in some regions and can be due to a variety of reasons, including chronic diarrhoea associated with infection or parasitism, foreign bodies or feed impaction due to poor teeth, dehydration, or following parturition, many of which cause increased straining (tenesmus) (Colahan et al. 1999). Other non-gastrointestinal causes can include overloading (the animal strains in the hindquarters to move the load) and prolonged recumbency from other illnesses. A study of 177 donkeys in Ethiopia (Getachew et al. 2012) showed over 83% of cases were associated with the presence of *Gasterophilus nasalis*.

A prolapsed rectum is a symptom of an underlying problem, not the problem itself!

Treatment This involves not only replacing the prolapse (Figure 11.7.1), but taking a good history and doing a detailed clinical examination to try to identify and treat the underlying cause. Many cases of prolapse are recurring and, unless the underlying cause is rectified, the animal's welfare will be severely compromised with repeated attempts at replacement. Provide analgesia and anti-inflammatory drugs.

How do you replace the prolapse?

- Ensure the animal is adequately restrained. This requires a competent handler, sedation and, ideally, epidural anaesthesia, (details in Section 7.2), particularly if the animal is still straining a lot or distressed.



Figure 11.7.1 An example of a case of rectal prolapse in a donkey.

11 THE GASTROINTESTINAL SYSTEM

- Tie back the tail and clean any faeces from the tail and perineal area.
- Wash the prolapse gently and remove as much necrotic tissue as possible. Applying table sugar will help decrease any oedema and allow for easier replacement back into the rectum (Myers and Rothenberger 1991).
- Gently push the rectal tissue back into the rectum manually, avoiding excessive pressure which could tear the delicate rectal mucosa; use lubrication to aid the process. If the animal continues to strain and the rectum keeps re-prolapsing, consider sedation, if this has not already been given, or even a caudal epidural (Michou and Leece 2012) if the animal appears to be in a lot of pain. Ensure that a caudal epidural has been administered before suturing the anus.
- Many rectal prolapses can be resolved through manual correction and do not require suturing. If this is not possible then a suture can be placed.

Ensure that the animal can pass droppings and that sutures are removed.

- Using a large needle and thick suture material (cattle vaginal tape is best if available), make a 'purse-string' suture (a single, continuous suture pattern with needle insertion at 4–6 points in a full circle around the rectum). Infuse local anaesthetic into the area first with a 24G needle.
- Gently pull the two ends of the suture so that the rectum closes. Leave a space large enough to insert 2–3 fingers into the hole (so the rectum will not prolapse back through the hole, but that soft faeces and gas can still escape).
- Treat for underlying colic symptoms, pain and infection, if necessary. Pass a nasogastric tube and give liquid paraffin or mineral oil (the same dose as for suspected obstruction in colic – Section 11.5 of this chapter) as this lubricates the faeces to allow for easy passage once the suture is removed. If parasites are suspected to be the underlying cause, do not give anthelmintics until the sutures are removed and faeces are passing normally, as this could cause recurrence of the prolapse or colic.



Figure 11.7.2 A replaced rectal prolapse being held in place by sutures.

Important: If the prolapse has been replaced and held in place with sutures, ensure that the sutures are removed after 48 hours (72 at most) (Colahan et al. 1999). Leaving in sutures will ultimately lead to the death of the animal, which is far worse than the original prolapse.

- Do not just rely on the owner to take the sutures out – aim to revisit the animal the next day, assess for colic symptoms and pain, and repeat the paraffin or other symptomatic treatment if necessary. If follow-up is not possible, ensure another member of the team or a trusted local health provider is able to revisit. The animal will require several days of soft laxative feed, plenty of water, and a reduced workload for the next week to ensure the prolapse does not recur.



Figure 11.7.3 Bandage preventing recurrence of prolapse.

Always remember to remove the sutures.

A novel treatment in the field, when suturing is not possible or not required (Chittora 2012, personal communication), is to use a bandage pack to keep the prolapse inside after manual replacement (Figure 11.7.3). As before, careful monitoring of the animal is required and analgesia and anti-inflammatory drugs administered. The owner needs to be advised of a soft diet to reduce straining until the symptoms have resolved.

Prevention: The underlying cause must be found and treated or repeated prolapse may occur.

Rectal tears/perforation

This is a serious condition in equids and can result in fatal peritonitis which requires euthanasia. The rectum consists of three layers. From the lumen side these are: mucosal (lining the rectal lumen), muscularis and serosal layers.

Causes

- Inappropriate rectal examination technique is the most common cause (Watkins et al. 1989) possibly due to an unrestrained animal, lack of lubrication and/or excessive force (see rectal examination in Section 11.5 of this chapter).
- Foaling difficulties – excessive straining by the mare, foal hooves tearing the rectal lining, or an explosive delivery
- Inappropriate or violent coitus

Diagnosis

- Suspect if signs of colic or peritonitis are observed, with a history of foaling, coitus or recent rectal examination.
- Abdominocentesis can reveal faecal material or other signs that a complete rectal rupture has occurred.

11 THE GASTROINTESTINAL SYSTEM

- Rectal examination is the only way to determine the size of the tear and whether it is full thickness or involves just the mucosa and/or muscularis layers.
- Rectal tears have been classified from Grades 1 to 4 depending on the depth of the tear: from Grade 1 involving just the mucosa (mildest form) to Grade 4 involving full thickness of the rectal wall leading to contamination of the peritoneal cavity with faeces (which is invariably fatal and calls for immediate euthanasia) (Watkins et al. 1989).

Treatment

- A full-thickness tear (through all three layers – mucosa, muscularis and serosa) will most likely result in fatal peritonitis (Grade 4), and humane euthanasia is recommended early to prevent suffering.
- Semi-thickness or simple mucosal tears may heal themselves if small (Grade 1). However, if the animal is presented with depression/colic/inappetance and a rectal perforation is the cause, the likelihood of healing is reduced.
- If the signs are mild and there is no pyrexia or excessive pain, advise complete rest for 1–2 weeks, feeding very soft food only, with paraffin included 2–3 times daily. Be sure to give pain relief, and antibiotics to avoid secondary infection in the colorectal wall. At the first signs of colic or peritonitis it is advisable to euthanase on humane grounds.
- Good first aid care as soon after the occurrence of the tear can improve the outcome (Watkins et al. 1989). Reduce the straining (consider epidural anaesthesia), remove faecal material and pack the tear with gauze swabs.
- Recommend a soft diet and mineral oil.
- Analgesia and anti-inflammatory drugs should be given.

Prognosis

This is usually poor for all but the most minor type of rectal tear.

Peritonitis is inflammation of the peritoneum that lines the peritoneal cavity.

Equids are much more susceptible to peritonitis than bovine animals.

Peritonitis can be described as:

- primary or secondary
- diffuse or localised
- peracute, acute or chronic
- septic or non-septic.

These descriptions relate to the presentation and underlying cause of peritonitis. Equids most commonly present with secondary peritonitis following damage to the gastrointestinal tract or abdominal wall (Dart and Bischofberger 2011).

Causes Peritonitis is commonly associated with bowel compromise (colic signs), parasitism and abdominal abscesses from infection with *Streptococcus* or *Rhodococcus* bacteria. Injuries which puncture the abdominal wall, rupture of the uterus during foaling, or rectal wall damage during examination, parturition or coitus are frequent causes of acute peritonitis.

Differential diagnoses for peritonitis should include various other causes of colic, as well as myopathies, laminitis, and pleuritis – anything which causes reluctance to move and increased pain, pyrexia and dullness in the animal.

Clinical signs and diagnosis History of trauma, colic or parasitism is extremely suggestive. Diarrhoea, weight loss, and recurrent colic combined with pyrexia are common signs in chronic cases. Systemic signs such as fever and increased heart and respiratory rates are indicative, as is reluctance to move, and ileus in the more critical cases. Severe cases associated with gut rupture show more grave signs of endotoxaemia, septicaemia, depression, acute cardiovascular deterioration, severe abdominal pain, sweating, tachycardia, and red to purple mucous membranes. In severe cases death occurs in 4–24 hours of the rupture.

Rectal exam may reveal 'gritty' peritoneal surfaces or thickening of intestines, as well as possible masses attributing to the peritonitis.

Abdominocentesis This helps achieve a definitive diagnosis (see Section 11.5. of this chapter, on colic).

Treatment Many cases of peritonitis cannot be treated and it is preferable to euthanase the animal. Any type of perforation falls into this category. However, for milder cases, it may be possible to treat symptomatically with fluids, anti-inflammatory drugs and antibiotic therapy; for secondary peritonitis always try to identify and treat the underlying cause (Dart and Bischofberger 2011).

Treatment is long term (> 3 weeks) and may not be ethically justified in working equids, especially if adequate follow-up and nursing care is not possible. Oral trimethoprim-sulphur tablets could be a useful alternative to injectable antibiotics to provide long-term therapy. However, remember the bacteria must be susceptible – if there is no improvement over the course of a week consider euthanasia.

11.9

Liver disease

The liver has an amazing compensatory capacity. In fact 80% of the liver has to be compromised before its functional capacity will start to fail (Pearson 1999).

Liver disease in working equids is relatively common.

The liver is the major metabolising organ of the body, with large amounts of blood passing through it, so it is a common site for secondary abscessation and pathology after an infection such as strangles, toxicity or an inadequate diet. Many drugs are also metabolised by the liver; it is important to administer the correct drugs in the correct amounts and advise owners of the seriousness of inappropriate drug administration.

Clinical signs of liver disease

Sub-clinical liver disease may be common in equids, but signs are only seen when cases are advanced (Gehlen et al. 2010).

Signs are general, variable and often non-specific and many of these common, generalised signs of illness in working equids have any number of underlying causes. It is important to remember the liver during examinations, and take a good history, to be successful in narrowing a case down to a liver problem.

- Depression
- Inappetance
- Weight loss
- Icterus (jaundice)
- Colic
- Neurological signs*: gait abnormalities and motor deficits (rare hepatic myelopathy and encephalopathy – Nout 2011)
- Pruritis and other skin conditions
- Diarrhoea or constipation
- Coagulopathies (dysfunction of blood clotting mechanisms)

*Signs of neurological (central nervous system – CNS) dysfunction can range from mild behavioural changes, which only the owner may notice, to more severe signs such as incoordination, yawning, aimless wandering and dullness.

Acute liver disease

Acute toxicosis is a common cause of sudden onset liver disease – caused by the ingestion/administration of hazardous plants or chemical materials, including drugs, tetanus antitoxin and mycotoxins found in bad feed (Pearson 1999).

Remember that, due to the compensatory mechanisms of the liver, what seems an acute onset liver problem could also be the sudden inability of the liver to cope with a chronic condition, or liver pathology due to a secondary cause.

Clinical signs Depression, lethargy, anorexia, colic, jaundice (Figure 11.9.1) and possible CNS signs (depression, head pressing, incoordination). The urine may be dark-coloured (high bilirubin). A history of the recent ingestion/administration of harmful substances is suggestive of an acute condition (Pearson 1999).

Diagnosis Usually based on clinical signs and history. A blood test for liver enzyme levels usually shows elevations in plasma or serum total bile acids, serum total bilirubin, AST, ALP, GGT, and bilirubin (see Section 4.5); **SDH and GLDH are liver specific**. Differential diagnoses include chronic liver failure/abscessation, chronic active hepatitis and other systemic disease causing anorexia/CNS signs/depression.



Figure 11.9.1 Yellow-coloured (icteric) mucous membranes of the eye.

Treatment The regime below can be used for any suspected liver disease. The aim of therapy is to be supportive until hepatic compensation and regeneration can occur which may take from weeks to months. Work out a timeline with the owner for indicators of improvement. Rest and appropriate feeding can be ongoing; however, sedation, vitamins and antibiotics are not recommended long term. Monitor the case closely, although euthanasia may be the only option if signs do not improve or deteriorate within the first few days despite initial therapy.

- In the first 24–48 hours, sedation with xylazine or another alpha2-agonist is recommended only if neurological signs result in manic behaviour (Pearson 1999). However, depression is more likely.

Remember the liver will have to metabolise any drug administered at this time, thus putting extra pressure on it.

- Ensure the animal is kept in a place where minimum harm can come to it from its decreased mental capacity.
- Provide a high-energy, low-protein diet. Aim to boost blood glucose levels without increasing the strain on the liver to break down proteins – a 5% dextrose IV infusion at 2 L/hour may be effective in the short term. Nasogastric intubation of food may be required initially if the animal is anorexic.
- B-complex vitamin administration IM
- Supportive mechanisms such as fluid therapy, pain relief or antibiotics if signs such as dehydration, colic or pyrexia are present. If available, metronidazole is a good antibiotic to use in liver disease as it can penetrate abscessation, otherwise, trimethoprim sulphur or a combination of penicillin/gentamicin is acceptable.
- Rest!

11 THE GASTROINTESTINAL SYSTEM

Prognosis For acute conditions, expect a rapid, maintained improvement in signs over 24–48 hours as the toxicity is flushed through the system and the liver returns to functional capacity. A downward trend in liver enzymes is also positive for hepatic function improvement; if possible take a blood sample again after 7–10 days. If blood analysis is not available, expect to see a rapid improvement in the animal's condition, a desire to eat and drink, as well as a substantial reduction in CNS impairment.

Failure/slow improvement, or return to clinical signs once therapy is stopped, is an indication that the damage to the liver is extensive or chronic, and prognosis is not good.

Chronic liver disease/chronic active hepatitis

The causes, diagnosis and treatment are similar to those for acute liver disease; however, a history of chronic weight loss is common.

The most common cause of chronic liver failure is from eating hepatotoxic plants over a sustained period of time, especially those containing pyrrolizidine alkaloids.

Become familiar with any plants in the local area which may contain these compounds; often the owners are aware of them already if they have other livestock. Other potential causes are infectious and immune-mediated (Pearson 1999).

Clinical signs A history of chronic weight loss is the most common differentiating factor between acute and chronic liver disease. Other signs are as above for acute disease (depression, neurological signs), although jaundice may not be as pronounced in the chronic case. If chronic disease has resulted in hypoproteinaemia, oedema may occur in dependent areas such as under the jaw/sternum, and polyuria/polydipsia may have been noticed by the owner. Skin lesions may be visible: photosensitisation/pruritis/dermatitis.

Diagnosis Clinical signs, history and elevations in liver enzymes and bile acids are suggestive. The blood results may not be as pronounced as expected since active liver inflammation may have ceased in the chronic case. Toxicosis from pyrrolizidine alkaloids can only be confirmed by liver biopsy (Pearson 1999) which is not practical. Inspection of grazing areas can aid diagnosis if plants are found containing pyrrolizidine alkaloids.

Treatment As for acute cases: treatment should be supportive and eliminate the cause. Response to treatment can help you to determine prognosis and sometimes to differentiate between acute and chronic conditions.

Prognosis Equids with chronic liver disease have a poor prognosis for survival and, as with acute liver failure, treatment is supportive only in the hope that liver function returns, which rarely happens. Owners should be warned of this before embarking on treatment.

Prevention of liver disease

Ideally, this is the best route. It is vitally important to create awareness in owners and equine health providers of the causes and, therefore, the prevention, of liver disease, especially as subclinical disease may be common and yet undetected until too late.

Removal and avoidance of toxic plants (pyrrolizidine alkaloids) and correct dosage of drugs are steps that can be taken (Pearson 1999).

Case study – Impaction colic caused by foreign material consumption

11.10

Location Matrouh, Egypt

Attending veterinarian Dr Ahmed Elrwany

History

A 7-year-old female donkey, presented by the owner. The donkey had not defaecated during the last 12 hours. Previous to this, defaecation was less than normal with small balls. The donkey last ate 12 hours previously and did not finish the whole feed. She last drank a small amount of water 12 hours previously. She was not pregnant, had no teeth problems and was last de-wormed a month ago. The owner had noticed colic signs in the donkey (rolling) 6 hours previous to presentation.

Clinical findings

- Dullness
- Rolling on the ground
- No defaecation, no urination
- Anorexic
- Pulse rate 56 beats per minute
- Respiratory rate 25 breaths per minute
- Rectal temperature 37.2°C
- Decreased gut sounds
- Normal mucous membrane

Diagnosis

Made from history and clinical signs, after full clinical examination. Careful rectal examination found that the rectum was completely blocked by foreign material. The donkey was suffering from impaction colic.

Differential diagnosis

Tympanitic or obstructive colic

Treatment

- Immediate pain control – Injection phenylbutazone 2.2 mg/kg IV
- The foreign material could not be brought out of the rectum on the first rectal examination, so oral water and laxatives were given as well as liquid paraffin 1.5 litres administered by nasogastric tube – to soften and lubricate the faeces.
- Further food was withheld, the donkey walked in-hand regularly to stimulate gut motility, and closely monitored.
- After 12 hours, a piece of foreign material appeared at the rectum that was carefully removed; it was a large piece of cloth (Figures 11.10.1 and 11.10.2).



Figure 11.10.1 Foreign material found in the rectum.

11 THE GASTROINTESTINAL SYSTEM

- Food was withheld a further 12 hours until no other foreign material was passed, only faeces, then small amounts of green fodder was offered 6 times per day for 4 days.
- Normal food was gradually re-introduced.

Prevention

Owner involvement, follow-up plan, good management/husbandry practices and take-home message.

- Feed high-quality hay that is not too mature and hard to digest.
- Feed small meals frequently, instead of large meals once or twice per day.
- Provide plenty of fresh clean water to drink at all times.
- Provide electrolytes to stimulate drinking and replenish any losses.
- Provide preventative care such as appropriate intestinal parasite control and regular dental check-ups.
- Make any changes to the diet, exercise or stabling gradually.
- Observe the donkey daily, looking for any changes in behavior that may indicate ill health.
- Be aware of the average number of piles of faeces that a donkey passes daily and the consistency.

Prognosis

The prognosis was good because, after rectal examination and removing the foreign material, the animal felt comfortable.

Discussion

Colic is a serious welfare issue and may lead to death of the animal. Regular good management/ husbandry practices and timely treatment reduces its severity and saves lives. This case of impacted colic was due to foreign material ingestion. The piece of cloth found on rectal examination was twisted with faecal material, totally blocking the rectum; the animal was not defecating and was in constant pain. After rectal examination and removal of these materials the animal was more comfortable and gradually recovered, showing the effectiveness of correct diagnosis and treatment.

Prevention of impaction colic

The following are potential risk factors:

- Dehydration, due to insufficient fresh water availability
- Animals eating straw bedding, plastic bags, rope and cloth – ensure the stabling/grazing area is clean
- Animals grazing on sandy soil may take in sufficient sand to cause impaction of the colon
- A sudden change in management, such as stabling
- A sudden drop in the amount of exercise



Figure 11.10.2 Foreign material removed from the rectum.

- Almeida, P.E., Weber, P.S.D., Burton, J.L., Zanella, A.J. (2008) Depressed DHEA and increased sickness response behaviors in lame dairy cows with inflammatory foot lesions. *Domest. anim. Endocrin.* 34, 89–99.
- Bertone, J.J. (2002) Clinical field efficacy and safety of N-Butylscopolammonium Bromide in horses. Proceedings of the 48th American Association of Equine Practitioners convention, Florida, USA. 48, 370–374.
- Boulot, S., Brun, J.P., Doreau, M., Martin-Rossett, W. (1987) Activites aliminentaires et niveau d'ingestion chez la jument gestante et al.laitante. *Repro. Nutr. Develop.* 27, 205–206.
- Carroll, C.L., Huntington, P.J. (1988) Body condition scoring and weight estimation of horses. *Equine Vet. J.* 20 (1), 41–45.
- Carson, K., Wood-Gush, D.G.M. (1983) Behaviour of Thoroughbred foals during nursing. *Equine Vet. J.* 15 (3), 257–262.
- Chittora, R. (2012) Discussion on rectal prolapse in working equids. [letter] (Personal communication, 21 August 2012).
- Colahan, P.T., Mayhew, I.G., Merritt, A.M., Moore, J.N. (1999) *Manual of Equine Medicine and Surgery*. Mosby, Inc. Missouri. pp. 17–22.
- Coleman, R.J., Mathison, G.W., Les Burwash, M.S. (1999) Growth and condition at weaning of extensively managed creep-fed foals. *J. Equine Vet. Sci.* 19 (1), 45–50.
- Crowell-Davis, S.L., Houpt, K.A. (1985) Coprophagy by foals: effect of age and possible functions. *Equine Vet. J.* 17 (1), 17–19.
- Dart, A.J., Bischofberger, A.S. (2011) Peritonitis in the horse: a treatment dilemma. *Equine vet. Educ.* 23 (6), 294–295.
- Dobromylskyj, P., Flecknell, P.A., Lascelles, B.D., Livingston, A., Taylor, P., Waterman-Pearson, A. (2000) Pain assessment. In: *Pain management in animals*, Eds: P.A. Flecknell and A. Waterman-Pearson, W. B. Saunders Ltd, London. pp. 53–80.
- Feary, D.J., Hassel, D.M. (2006) Enteritis and Colitis in horses. *Vet. Clin. N. Am. – Equine.* 22, 437–479.
- Finkler-Schade, C. (2007) Development and nutrition of the foal. *Pferdeheilk.* 23, 569–576.
- Gehlen, H., May, A., Venner, M. (2010) Liver disease in horses. *Pferdeheilk.* 26 (5), 668–679.
- Getachew, A.M., Innocent, G., Trawford, A.F., Reid, S.W., James, and Love, S. (2012) Gasterophilosis: a major cause of rectal prolapse in working donkeys in Ethiopia. *Trop. Anim. Health. Prod.* 44 (4), 757–762.
- Gonçalves, S., Julliand, V., Leblond, A. (2002) Risk factors associated with colic in horses. *Vet. Res.* 33 641–652.
- Grindem, C.B., Fairley, N.M., Uhlinger, C.A., Crane, S.A. (1990) Peritoneal fluid values from healthy foals. *Equine Vet. J.* 22 (5), 359–361.
- Hague, B.A., Honnas, C.M. (1998) Traumatic dental disease and soft tissue injuries of the oral cavity. *Vet. Clin. N. Am. Equine Pract.* 14 (2), 333–347.

11 THE GASTROINTESTINAL SYSTEM

- Hallowell, G.D. (2008) Retrospective study assessing efficacy of treatment of large colonic impactions. *Equine Vet. J.* 40 (4), 411–413.
- Henneke, D.R., Potter, G.D., Kreider, J.L., Yeates, B.F. (1983) Relationship between condition score, physical measurements and body fat percentage in mares. *Equine Vet. J.* 15 (4) 371–372.
- Hillyer, M. (2004) A practical approach to diarrhoea in the adult horse. *In Practice.* 26 (1), 2–11.
- Letchworth, G.J., Rodriguez, L.L., Del C. Barrera, J. (1999) Vesicular stomatitis. *Vet. J.* 157, 239–260.
- MacAllister, C.G., Morgan, S.J., Borne, A.T., Pollet, R.A. (1993) Comparison of adverse effects of phenylbutazone, flunixin meglumine, and ketoprofen in horses. *J. Am. Vet. Med. A.* 202 (1), 71–77.
- MacLeay, J.M., Ames, T.R., Hayden, D.W., Tumas, D.B. (1997) Acquired B lymphocyte deficiency and chronic enterocolitis in a 3-year-old Quarter horse. *Vet. Immunol. Immunopath.* 57, 49–57.
- McGorum, B.C., Pirie, R.S. (2010) Antimicrobial associated diarrhoea in the horse. Part 2: Which antimicrobials are associated with AAD in the horse? *Equine vet. Educ.* 22 (1), 43–50.
- Mair, T.S., Pearson, G.R., Divers, T.J. (2006) Malabsorption syndromes in the horse. *Equine vet. Educ.* 18 (6) 299–308.
- Maranhão, R.P.A., Palhares, M.S., Melo, U.P., Rezende, H.H.C., Braga, C.E., Silva Filho, J.M., Vasconcelos, M.N.F. (2006) Most frequent pathologies of the locomotor system in equids used for wagon traction in Belo Horizonte. *Arq. Bras. Med. vet. Zoo.* 58, 21–27.
- Michou and Leece (2012) Sedation and analgesia in the standing horse 2. Local anaesthesia and analgesia techniques. *In Practice.* 34, 578–587.
- Myers, J.O., Rothenberger, D.A. (1991) Sugar in the reduction of incarcerated prolapsed bowel. *Diseases of the colon and rectum.* 34 (5), 416–418.
- Naylor, J.M. (1979) Colostral immunity in the calf and the foal. *Vet. Clin. N. Am. Equine Prac.* 1, 169–178.
- Naylor, R.J., Dunkel, B. (2009) The treatment of diarrhoea in adult horses. *Equine vet. Educ.* 21 (9), 494–504.
- Nolen-Walston, R., Paxson, J., Ramey, D.W. (2007) Evidence-based gastrointestinal medicine in horses: it's not about your gut instincts. *Vet. Clin. N. Am. – Equine.* 23, 243–266.
- Nout, Y.S. (2011) Gait deficits in liver disease: hepatic encephalopathy and hepatic myelopathy. *Equine vet. Educ.* 23 (1), 11–13.
- Orsini, J.A. (2011) A fresh look at the process of arriving at a clinical prognosis Part 2: colic. *J. Eq. Vet. Sci.* 31 (7), 370–378.
- Ousey, J.C., Prani, S., Zimmer, J., Holdstock, N., Rossdale, P.D. (1997) Effects of various feeding regimes on the energy balance of neonates. *Am. J. Vet. Res.* 58, 1243–1251.
- Pearson, E.G. (1999) Liver disease in the mature horse. *Equine vet. Educ.* 11 (2), 87–96.
- Pringle, R.O. (1871) Purdon's veterinary handbook. The diseases of horses, cattle, sheep, swine, dogs and poultry, their causes, symptoms and treatments. 2nd Ed. William Blackwood and sons, Edinburgh and London. p. 10.
- Proudman, C.J. (2003) Diagnosis, treatment, and prevention of tapeworm-associated colic. *J. Eq. Vet. Sci.* 23 (1) 6–9.

- Proudman, C.J., Trees, S. (1999) Tapeworms as a cause of intestinal disease in horses. *Parasitology Today*. 15 (4) 156–159.
- Reeves, J.R., Salman, Mo. D., Smith, G. (1996) Risk factors for equine acute abdominal disease (colic): Results from a multi-center case-control study. *Prev. Vet. Med.* 26, 285–301.
- Scantlebury, C.E., Archer, D.C., Proudman, C.J., Pinchbeck, G.L. (2011) Recurrent colic in the horse: Incidence and risk factors for recurrence in the general population. *Equine Vet. J.* 43 (s39) 81–88.
- Stoneham, S.J. (2005) How to feed the sick neonatal foal. In, *The 1st BEVA and Waltham nutrition symposia*. Eds: Harris, P.A., Mair, T.S., Slater, J.D., and Green, R.E. Equine Veterinary Journal Ltd. Cambridge, England. pp. 33–37.
- Taylor, S.D., Pusterla, N., Vaughan, B., Whitcomb, M.B., Wilson, W.D. (2006) Intestinal Neoplasia in Horses. *J. Vet. Intern. Med.* 20, 1429–1436.
- Tell, A., Egenvall, A., Lundstrom, T., Wattle, O. (2008) The prevalence of oral ulceration in Swedish horses when ridden with bit and bridle and when unriden. *Vet. J.* 178, 405–410.
- Vall, E., Ebangi, A.L., Abakar, O. (2003) A method for estimating body condition score (BCS) in donkeys. *Working animals in agriculture and transport: a collection of some current research and development observations*. 6, 93–102.
- Venner, M., Ohnesorge, B. (2001) Glucose and D-xylose absorption test for diagnosis of malabsorption in the horse. *Tierarztl. Prax. G. N.* 29 (4), 256–259.
- Watkins, J.P., Taylor, T.S., Schumacher, J., Taylor, J.R., Gillis, J.P. (1989) Rectal tears in the horse: an analysis of 35 cases. *Equine Vet. J.* 21 (3), 186–188.
- Weese, J.S., Staempfli, H.R., Prescott, J.F. (2001) A prospective study of the roles of *Clostridium difficile* and enterotoxigenic *Clostridium perfringens* in equine diarrhoea. *Equine Vet. J.* 33 (4), 403–409.
- Weishaupt, M.A., Wiestner, T., Hogg, H.P., Jordan, P., Auer, J.A. (2004) Compensatory load redistribution of horses with induced weight-bearing hindlimb lameness trotting on a treadmill. *Equine Vet. J.* 36, 727–733.
- Widman, E. (2008) How to deal with the choking horse. *J. Eq. Vet. Sci.* 28 (9) 504.

Further reading

- Bezdekova, B. (2012) Esophageal disorders of horses: a review of literature. *Pferdeheilk.* 28 (2), 187–192.
- Hague, B.A., Honnas, C.M. (1998) Traumatic dental disease and soft tissue injuries of the oral cavity. *Vet. Clin. N. Am. Equine Pract.* 14 (2), 333–347.
- Hillyer, M. (2004) A practical approach to diarrhoea in the adult horse. In *Practice*. 26 (1), 2–11.
- National Research Council (2007) *Nutrient requirements of horses*. 6th Ed. National Academy of Sciences, Washington, USA.