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Satellite Article

Nonstrangulating obstruction of the small intestine in the horse

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Introduction

Abdominal disease in the horse has been a significant concern for man from the time the species was first domesticated. Although colic by definition means abdominal pain, the term has evolved to define a condition rather than a clinical sign.

Treatment of colic in horses has been documented in texts and journals since the early 1800s. In the early years of the veterinary profession most patients suffering serious abdominal pain succumbed to the condition or were humanely destroyed. Most episodes of abdominal pain that are the result of a disorder of the gastrointestinal tract recover spontaneously or respond to medical therapy but a small percentage of cases require surgical intervention. Sir Frederick Hobday of the Royal Veterinary College was performing some of the attempts at exploratory laparotomy in the treatment of colic in 1897. Since the 1970s, great advances have been made in the understanding of the aetiology and pathogenesis of gastrointestinal disease in the horse; and many advancements made in successful treatment.

This article accompanies the case report by Jansson (2000) on spontaneous correction of a nonstrangulating ileal obstruction caused by a pedunculated lipoma in a 14-year-old pony.

Aetiology

Nonstrangulating obstruction of the small intestine as a disease entity can be divided into 2 broad categories:

- Functional obstruction related to ileus
- True obstruction related to stricture or impaction.

Ileus is an adynamic condition that can occur throughout the length of the intestine or in isolated segments (Bertone 1995). **Inhibition of intestinal motility** has been proposed to be a function of spinal and peripheral reflex arcs of the sympathetic nervous system. It has been reported that malfunctions of the myenteric plexus of the small intestine also results in intestinal paralysis (Allen *et al*. 1989; Burns *et al*. 1990). Propulsive motility is increased under parasympathetic, and reduced with sympathetic, influence. Stimulation of the spinal reflex has been shown to occur with both intra-abdominal and extra-abdominal pain as well as in severe infection (Neeley and Catchpole 1971). **In general terms, any painful condition or condition that results in sympathetic stimulation can induce adynamic ileus.**

Alterations in calcium and potassium homeostasis may also play a role in development of ileus. Hyperkalaemia causes an increase in the resting membrane potential. As this potential approaches or equals the threshold potential, a depolarising block occurs, resulting in smooth muscle paralysis. Hypokalaemia can be associated with intestinal hypermotility. Hypercalcaemia results in decreased excitability of smooth muscle and in extreme degree results in ileus. Hypocalcaemia promotes a reduction in the contractile force exhibited by gastrointestinal smooth muscle resulting in diminished propulsive motility (Feldman 1987; Dart *et al*. 1992).

True obstruction of the small intestine is an infrequent occurrence. In one study small intestinal obstruction represented only 3.2% of the total colic cases reviewed and was most often caused by impaction of the ileum (White and Lessard 1986). Other work indicates ileal impaction represents 0.5–10.8% of acute abdominal disease cases (Edwards 1981; Huskamp 1982; Parry 1983; Kersjes *et al*. 1988; Baxter and Broome 1989). Infrequently, ingested foreign materials, such as wood shavings, synthetic hay nets, binder twine, or small hard fruit have been reported as a cause of small intestinal obstruction (Wilson and Scruggs 1992). Neoplasia, fibrosis, abscess, trichophytobezoar, choleliths, and ascarid impaction have also been reported to result in intestinal blockage (Rumbuagh *et al*. 1978; Turner 1986; Green and Tong 1988; Rook *et al*. 1991; Laverty *et al*. 1992; Traub-Dargatz *et al*. 1992; Kasper and Doran 1993; Kraus-Hansen 1995).

Ileal impaction and obstruction is more commonly seen in the south-eastern part of the USA and is proposed to be related to consumption of Coastal Bermuda grass hay commonly available in that region (Doran *et al*. 1985; Embertson *et al*. 1985). In this region, a significantly higher rate of ileal impaction occurs during the fall of the year when the crude fibre content of the forages increases and digestibility decreases (Hanson *et al*. 1998). Muscular ileal hypertrophy can also result in small intestine obstruction (Rooney and Jeffcott 1968; Lindsay *et al*. 1981). Proposed mechanisms include alterations in parasympathetic nervous system function resulting in hypertrophy of the musculature, and chronic hypertonicity

TABLE 1: Patient history

- **General history**
  1. Environment
  2. Diet
  3. Use of the patient
  4. Medical history
  5. Parasite control programme
- **Recent data**
  1. Onset of signs
  2. Most recent consumption of feed or water
  3. Changes in diet
  4. Changes in medication, housing, use or activity
  5. Changes in companions
  6. Most recent anthelmintic therapy
  7. Reproductive status
  8. Trauma or injury
  9. Potential for foreign body ingestion
- **Presenting complaint data**
  1. Degree and progression of pain
  2. Signs observed
  3. Treatments administered
  4. Response to treatments
  5. Last bowel movement
  6. Character of faeces

TABLE 2: Physical examination

- Nasogastric intubation – volume, gas, fluid, colour, pH
- Mucous membranes – colour, capillary refill time (CRT)
- Blood work – PCV, total solids, electrolytes, calcium, bicarbonate
- Pain – severity, intermittent or continuous
- Heart rate
- Respiration rate
- Body temperature
- Attitude
- Physical condition or concurrent injuries
- Abdominal auscultation – borborygmi, rate, intensity
- Abdominal distention
- Rectal examination – distended bowel, displacement
- Abdominal ultrasound – distended bowel, intussusception, fluid in the abdominal cavity, thickened bowel wall
- Abdominocentesis – colour, character, cytology, total solids

of the ileocaecal valve (Chaffin *et al*. 1992). It has been proposed that a high parasite load in the caecum may also result in hypertonicity of the ileocaecal valve with subsequent hypertrophy of the ileum (Proudman *et al*. 1998; S.R. Hance, personal communication).

Obstruction of any form results in an inhibition of the oral to aboral movement of ingesta. From the point of the obstruction orad, ingesta collects as the nonaffected intestine proximal to the blockage continues to propel ingesta aborally. Retrospective studies of ileal impaction treated surgically indicate the long-term survival rate (>1 year) ranges from 31 to 71% (Doran *et al*. 1985; Embertson *et al*. 1985; Parks *et al*. 1989; Hanson *et al*. 1998).

Clinical features

In the early stages of obstruction, intestinal motility proximal to the obstruction increases as slight distention stimulates peristalsis (MacHarg 1986). As distension increases, intestinal spasms develop, resulting in intermittent abdominal pain. In later stages of the disease, distention of the intestine becomes sufficient to stimulate stretch pain receptors in the intestinal wall causing a continuous and substantial degree of abdominal discomfort. The intensity of pain is proportional to the magnitude of tension placed on the bowel wall (Watson and Stodeman 1974).

Distension results in increased rate of secretion of fluid into the lumen and decreased absorption of fluids from the lumen (Kohn 1982; Allen 1988). As the intraluminal pressure equals or exceeds the venous portal pressure, the veins, venules and lymphatics that drain the tissues of the intestine collapse causing an increase in capillary pressure (Granger and Shepard 1979). Increased capillary pressure results in movement of fluid from the lumen of the capillaries into the interstitial space and subsequently the intestinal lumen and abdominal cavity (Kohn 1982). Research has shown that net production of gastric reflux fluid from an obstructed bowel can be up to 0.3 l/100 kg bwt/h (Puotunen-Reinert and Huskamp 1986).

Distention of the intestine also results in a reduction in blood flow to the intestinal tissues and subsequent ischaemic injury (Kohn 1982; Snyder 1989; Dabareiner *et al*. 1993; Kooreman *et al*. 1998). Oxygen deprivation causes a decrease in oxidative phosphorylation resulting in reduced ATP production. Intestinal mucosal epithelial cells have a very high metabolic rate. Inadequate supply of ATP for these tissues results in the utilisation of anaerobic glycolysis for energy production. Application of this metabolic pathway results in intracellular acidosis, which further inhibits ATP production. As energy production becomes insufficient to meet the cellular demands, cellular swelling occurs due to the inability of energy dependent membrane ion pumps to maintain a normal ion balance. As cellular swelling increases, structural damage occurs to cellular membranes resulting in the inability of the cell as a whole to maintain homeostasis. This alteration in cell function results in leakage of lysosomal and other degradative enzymes causing autolytic destruction of cellular organelles. These changes result in irreversible cell damage and cell death (Moore *et al*. 1995).

Symptoms of small intestine obstruction are highly variable between horses, making diagnosis sometimes difficult. Horses that are very stoic may present with very few or very mild signs in the face of a severe lesion. Others may react violently to mild abdominal pain. In general, abdominal pain is manifested by depression, anxious behaviour, sweating, bruxism, groaning, looking at or biting at the flanks, laying down, dog sitting, pawing at the ground, rolling, kicking at the abdomen with the hind limbs, or stretching.

Diagnosis

With the colic patient, triage should be performed before

TABLE 3: Packed cell volume (PCV) and total protein (TP) values in relation to dehydration

PCV (%)	43–50	Slight
TP (g/l)	70–82	Dehydration (6%)
PCV (%)	50–57	Moderate
TP (g/l)	83–95	Dehydration (8%)
PCV (%)	>57	Severe
TP (g/l)	>95	Dehydration (10%)

TABLE 4: Normal equine peritoneal fluid values

Subjects studied	20 Horses[a] and ponies	13 Horses[b]	17 Foals[c]
Total erythrocyte count (cells/μl)	0	0	0
Total nucleated count (cells/μl)	15–10,100[d] ($1.5–10x10^9/_1$)	1890–4610 ($1.9–46x10^9/_1$)	60–1420 ($6–14x10^9/_1$)
Differential cell count			
Neutrophils %	22–82	24–62	2–94
Lymphocytes %	1–19	5–36	0–7
Large mononuclear cells %	19–68	17–50	5–98
Eosinophils %	0–5[d]	1–6	0–4[e]
Total solids g/l	2.0–15	7.0–11	14–19
Specific gravity	1.008–1.012	1.000–1.015	1.012–1.015

[a]Bronlow 1979; [b]Nelson 1979; [c]Grindem 1990; [d]19 horses (95%) had total nucleated cell count of 1500–7600 cells/μg; [e]16 foals (94%) had 0% eosinophils.

embarking on an in-depth diagnostic work-up. In severe cases of intestinal obstruction, gastric reflux can result in distention of the stomach to the degree that gastric rupture can occur. With gastric distention the patient is often in significant pain. Since gastric rupture could be imminent, **it is imperative that the stomach be immediately decompressed through nasogastric intubation**. In some patients, it has been proposed that distention of the stomach results in spasm of the cardiac sphincter making it difficult or impossible to pass a nasogastric tube into the stomach (J. Schumacher, personal communication). Infusion of 30 ml 2% lidocaine solution followed by 60 ml water through the nasogastric tube to deliver the lidocaine at the cardiac sphincter may result in relaxation of the sphincter and successful passage of the tube (R.H. Purohit, personal communication). The net volume of fluid aspirated from the stomach and time of collection should be noted for later assessment of reflux production rate. The normal capacity of the mature equine stomach is 8–20 l (mean = 18 l) (Nickel *et al.* 1979). Net recovery of less than 2 l fluid should not be considered significant, but repeat recovery of volumes greater than 2 l fluid at 1 to 2 h intervals indicates a marked alteration in intestinal function.

Pulse quality, mucous membrane colour, capillary refill time and heart rate should be assessed to determine the

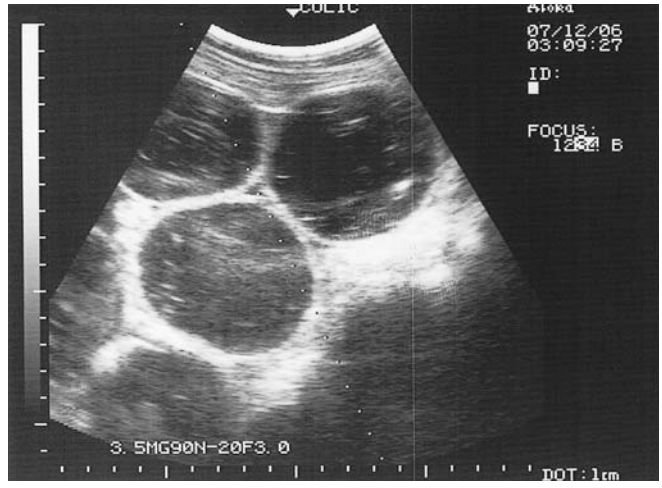

Fig 1: Abdominal ultrasound: cross section of loops of distended small intestine.

quality of peripheral perfusion. If necessary, i.v. fluids should be administered immediately at sufficient rate and volume to combat the onset of cardiovascular collapse.

Patient history is an integral component of diagnosis after the immediate needs of the patient have been met. The veterinarian should prompt the handler or owner with specific but nonleading questions to ensure the collection of accurate information. Responses must be carefully interpreted, as errors in management and care of the animal are not generally professed. Since quality historical data is instrumental in making an accurate diagnosis care and attention to detail in this area are essential. The history can be collected in 3 parts (a) general history, (b) recent data, (c) presenting complaint data (**Table 1**).

After collection of history, a physical examination should be performed. A thorough examination is essential as decisions for treatment and prognosis are made utilising this examination data (**Table 2**). Physical examination often reveals tachycardia, tachypnoea, increased or decreased borborygmi, distended abdomen, increased capillary refill time (CRT), pale mucous membranes and dehydration. **It is important to remember that** diagnosis and prognosis cannot be made on the individual components of a physical examination. The individual parameters must be fitted together like the pieces of a puzzle in an attempt to develop a more lucid picture of the situation at hand.

Tachycardia is often the result of the response to pain and/or cardiovascular shock. Cases of simple obstructive disease of the small intestine generally have slight elevations in heart rate in the range 40–70 beats/min, but more severe cases can result in heart rates greater than 100. **Prompt and aggressive therapy is indicated with heart rates greater than 80 beats/min**.

Cardiovascular shock can be a result of the effects of circulating endotoxin (Moore 1988), reduced circulating blood volume due to dehydration, and/or inhibition of venous return related to abdominal distension. Other signs of shock include increased CRT and discoloration of

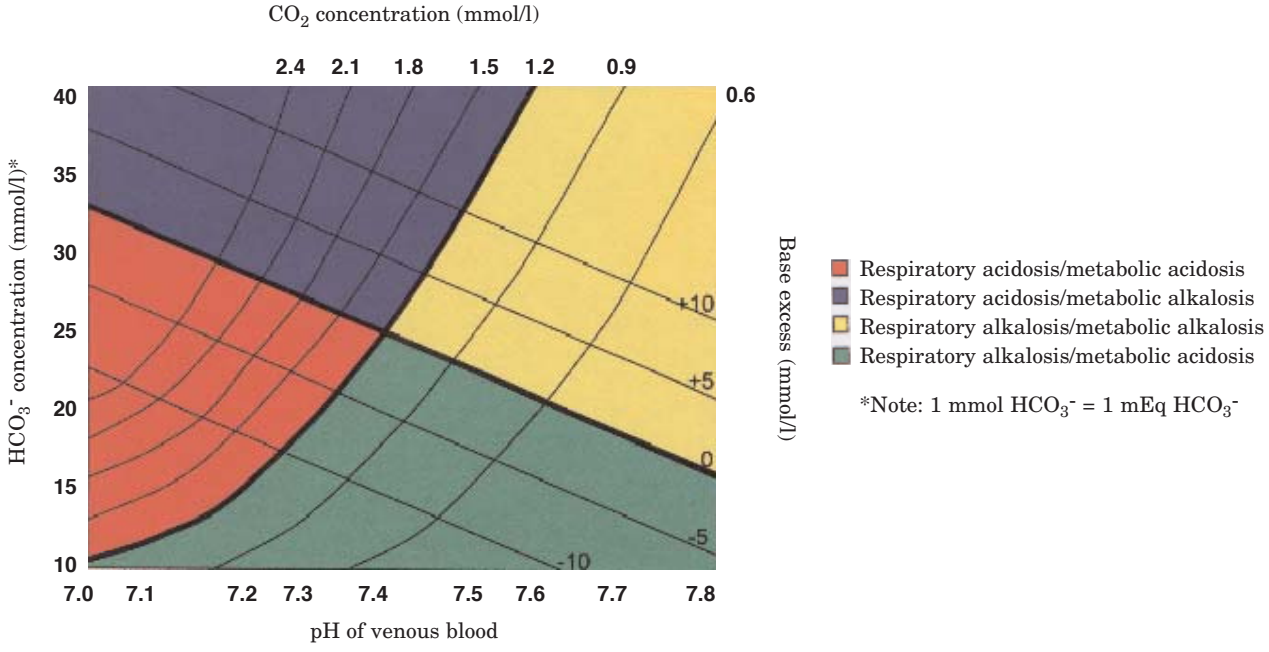

Fig 2: Davenport diagram.

the gums. Normal CRT is <2 s. Horses with moderate reduction in peripheral perfusion have CRT 3 or 4 s and severely reduced perfusion results in CRT of >5 s. Mucous membrane colour is normally pale pink. Patients suffering from endotoxaemia can develop bright red mucous membranes. During severe shock mucous membranes become dark red to cyanotic.

Dehydration can be assessed roughly by the time-honoured skin pinch test or through interpretation of the packed cell volume (PCV) and total plasma protein (TP) values. The skin pinch test is difficult to interpret with minor degrees of dehydration. Age and body condition of the animal can also significantly affect the accuracy of the test. **PCV and TP analysis has proven to be a more repeatable and accurate test of hydration status** (**Table 3**).

Auscultation of the abdomen is performed using a stethoscope in a 4-quadrant system. The quadrants consist of the upper and lower paralumbar regions on the left and right side of the patient. Each quadrant is auscultated for several minutes to determine the degree of intestinal motility. The loudest and deepest sounds are associated with contractions of the large intestine and caecum. Contractions in these structures normally occur on a 2–4 min cycle, but variations occur relative to recent consumption of feed. Auscultation of the small intestine is more difficult as this organ can function while producing very little sound. Sounds generally associated with the small intestine tend to be more fluid in nature and occur every 15 to 45 s with variations in frequency related to recent consumption of feed. In general, pain inhibits gastrointestinal motility by activating inhibitory reflexes

TABLE 5. Gastric motility stimulators

Drug	Dose	Area of action	Side effects
Cisapride[a]	0.1 mg/kg i.m. or i.v. q. 8 h 0.15 mg/kg *per rectum* q. 6 h	Small and large intestine	Mild tachycardia, mild colic
Erythromycin[b]	0.1–1.0 mg/kg i.v. q. 12 h Administer as an every other day treatment	Entire GI tract	Diarrhoea
Lidocaine[c]	Loading dose 1.3 mg/kg i.v. followed by continuous infusion of 0.05 mg/kg/min	Entire tract	None
Yohimbine[d]	0.075–0.25 mg/kg i.v. q. 3 h	Entire tract	CNS effects

[a]King and Gerring 1988; Velden and Klien 1993; [b]Masri *et al*. 1991; [c]Rimbach *et al*. 1990; [d]Roberts and Argenzo 1986.

in the digestive tract (Furness and Costa 1974; Nadrowski 1983). **As a result, a painful lesion in the small intestine can affect motility of the entire digestive tract.**

Transrectal examination of the caudal abdominal cavity may reveal loops of distended small intestine in any or all of the caudal abdominal quadrants. Character of faeces removed from the rectum should be noted as it can provide useful insight into the disease process. Mucus- or fibrin-covered faeces may be consistent with enteritis or prolonged faeces retention compatible with the early stages of impaction type diseases. The presence of sand may indicate sand impaction and a simple test is to place a handful of faeces in a rectal sleeve and mix with water. The sleeve is then hung up and the mixture allowed to settle. **Any sand present settles into the fingers of the sleeve becoming easily observed and quantified**.

Abdominal ultrasound has become a useful diagnostic tool in assessment of colic (Klohnen *et al*. 1996). Utilising a 3.5 MHz transducer and isopropyl alcohol applied to the skin as a coupling medium, loops of distended small intestine are observed easily and their diameter measured and degree of motility assessed (**Fig 1**). Thickness of the intestinal wall is also assessed readily as well as the presence and location of free fluid in the abdominal cavity. Ultrasound can also be extremely helpful in selecting an abdominocentesis site that will provide a greater potential for recovery of abdominal fluid.

Abdominal fluid analysis allows significant insight into the degree of compromise of the intestinal tissues (Freden *et al*. 1998). Normal fluid should be straw-coloured with no turbidity and a low total protein content (**Table 4**). There should be no bacteria or faecal material present in the fluid. Identification of these constituents indicates either enterocentesis or leakage/rupture of the digestive tract. Excursions from normal values indicate compromised tissues within the abdomen. The severity of intestinal compromise is related to degree of deviation from normal values.

Treatment

Medical management of nonstrangulating obstruction of the small intestine includes i.v. fluids, analgesics, intestinal motility stimulators, prevention of oral consumption of feed and water, and preemptive laminitis therapy.

As long as there is significant gastric reflux production, consumption of food and water must be prohibited and i.v. fluids must be administered. The i.v. maintenance fluid requirement for the normal mature horse at rest is approximately 3 ml/kg bwt/h. However, adjustments in this administration rate must be made to compensate for ongoing fluid losses, such as gastric reflux. **The volume of i.v. fluids required for elimination of hydration deficit can be calculated using the following formula:**

- bwt (kg) x % dehydration (expressed as a decimal) = litres i.v. fluids required

Example: A 500 kg horse that is 8% dehydrated, requires

- 500 kg x 0.08 = 40 l i.v. fluid to address fluid deficit.

Patients with severe volume deficits may receive 20–30 l of balanced electrolyte solution/h until the deficit is corrected. Serial PCV/TP analysis should be performed at 6 to 12 h intervals to assess hydration status with adjustments made to maintain proper levels of hydration (**Table 3**).

Acid-base balance is most accurately assessed through blood gas analysis. **Hypovolaemia** and increased metabolic rate usually result in metabolic acidosis. In general, immediate and complete correction of acid-base derangement is not desirable as normalisation of hydration status often results in correction acid-base derangement. **Decision on acid-base therapy should be made after adequate rehydration of the patient has occurred**. Venous blood gas analysis is a more accurate reflection of the state of the cellular environment in comparison to arterial blood gas values (DiBartola 1992). Using the results of blood gas analysis in conjunction with the **Davenport Diagram**, the type and severity of acid-base derangement can be determined (**Fig 2**). Correction of bicarbonate deficit should be considered when the base deficit exceeds 10 mmol/l (NB: 1 mmol HCO_3^- = 1 mEq HCO_3^-). Initially half the calculated bicarbonate deficit should be administered over a 3 or 4 h period. **After the initial treatment**, recheck the acid-base status to determine if further therapy is required. **To determine the quantity of bicarbonate required for treatment, utilise the following formula:**

- bwt (kg) x base deficit (mmol/l) x 0.3 = quantity of HCO_3^- to administer (mmol)

Example: A 500 kg horse with a base deficit of 19 mmol/l

- 500 kg x 19 mmol/l x 0.3 = 285 mmol HCO_3^- to be administered to eliminate the base deficit.

Remember, only half the dose should be administered before the blood gas analysis is rechecked, so half of 285 mmol = 142.5 mmol. If an 8.4% (1 mmol/ml) sodium bicarbonate solution is used as the HCO_3^- source, then 142.5 ml of the 8.4% solution should be added to the replacement fluids.

Serum electrolyte analysis is a valuable diagnostic tool as metabolic derangement can result in response to a variety of influences. Serum electrolyte analysis and supplementation should not occur until normal hydration levels have been achieved. Potassium is an intracellular ion and **decreases** in serum potassium are to be expected with anorexia and diarrhoea. **Increases** in serum potassium are seen in acidaemia as hydrogen ions are exchanged by the cell for potassium ions in an attempt to correct intracellular pH. The following formula can be used to calculate the quantity of potassium needed for deficit correction:

- desired serum K^+ (mmol/l) - measured serum K^+ (mmol/l) x bwt (kg) x 0.3 = quantity of potassium to administer

Example: 500 kg horse with a measured serum potassium of 2.1 mmol/l and a desired serum potassium of 3.5 mmol/l

- 3.5 mmol/l - 2.1 mmol/l x 500 kg x 0.3 = 210 mmol of potassium administered.

*Note: 1 mmol K^+ = 1 mEq K^+

If a 2 mmol/ml KCL solution is used for supplementation, then 105 ml of the KCL solution should be added to the replacement fluids. **The administration rate should not exceed 0.5 mmol K^+/kg bwt/h. The replacement fluids should not contain more than 60 mmol/l of potassium. Greater concentrations will cause pain and phlebitis at the catheter site**.

Hypocalcaemia is also commonly seen with anorexia. Calcium therapy may be instituted when serum calcium levels drop to 0.5 mmol/l (2 mg/dl) below low normal values. The authors generally add 500 ml 23% calcium gluconate to the initial replacement fluids and, as needed, thereafter. This volume of calcium gluconate should be administered over no less than a 15 min period. Retesting of postadministration calcium levels should be used to determine further therapy. Calcium containing solutions are not compatible with solutions that contain bicarbonate. **Combining these 2 components will result in formation of an insoluble precipitate.**

Analgesics are administered to reduce abdominal pain and to help inhibit spinal reflex-induced ileus. **Flunixin meglumine** is an NSAID that can be used to control moderate visceral pain and also bind endotoxin. The analgesic/anti-inflammatory dose is 1.1 mg/kg bwt i.v. q. 12 h or 0.55 mg/kg bwt i.v. q. 6 h. The endotoxin binding dose is 0.25 mg/kg bwt i.v. q. 6 h (Semrad *et al*. 1987; Moore 1988). **Prolonged high dose use of this drug has been shown to promote gastric ulceration and subsequent reduction in intestinal motility.**

Xylazine hydrochloride is an α2-adrenergic agonist and a potent sedative and analgesic. It is often used to treat acute episodes of abdominal pain. The duration of action ranges from 15 to 45 min after i.v. administration, depending on the severity of abdominal disease (Muir 1991). Common i.v. dosages range from 0.3 to 0.7 mg/kg bwt. This range of dose produces mild to significant levels of analgesia and sedation. Administration i.m. may also be used **with a longer duration of effect with slower onset.** Intramuscular (i.m.) doses range from 0.6–1.4 mg/kg bwt and produce approximately the same depth of sedation and analgesia as the lower i.v. doses.

Detomidine hydrochloride is also an α2-adrenergic agonist and a potent sedative and analgesic. In the authors' experience, the duration of action obtained with detomidine is approximately 2 or 3 times longer than that seen with xylazine. Common dosages range from 0.006 to 0.02 mg/kg bwt i.v. This range produces mild to significant levels of analgesia and sedation. This drug may also be administered i.m. at a dose twice the i.v. dose to produce the same degree of analgesia and sedation with slower onset of action and increased duration of effect.

Both xylazine and detomidine should be used with caution with patients suffering from cardiovascular shock. Administration causes a reduction in blood pressure that could lead to collapse in some patients. The α2-adrenergic agonists have also been shown to depress intestinal motility (Rutkowski *et al*. 1991). **Reversal of the effects of both drugs** can be produced by administering Yohimbine, an α2-adrenergic antagonist at 0.075 mg/kg bwt i.v. (Muir 1991; Plumb 1995).

Butorphanol tartrate, a synthetic opiate partial agonist, is often administered concurrently to potentate the analgesic effects of both xylazine and detomidine. Common dosages range from 0.01 to 0.05 mg/kg bwt i.v. **Butorphanol is compatible in the same syringe with xylazine or detomidine.**

Numerous **motility stimulators** have been utilised with variable success in the treatment of paralytic ileus (**Table 5**) (Dart and Hodgson 1998). **Lidocaine hydrochloride** i.v. is showing promise in treating low level abdominal pain and ileus. Two theories have been proposed as to the mechanism of action. Several studies have shown that lidocaine may have a direct stimulatory effect on intestinal smooth muscle (Feinstein and Paimre 1969; Wood and Harris 1972; Tansy and Ganse 1972; Bortoff and Muller 1999). Other work indicates that systemically administered lidocaine blocks pain associated with ileus therefore blocking the inhibitory reflex and thereby releasing spontaneous myogenic activity (Furness and Costa 1974; Nadrowski 1983). Dosage of lidocaine i.v. consists of a loading dose of 1.3 mg/kg bwt administered over a 3 or 4 min period followed by a continuous infusion of 0.05 mg/kg bwt/min. The continuous infusion has been administered for up to 96 h with no negative impact on any body systems (G.A. Meyer, unpublished data). The present authors normally add 550 ml of 2% lidocaine to 5 l of balanced electrolyte solution resulting in a 2 mg/ml solution. This solution is then administered via a 15 drop/ml calibrated drip set after administration of the loading dose.

Laminitis is a potentially fatal complication of GI derangement. The mechanism of induction of laminitis is not clear and appears multifactoral, but endotoxin has been implicated with the disease (Moore 1979). Through whatever mechanism, a reduction of blood flow to the laminae occurs, resulting in ischaemic necrosis and subsequent physical breakdown of these tissues (Ekfalck *et al*. 1992). To date, the only truly effective treatment for laminitis is prevention. The present authors routinely apply the following pre-emptive treatment protocol. Heparin administered at a rate of 60 iu/kg bwt i.v. or sub cut. q. 12 h (R.E. Embertson, personal communication). In association with heparin administration, PVC is monitored q. 12 h and the heparin discontinued when PCV drops to 20% or the risk of laminitis passes. Acepromazine or nitroglycerine paste is also concurrently administered for their vasodilative properties. Acepromazine is dosed at 0.03 mg/kg bwt i.m. or sub cut. q. 8 h (Walker and Geiser 1986; Hunt *et al*. 1994).

Nitroglycerine 2% paste is dosed for a 450 kg horse as a 2 or 3 cm strip as it is dispensed from the tube. The

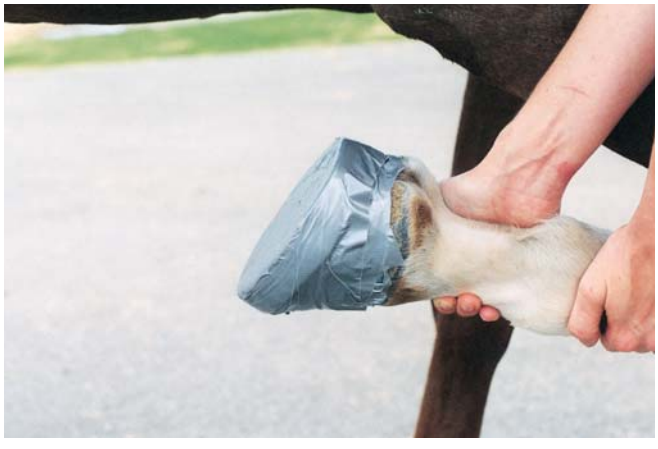

Fig 3: Application of foam pads. Duct tape is applied to fix the pad to the foot.

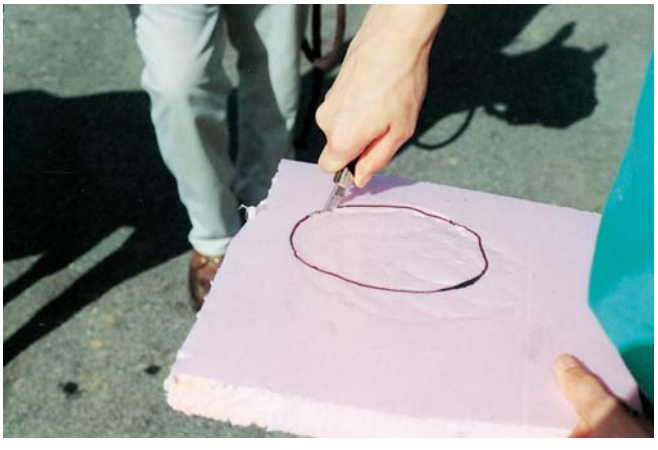

Fig 4: Fabrication of foam pads. The foot is traced and the pad trimmed with a knife.

paste is applied to the skin of the proximal medial hindlimb in the area of minimal hair growth q. 12 h. Because nitroglycerine is a systemically acting drug, the accepted practice of application to the coronary band has been found by these authors to offer no advantage. Sole and frog support is also promoted by the application of conforming Styrofoam pads affixed to the bottom of the feet (**Fig 3**). These pads are fabricated from 122 x 244 x 2.5 cm (4' x 8' x 1") foam insulating board obtained from local building material suppliers (**Fig 4**).

Surgery

The decision for surgical treatment of small intestinal disease is often based on several different criteria. The degree of pain and response to analgesics, physical examination parameters, transrectal findings, and abdominal fluid analysis must all be carefully considered in assessing the need for exploratory surgery. Cardiovascular and toxic shock are the ultimate reasons for death in horses suffering intestinal disease (Nguhiu-Mwangi 1986). The cascade of pathological events that lead to shock must be interrupted before the process becomes irreversible. Delaying the decision for surgery while waiting for physical examination, abdominal fluid character or laboratory parameters to degrade can result in a patient that poses an extreme anaesthetic risk and one that will often require longer and more intensive postoperative therapy.

Manifestation of pain is the most valuable single tool in assessment of the need for surgery. Patients displaying severe unrelenting pain that is nonresponsive to analgesics or is responsive for only a short time must always be considered to be surgical candidates. Most equine surgeons agree that occasionally to err on performing exploratory surgery on a patient with nonsurgical lesions is far better than to lose a patient because the decision for surgical intervention was made too late.

Surgery to relieve obstructive lesions of the small intestine most commonly involves manual reduction of feed impaction. Less frequently, obstruction of the small bowel requires entering the lumen of the intestine to relieve the blockage.

Manual breakdown of impactions without opening the lumen of the bowel is the most desirable means of resolution of obstruction. **Infusion of lubricants** such as mineral oil, carboxymethylcellulose, the surfactant diocytl sodium succinate (DSS), physiological saline, or

balanced electrolyte solution into the lumen of the intestine via a 18 gauge needle can be helpful in manual reduction of impactions.

Those obstructions that are not manually reducible require an enterotomy for removal. Sections of nonviable bowel require resection and anastomosis. Ileal hypertrophy may be treated by ileal bypass or jejunocaecostomy. **The use of stapling devices in performing enterotomies or resection and anastomosis has significantly reduced the time required for these procedures** (van der Velden and van der Gaag 1987). Care must be given in evaluation of the thickness of the bowel when utilising stapling equipment. **Excessive wall thickness can result in insufficient crimping of the staples** causing postoperative leakage or failure of the enterotomy or anastomosis site (Anon 1980). When closing oedematous bowel, the more time-consuming hand-suturing techniques may result in a superior closure.

Common complications associated with gastrointestinal surgery include formation of interintestinal adhesions, abscessation, septic or sterile peritonitis, postoperative ileus, incision line infections, incision line herniation, postoperative diarrhoea, or laminitis.

The authors have had good success in reducing the incidence of interintestinal adhesion formation by utilising copious intra-abdominal lavage with balanced electrolyte solution in conjunction with heparin therapy. The intra-abdominal lavage is followed by infusion of 1 litre balanced electrolyte solution containing 20,000 iu sodium heparin, just prior to closure of the incision. Postoperatively, heparin is also administered at a rate of 60 iu/kg bwt i.v. q. 12 h for 48 h or until the PCV drops to 20% or below. **Omentectomy** is also showing great promise in reducing intraabdominal adhesion formation. In one study a substantial reduction in incidence of small intestine adhesion formation was recorded when omentectomies were performed (Kuebelbeck *et al*. 1998).

Abscessation and or **septic peritonitis** is generally related to contamination of the abdominal cavity with foreign material. Leakage of anastomosis sites or spillage of intestinal contents into the abdomen during enterotomy procedures is often implicated. Rinsing the enterotomy site prior to incising the bowel, with sterile balanced electrolyte solution containing heparin at a concentration of 10,000 iu heparin/l solution significantly reduces the adherence of ingesta to serosal surfaces. This technique significantly enhances the ability to rinse away contaminants from the enterotomy site, after completion of the enterotomy.

Conclusion

Small intestinal obstructive disease poses a significant diagnostic and therapeutic challenge. Derangement of numerous organ systems is often associated with the primary disease. **Prompt and aggressive diagnosis and treatment has been shown to greatly reduce morbidity and mortality.**

Although much has been learned about the treatment of colic in recent years, **adynamic ileus** still remains a major obstacle in both medical and postoperative surgical treatment. In time, the hard work of the research community will result in solutions to many of the dilemmas faced by the practising veterinarian. Until these solutions are realised, horse owners and veterinarians alike will have many sleepless nights.

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