

DISEASES OF THE GASTROINTESTINAL TRACT

(Notes Courtesy of Dr. L. Chris Sanchez, Equine Medicine)

The objective of this section is to discuss major gastrointestinal disorders in the horse. Some of the disorders causing malabsorption will not be discussed in this section as they are covered in the “chronic weight loss” portion of this course. Most, if not all, references have been removed from the notes for the sake of brevity. I am more than happy to provide additional references for those of you with a specific interest.

Some sections have been adapted from the GI section of Reed, Bayly, and Sellon, Equine Internal Medicine, 3rd Edition.

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DIAGNOSTIC APPROACH TO COLIC IN ADULT HORSES

The described approach to colic workup is based on the “10 P’s” of Dr. Al Merritt. While extremely hokey, it hits the highlights in an organized fashion. You can use whatever approach you want. But, find what works best for you then stick with it.

1. PAIN – degree, duration, and type
2. PULSE – rate and character
3. PERFUSION – mucous membranes, skin tent, jugular fill, etc.
4. PERISTALSIS – gut sounds, fecal production
5. PINGS – simultaneous auscultation/percussion
6. PASSING A TUBE – amount and character of reflux, if present
7. PALPATION – rectal exam
8. PAUNCH – a word for obvious abdominal distention that begins with “P”
9. PCV/TP
10. PERITONEAL FLUID

PHYSICAL EXAMINATION

1. PAIN

One should consider the severity and duration of pain, as well as the animal’s response to analgesic medication. As part of the history, it is important to ascertain the quantity and type of medication administered by the owner prior to your arrival.

Generally, continuous pain which is not responsive to analgesics more likely originates from a surgical lesion. The one caveat to this is moderate to severe pain which is relieved by gastric decompression, suggestive of proximal enteritis or moderate pain in horses with ileal impaction. Those few horses that continue to exhibit violent signs of pain (ie. throwing themselves down, violently rolling, etc. – not just pawing or lying quietly) despite NSAID therapy and increasing amounts of sedation generally have a surgical lesion and necessitate PROMPT referral. Truly, pain is one of the most important clinical signs when differentiating between surgical and medical causes of colic and, along with cardiovascular status, indication for referral.

Intermittent pain is usually not as serious, though it clearly does not preclude diagnosis of a surgical lesion. Many horses with large colon displacements, and some with strangulation of a small piece of small intestine, will display only moderate but persistent signs of pain. The abrupt cessation of pain followed by clinical signs of shock should raise the concern of intestinal rupture.

2 & 3. PULSE and PERFUSION

Cardiovascular status is another telling indicator of the need for referral and, along with pain, one of the primary indications for surgery. Moderate (>50) to marked (>80) tachycardia should always raise concern and heighten one’s sense of urgency during an examination. Always repeat the heart rate in a few minutes, especially if the horse has just been moved in/out from a field or stall, or if it has just come off the van after transport. A heart rate consistently 50 or greater should raise concern in any horse. Endotoxemia can contribute to tachycardia as well as poor systemic perfusion.

Toxic horses will often have darker pink, progressive to purple, mucous membranes. Alternatively, horses with simple dehydration will often have pink to pale pink membranes which are dry or tacky to the touch, often with a delayed CRT.

4 & 5. Abdominal auscultation (PERISTALSIS and PINGS)

These should be considered together during abdominal auscultation. The frequency, intensity, and duration of sounds should be considered and characterized for each quadrant of the abdomen. But, be careful not to read too much into these findings. Horses with strangulating lesions can have normal or increased borborygmi early in the course of disease. A complete absence of gut sounds or the presence of only short, muffled sounds often indicates ileus, but will not be specific as to cause. The re-appearance of gut sounds after a previous absence should be considered as a positive finding. In cases with marked large intestinal distention but not a surgical option, pings can be used to identify a location for enterocentesis (i.e. cecal trocharization).

Another indicator of peristalsis is fecal production and time to appearance of mineral oil (normally 12-24 hours after administration). If feces are present, either on the ground or in the rectum upon palpation, one can perform a "sand test" by inverting the feces in a rectal sleeve and adding water. While a negative sand test does not rule out sand as a component of an impaction, a positive one certainly supports it.

6. PASSING A TUBE

This is one of, if not the most, important procedures when evaluating a horse with colic. Many, if not most, colics treated in the field will resolve with a single dose of flunixin (with or without enteral therapy). But, checking for reflux prior to administering any enteral therapy requires only a few minutes and can provide valuable information. If an animal has moderate to marked tachycardia or moderate to severe signs of pain, passage of a nasogastric tube should be performed early in the examination process. If a significant amount of reflux is obtained, it may well have been a life-saving procedure. Otherwise, the tube can always be left in place for the remainder of the exam for subsequent administration of enteral therapy, if necessary.

If reflux is obtained, one should consider both the amount and character of the fluid obtained. In animals with suspected proximal enteritis, additional testing for *Clostridium* or *Salmonella* can be performed. Repeated large volumes of gastric reflux suggest ileus of at least the proximal small intestine.

7. RECTAL PALPATION

Findings from rectal palpation can often provide very useful diagnostic and prognostic (especially surgical vs. medical) information. However, a normal rectal examination does not imply the absence of serious disease. The most important questions to answer when performing a rectal examination are:

- Is visceral distention present?
- If so, which segment (i.e. large colon, small colon, cecum, or small intestine) is distended?

- What is the nature (i.e. fluid, feed, gas, solid object) and severity (mild, moderate, severe) of the distention?

Answers to these basic questions will provide diagnostic and/or therapeutic information for the majority of horses with colic, even if a specific lesion is not identified. Similar to the physical exam, find a systematic approach that works for you, then stick to it. Don't conclude an exam because you find an abnormality- the easiest way to miss something is to perform an incomplete exam.

Other things to consider:

- Small intestine is usually not palpable in the normal horse.
- Failure to locate the pelvic flexure is not uncommon in normal horses.
- Mild to moderately distended loops of small intestine may signify duodenitis/proximal jejunitis, whereas larger, more tightly distended loops may indicate a strangulating lesion or an ileal impaction. But, this distinction is rarely straightforward. Ultrasonographic assessment can be extremely helpful.
- With left dorsal displacement (nephrosplenic entrapment), one can palpate the colon as it passes over the ligament, immediately lateral to the caudal pole of the left kidney. The character of the colon can vary, depending upon the degree of distention. If the colon is located high on the left side, but not definitively over the ligament, it probably isn't truly entrapped.
- Gas-distended large colon is just that. Rectal findings, in conjunction with clinical findings, can be suggestive of right dorsal displacement of the large colon. But, many horses with suspected displacement can resolve with medical therapy – tough to say if they were truly displaced or just distended; probably the latter.
- ALWAYS sweep the inguinal rings. Small intestine entering the ring is indicative of inguinal hernia.
- In a pregnant mare, the body of the uterus should be carefully palpated for any evidence of uterine torsion which is usually cranial to the cervix.
- A firm mass in the abdomen (intussusception, abscess, lipoma, or intramural lesion) or within the intestinal lumen (enterolith or impaction) can provide an immediate diagnosis (or at least a short list of differentials).
- Palpation of gritty serosal surfaces or serosal crepitus is highly suggestive of a ruptured viscus.
- Small colon impactions frequently result in an edematous, sometimes gritty rectum. This can also cause some serosanguinous discharge.

While most veterinarians perform rectal exams as part of their routine colic examination, some choose not to do so in every case. One should always perform a rectal exam if: repeated examination is necessary, the signs of pain or perfusion are moderate to severe, the animal displays gross abdominal distention, any gastric reflux is obtained, or if any other “red flags” are raised during the initial physical examination.

The most important thing to consider when performing a rectal examination is the safety of both patient and veterinarian. Risk factors for rectal tears include: small size,

male sex, Arabian breed, colic, inadequate restraint, excessive straining, and inadequate lubrication. Thus, always use plenty of lube, have a competent handler, and use ample sedation and/or a twitch. If an animal combines several risk factors (ie. a 375 kg Arab stallion presented for colic), always take care to make the procedure as safe as possible. Buscopan (0.1-0.3 mg/kg IV) will greatly improve the rectal exam due to its antispasmodic effects. Just remember that it also causes a significant, transient tachycardia. A lidocaine enema (15-60 ml of 2% lidocaine, q/s to 60 ml total volume) can also facilitate the procedure by increasing rectal compliance.

8. Abdominal distention (PAUNCH)

Abdominal distention should always be evaluated in the context of the horse's normal appearance. The owner's opinion is often very useful in this analysis. The source of the distention, identified on rectal exam, will typically provide more useful information, but this can be useful for evaluation of disease progression.

9. PCV/TP

The last two procedures on this list certainly are not necessary for the majority of horses with colic examined and/or treated in the field. Depending upon the practice and location, some veterinarians will have a microcentrifuge in their truck, but many will not. If an animal displays clinical signs of endotoxemia and/or dehydration, a "quick and dirty" PCV can be obtained by filling a purple (or green) top tube and letting it sit upright. The red blood cell should settle out within the time necessary to perform a physical exam, rectal exam, pass a nasogastric tube and relay one's findings to the owner. This will provide a rough estimate of the PCV and, if a refractometer is available, the total solids.

In a hospital setting, PCV/TP can provide a cheap, effective means of monitoring an animal's hydration status prior to and during intravenous and/or enteral fluid administration. While a PCV >60 is certainly a poor prognostic indicator, it does not mean that such a horse can't survive following aggressive medical (and often surgical) therapy. Combining PCV/TP with venous lactate and physical findings can give a reasonable indicator of peripheral perfusion.

10. PERITONEAL FLUID

Analysis of peritoneal fluid can often be extremely helpful, especially when differentiating between strangulating and non-strangulating small intestinal lesions and documenting cases of gastrointestinal perforation. One thing to remember: if the results of the test will not alter your treatment plan, it's probably best not to perform the test. This is likely true for the majority of colics, and the procedure does have a small amount of inherent risk for both the horse (enterocentesis) and veterinarian (kick). Thus, the recommended indications for performing an abdominocentesis in the field are limited. Normal equine peritoneal fluid should be translucent and very pale yellow to amber, with a total protein of < 2 g/dL and WBC <5,000 -10,000/ μ L. A few points to remember:

- Typically, strangulating lesions will result in a serosanguinous tap, while non-strangulating lesions will typically result in a normal tap or one with a slight to moderate increase in protein with normal WBC.
- Horses with strangulated small intestine can have normal peritoneal fluid early in the disease process.
- A serosanguinous tap, while suggestive of a strangulating lesion or otherwise compromised bowel, can result from blood contamination, a splenic tap, or hemoperitoneum. Platelets and hemosiderin-laden macrophages can differentiate.
- Ingesta or other feed particles in a tap can result from either enterocentesis or a ruptured viscus. Ultrasound can be extremely helpful to determine the presence of a specific pocket of fluid or to characterize the fluid present. An “oily” looking tap can result from lipid material and does not automatically indicate free mineral oil within the peritoneal cavity.

ANCILLARY DIAGNOSTICS

Ultrasound: Transabdominal ultrasonography can offer very useful information in the acute abdomen. Typically, a 3-7 mHz curvilinear transducer is used. As with all ultrasonographic assessments, the quality of the equipment and experience of the operator will affect the usefulness of the procedure. But, any machine used for routine reproductive exams will allow for a basic colic exam (presence of fluid, identification of SI distention, possibly SI wall thickness). Transrectal ultrasonography is not commonly used in cases of acute colic. The following basic parameters should be assessed:

- Objective measurement of small intestinal distention. This can be especially useful early in the disease process.
- Objective measurement of intestinal wall thickness, including duodenal size, thickness, and motility.
- Amount and character of peritoneal fluid.
- Estimation of gastric size.
- Estimation of SI and large colon contents (i.e. gas vs. fluid)
- Right dorsal colon wall thickness.
- Identification of the spleen and kidney in the left flank. While failure to identify the spleen does not confirm a nephrosplenic entrapment, identification of both makes that diagnosis MUCH less likely.
- Identification of a target-like lesion typical of an intussusception.

Radiology: Abdominal radiographs in the adult horse are limited to hospital facilities with specialized equipment. Even so, radiographs are essentially helpful for two major diagnoses: sand and enterolithiasis. Remember that the lack of a radiographically evident enterolith does not rule out the diagnosis, and some enteroliths can be difficult to differentiate from sand within the colon.

Endoscopy: Gastroscopy is usually not indicated in cases of acute colic, but more commonly chronic or recurrent colic, poor performance, decreased grain consumption, etc. The stomach and proximal duodenum of foals and adult horses can be easily examined with appropriate equipment. A 2-meter scope will allow access to the stomach of smaller horses, but not a complete examination of the stomach in average sized adults. A 3-meter scope is necessary for complete examination of the body of the stomach, gastric antrum, pylorus, and proximal duodenum. This is important as antral lesions can exist without other obvious squamous or glandular ulceration, and these lesions can easily be missed without a thorough examination. In order to facilitate a thorough exam, adequate fasting is essential: usually at least 12, preferably 16-18 hours off feed. Some clinicians withhold water prior to gastroscopy.

SPECIFIC DISEASE PROCESSES

Diseases of the upper gastrointestinal tract, foals, and causes of enterocolitis are discussed elsewhere in these notes. Specific disorders and their associated surgical corrective procedures are covered in detail in the Equine Surgery elective.

MEDICAL MANAGEMENT OF COLIC IN ADULT HORSES

Most cases of equine colic will resolve with medical therapy. The three hallmarks of medical management include analgesia (NSAIDs, sedatives, opiates), fluid therapy (laxatives, oral fluids, IV fluids), and husbandry (fasting, preventing self-injury, etc.).

While severe or persistent pain indicates the need for referral, a basic guide to medical therapy involves the following: Withholding feed but allowing free choice access to water (for new clients, one must often clarify that “no feed” includes grain, hay and grass); oral laxatives (such as mineral oil); analgesics (flunixin ± xylazine). After monitoring the initial response to therapy, additional enteral therapy (fluids and/or laxatives) can be provided. Intravenous fluid therapy can be added in horses with clinical signs of dehydration. One of the keys is not to re-introduce feed until the horse has passed a significant quantity of feces and/or mineral oil (often used as a marker of intestinal transit).

PAIN MANAGEMENT

Pain control is the single-most important therapeutic factor when treating colic in horses. Regardless of the initial cause of colic, pain itself can result in ileus, and providing pain relief can break the cycle of motility inhibition. While many of the analgesic agents disrupt motility, the benefits of analgesia typically outweigh the negative consequences. Analgesic therapy alone often provides sufficient relief for horses with spasmodic colic. For with non-strangulating obstructions, analgesia provides important adjunctive therapy. Continuous pain despite analgesic therapy should always raise concern and prompt referral as this is an important indicator of the need for surgical intervention. Dosages for commonly used drugs are presented below.

Non-steroidal anti-inflammatory drugs (NSAIDs): NSAIDs are usually the initial therapy of choice for visceral pain. They work by either selectively or non-selectively inhibiting cyclooxygenase production therefore predominantly target inflammatory-mediated visceral pain. Because inflammation can lead to a vicious circle of nociceptive up-regulation, these agents are extremely useful, regardless of the primary insult. Selectivity between COX isoforms varies between agents and dosages, and species, and expression of the isoforms varies between species and tissue. Flunixin meglumine, ketoprofen, and phenylbutazone are non-selective inhibitors (or some such as flunixin are slightly COX-1 specific), whereas carprofen and etodolac are somewhat COX-2 selective. Firocoxib is extremely COX-2 selective in the horse. Etodolac has been shown to inhibit epithelial restitution following ischemia-reperfusion injury in *in-vitro* studies of equine intestinal mucosa to a lesser degree than does flunixin. But, flunixin and etodolac each inhibit recovery of intestinal barrier function in jejunal mucosa after ischemia and a longer (18 hours) period of reperfusion. Evaluation of firocoxib is forthcoming. All NSAIDs have inherent negative effects, and the key to safe and effective administration often lies in their judicious use.

α -2 adrenergic agonists: Alpha-2 adrenergic agonists, such as xylazine, romifidine and detomidine, can provide excellent sedation, analgesia, and muscle relaxation for horses with severe abdominal pain. These drugs have a relatively short duration of action (longer for detomidine). The major side effects include inhibition of motility throughout the gastrointestinal tract as well as systemic and gastrointestinal alterations in hemodynamics. These agents are often combined with an opioid, such as butorphanol, in order to give more balanced analgesia; however this combination can create a synergistic inhibition of motility. It is important to note that the sedative effects typically outlast the analgesic effects of these drugs.

Opioids: Opioids, typically butorphanol, are most commonly used in conjunction with another agent, such as an α -2 agonist. Bolus dosing is obviously more practical in the field, whereas CRI can be extremely useful in a hospital setting. As with the α -2 agonists, opioids have well-known disruptive effects on gastrointestinal motility. These effects appear to be reduced substantially when butorphanol is administered as a CRI.

Sedatives: Other sedative agents, such as chloral hydrate, can be used as an alternative to the α -2 agonists in certain situations. Chloral hydrate is a very powerful sedative and should be used only with a clear diagnosis (ie. small colon impaction) or without a surgical option. Dosage is 10g (for a 450-500 kg horse) as a 12% solution slowly (over 5 min) IV, preferably with a catheter as the agent is very caustic peri-vascularly. The dosage can be titrated in 5 g increments and repeated in 6-8 hours, if necessary.

Anti-spasmodics: N-butylscopolammonium bromide (Buscopan®) has both anticholinergic and antispasmodic properties and has been commercially available for the treatment of spasmodic colic in Europe for many years (in conjunction with

dipyron). This drug is also useful for facilitating rectal examination. Two important facts – Buscopan causes tachycardia and it is very short-acting (20 minutes).

Decompression: Because much of the pain associated with colic in horses is caused by gaseous distention, decompression can often serve as a viable method of analgesia. By far the most common application is gastric decompression via a nasogastric tube. For horses with severe large colon and/or cecal distention for which surgery is not an option, cecal enterocentesis is a viable option. This procedure can be performed in the right paralumbar fossa, with a specific location identified by simultaneous auscultation and percussion. After a surgical prep and local anesthesia, a 14 ga, 5.5" catheter is inserted into the cecum. Preferably, an extension set is attached to the catheter and inserted in a cup of water at a level below the insertion point. This allows for easy identification of gas expulsion and prevents inflow of air into the viscus. Once gas flow has ceased, inject a small amount of gentamicin (3-5 ml) prior to removal to prevent contamination of the abdominal wall with any aspirated gastrointestinal contents. This procedure is certainly not without risk and can result in peritonitis. Thus, it should only be performed if surgical therapy is not an option. Also, it rarely helps as much as one would think.

Alternate analgesia: Intravenous lidocaine has been used in horses both as an analgesic and as a treatment/preventative for post-operative ileus. Lidocaine has been used in this fashion for years to treat human patients with neuropathic pain, and will decrease the MAC for halothane in horses. Some data support its somatic, but not visceral, analgesic properties in normal horses, and IV lidocaine has also been shown to attenuate the effect of ischemia-reperfusion injury in the equine jejunum. Side effects of lidocaine infusion include CNS excitement, ataxia, seizures, collapse, and muscle fasciculations. These side effects rarely occur at the dosage provided, but stress the importance of an accurate drug delivery as they are well-correlated with plasma lidocaine concentrations. These effects are transient and horses typically recover within minutes after discontinuation of the drug due to its short half-life.

Alternative therapies, such as acupuncture, have been used in Eastern countries for many years to treat colic pain in horses. One trial using a duodenal distention method did not demonstrate an analgesic effect of acupuncture at the Guan-yuan-shu (similar to BL-21) acupoint. However, this study only evaluated a single-point therapy which is not typical of the Traditional Chinese approach to colic pain therapy.

FLUID THERAPY

Regardless of the initial cause of gastrointestinal disease, dehydration frequently results. A thorough discussion of fluid therapy is provided elsewhere in these notes.

Oral fluids: Oral (or enteral) fluid therapy provides an effective, practical and economical alternative to IV fluid therapy for horses with colonic impactions. Enteral fluids can be administered in either bolus or continuous fashion via nasogastric tube.

Continuous administration mitigates the practicality of this route, but not necessarily the expense. Always check for reflux prior to administration, and never administer enteral fluids to a horse with more than 1-2 liters of reflux.

Normal horses will tolerate up to 6-8 liters of water every 30 minutes for several doses, but this rate should not be attempted in horses with colic. Large volumes of fluid should be administered via gravity flow (i.e. funnel). I have good luck with enteral fluids (either alone or in conjunction with IV fluids) and usually give 6-8 L every 4-6 hours, alternating balanced electrolyte solution and either plain water or water with MgSO₄ (with MgSO₄ given no more frequently than once a day). Solutions should have an osmolality less than 800 mOsm/L to avoid hypertonicity. Two easily made enteral fluid solutions are presented below; both approximately isotonic. If a horse is drinking well, another option is to provide electrolyte paste, either a commercially available product or the slurry suggested in the fluid/electrolyte therapy notes. This option is best used once an impaction has passed and offers an effective means to increase water consumption in horses predisposed to impaction colic, especially if given at times of increased risk such as travel or changes in the weather.

Intravenous fluids: For the severely dehydrated horse, IV fluids provide the most rapid method to restore circulating blood volume. With marked dehydration and shock, fluid therapy with either synthetic colloid (i.e. hetastarch; 10 ml/kg) or hypertonic saline (2-4 ml/kg) can provide rapid restoration of plasma oncotic pressure. Each should be followed with a crystalloid replacement solution.

For horses with moderate dehydration, a reasonable goal is to replace approximately half of the calculated fluid deficit within the first 1-2 hours, with replacement of the remaining deficit (plus maintenance and ongoing losses) over the next 12-24 hours. Any polyionic replacement solution (LRS, Plasmalye A, Normosol-R, etc) can serve this purpose. Research suggests that “overhydration”, or provision of more than twice maintenance without ongoing losses, does not cause a further increase in cecal or fecal water content (vs. 2x maintenance) and causes a more pronounced rebound dehydration following the abrupt discontinuation of IV fluids.

LAXATIVES

Mineral oil is the most common laxative used in the treatment of equine colic. Mineral oil is commonly dosed at 0.5-1 gallon via NGT in an adult horse. Additional water can be administered to a total volume of 6-8 liters as long as no reflux is obtained. Mineral oil offers the additional bonus of providing an estimate of gastrointestinal transit time as it is usually easily visible on a horse’s perineum and/or hind limbs. Oil should typically appear within 12-24 hours following administration. One word of caution is that oil can pass around an incomplete obstruction, or “over the top” of an impacted cecum.

Other laxatives include magnesium sulfate (Epsom salts; 0.5-1 g/kg in 8 L water) and dioctyl sodium succinate (DSS). A recent experimental trial showed that MgSO₄ did not provide additional benefit versus balanced electrolyte solution given enterally

and was more likely to cause electrolyte disturbances. However, the rate of enteral fluid administration (5L/hour, continuous) in that study was clearly more than what is practical in a field situation. DSS can be irritating to the colon, and its use has likely declined over time. One should not use either substance in a significantly dehydrated horse as they can exacerbate dehydration via fluid shift to the colon.

Psyllium mucilloid is a bulk laxative often used in the treatment of sand impactions (usually 16 oz in 0.5-1 gallon mineral oil once a day for 3-4 days). Psyllium in water forms a highly viscous gel very quickly (and clog a pump), hence the reason to mix it with mineral oil for nasogastric administration. Psyllium administration was shown to have no benefit in dispersion of sand from the cecum of cannulated ponies.

DISEASES OF THE ORAL CAVITY

Diseases of the oral cavity predominantly include dental disease, foreign bodies, neoplasia, infectious diseases (mostly vesicular stomatitis), and ulceration from NSAIDs or locally caustic agents. Congenital and developmental disorders include cleft palate, wry nose, brachygnathism/prognathism, and disorders of eruption. These problems are beyond the scope of this course and are covered primarily in the surgery elective.

Dysphagia can be classified anatomically as prepharyngeal, pharyngeal, or postpharyngeal (esophageal). Prepharyngeal dysphagia is expressed clinically by dropping food (quidding) or water from the mouth, hypersalivation, reluctance to chew, or difficulty in prehension. Pharyngeal or esophageal dysphagia is characterized predominantly by coughing and nasal discharge consisting of food, water, or saliva. Dysphagia can be caused by pain, muscular, neurologic, or obstructive disorders. Neurologic causes of dysphagia include diseases affecting the forebrain, brainstem, or peripheral cranial nerves controlling prehension (V_m, V_s, VII, and XII), transfer of the food bolus to the pharynx (V_s and XII), or swallowing (IX and X). Recent evidence suggests that, while stimulation of cranial nerve IX stimulates swallowing, bilateral blockade of that nerve does not prevent normal swallowing of either liquid or solid material. Diagnosis and treatment of those diseases will be discussed in detail in the neurologic disease section.

Salivary glands in the horse are paired and include the parotid, mandibular, and polystomatic sublingual glands. The parotid gland is the largest and can secrete saliva at up to 50 ml/minute, with daily secretion up to 12 L in a 500 kg horse. Parotid secretion occurs only during mastication and can be blocked with atropine or anesthesia of the oral mucosa. Parotid saliva is relatively hypotonic, with average daily ion concentrations (mEq/L) as follows: Na⁺ 55, K⁺ 15, Cl⁻ 50, HCO₃⁻ 52. Salivary diversion will typically not result in acid-base or electrolyte abnormalities for several days, at which point horses will become slightly hyponatremic (day 3), markedly hypochloremic (day 3), mildly hypokalemic (day 10), and mildly alkalemic (day 10). Concentrations of sodium, chloride, and bicarbonate ions increase at high rates of flow.

DISEASES OF THE ESOPHAGUS

The equine esophageal mucosa consists of keratinized stratified squamous mucosa with no digestive or absorptive function. The submucosa confers elasticity to the esophageal wall. The tunica muscularis transitions from striated skeletal muscle in the proximal two thirds to smooth muscle in the distal third. The striated skeletal muscle is innervated by the pharyngeal and esophageal branches of the vagus. Parasympathetic fibers of the vagus innervate the smooth muscle; sympathetic innervation of the esophagus is minimal. During the esophageal phase of swallowing, the upper esophageal sphincter closes immediately, the lower esophageal sphincter (LES) opens, and food passage is propelled via esophageal peristalsis. Gastric distention causes mechanical constriction of the LES while also triggering a vagal reflex that increases LES tone. These factors normally prevent spontaneous reflux.

Esophageal obstruction (Choke): Causes include primary impaction with feed (typically roughage), bedding, or other material. Predisposing factors include poor dentition, prior esophageal trauma, gulping food, dry or improperly moistened feeds with predisposition to enlarge when moistened (i.e. beet pulp), mild dehydration, and chronic debilitation. Secondary impaction can result from intraluminal (foreign body), extramural (neoplasia, vascular ring anomaly, granuloma), intramural (abscess, granuloma, neoplasia, cyst, diverticulum, stenosis), or functional (dehydration, exhaustion, pharmacologic, primary megaesophagus, esophagitis, autonomic dysautonomia, vagal neuropathy, sedation) disorders.

Clinical signs of esophageal obstruction include those previously described for postpharyngeal dysphagia, including nasal regurgitation of feed, saliva, or water. Other signs include coughing, gagging or retching, ptyalism, nervousness, and an extended neck carriage. A thorough physical exam is important to rule out not only the extent of the lesion but also to rule out other causes of dysphagia. Diagnosis and treatment of esophageal obstruction are initially somewhat similar, as the primary goal is to relieve the obstruction. The most common sites of impaction are those of natural narrowing of the lumen, such as the cervical esophagus, thoracic inlet, base of the heart, and terminal esophagus. For cervical obstructions, palpation and ultrasonography can be useful to identify the site and extent of the lesion. Endoscopic evaluation is useful prior to resolution in order to identify a suspected foreign body and critical after resolution in order to identify mucosal integrity as well as any primary underlying conditions. Foreign bodies may be retrieved with endoscopic guidance.

During relief of the obstruction, sedation is critical not only to reduce esophageal spasm and pain but also to keep the head lowered to reduce the risk of aspiration. Intravenous acepromazine, xylazine, or detomidine can be used either alone or in conjunction with butorphanol. In addition, IV oxytocin (0.1-0.2 mg/kg) or intraluminal lidocaine (15-30 ml of 2%) can be used for additional esophageal relaxation (oxytocin will only affect the proximal 2/3). Simple obstructions can be relieved with gentle pressure from a nasogastric tube. External manipulation can be helpful if a cervical obstruction is identified. For more difficult cases, careful lavage with water is often

necessary to gradually break down the obstruction. This can be accomplished with either a typical uncuffed nasogastric tube or a cuffed endotracheal tube placed within the esophagus, with the goal to prevent flow of water and/or feed material back into the pharynx and/or trachea. With the cuffed esophageal tube in place, that tube can serve as the sole source of lavage, or a smaller tube can be placed within the lumen for infusion, with the remaining lumen used for efflux. This procedure is typically done in a standing, well-sedated animal, but can be performed under general anesthesia in order in refractory cases (be cognizant of the risk of pneumonia). In refractory cases, especially ones in which concurrent dehydration is significant, a period of IV fluid support combined with strict restriction of food, water, and bedding can allow softening of the impaction. In very rare cases, esophagostomy may be required.

Systemic support is critical following relief of the impaction. Correction of dehydration and acid-base or electrolyte disturbances should be considered, as well as treatment and/or prevention of aspiration pneumonia. Local treatment of the esophagus depends upon endoscopic evaluation. If any abnormalities are noted, follow-up evaluation is critical. Possible sequelae include mucosal injury, dilation proximal to the original site of obstruction, stricture or diverticulum formation, megaesophagus, rupture, and esophagitis, each of which can lead to re-obstruction. With any degree of mucosal injury, risk of re-obstruction is high during the initial 24-48 hours. Judicious use of NSAIDs can reduce esophageal inflammation, but the risk of NSAID-induced mucosal injury should be considered. Sucralfate (20 mg/kg PO q 6-8 hours) may improve mucosal healing.

Strict dietary restriction is critical to a successful outcome. All food should be withheld for at least 24 hours, and up to 48-72 hours in cases of severe mucosal injury. A soft gruel of pelleted feed is frequently the best initial diet; this can be gradually transformed to a mash, with subsequent gradual return to a normal diet over 1-3 weeks assuming good clinical improvement. Feeding affected horses on the ground during the initial phase can also reduce the risk of aspiration.

The most important immediate complications of choke are aspiration pneumonia and esophageal rupture. Immediate broad-spectrum antimicrobial therapy is indicated in any horse in which aspiration is deemed possible. Esophageal perforation typically results in extensive cellulitis and can result in pneumomediastinum and pneumothorax. Treatment includes opening the wound, extensive debridement, broad spectrum antimicrobial and anti-inflammatory therapy, tetanus prophylaxis, and esophageal rest. Other complications include Horner's syndrome, permanent fistulae, and left laryngeal hemiplegia. Prognosis with rupture is poor-grave.

Miscellaneous esophageal disorders: Esophagitis involves inflammation of the esophagus with or without ulceration. Esophagitis is a secondary event; diagnosis and treatment should be aimed at identifying and treating the primary cause. Reflux esophagitis typically occurs secondary to functional esophageal disorders or delayed gastric emptying (severe gastric ulceration, ileus, functional gastric outflow obstructions, etc.). Other causes of esophagitis include trauma, infection, and chemical

injury. For reflux esophagitis, reduction of gastric acid is critical. Sucralfate may enhance mucosal healing, but these effects have not been proven.

Megaesophagus is typically acquired in the horse and can occur following repeated obstruction, extramural masses, neurologic disorders resulting in vagal neuropathy, or pleuropneumonia (also resulting in vagal neuropathy). Other causes include equine dysautonomia, myasthenia gravis (not yet reported definitively in the horse), or systemic disorders. Idiopathic megaesophagus has also been reported in young horses. Endoscopy and contrast radiography can aid the diagnosis. Treatment is aimed at the underlying disorder.

Esophageal strictures are typically caused by pressure necrosis during an esophageal obstruction, but they can also occur secondary to direct or indirect trauma or as congenital lesions. Strictures can originate from the mucosa, submucosa, muscularis, adventitia, or all layers of the esophagus. Following esophageal obstruction, maximum luminal reduction occurs within 30 days, but remodeling continues for up to 60 days. Thus, medical management is warranted for at least 60 days with anti-inflammatory and antimicrobial therapy in addition to dietary management. Possible strategies to increase luminal diameter include bougienage and surgical therapy. Surgical management can include esophagostomy with fenestration, resection and anastomosis, esophagomyotomy, and patch grafting.

Various congenital esophageal disorders have been reported, but all are rare, have a clinical presentation similar to that of esophageal obstruction, and are diagnosed via endoscopy and/or contrast radiography. The predominant neoplasm of the esophagus is squamous cell carcinoma, but leiomyosarcoma has also been reported.

DISEASES OF THE STOMACH

EQUINE GASTRIC ULCER SYNDROME

Peptic ulcer disease is defined as erosions or ulcers of any portion of the gastrointestinal tract normally exposed to acid. Mucosal damage can include inflammation, erosion (disruption of the superficial mucosa), or ulceration (penetration of the submucosa). In severe cases, full-thickness ulceration can occur, resulting in perforation. The orad portion of the equine stomach is lined by stratified squamous mucosa similar to that lining the esophagus. The aborad portion of the stomach is lined with glandular mucosa, and the distinct junction between the two regions is deemed the *margo plicatus*. Ulceration can occur in either or both gastric regions although different clinical syndromes and pathophysiologic mechanisms apply. As a result, the broad term "Equine gastric ulcer syndrome" (EGUS) has been used to encompass the wide array of associated clinical syndromes.

Prevalence: The prevalence of gastric ulceration has been reported for a variety of breeds and usages, however most data involve Thoroughbreds or Standardbreds in race training. The prevalence of squamous ulceration in horses in race training varies from 70 to 95%, and can be as high as 100% when limited to animals actively racing. In recent

years, horses performing in other disciplines have also been evaluated, including active show horses (58% prevalence), endurance horses (67%), western performance horses (40%), Thoroughbred broodmares (67% pregnant; 77% non-pregnant) and non-racing performance horses (17% pre-competition; 56% post-competition). In one large retrospective study (3715 adult horses from 1924-1996) evaluating incidence of gastric ulceration identified at necropsy, an overall prevalence of 10.3% was found, with the highest prevalence in Thoroughbreds (including Arabians) and Standardbred trotters (19%). Horses in a university riding program also demonstrated low squamous ulceration prevalence (11%). Approximately 49% of horses presented to a referral hospital for colic had evidence of gastric ulceration. The reported prevalence of gastric ulceration in foals varies from 25-57%.

Many earlier studies investigating prevalence of gastric ulceration do not differentiate between non-glandular and glandular lesions and many evaluate only the non-glandular region of the stomach, but this trend is changing. In a recent study of 162 horses in a hospital setting, 58% had antral or pyloric erosions or ulcerations, 58% had squamous mucosal lesions, and 8% had lesions involving the glandular body. In other studies, 56% of Thoroughbreds in which the pylorus was examined had glandular ulceration and 47% of racehorses (Thoroughbred and Standardbred) in which the pylorus was evaluated had ulceration in that location. In the former, all horses with glandular lesions also had squamous disease, whereas such an association was not seen in the latter study. In endurance horses with a 67% overall lesion prevalence, 57% had squamous ulceration whereas 27% had glandular disease. In a post-mortem evaluation, lesions were most commonly located in the squamous mucosa along the margo plicatus, followed by the glandular body, proximal squamous mucosa, and antrum.

Pathophysiology: An imbalance between inciting and protective factors in the mucosal environment can result in ulcer formation. The major intrinsic factors promoting ulcer formation include hydrochloric acid (HCl), bile acids, and pepsin, with HCl the predominant factor. Various intrinsic factors protect against ulcer formation such as the mucus-bicarbonate layer, mucosal perfusion, mucosal prostaglandin E₂ and epidermal growth factor production, and gastroduodenal motility. In humans, extrinsic ulcerogenic factors include NSAIDs, *Helicobacter pylori*, stress, changes in diet, and gastrointestinal disorders, especially those resulting in delayed gastric emptying. In human neonates, physiologic stress associated with a major primary illness seems to be strongly associated with gastric ulcers. Many of these factors are believed to be important in horses; but, clear evidence of an infectious agent has not been identified.

The specific factors involved in injury as well as protective mechanisms vary regionally. The pathophysiology of squamous mucosal ulceration in the horse appears similar to that seen in gastroesophageal reflux disease (GERD) in humans. Excess acid exposure is the predominant mechanism responsible for squamous ulceration, although many details remain unclear. HCl is secreted by parietal cells in the gastric glands via a hydrogen-potassium adenosine triphosphatase (H⁺,K⁺-ATPase) pump on the luminal side. Horses secrete acid continuously.¹ A protocol of repeated 24-hour periods of

fasting and feeding has been shown to induce squamous erosion and ulceration and concurrent administration of ranitidine reduces lesion severity.

The predominant stimuli to hydrochloric acid secretion are gastrin, histamine, and acetylcholine. Gastrin is released by G cells within the antral mucosa, while histamine is released by mast cells and enterochromaffin-like (ECL) cells in the gastric gland. Histamine binds to type 2 receptors on the parietal cell membrane, causing an increase in cAMP, resulting in activation of the proton pump. Gastrin and acetylcholine can also stimulate histamine release directly, and histamine is the primary stimulant of acid production. Gastric acid secretion by parietal cells is primarily inhibited by somatostatin, released by fundic and antral D cells. Gastric acid secretion also is inhibited by epidermal growth factor (EGF), a peptide produced in saliva.

In esophageal squamous mucosa, intercellular tight junctions and bicarbonate secretion are the major protective factors involved in other species, although squamous bicarbonate secretion had not been documented in the horse.^{2,3} The principal barrier is a glycoconjugate substance secreted by cells in the *stratum spinosum*, with a contribution from the tight junctions in the *stratum corneum*. This barrier function is considered weak at best. In horses, the normal gastric fill line rests just below the lower esophageal sphincter, so only the squamous mucosa along the lesser curvature adjacent to the *margo plicatus* theoretically receives exposure to acidic gastric contents on a regular basis. Not surprisingly, this is the most common location of squamous ulceration.

Bile salts and pepsin have been implicated as contributing factors to ulcer disease in many species, and could play a role in squamous ulceration as duodenogastric reflux occurs normally in the horse. A synergistic ulcerogenic effect of bile salts and acid have been documented in some studies but not others. These differences may be pH-related and stresses the importance of acid exposure in squamous ulcer disease.

Several mechanisms help protect the glandular mucosa from acid injury. The mucus-bicarbonate layer serves to titrate H⁺ ion from the gastric lumen to CO₂ and H₂O. Cellular restitution and E series prostaglandins, which enhance mucosal blood flow and mucus/bicarbonate secretion in the glandular mucosa, have not been documented in squamous epithelium.⁴ Of these mechanisms, mucosal blood flow is likely the most important contributor. Nitric oxide is a key regulator of mucosal blood flow and prostaglandin synthesis, thus may play a role in mucosal protection.

Risk factors: Many aspects of diet and management have been associated with the development of non-glandular ulceration in adult horses, several of which have already been discussed. These include diet, feeding patterns, exercise, stall confinement, transportation, and administration of non-steroidal anti-inflammatory drugs.

Horses in race training have a high incidence of gastric ulceration and are frequently fed high-concentrate, low-roughage diets. A diet high in concentrate and either high or low in forage, in conjunction with stall confinement but no exercise, was shown to induce non-glandular ulceration within 2 weeks. In one study, higher volatile fatty acid concentrations, higher gastric juice pH, and lower number and severity of nonglandular ulceration were documented after feeding an alfalfa hay-grain diet when

compared to a bromegrass hay diet. However, many factors differed between the diets, such as digestible energy, bulk, crude protein, and mineral content (especially calcium). These findings were recently supported by a study in which an alfalfa hay/pelleted concentrate diet significantly reduced ulcer severity scores and/or prevented ulcer development relative to a coastal hay/concentrate diet in horses managed with dry lot housing and regular exercise. Intermittent feeding has clearly been shown to induce non-glandular ulceration and is a consistent model of ulcer induction.

The pathophysiological correlation between exercise and squamous ulcer disease has not yet been defined despite the high prevalence of ulceration in performance horses. Non-glandular ulceration can develop within 8 days of exercise varying from light halter to active race training, and time in work was found to be a risk factor for squamous ulceration in racing Thoroughbreds. Elevations in post-feeding serum gastrin concentration have been demonstrated after treadmill exercise. During treadmill exercise at gaits faster than a walk, proximal gastric size decreases in conjunction with an increase in intra-abdominal pressure, resulting in a simultaneous decrease in proximal gastric pH. Both proximal gastric size and pH return to baseline levels as soon as the horse returned to a walk, and the resultant theory was that gastric contracture could result in increased acid exposure to the squamous mucosa by raising the level of liquid gastric contents.

Stall confinement and transport have been shown to induce non-glandular ulceration. But, a distinct mechanism has not been determined. A recent study did not detect a difference in pH in the proximal or ventral stomach in response to three different environmental situations (stall confinement alone, stall confinement with a horse in an adjacent stall, and paddock turnout), each for 24 hours. This work suggests that increased acid exposure to the proximal stomach alone is not causative.

Several studies have failed to document a correlation between NSAID administration and naturally occurring ulcer disease. However, NSAID administration is a well-known cause of gastric ulceration under experimental conditions. NSAID-related ulceration is typically described as predominantly glandular in nature, although nonglandular ulceration can also occur by a mechanism that has not yet been fully characterized. NSAIDs cause a decrease in prostaglandin E₂ synthesis due to inhibition of the cyclooxygenase (COX) pathway. Therefore, a resultant decrease in glandular mucosal protection, most notably via decreased mucosal blood flow and mucus production, is the most likely mechanism of action. In one study, however, phenylbutazone administration resulted in ulceration of the glandular mucosa at the pyloric antrum but did not significantly alter mucosal prostaglandin E₂ concentration.

Other risk factors associated with gastric ulceration include sex and age, and the reported prevalence of gastric ulcers has increased over time. In one study, the frequency of gastric ulceration increased from <6% prior to 1945 to approximately 18% after 1975. The association between sex or age and ulceration has not been consistent between studies. Crib-biting has also been discussed as a risk factor. In one study, difficulty maintaining weight and playing of a radio were also associated with risk of ulceration. While the former is easily explained, the latter is not!

Clinical syndrome: Neonatal foals: Clinical signs typically associated with gastric ulceration in foals include poor appetite, diarrhea, and colic. Many foals probably never exhibit clinical signs, and few will not exhibit clinical signs until ulceration is severe or fatal perforation has occurred. Glandular ulceration is typically considered the most clinically significant type of disease in this population, although squamous ulceration has recently been identified similarly. The physiologic stress of a concurrent illness has been associated with gastric ulceration in foals. Retrospectively, 14 (23%) of 61 foals up to 85 days of age with a clinical disorder were found to have glandular lesions, and prospectively 8 (40%) of 20 foals up to 30 days of age with a clinical disorder had glandular ulceration. By contrast, only 4-9% of clinically normal foals examined in endoscopic surveys had glandular lesions.

Critically ill neonatal foals can have a markedly different pH profile compared to clinically normal foals, potentially due to alterations in gastric motility and acid secretion. Gastric ulceration was not identified in any animals at necropsy in that study; however ulceration has been documented in a similar population. Thus, factors other than acid exposure, most notably mucosal perfusion, may play an important role in the "stress" related ulceration seen in neonates. Gastric ulceration and rupture in the hospitalized neonatal population appears to occur less commonly now than in previous reports, despite a decline in the use of ulcer prophylaxis in two reports. Advances in overall neonatal care, especially supportive care, have likely contributed to this decline.

Clinical syndrome: Sucklings/weanlings: In suckling foals less than 50 days, lesions typically originate in the squamous mucosa adjacent to the margo plicatus along the greater curvature. Such lesions can be seen in foals as young as 2 days and have been observed in 50% of foals less than 50 days. Histologic examination of these lesions has revealed disruption of the epithelial layers of the mucosa and a neutrophilic infiltration. Another phenomenon that occurs in young foals is the shedding, or desquamation, of squamous epithelium, which appears as flakes or sheets of epithelium. Desquamation occurs without ulceration in up to 80% of foals less than 35 days of age, and this process is not typically associated with clinical signs.

In older foals, lesions become more prevalent in the squamous mucosa, particularly along the lesser curvature. Squamous lesions also are found in the fundus. These lesions can be very severe, and are often associated with clinical signs such as diarrhea, poor appetite, and poor growth and body condition. Diarrhea is the most frequent clinical sign, and is associated with more diffuse squamous erosion or ulceration than occurs in asymptomatic foals. In some foals, poor growth, rough hair coat, or a "potbelly" appearance occur in conjunction with moderate to severe squamous ulceration. Colic or bruxism can occur with severe or diffuse squamous ulceration.

Gastroduodenal ulcer disease (GDUD) occurs almost exclusively in suckling and early weanling foals. Clinical signs of duodenal ulceration are similar to that described for gastric ulceration (bruxism, colic, ptyalism, diarrhea), but the consequences are often more severe. Lesions occur primarily in the proximal duodenum, ranging from diffuse

inflammation to severe ulceration. Affected foals typically have severe squamous and/or glandular ulceration as well, and can have reflux esophagitis, due to associated delayed gastric emptying. Determination of gastric outflow obstruction is one of the most important factors in formulating a proper therapeutic plan (medical vs. surgical therapy) and prognosis. Complications can include gastric or duodenal rupture, pyloric or duodenal stricture, and ascending cholangitis, and aspiration pneumonia. If an outflow obstruction is diagnosed or suspected, GDUD becomes a surgical disease.

The GDUD syndrome can occur in outbreaks and is most commonly identified in intensive breeding operations. The cause of duodenal lesions in foals is not known. One theory is that the problem begins with diffuse duodenal inflammation that can coalesce down to a focal area of ulceration. A temporal relationship between GDUD and rotaviral diarrhea has been suggested, but an infectious etiology remains unproven. Although lesion location and severity associated with rotaviral infection varies among species, duodenal ulceration has not been reported.

Clinical syndrome: Yearlings and adult horses: Clinical signs attributable to EGUS in older horses are variable and classically include anorexia, and chronic or intermittent colic of varying severity. Many horses with endoscopic evidence of disease may appear to be clinically normal or have vague signs that include decreased consumption of concentrates, postprandial episodes of colic, poor performance, poor quality haircoat, and decreased condition or failure to thrive. Diarrhea is not typically associated with gastric ulceration in adult horses, although ulceration can occur concurrently with other causes of diarrhea. Neither presence nor type of clinical signs was correlated with prevalence or severity of ulceration. Horses actively racing appear more likely to have squamous ulceration than those solely in training. In some studies of horses in race training, neither prevalence nor severity of ulceration was associated with age or sex, whereas in others, ulcer severity increased with age.

Lesions occur predominantly in the squamous mucosa, particularly adjacent to the margo plicatus, but glandular and antral involvement have become increasingly apparent. In severe cases, lesions can extend dorsally into the squamous fundus. Clinically relevant lesions typically affect a greater portion of the squamous mucosa and can be deep enough to cause bleeding, but a correlation between lesion severity and clinical signs is often not apparent. Bleeding from ulcers in the gastric squamous mucosa is typically not associated with anemia or hypoproteinemia.

Diagnosis: Although a diagnosis of EGUS can be suspected based upon clinical signs and response to treatment, the only current method of confirmation is via gastroscopy, which can easily be performed in the standing horse or foal with mild sedation. In adult horses, a 3-meter endoscope allows inspection of the entire stomach, pylorus, and proximal duodenum. Shorter scopes will permit examination of the gastric body and fundus, but not the pyloric antrum in most cases. Proper fasting and a thorough exam are critical for an accurate diagnosis, as antral disease can occur in the absence of squamous disease. A maximum external diameter of 9 mm should be used for neonatal

foals. Numerous scoring systems for lesion severity have been described, but a recent consensus has been published by the Equine Gastric Ulcer Council. This system appears as accurate and more user-friendly than previous systems. Urine and blood sucrose absorption testing have recently been evaluated as a measure of gastric mucosal permeability in horses with EGUS. Also, serum α_1 -antitrypsin was detectable more frequently in foals with gastric ulceration than normal foals. These blood tests could provide economical, less invasive screening, but further evaluation is necessary prior to their widespread use.

Duodenoscopy is used to diagnose duodenal ulceration or inflammation. Diffuse reddening or inflammation may be the only recognizable lesion in cases of early duodenal disease. In older foals with GDUD, detection of gastric outflow obstruction is critical to the therapeutic plan and appropriate prognosis. Abdominal radiography without contrast in foals with outflow obstruction typically reveals a distinctly enlarged, gas-filled stomach. Liquid barium contrast will either have markedly delayed (with incomplete obstruction) or no (complete obstruction) outflow. Clinically, foals with outflow obstruction will develop reflux after suckling, or marked reflux even with limited to no suckling if the duodenal obstruction is distal to the common bile duct.

Treatment: Because acid has been implicated as the most important pathophysiological component of squamous ulcer disease, most anti-ulcer therapy centers on acid suppression. If gastroendoscopy is unavailable, treatment efficacy can be based on improvement clinical signs, which are often vague or nonspecific. Signs of colic that result from gastric ulcers often resolve within 48 hours. Improvements in appetite, bodily condition, and attitude can be noted within 1 to 3 weeks. If improvement in clinical signs is not observed, either treatment has not been effective or gastric ulceration was not the primary problem. Typically, a treatment period of at least 2-4 weeks, followed by prophylaxis depending upon the animal's exercise and environmental status, is recommended. The principal therapeutic options for ulcer treatment include H₂ antagonists (cimetidine, ranitidine, famotidine, nizatidine), proton pump inhibitors (PPI; omeprazole, pantoprazole, rabeprazole, esomeprazole), the mucosal adherent sucralfate, and antacids.

The H₂ antagonists suppress hydrochloric acid secretion through competitive inhibition of the parietal cell histamine receptor. Use of H₂ antagonists has been successful in raising gastric pH and resolving gastric lesions in both foals and adult horses. Clinical and experimental evidence has demonstrated greater individual variability with lower dosages of H₂ antagonists. Thus, dosage recommendations are based upon levels necessary to increase gastric pH and promote ulcer healing in a majority of horses. Famotidine has been used less extensively in the horse, but a dose of 10 to 15 mg/kg/day has been recommended. Although clinically normal foals respond predictably to ranitidine, sick neonates have shown variability in pH response to intravenous ranitidine, with a much shorter duration of action and, in some cases, no noticeable response. Currently, cimetidine and ranitidine are available in injectable, tablet, and liquid forms. Famotidine and nizatidine are available in tablets.

Proton pump inhibitors (PPIs) block secretion of H⁺ at the parietal cell membrane by irreversibly binding to the H⁺, K⁺-ATPase proton pump of the cell. These agents have a prolonged antisecretory effect, which allows for once-daily dosing. Omeprazole, the first PPI to be developed, is the only currently approved agent for the treatment of EGUS. Omeprazole has demonstrated efficacy in the healing of NSAID-induced ulcers in horses as well naturally-occurring cases of EGUS in horses and foals. Omeprazole has been shown to eliminate or reduce the severity of gastric ulcers in Thoroughbreds maintained in race training, and has been shown superior to ranitidine. The available equine preparation of omeprazole (GastroGard™, Merial, Ltd., Duluth, GA) is recommended at a dose of 4 mg/kg orally every 24 hours for ulcer healing. An increase in gastric pH and a decrease in acid output are evident 5-8 hours after omeprazole paste administration. Treatment with 1, 2 or 4 mg/kg orally every 24 hours has been shown to decrease or prevent disease or the recurrence of disease in animals maintained in training. Omeprazole (4 mg/kg) also has demonstrated efficacy for raising intragastric pH in clinically normal or critically ill foals and for ulcer healing in foals.

The powder form of omeprazole is rapidly degraded in an acidic environment, thus an enteric-coated capsule or a specially-formulated paste must be used to allow delivery of the active drug to the small intestine for absorption. Compounded oral preparations have shown limited to no efficacy in pharmacodynamic and clinical trials, and GastroGard was the only product (including buffers, H₂-receptor antagonists, sucralfate, and compounded preparations) that decreased the odds of gastric ulceration in a population of racehorses. Omeprazole appears more effective for ulcer healing than either ranitidine or cimetidine. A compounded intravenous preparation of omeprazole (0.5 mg/kg) has been shown to raise gastric juice pH and decrease the number of non-glandular lesions in horses. From a regulatory standpoint, omeprazole (4 mg/kg) does not appear to affect quantitative measurements of performance in standardbreds.

Other PPIs have been recently developed for use in humans, including rabeprazole, lansoprazole, esomeprazole, and pantoprazole. In the treatment of GERD in humans, esomeprazole has demonstrated a higher rate of healing at 4 and 8 weeks when compared to omeprazole, but rabeprazole, lansoprazole, and pantoprazole have similar efficacy. Pantoprazole given by either intravenous or intragastric routes (1.5 mg/kg) raised intragastric pH in clinically normal neonatal foals.

Sucralfate is effective in the treatment of peptic ulcers and prevention of "stress-induced" ulcers in humans. The mechanism of action likely involves adherence to ulcerated mucosa, stimulation of mucus secretion, enhanced prostaglandin E synthesis, and concentration of growth factor at the site of ulceration, although the prostaglandin effects may not play an important role in ulcer healing. These are all factors relevant to glandular mucosa, and the efficacy of sucralfate in treating ulcers in the equine gastric squamous mucosa remains undetermined. In one study, sucralfate did not promote subclinical ulcer healing in foals, relative to corn syrup. In another, simultaneous sucralfate administration significantly reduced the total area of oral ulceration and gastric epithelial necrosis experimentally induced by phenylbutazone, but did not significantly reduce the number of squamous or glandular ulcers in foals. In humans,

sucralfate provides protection against stress-induced ulcers with a decreased risk of pathogenic gastric colonization. The efficacy of sucralfate in an alkaline pH is controversial, but appears likely.

The use of antacids in the treatment of gastric ulcers has not been critically examined in the horse. Research in horses has shown that 30g aluminum hydroxide/15 g magnesium hydroxide will result in an increase in gastric pH above 4 for ≈2 hours. Thus, although antacids may be useful for the treatment of ulcers in adult horses, a dose of 180-200 ml at least every 4 hours is necessary.

The use of synthetic prostaglandin E₁ analogs, such as misoprostol, has been effective in the treatment of gastric and duodenal ulcers in humans, and the proposed mechanism of action involves both inhibition of gastric acid secretion and mucosal cytoprotection. In horses, misoprostol has been shown to increase gastric pH and ameliorate deleterious effects of flunixin on mucosal recovery after ischemic injury in vitro. Misoprostol is contraindicated in pregnant mares.

Prokinetic drugs should be considered in foals with GDUD when delayed gastric emptying without a physical obstruction is suspected. Bethanechol and erythromycin have been shown to increase the rate of gastric emptying in horses. In cases of acute gastric atony, bethanechol 0.025 to 0.030 mg/kg, SC every 3 to 4 hours has been effective in promoting gastric motility and emptying, followed by oral maintenance dosages of 0.35 to 0.45 mg/kg three to four times daily. Adverse effects can include diarrhea, inappetance, salivation, and colic, but are infrequent and mild.

For foals with severe GDUD that have developed duodenal stricture, surgical therapy is necessary with either a gastroduodenostomy or gastrojejunostomy, depending upon stricture location. Prognosis can be good, but these animals require a serious financial commitment, as intensive peri-operative medical therapy is critical for a successful outcome. In one recent report, 98% of surgically treated foals survived to discharge from the hospital, with 68% survival 8 months following discharge. In that study, of the surviving Thoroughbreds of racing age, 71% had started a race. In another, short-term survival was reported as 80% for those foals treated surgically and 50% for those treated medically.

A variety of nutraceuticals are currently marketed for the treatment and/or prevention of EGUS, but no objective data are available regarding their efficacy.

Prevention: Because gastric perforation due to glandular ulcer disease has been reported in hospitalized neonates, many clinicians routinely use prophylactic anti-ulcer therapy in this population. Because some critically ill foals have a predominantly alkaline gastric pH profile, and because gastric acidity may be protective against bacterial translocation in neonates, the need for prophylactic ulcer therapy is controversial. In critically ill human neonates, while intravenous ranitidine therapy raises gastric pH and gastric bacterial colonization, it does not increase the risk of sepsis. In a retrospective study of 85 hospitalized foals <30 days of age, no difference in the frequency of gastric ulceration at necropsy was found between those foals that received prophylactic treatment for gastric ulcers and those that did not. Due to the

retrospective nature of that study, specific details regarding lesion location and severity were not available, however none of the foals in the study died as a result of gastric ulcer disease. Thus, many clinicians no longer recommend routine ulcer prophylaxis in all ill neonates. Exceptions could potentially include those foals requiring significant doses of NSAIDs for painful orthopedic disorders.

In adults, dietary and environmental management can help prevent gastric ulceration. Pasture turnout and continuous access to good quality forage, especially alfalfa, are currently recommended. For horses at high risk, the best proven pharmacologic approach to prevention involves administration of omeprazole 1-2 mg/kg orally every 24 hours.

OTHER DISORDERS OF THE STOMACH

Pyloric stenosis and delayed gastric emptying: Pyloric stenosis is a structural resistance to gastric outflow. Congenital pyloric stenosis has been reported in foals and one yearling and results from hypertrophy of the pyloric musculature.^{6,7} Acquired pyloric stenosis can result from neoplasia or duodenal ulceration.⁸⁻¹⁰ Clinical signs are dependent upon the degree of obstruction but include abdominal pain, salivation, and teeth grinding. Complete or near complete obstruction can result in gastric reflux and reflux esophagitis. In foals with congenital pyloric hypertrophy, clinical signs may begin with the consumption of solid feed. In foals, a presumptive diagnosis can be made via gastric endoscopy and radiography (plain and contrast studies). Depending upon the cause and severity of disease, gastric endoscopy may provide a presumptive diagnosis in the adult horse. Measurement of gastric emptying can aid the diagnosis. Several methods of measurement are currently available, including nuclear scintigraphy, acetaminophen absorption, and post-consumption [¹³C]octanoic acid blood or breath testing.^{11,12} During an exploratory laparotomy, a distended stomach and thickened pylorus are accompanied by a relatively empty intestinal tract.

If complete obstruction is not present, medical therapy with a prokinetic such as bethanecol can increase the rate of gastric emptying. Phenylbutazone and cisapride have also been shown to attenuate the delay in gastric emptying caused by endotoxin administration.¹³ Surgical repair is necessary for definitive treatment of complete or near-complete obstruction and consists of either gastroenterostomy or pyloroplasty.¹⁴

Gastric dilatation and rupture: Gastric dilatation can be classified as primary, secondary, or idiopathic. Causes of primary gastric dilatation include gastric impaction, grain engorgement, excessive water intake after exercise, aerophagia, and parasitism.¹⁵ Secondary gastric dilatation occurs more commonly and can result from primary intestinal ileus or small or large intestinal obstruction. Time to development of reflux is proportional to the distance to the segment involved, with duodenal obstruction resulting in reflux within 4 hours. Clinical signs of gastric dilatation include those associated with acute colic and, in severe cases, nasal discharge. Associated laboratory abnormalities include hemoconcentration, hypokalemia, and hypochloremia.

The most common reported cause of gastric rupture in horses varies between reports. In one study of 54 horses, secondary gastric rupture was most common (65%), usually due to small intestinal obstruction, with primary gastric dilatation and idiopathic rupture occurring almost equally. In another retrospective study, 60% of the gastric rupture cases were classified as idiopathic. Reported risk factors are non-specific and include feeding grass hay, not feeding grain, gelding, and a non-automatic water source. Nasogastric intubation does not preclude the possibility of gastric rupture, and the amount of reflux obtained prior to rupture is variable.

Regardless of the initiating cause, gastric rupture usually occurs along the greater curvature. In horses with rupture due to gastric dilatation, tears in the seromuscular layer are frequently larger than the corresponding tears in the mucosa, indicating that the seromuscularis likely weakens and tears prior to the mucosa. In contrast, horses or foals with gastric rupture secondary to gastric ulceration usually demonstrate full-thickness tears of equal size in all layers. Gastric rupture is usually fatal due to widespread contamination of the peritoneal cavity, septic peritonitis and septic shock. Surgical repair is thus limited, but has been reported for partial thickness tears; in one case of a combined tear of the mucosa and muscularis with only a focal serosal tear, a full thickness repair was performed with a favorable outcome.

Gastric impaction: Gastric impaction can result in either acute or chronic signs of colic. Although a specific cause is not always evident, ingestion of coarse roughage (straw bedding, poor quality forage), foreign objects (rubber fencing material), and feed that may swell after ingestion or improper mastication (persimmon seeds, mesquite beans, wheat, barley, sugar beet pulp) have been implicated.^{16,17} Possible predisposing factors include poor dentition, poor mastication and rapid consumption of feedstuffs, and inadequate water consumption. Clinical signs can vary from anorexia and weight loss to acute colic. In severe cases, spontaneous reflux may occur. In cases presenting with acute colic, a diagnosis is often made via ultrasonography or during exploratory celiotomy. Identification of a severely enlarged stomach with ultrasound in the absence of reflux is suggestive. In animals not exhibiting signs of colic warranting surgical intervention, an endoscopic finding of a full stomach after a normally adequate fast (18-24 hours) can often confirm the diagnosis. In addition to pain management, specific treatment consists of gastric lavage via nasogastric intubation or massage and injection of fluid to soften the impaction during laparotomy.^{16,18}

Miscellaneous disorders: Non-ulcerative gastritis rarely occurs in the horse; however a single case of emphysematous gastritis due to *Clostridium perfringens* has been reported. The most common gastric neoplasia is squamous cell carcinoma. Clinical signs are typical of chronic recurrent colic, and, often most importantly, weight loss. The diagnosis is confirmed with gastroscopy, and the tumor can occasionally be imaged with transabdominal ultrasonography in the left cranial abdomen. Treatment of affected cases is typically not warranted, as surgical exposure is limited, and tumor size is typically considerable by the time of diagnosis.

Clinical signs and Laboratory findings: The biggest challenge with DPJ is differentiation from small intestinal strangulating obstruction. The most characteristic clinical findings with DPJ include moderate to severe pain which often improves after gastric decompression; large volumes of gastric reflux; clinical signs of endotoxemia (including fever); small intestinal distention evident on rectal palpation and ultrasonographic examination. Ultrasonographic findings in horses with DPJ typically include gastric, duodenal, and SI distension with normal or increased SI wall thickness. Horses with obstructive SI lesions usually show consistent signs of abdominal pain. In contrast, signs of colic typically subside after gastric decompression in horses with DPJ. They are replaced by lethargy and general malaise.

Abnormal clinicopathologic findings include hemoconcentration, neutropenia, acidemia, prerenal azotemia, hyponatremia, hypochloremia, hypokalemia, and elevated hepatic enzymes. Typically, peritoneal tap findings include a mild to moderate increase in WBC (up to 20,000) with a moderate to marked increase in total solids (up to 5 g/dl), but the nucleated cell count has been reported to vary widely in some cases. Strangulating lesions typically result in more severe changes in the peritoneal fluid, typically serosanguinous fluid with increased protein, white blood cells, and red blood cells. Horses with intraluminal obstructions, such as ileal impaction, typically have grossly normal peritoneal fluid with a normal or mildly elevated protein. Biochemical abnormalities may include hyponatremia, hypochloremia, hypokalemia, prerenal azotemia, and elevated hepatic enzymes (GGT, AST, and ALP). The loss of enteric bicarbonate through reflux along with poor tissue perfusion and hypovolemia can lead to metabolic/lactic acidosis with a high anion gap.

Although there are differences between DPJ and small intestinal obstructive lesions, there is no single distinguishing feature and all data must be considered collectively.

Treatment and Prognosis: Treatment should center primarily on supportive care with fluid therapy and gastric decompression. Tachycardia and signs of colic do not always precede the need for gastric decompression, and repeated drainage should be performed until the amount of reflux decreases significantly. Decompression should occur initially at least every 2 hours. With decreasing amounts of accumulated reflux, the interval can be decreased to 4 hours. The presence of an indwelling nasogastric tube alone does not prevent gastric rupture, nor does the removal of fluid via nasogastric tube rule out a gastric rupture in a particular horse.

Initial fluid replacement requirements can be substantial and may necessitate a combination of colloid and crystalloid replacement. Maintenance requirements can normally be met with crystalloids once an animal has been stabilized. The rate and type of fluid therapy will depend upon the continued rate of loss via reflux. This is most easily accomplished by calculating the total daily (at least) volume of reflux obtained and compare this amount to the total volume of fluid delivered, while also accounting for maintenance requirements. Achieving and maintaining adequate hydration and normal electrolyte status is critical for a return to normal intestinal function. NSAIDs

(typically flunixin) should be used for analgesic as well as anti-inflammatory therapy as long as renal function remains normal. Additional anti-endotoxic therapy, such as polymixin B sulfate, should be considered. The use of systemic antibiotics is controversial. In the presence of profound neutropenia ($<1000/\mu\text{L}$) or peritonitis, broad spectrum coverage with penicillin and gentamicin (or enrofloxacin in the face of azotemia) is recommended. When cases either deteriorate or do not improve with several days of medical therapy or if a strangulating lesion cannot be ruled out, surgical exploration should be considered. Surgical exploration can offer manual decompression of the small intestine and rule out any physical obstruction. In protracted cases or in horses with elevated triglycerides, supplemental parenteral nutritional support should be considered.

Normal (healthy) intestine is necessary for optimum performance of prokinetic agents in horses, thus most prokinetics likely are ineffective in cases of DPJ. Inflamed jejunal tissue has been shown to have downregulation and decreased production of motilin receptors, which may alter the prokinetic response to erythromycin. Lidocaine has shown promise - although the exact mechanism of action is unknown, the beneficial effects in horses with ileus are likely due to anti-inflammatory effects as a direct prokinetic effect has not been demonstrated. Therapy with either erythromycin lactobionate or bethanechol can also be considered in protracted cases.

With prompt medical therapy, horses with DPJ generally carry a good prognosis, but can have high costs associated with therapy. Horses treated surgically have a similar prognosis. Potential complications include laminitis, thrombophlebitis, peritonitis, cholangiohepatitis and adhesions. Development of the first three complications will be evident immediately, although they may occur at any point in the first few days of illness. Adhesions can either cause a protracted initial course of disease or recurrent bouts of colic after initial recovery. Horses with a prolonged disease course can develop pharyngitis and/or esophagitis secondary to either repeated nasogastric intubation or indwelling nasogastric tubes. In animals with severe electrolyte abnormalities, cardiac arrhythmias can also occur.

MISCELLANEOUS INFLAMMATORY BOWEL DISORDERS

Several other factors can lead to small intestinal inflammation in the horse. These include inflammatory bowel diseases (IBD; lymphocytic/plasmacytic enteritis, granulomatous enteritis, Multisystemic Eosinophilic Epitheliotropic Disease (MEED), eosinophilic enteritis, lymphosarcoma) and proliferative enteropathy (infection with *Lawsonia intracellularis*). Causes of IBD in horses are similar to that reported in most other species. These disorders are typically, but not always, associated with maldigestion and/or malabsorption. Thus, they often cause weight loss, ill thrift, and hypoproteinemia. Many can also cause inappetence and intermittent colic, occasionally without signs of malabsorption.

Tests of intestinal function: Intestinal function tests can provide a practical and inexpensive means to assess the absorptive capability of the small intestine. For clinical

practice purposes this is limited to carbohydrate absorption tests, which are easy to perform. The noninvasive breath hydrogen test used to assess carbohydrate malabsorption in human beings has not proved reliable in equine studies. Results of absorption test require cautious interpretation as pathologic changes in the mucosa and submucosa must be extensive and widely distributed to greatly affect the peak plasma concentration and shape of the curve. Few commercial laboratories conduct xylose assays, while glucose concentrations are routinely monitored in most referral and many primary care facilities.

The oral glucose tolerance test (OGTT) is typically performed by administering 1g glucose per kg body weight as a 20% solution via nasogastric tube following an overnight fast (14-16 hours). Blood is collected into heparinized tubes prior to glucose administration and at 30-minute intervals for 180 minutes; if possible, the test is continued at 60-minute intervals until 360 minutes. An alternative protocol is to continue sampling at 30-minute intervals for 240 minutes. Food is withheld during the sampling period, but water is allowed at all times. Maximum plasma glucose concentration (>85% increase above baseline) is reached by 120 minutes in healthy horses. The immediate dietary history, gastric emptying rate, intestinal transit, age, and hormonal effects of the horse influence glucose peak and curve shape. Prolonged fasting (≥ 24 hours) results in a delayed and slightly lower peak plasma glucose concentration. Results from the OGTT can also be affected by physiologic stress, content of non-structural carbohydrate and fat in the diet and other disorders, such as polysaccharide storage myopathy.

The xylose absorption test is performed as per the OGTT, except 0.5 g D-xylose per kilogram body weight is administered as a 10% solution via nasogastric tube. The pre-test period of fasting and timing of sample collection is identical. One should contact the laboratory to which samples will be sent prior to collection to ensure that heparinized plasma is acceptable for their laboratory. Plasma D-xylose should peak between 20 and 25 mg/dl between 60 and 90 minutes following administration, with occasional peaks as late as 120 minutes. D-xylose absorption testing is not confounded by hormonal effects or mucosal metabolism as is glucose, but is altered by diet, length of fasting (which could also be influenced by recent appetite or degree of cachexia), and age. Gastric emptying rate, intestinal motility, intraluminal bacterial overgrowth, and renal clearance can affect curve shape.

Alimentary Lymphosarcoma: Alimentary lymphosarcoma of the horse may represent a primary neoplasia of the gut associated lymphoid tissue with significant cellular infiltration of the small intestine and associated lymph nodes with minimal large intestinal or systemic involvement. Young horses (2-4 years) are most commonly affected, but horses of all ages are at risk. No breed or sex predilection has been documented, and disease prevalence is unknown. Despite the progressive nature of disease, onset of clinical signs can be rapid. As with all adult cases of chronic inflammatory bowel disease, antemortem diagnosis is by a process of exclusion and usually is confirmed post mortem. Frequent abnormalities include anemia,

thrombocytopenia, neutrophilia or neutropenia, hypoalbuminemia with hyperglobulinemia, resulting in either a normal elevated serum protein. Lymphocytosis is rare. One may palpate intraabdominal masses, mainly enlarged mesenteric lymph nodes, rectally. Abdominocentesis or duodenal or rectal biopsy can provide a diagnosis, but are not a sensitive indicator of disease. Carbohydrate absorption tests usually reveal partial to total malabsorption. Early confirmation of a suspected diagnosis necessitates exploratory laparotomy with biopsy. Prognosis is poor, especially as most horses are presented in an advanced state of disease. Immunosuppressive drugs or chemotherapy may afford temporary improvement, but long-term outcome is unaffected.

Granulomatous Enteritis: Granulomatous enteritis is a chronic wasting condition. Most affected horses are young (2-3 years) and Standardbreds are over-represented.. Prevalence is low. The condition is sporadic and has an insidious onset, and the course can be protracted. Significant diagnostic features include anemia, slight increases or decreases in WBC, hypoalbuminemia, normal or decreased serum protein, occasional increases in ALP, normal GGT, enlarged mesenteric lymph nodes, and partial or complete malabsorption. Small intestine is usually most severely affected. The cause of granulomatous enteritis is unknown. The condition may represent a granulomatous hypersensitivity reaction, and immune-mediated responses to dietary, parasitic, or bacterial antigens may be important initiating factors. Treatment with a variety of drugs, particularly corticosteroids, has not affected the long-term outcome in the majority of cases. Surgery may be indicated with localized disease.

Multisystemic Eosinophilic Epitheliotropic Disease: MEED encompasses disorders characterized by a predominant eosinophilic infiltrate in the gastrointestinal tract, associated lymph nodes, liver, pancreas, skin, and other structures. The disorders include chronic eosinophilic gastroenteritis, eosinophilic granulomatosis, chronic eosinophilic dermatitis, and probably basophilic enterocolitis. Although prevalence is low, MEED appears to be more common than granulomatous enteritis. Most affected horses are young (2 to 4 years) and Standardbreds and Thoroughbreds predominate. The condition is sporadic, has an insidious onset, and often a protracted course. Diarrhea is common. Severe skin lesions with exudative dermatitis and ulcerative colitis are prominent and frequently are the principal presenting complaint. Despite extensive tissue eosinophilia, systemic eosinophilia is rare and hematologic values are usually unremarkable. Notable features include hypoalbuminemia and elevations in GGT and ALP. Partial or complete malabsorption is common. Morphologic changes are less pronounced in the small intestine than in the large intestine. Small intestinal lesions predominate segmentally in the proximal duodenum and distal ileum and gastric fundic hyperkeratosis is also possible. Abundant fibrosis is a feature of all affected tissues. The cause of MEED is unknown; infectious agents have not been identified. Biopsies of the rectal mucosa, skin, liver, intestinal tract, and lymph nodes may assist in diagnosis. Unlike the other conditions, MEED has definitive liver and pancreatic involvement; thus maldigestion may contribute to weight loss. Treatment

has been attempted with antibiotics, corticosteroids, and larvicidal anthelmintics. Although some horses can improve briefly, the long-term prognosis is poor.

Eosinophilic enterocolitis: Idiopathic eosinophilic enterocolitis affects segmental lesions in the small or large intestine, inducing signs of colic, often requiring surgical intervention. This problem may not involve malabsorption and does not have multisystem involvement. Because the problem is often associated with signs of colic and not signs of malabsorption, eosinophilic enterocolitis differs from the other conditions discussed in this section and is often diagnosed at the time of surgery. It carries a much better prognosis than the other inflammatory bowel diseases. Eosinophilic enteritis can also affect the proximal small intestine. In those cases, diagnosis is by duodenal biopsy; treatment with corticosteroids may result in improvement.

Lymphocytic-Plasmacytic Enterocolitis: No specific clinical or clinicopathologic features differentiate this condition antemortem from other forms of IBD. In a retrospective study of 14 horses, carbohydrate absorption was abnormal or delayed in 9 of 12 horses, consistent with the predominance of small intestinal pathologic changes. Rectal biopsies were abnormal in 3 of 7 horses, two of which were reported as having lymphocytic-plasmacytic proctitis. Prognosis is poor. Treatment has been unsuccessful, probably because of the advanced nature of the condition at the beginning of treatment. If diagnosed early, with duodenal or rectal biopsy, corticosteroid therapy may result in improvement, which may be temporary.

Proliferative enteropathy (PE): PE typically affects weanling foals from 3 to 8 months of age and has been reported in North America, Europe, and Australia, causing disease in individuals or outbreaks of multiple affected animals on the same premise. PE has been reported uncommonly in yearlings and adult horses. The disease affects many other species, namely swine, and is caused by *Lawsonia intracellularis*, an obligate intracellular bacterium found in the cytoplasm of proliferative crypt epithelial cells of the jejunum and ileum. The source of infection is typically not determined. Recent weaning appears to be a common risk factor.

Clinical signs include depression, rapid and significant weight loss, edema, diarrhea, and colic. Poor body condition, a rough hair coat, and potbelly appearance are also reported. One should note that not all clinical signs are present in all cases, and diarrhea has only been noted in approximately half of reported cases. Other problems often were concurrent, including respiratory tract infection, dermatitis, intestinal parasitism, and gastric ulceration. The most significant laboratory finding is profound hypoproteinemia, predominantly characterized by hypoalbuminemia, but panhypoproteinemia can also occur. Leukocytosis and hyperfibrinogenemia are also common, with occasional alterations in electrolytes (hyponatremia, hypokalemia, hypochloremia) and elevated serum creatine kinase concentrations. Abdominal ultrasound commonly reveals increased small intestinal mural thickness.

One can suspect PE based upon clinical signs and severe hypoalbuminemia in a weanling foal with exclusion of common enteric infections. Fecal polymerase chain reaction for bacterial DNA or serum immunofluorescence assay or immunoperoxidase monolayer assay (IMPA) for antibodies against the organism can provide additional support for the diagnosis. The IMPA may be more sensitive, and typically titers $\geq 1:60$ are considered positive. Submission of both tests is recommended. Although both tests are quite specific, both also lack sensitivity, especially early in the course of disease (serology) or with prior antimicrobial therapy (fecal PCR). Of note, fecal PCR can become negative in affected foals within 4 days of antimicrobial therapy. Proliferative enteropathy is not typically associated with abnormal carbohydrate absorption test results. In cases with diarrhea, infectious causes should be ruled out. A definitive postmortem diagnosis can be confirmed by identifying characteristic mural thickening and intracellular bacteria within the apical cytoplasm of proliferating crypt epithelial cells using silver stains, PCR, and/or immunohistochemical testing.

Reported antimicrobial therapy includes erythromycin, alone or with rifampin, azithromycin, clarithromycin, oxytetracycline, doxycycline, metronidazole, or chloramphenicol. Recent reports appear to favor the use of intravenous oxytetracycline, followed by oral doxycycline, with apparent success. Duration of therapy is typically 2 to 4 weeks. Affected foals often need supportive therapy including crystalloid fluid and electrolyte replacement and often colloid support with either hydroxyethyl starch or plasma. Non-steroidal anti-inflammatory therapy can be used as needed for significant pyrexia. Corticosteroid therapy is not indicated, as inflammation is not a significant pathologic finding. Response to therapy has been good, with reported survival rates between 82 and 93%. Rapid improvement in clinical signs even within 24 hours preceded the rise in plasma protein concentration.

ACUTE COLITIS IN ADULT HORSES

Etiology: Principal infectious agents often associated with colitis in horses include *Salmonella* spp, *Neorickettsia* (formerly *Ehrlichia*) *risticii* (Potomac Horse Fever, Equine monocytic ehrlichiosis), *Clostridium difficile*, and *Clostridium perfringens*. *Aeromonas* spp are often isolated from horses with diarrhea, but their significance has not been determined. Parasites are not typically associated with acute diarrhea in adult horses, with the exception of larval cyathostomiasis in Europe and the northern part of the United States and Canada. The most common cause of outbreaks of colitis in horses is salmonellosis. Outbreaks of Potomac horse fever and clostridial colitis are rare, although the latter may occur as a clustering of cases of foals or hospitalized horses.

Non-infectious causes of acute diarrhea include dietary changes (composition or quantity), toxicity (e.g., heavy metals, phenylbutazone or cantharadin), and antibiotic-associated diarrhea. While antimicrobial-associated diarrhea can be caused by *Salmonella* or *Clostridium difficile*, an infectious cause is not always identified.

Epidemiology: Salmonellosis is generally considered to be the most common cause of infectious enterocolitis in adult horses, but reports from northern California suggest

that *Clostridium difficile* is most common in that region. The reported prevalence of infection with *Salmonella* has been variable, ranging from less than 1% to 70%. Prevalence of *Salmonella* is increased: 1) amongst horses with diarrhea; 2) when a highly sensitive method is used for identification (such as polymerase chain reaction (PCR) or culture of intestinal contents); or 3) during the warmer months of the year (summer and autumn). Most outbreaks occur during this period, particularly summer in Florida. Approximately 15-20% of horses presented to the VMC with gastrointestinal disease have at least one fecal sample from which *Salmonella* spp. are isolated. Over the past several years, we have seen peaks in *Salmonella* shedding in the spring and summer months. Risk factors for enteric salmonellosis include transportation, change in diet, antimicrobial treatment, surgery, common nasogastric tubes, wet and dark conditions, and other gastrointestinal disorders (e.g., impaction colic). Foals are at risk for *Salmonella* bacteremia although this rarely occurs in adult horses. Mares are commonly considered the source of infection, although they rarely develop clinical signs.

Reported risk factors for Potomac horse fever include frequency of administration of fly spray, residence on a farm with history of PHF, residence in a region of high-prevalence of seropositivity, and travel to or riding in specific risk regions. The disease is seasonal in nature (most cases occur from May to October) and often occurs in close proximity to a water source. Evidence to date indicates that the probable vector of *N. risticii* is a trematode of freshwater snails. This same trematode, *Acanthatrium oregonense*, is endemic in bats. The metacercariae of these flukes have a broad host range and other metacercariae-infected insects shown to harbor *N. risticii* DNA include species of caddis flies (*Trichoptera*), mayflies (*Ephemeroptera*), damselflies (*Odonata* and *Zygoptera*), dragonflies (*Odonata* and *Anisoptera*) and stoneflies (*Plecoptera*). The exact mechanism of transmission to horses is unknown, but the most recent evidence suggests accidental ingestion of aquatic insects harboring *N. risticii*-infected metacercariae. Once transmitted, the organism can infect equine monocytes, mast cells, and intestinal epithelial cells. The risk factors for clostridial enterocolitis include young age, recent use of antimicrobials, management factors, and hospitalization.

Signalment and History: Young horses may be more likely to develop certain forms of colitis (clostridial), and performance horses may be more likely to receive NSAIDs for musculoskeletal disorders resulting in right dorsal colitis. Feeding history may indicate change in diet, or feeding alfalfa hay that would increase suspicion of cantharidin toxicosis. If multiple animals are affected, infectious or toxic causes are more likely.

The most common cause of outbreaks of colitis in horses is salmonellosis. Outbreaks PHF and clostridial colitis are rare, although the latter may occur as a clustering of cases of foals or hospitalized horses. Intoxication with cantharidin, hoary alyssum (*Berteroa incana*), heavy metals, mycotoxins, or other intestinal toxicants should be considered. Visiting the farm and inspecting pastures may be necessary. It is unusual for multiple horses to be affected with cyathostome-associated colitis.

The owner also should be queried with regard to duration and character of clinical signs, history and duration of fever, and medications administered. Although

antibiotic-associated diarrhea generally occurs within 1 week of treatment initiation, onset may occur up to several days after cessation of treatment.

Clinical Signs & Lab Findings: Diarrhea is the most commonly observed clinical sign. Not all horses with enterocolitis will develop diarrhea. This is particularly true if the predominate area of disease is small intestinal or focal within the cecum or large colon. The consistency, color, or odor of diarrhea does not indicate a particular cause. Hemorrhagic diarrhea is uncommon. Abdominal pain of variable intensity is common and often precedes diarrhea.

Potomac horse fever can present with or without diarrhea. Occasionally, pyrexia (often extreme) and depression can be the only presenting signs, and fecal consistency with this disease can vary from normal to profuse, watery diarrhea.

Most horses with infectious enterocolitis are febrile. Endotoxemia and dehydration frequently occur, and can be reflected by drying and darkening of the mucous membranes, prolongation of the capillary refill time, and an elevation in pulse and respiratory rates. Horses with right dorsal colitis often have normal feces but hypoproteinemia. Ventral edema can be found in any disease causing an enteric vasculitis or protein-losing enteropathy. Auscultation and percussion of the abdomen reveal increased borborygmi and tympany, although in peracute or acute cases gut sounds are reduced or absent.

Sequelae of enterocolitis include laminitis, hemodynamic renal failure, thrombophlebitis, fungal pneumonia, cholangiohepatitis, and peritonitis. The latter 3 complications are rare, but offer an extremely poor prognosis.

Abnormal lab findings include acidemia, hyperlactemia, hyponatremia, hypochloremia, hypokalemia, hypocalcemia and hypoalbuminemia. Total calcium may be low as a result of decreased intake and loss of protein-bound calcium; decreased ionized calcium may be associated with endotoxemia, may persist for several days, and may result in clinical signs such as synchronous diaphragmatic flutter. Extremely low total or ionized calcium is consistent with cantharadin toxicosis. Neutropenia with toxicity and left shifting is common, particularly early in the course of the disease. Pre-renal azotemia is seen in most horses and rarely exceeds 7.0 mg/dl. The serum creatinine should decrease by approximately 50% every 24 hours with appropriate fluid therapy. Elevated liver enzymes (AST and GGT) may be observed and is presumably caused by endotoxemia and hypoperfusion of hepatic parenchyma. Presence of fecal leukocytes may help to attribute colitis to an invasive pathogen. A definitive diagnosis is not always possible, and co-infections can exist.

Diagnostic tests for feces:

- **Aerobic culture:** The main organisms of interest are salmonellae. These cultures require special media and antigens for serogroup identification, and are readily available through most, if not all, commercial laboratories. Multiple cultures are preferable (ideally 5 cultures >12 hours apart), and recovery of pathologic

organisms can be difficult when feces are very watery. Culture of a rectal mucosal biopsy sample can improve recovery.

- Polymerase chain reaction (PCR): PCR has been reported as a more sensitive method of detecting salmonella in feces relative to culture. The diagnostic significance of those horses positive by PCR but negative by culture of multiple fecal samples remains to be determined.
- Anaerobic culture: Clostridial organisms are primarily of interest. Strict anaerobic handling of the feces is critical to successful culture, especially for *C. difficile*. Recovery of *C. difficile* organisms is dramatically reduced after storage for 72 hours in aerobic conditions at 4°C. Because clostridial organisms can be cultured from the feces of some normal horses, toxin detection is preferred for a diagnosis of clinically relevant disease. Genotyping of *C. perfringens* isolates is also commercially available.
- Toxin analysis: Commercial assays (ELISA) are available for *C. difficile* toxins A and B as well as the enterotoxin of *C. perfringens* (CPE). Previous work has shown that *C. difficile* toxins are more stable in aerobic conditions than the organisms themselves. Many diagnostic laboratories will perform toxin testing, and some will provide packages including both culture and toxin analysis.
- Sand sedimentation: Easily performed with a rectal sleeve and water.
- Floation and McMaster's quantification if necessary for parasites.
- Microscopic analysis: Gram stain is useful for identification of increased fecal leukocytes and as an initial screen for Clostridial organisms (Gram positive rods). Cyathostome larvae are best detected by direct examination of feces. If cantharidin toxicosis is suspected the feces should be examined for parts of *Epicauta* beetles. The hay should also be checked for beetles, and cantharidin concentrations can be measured in blood and urine.

Diagnostic tests on whole blood or serum: *Neorickettsia risticii*: Although an enzyme-linked immunosorbent assay (ELISA; serum) has been described, most laboratories use an immunofluorescent assay (IFA; serum) or PCR (whole blood in EDTA). Diagnosis can be confirmed by PCR, single IFA >1:800 or via paired serum samples collected at a 5-7 day interval. Vaccination for PHF results in positive titers for up to 6-9 months. Natural seroprevalence has been reported from 16-33%, typically with <1:320. PCR offers the advantage of excellent sensitivity without the potential for interference from vaccination, thus is the test of choice.

Ancillary diagnostic tests: Additional tests include abdominal radiography (sand, enteroliths), rectal biopsy (salmonellae, cyathostomes, or inflammatory infiltrate), abdominal ultrasonography, cantharidin analysis (urine, serum, or gastric contents), and laparoscopy.

Treatment: Fluid therapy: Intravenous therapy is usually needed for horses with acute colitis. Acidemia is frequently corrected by restoring plasma volume with a balanced

polyionic solution (LRS, Normosol-R, etc.). In the face of severe dehydration, hypertonic saline can be used to restore circulatory volume, followed by administration of isotonic fluids. Many horses with severe colitis become hypoproteinemic and/or develop endotoxemic shock. Thus, they may benefit from administration of colloids. Plasma is the colloid of choice and, if possible, should be administered to maintain TP > 4 g/dl. Although it is generally economically impractical to administer plasma in volumes to replace a decrease of even 1-2 g/dl of plasma protein, smaller volumes (1-3 L) may exert beneficial effects because of other protein constituents (e.g., AT III, fibronectin). Oncotic pressure can be restored with synthetic colloids such as hetastarch (maximum 10 ml/kg/day). If oncotic pressure is restored, CVP can be used to determine crystalloid fluid rate. Practically, this is rarely done and physical exam parameters, acid-base status, and venous lactate are used to aid therapy.

Anti-endotoxin: Non-steroidal anti-inflammatory drugs (NSAID) may be indicated for analgesic and anti-endotoxic effects. Use of these drugs should be avoided in horses with NSAID toxicity, severe dehydration, or azotemia. Pentoxifylline (8 mg/kg; q. 12 hrs; PO), a rheologic agent, has been demonstrated to decrease platelet aggregation and reduce the release of TNF α and IL-1 from endotoxin-stimulated equine macrophages *in vitro*. Administration of aspirin (10-20 mg/kg or 60-90 grains total q. 48 hrs; PO) may help if a hypercoagulable state is suspected. Polymyxin B (6,000 units/kg IV diluted in 1 L; q. 12) is an antimicrobial that binds circulating endotoxin. Polymyxin is nephrotoxic and should not be used in the face of significant azotemia.

Anti-toxin: DTO-smectite (Biosponge) has been shown to bind *C. difficile* toxins A, B, and C as well as CPE, thus is recommended for antibiotic-associated or other clostridial diarrhea. Activated charcoal (1 to 3 g/kg as a slurry 1-2 x/day), bismuth subsalicylate (2 to 4 L), or mineral oil (2 to 4 L) may help to bind lipid soluble toxins.

Antibiotics: Antimicrobials should be avoided in adult horses with acute colitis unless there exists clinical or laboratory evidence of bacteremia or immunosuppression (i.e. neutropenia <1000). Metronidazole can be used for clostridial colitis, although approximately one third of recent *Clostridium difficile* isolates from California are resistant. Oxytetracycline (6.6 mg/kg IV q24 hours for 3-5 days) is the treatment of choice for PHF. Horses will typically demonstrate dramatic improvement within 24-48 hours. Antimicrobials that have been used frequently to treat salmonellae include chloramphenicol, and enrofloxacin. These agents are normally reserved for cases in which diarrhea does not respond to initial supportive care or from which positive fecal cultures continue beyond resolution of clinical signs. Normally, a long (3-4 week) course of enrofloxacin is necessary to achieve negative cultures, and some horses will continue to shed after such a course.

CHRONIC DIARRHEA IN ADULT HORSES

Chronic diarrhea is usually defined as diarrhea lasting greater than four weeks duration. Fecal consistency can vary widely. While many specific diseases can result in

chronic diarrhea, an inciting cause frequently remains elusive. Occasionally, problems of a non-gastrointestinal nature result in diarrhea, such as hepatic disease or abdominal abscessation. More common infectious causes of chronic diarrhea include chronic salmonellosis and parasitism with large and/or small strongyles. Recently, the spirochete *Brachyspira pilosicoli* was implicated in a herd outbreak of chronic diarrhea in weanling age horses. Non-infectious inflammatory causes include granulomatous enteritis or colitis, neoplasia (mainly lymphosarcoma), sand irritation, and right dorsal colitis. Non-inflammatory causes encompass a range of problems with the common theme of disruption of the large colonic intestinal flora. This may or may not be related to a dietary disruption, and many of these horses have few other clinical signs. Regardless of the inciting cause, horses with chronic diarrhea remain very difficult to treat and afford a guarded prognosis.

Diagnostic tests: A minimum database for the individual horse with chronic diarrhea typically includes CBC with fibrinogen, serum biochemical profile, venous blood gas analysis, rectal exam, abdominal ultrasound, and analysis of peritoneal fluid. Results are commonly normal.

Comprehensive fecal analysis should include assessment for parasites (grossly and by fecal flotation/McMasters quantification), aerobic culture for Salmonella (5 samples at a minimum 12 hour interval), water suspension for sand, unstained wet mount for protozoa and parasites, new methylene blue stain for fecal leukocytes, and Gram stain to get a ratio of gram-positive to gram-negative bacterial flora. Rectal biopsy is a simple, relatively noninvasive procedure. Two samples should be taken and submitted for culture (salmonella) and histopathology (inflammatory bowel diseases).

Horses in good general body condition with soft (cowpie), low volume feces may have a problem based in the small colon. These horses typically are bright, alert and responsive and have a stable water and electrolyte status. A rectal biopsy is often helpful. Conversely, horses with high volume, watery diarrhea most likely have significant large colon dysfunction. These animals are frequently in poor body condition and have a precarious water and electrolyte balance. Fecal analysis is a critical diagnostic test in these animals. Many horses with chronic diarrhea have normal CBC, peritoneal fluid and xylose absorption results, no fecal evidence of parasitosis, negative fecal culture, and absence of fecal leukocytes.

Treatment: If a specific diagnosis is achieved, directed therapy should be initiated. In all cases, free choice access to fresh water is critical to maintenance of hydration. Many horses will consume balanced, isotonic electrolyte-spiked water, and such a solution should be offered in addition to fresh water. In lieu of such a solution, access to a salt or mineral block can serve as a substitute source of electrolyte replacement. Typical feeding recommendations include good quality grass hay with limited legume hay and concentrate intake or a complete pelleted feed. Dietary changes alone are unlikely to provide a cure, but can often provide improvement.

Additional therapy can include:

- Enrofloxacin (7.5 mg/kg QD PO for 28 days) for chronic salmonellosis unresponsive to supportive therapy.
- Fenbendazole - 10 mg/kg PO for 5 days for suspected larval cyathostomiasis.
- Psyllium mucilloid (12-14 oz; 340-400 g) in 1 gallon mineral oil by NG tube SID for 3-5 days for horses with sand.
- Prednisolone 1 mg/kg PO q12-24 hours in horses with confirmed or suspected infiltrative bowel disease and negative cultures for *Salmonellae*.
- A complete pelleted feed (such as Equine Senior), psyllium, and oral metronidazole has recommended for horses with right dorsal colitis. In addition, one can add sucralfate and/or misoprostol.
- Transfaunation. Exact recommendations for the transfaunation procedure are sparse in the veterinary literature, as are reported benefits. Typically, cecal liquor is obtained either from an animal recently euthanized for non-gastrointestinal reasons or from an animal implanted with a cecal cannula. Recipients can be pre-treated with acid-suppressing agents to enhance viability of transplanted bacteria and protozoa as they pass through the gastric environment. Fresh feces can be used alternatively. The efficacy of such treatment has not been validated.

Prognosis: Regardless of the inciting cause, once a horse has diarrhea for at least a month, the prognosis for complete recovery is guarded, and the prognosis worsens as the course of disease progresses.

PERITONITIS

Peritonitis refers to inflammation of the mesothelial lining of the peritoneal cavity and is typically caused by mechanical, chemical, or infectious insult to the parietal peritoneum.

Etiology and Clinical Findings: Most cases of peritonitis occur secondary to a gastrointestinal event such as perforation of any portion of the gastrointestinal tract, intestinal ischemia, DPJ, colitis, neoplasia, verminous arteritis, intestinal mural abscess, or other causes. Iatrogenic causes include rectal tear, enterocentesis, castration, and abdominal surgery. Other causes include traumatic events (including uterine or vaginal perforation during foaling or breeding), mesenteric abscess (including those associated with *Streptococcus equi* subsp. *equi*), cholelithiasis, and others. Causes specific to the young foal include rupture of the urinary bladder or urachus, omphalitis and/or omphalophlebitis, sepsis, and *Rhodococcus equi* abscessation.

Organisms associated with gastrointestinal rupture include a mixed population of gram positive and gram negative aerobic and anaerobic organisms, typically with no clear predominance. Enterobacteriaceae, *Streptococcus* spp., and *Staphylococcus* spp. have been most commonly isolated from peritoneal fluid samples. Common anaerobes

include *Bacteroides*, *Clostridium*, and *Bacillus* species. In foals, peritonitis is more commonly associated with *Streptococcus* and *R. equi* infections. Several case series involving peritonitis associated with *Actinobacillus equuli* have been reported. These were initially reported in Australia, but one case was recently reported from the UK.

In most cases of peritonitis, clinical signs are variable, but include fever, depression and abdominal pain. Individuals can also present with diarrhea or weight loss. Based upon severity and localization, signs can also include those of endotoxemia and shock. In cases of *A. equuli* peritonitis, clinical signs in most horses included depression, inappetence, lethargy and mild to moderate abdominal pain acutely or weight loss in a chronic form. Post-partum mares with peritonitis secondary to a uterine perforation typically present with fever and depression, with or without abdominal pain.

Diagnosis: Definitive diagnosis is based upon an elevated TNCC in peritoneal fluid (>10,000 cells/ μ L). Culture of peritoneal fluid should be performed in all suspected cases, but this procedure has a low sensitivity, with only 9.5% to 32.5% of samples yielding positive growth. Total cell count can be increased following enterocentesis, abdominal surgery, or open castration. Thus, additional parameters must be considered in these populations. Abundant hypoechoic or variably echogenic peritoneal fluid evident upon abdominal ultrasound examination, fever, depression, and abdominal pain can all support the diagnosis. A decrease in peritoneal fluid pH (< 7.3) or glucose (< 30 mg/dl) or elevated peritoneal lactate (relative to serum) can suggest the presence of septic peritonitis. Peritoneal fluid cytology will typically reflect a septic process, with abnormalities ranging from the presence of bacteria or plant material to degenerate neutrophils. If gastrointestinal contents or plant material are evident, one should take care to differentiate between gastrointestinal rupture and enterocentesis. In post-foaling mares, the percentage of neutrophils in the peritoneal fluid can be elevated for up to 7 days, but the total protein and TNCC should remain within normal limits.

Therapy: Treatment of horses with peritonitis should begin with identification and correction of the underlying problem, if possible. If a gastrointestinal source is suspected, an exploratory celiotomy is likely indicated. Supportive care is also critical to the treatment protocol. This should include correction of fluid deficits, acid-base and electrolyte imbalances, and colloid oncotic pressure. Anti-inflammatory and anti-endotoxic therapy are also clearly of benefit. Additional analgesic and prokinetic drugs should be provided if necessary.

Antimicrobial therapy is critical to the management of septic peritonitis. Broad-spectrum coverage should be instituted pending results of peritoneal fluid culture and sensitivity. If positive results are obtained, therapy can be adjusted accordingly. A typical initial regimen includes penicillin, gentamicin, and metronidazole to cover gram-positive, gram-negative, and anaerobic spectrums, respectively. Metronidazole should be included if anaerobic involvement is suspected due to the known resistance

of multiple *Bacteroides* species to penicillins. Enrofloxacin can replace gentamicin in the treatment regimen because the lipophilic nature of this compound can provide increased penetration to the peritoneum. While *A. equuli* is typically sensitive to either penicillin or trimethoprim-sulfonamide combinations, initial broad-spectrum coverage with penicillin and gentamicin is suggested pending culture results due to resistance of some isolates.

Abdominal drainage and lavage can help remove excess fluid, foreign materials, fibrin, and bacterial products from horses with peritonitis, and post-operative lavage has been shown to decrease the incidence of experimentally-induced abdominal adhesions. Open surgical exploration provides the most effective and thorough examination of all peritoneal surfaces and is recommended if gastrointestinal perforation or ischemia is suspected or in any other case in which correction of a primary lesion is indicated. A ventral abdominal drain can either be placed at the time of surgery or in the standing horse with sedation and local anesthesia.

Peritoneal lavage is typically performed with 10-20 liters of a balanced isotonic electrolyte solution twice a day until the lavage solution becomes clear, or until the catheter becomes clogged with fibrin or omentum. Hypertonic solutions should be avoided, as they can result in fluid shifts into the peritoneum; povidone iodine should also be avoided as concentrations as low as 3% can induce peritoneal inflammation. Other agents such as antibiotics or heparin have been suggested as lavage additives, but data demonstrating their benefit are not currently available. Lavage with a plain isotonic solution did not alter the pharmacokinetics of gentamicin administered systemically. Thus, alteration of antimicrobial dosing does not appear necessary.

Prognosis: The prognosis is grave for peritonitis associated with gastrointestinal rupture. Reported survival rates for horses with peritonitis vary, but can be as high as 59.7%. Some of the variability in reported survival percentages can be related to inclusion criteria, mainly whether or not horses with gastrointestinal rupture were included. Septic peritonitis following abdominal surgery is reportedly associated with high mortality (56%). Peritonitis associated with *A. equuli* carries a very favorable prognosis, and all horses in these reports responded to medical therapy if attempted.

APPENDICES

EGUS lesion scoring system (as per EGUS Council)

Lesion Grade	Description
Grade 0	Intact epithelium with no appearance of hyperemia or hyperkeratosis
Grade 1	Intact mucosa with areas of reddening or hyperkeratosis (squamous)
Grade 2	Small single or multifocal lesions
Grade 3	Large single or multifocal lesions or extensive superficial lesions
Grade 4	Extensive lesions with areas of deep ulceration

Enteral fluid solutions made easy

	Unit	Soln. 1	Soln. 2
Tap water	liters	9	8
Table salt (NaCl)	oz. (approx. in urine cup)	1	1
Lite salt (1:1 NaCl:KCl)	oz. (approx. in urine cup)	1	0.25
Baking soda (HCO ₃)	oz. (approx. in urine cup)		0.75

Commonly used GI drugs

Class	Drug	Dosage (mg/kg)	Route	Interval (h)	Comments
NSAIDs	Flunixin	0.25-1.1	IV, PO	8-24	Avoid max. dose > 2x/day
	Ketoprofen	2.2	IV	24	
	Phenylbutazone	2.2-4.4	IV, PO	12-24	Avoid extravascular
	Carprofen	0.7	IV, PO	24	
	Etodolac	2.3	IV, PO	12	
α-2 Agonists	Xylazine	0.2-1.1	IV, IM	prn	
	Detomidine	0.005-0.04	IV, IM	prn	
	Medetomidine	0.004-0.01	IV	prn	
Opioids	Butorphanol	0.02-0.1	IV, IM	3-4	rarely give >0.05 mg/kg bolus
	Butorphanol	13-23 µg/kg/hr	IV	CRI	after 18 µg/kg bolus
	Morphine	0.1	epidural	8-16	qs to 30 ml w/ saline
Other	Lidocaine (2%; 20 mg/ml)	3 mg/kg/hr (75 ml/hr for 500kg)	IV	CRI	after 1.3 mg/kg bolus over 10-15 min
	Buscopan	0.3	Slowly IV	Once	Tachycardia; less for rectals
H2-Receptor Antagonists	Ranitidine	6.6	PO	8	
		1.5-2	IV	6	
	Cimetidine	20-25	PO	6	
		6.6	IV	6	
	Famotidine	2.8	PO	12	
0.3		IV	12		
Proton Pump Inhibitors	Omeprazole	0.5	IV	24	
		1-2	PO	24	Prevention
		4	PO	24	Treatment
	Pantoprazole	1.5	IV, PO	24	
Other	Misoprostol	0.005	PO	8	
	Sucralfate	20-40	PO	8	
Prokinetics	Erythromycin lactobionate	0.1-1.0	IV	6-8?	Motilin agonist
	Domperidone	0.2	IV		DA2 antagonist
	Bethanechol	0.025	SQ	3-4	Cholinomimetic
	Neostigmine	0.025	IV		Cholinomimetic