Chapter 2
Diseases of the Equine Gastrointestinal Tract
Timothy H. Ogilvie

COLIC is a nonspecific term referring to abdominal pain. In this chapter, its usage shall be restricted to gastrointestinal pain.

A. Determining the severity of the colic. Colic is usually sporadic in occurrence and may be mild or severe, acute or chronic. Repeated bouts in the same individual are not uncommon.

1. Patient profile and history
   a. Some intestinal problems that produce colic appear to be age related. For example, meconium impactions are restricted to neonatal foals, whereas feed impactions occur more frequently in older horses.
   b. An accurate history is essential in defining possible etiologies and pathophysiologies of medical colics. Retrospective information should include parasite control measures, pasture size, and stocking rates. The use and work schedule for the horse should be explored, as should any changes in environment or feeding. A past and present medical history is important for diagnostic purposes as well as interpretation of presenting clinical findings.

2. Clinical findings (Table 2-1). A complete physical examination should be attempted in all cases of colic to determine the site and cause of gastrointestinal pain as well as ruling out conditions that mimic gastrointestinal pain. The examination should be performed without sedation in tractable patients. If sedation is necessary, it should be administered only after a complete general examination has been performed because sedation will affect clinically important findings.
   a. Attitude. Colics produce attitudinal changes in the horse. Mild colics (e.g., large and small colon impactions) cause slight to moderate depression. Colics producing severe depression and toxemia often result from strangulation obstructions, which are not medically manageable. Medical colics may produce severe pain and anxiety, as in the case of gastric dilatation. The horse will often continue to eat with mild colics.
   b. Pain and anxiety is manifested as straining, pawing, stretching, and sweating. It is important to determine if the pain is continuous or intermittent, static or changing in intensity, responsive or unresponsive to medication.
   c. Temperature. Rectal temperature readings are usually normal to slightly elevated with medical colics. Subnormal temperatures should alert the examiner to the possibility of terminal shock and toxemia. High temperatures are associated with infectious or septic conditions. Temperatures may be normal if there has been use of antipyretic drugs (e.g., dipyrone, phenylbutazone, flunixin meglumine).
   d. Respiratory rate. Respiratory rates usually increase in proportion to the amount of pain. Abdominal pressure creates a rapid, shallow respiratory rate and pattern. A metabolic acidosis associated with tissue devitalization causes an increase in the respiratory rate.
   e. Evaluation of circulatory status
      (1) Pulse rates reflect the nature of the colic. In the adult horse, the interpretation of the pulse rate is shown in Table 2-2.
      (2) Pulse quality should also be evaluated. A strong, full pulse (rather than a weak, thready pulse) is reflective of a mild and medically responsive colic.
      (3) Capillary refill is normal with medically responsive colics and increases with surgical colics (as a result of vascular compromise).
      (4) Normal mucosal color is reflective of normal circulatory status and mild or early colics. Congested mucous membranes indicate vascular compromise, fluid loss, or shock.
f. Digestive system examination

(1) Abdominal contour is usually normal with medical colics. Distention is not a feature of serious small intestinal obstruction and most commonly is observed with large intestinal problems that are usually surgical in nature.

(2) Sharp molar teeth, reflective of poor dental occlusion or improper husbandry, may predispose horses to impaction colics.

(3) Abdominal auscultation should be carried out in a comprehensive, systematic way. Normal to increased hypermotility indicates early intestinal distention or entitis. Hypermotility, which results from ischemia or longstanding intestinal obstruction, may also be an initial response to gut ischemia.

(4) Method of examination

(a) During an esophageal examination, use the largest tube possible and a gentle technique, being careful to avoid esophageal perforation. To retrieve reflux, the tube may be primed with a bolus of warm water and gravity flow or suction used. The pH and composition of any fluid should be determined. Low pH fluid (4–5) indicates a gastric source, whereas a higher pH (6–7) indicates that the fluid is from the small intestine. Previously administered medications may be found in the reflux (e.g., mineral oil).

(b) A rectal examination, performed on patients of adequate size, is carried out in a systematic way, identifying normal and abnormal palpable structures. Feces may be present or absent in the rectum, a finding that is not indicative of the colic type. Firm and mucus-covered feces may point to an impaction colic. Sand in the feces is a special case of impaction colic. Sand in the feces is a special case of impaction colic. Sand in the feces is a special case of impaction colic.

(5) Abdominocentesis often is performed as part of the initial database of colic evaluation. Either a midline or paramedian site is acceptable, and the technique is considered a minor surgical procedure.

(a) A point 10–30 cm caudal to the xiphoid is chosen, and after the skin has been aseptically prepared and desensitized with a local anesthetic, either

### TABLE 2.2. Interpretation of Pulse Rate*

<table>
<thead>
<tr>
<th>Pulse Rate (beats/minute)</th>
<th>Mild Colic</th>
<th>Severe Colic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>30–39</td>
<td>40–59</td>
</tr>
<tr>
<td>Mild</td>
<td>40–59</td>
<td>60–79</td>
</tr>
<tr>
<td>Moderate</td>
<td>60–79</td>
<td>80–99</td>
</tr>
<tr>
<td>Serious</td>
<td>80–99</td>
<td>100+</td>
</tr>
</tbody>
</table>

* Foals will have relatively higher rates than adult horses.

<table>
<thead>
<tr>
<th>TABLE 2-3. Laboratory Findings With Normal Equine Peritoneal Fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
</tr>
<tr>
<td>-----------------</td>
</tr>
<tr>
<td>Total white blood cells ($\times 10^9/L$)</td>
</tr>
<tr>
<td>Neutrophils (%)</td>
</tr>
<tr>
<td>Lymphocytes (%)</td>
</tr>
<tr>
<td>Macrophages (%)</td>
</tr>
<tr>
<td>Eosinophils (%)</td>
</tr>
<tr>
<td>Red blood cells ($\times 10^9/L$)</td>
</tr>
<tr>
<td>Total protein (g/L)</td>
</tr>
<tr>
<td>Fibrinogen (g/L)</td>
</tr>
<tr>
<td>Specific gravity</td>
</tr>
<tr>
<td>Color</td>
</tr>
<tr>
<td>Turbidity</td>
</tr>
</tbody>
</table>

### TABLE 2.4. Gross Observation of Abnormal Peritoneal Fluid Samples

<table>
<thead>
<tr>
<th>Fluid Samples</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flocculent fluid, no odor</td>
<td>Bacterial and toxic peritonitis seen in early infective disease</td>
</tr>
<tr>
<td>Serosanguineous, no odor</td>
<td>Leakage of RBCs, toxins, and bacteria from necrotic bowel into peritoneal cavity</td>
</tr>
<tr>
<td>Sanguineus, malodorous with fecal material</td>
<td>Associated with parietal pain</td>
</tr>
<tr>
<td></td>
<td>Confirms presence of ruptured viscus</td>
</tr>
<tr>
<td>Frank blood in abdomen</td>
<td>Rectal tear</td>
</tr>
<tr>
<td></td>
<td>Rarely, blood-tinted fluid is present</td>
</tr>
<tr>
<td></td>
<td>Usually when blood vessel is entered or splenic parenchyma is penetrated</td>
</tr>
<tr>
<td></td>
<td>Rarely neoplasms, such as hemangiosarcoma, may cause abdominal hemorrhage</td>
</tr>
</tbody>
</table>

RBCs = red blood cells.
2. Large colon impaction

a. Patient profile and history. This is one of the most common colics encountered in practice. Large colon impaction may be age, feed, or management-related and occur with some repeatability in certain horses. Horses may have a history of dental problems, recent deworming, or feed or management changes.

b. Clinical findings. The clinical findings are consistent with a medical colic. There is often slight depression and anorexia. There are no abnormalities of temperature, pulse, and respiration (TPR), but there is evidence of periodic visceral pain when the horse stretches and looks at its flank. There is decreased fecal output, and feces are small, firm, and covered with mucus. Fecal composition may indicate the nature of the impaction (e.g., grain, sand). Rectal examination may reveal the site and the degree of the impaction. For example, the pelvic flexure is a common site of large colon impactions. On gastric intubation, there is no reflux of stomach contents. Abdominal auscultation reveals a generalized decrease in borborygmi.

c. Etiology and pathogenesis

(1) Physical agents

(a) Feed-related. Course roughage may predispose the horse to improper digestion of feedstuffs with a resultant impaction.

(b) Water-related. Insufficient amounts of water create a dry ingesta prone to impaction.

(c) Poor teeth. Similar to poorly digestible feeds, improper mastication causes some impactions.

(2) Parasitic agents. Migrating larval forms of *Strongylus vulgaris* interfere with circulation and innervation of various parts of the large intestine, which affects gut motility and leads to impactions.

(3) Extraluminal or intraluminal agents. Extraluminal events (e.g., abscesses, neoplasms, adhesions) or intraluminal masses (e.g., enteroliths) produce impaction colics. The majority of these, however, result in chronic, unre sponsive colics that must be surgically managed.

d. Diagnostic plan. The clinical findings often are enough to diagnose the condition of a large intestinal obstruction. The response to therapy also is a valuable diagnostic aid.

e. Laboratory tests. Hematology and clinical pathology findings are normal. Abdominocentesis, although usually not warranted, yields fluid of normal characteristics.

f. Differential diagnoses. Differential diagnoses to consider when presented with a large intestinal obstruction include early surgical colics (e.g., strangulating obstructions, nonstrangulating small intestinal obstructions), gastric ulcers, chronic *salmonella* colitis, chronic liver disease (cholelithiasis), and urolithiasis.

g. Therapeutic plan

(1) Analgesics. Analgesics may be indicated if discomfort levels of the horse warrant. All of the following agents may be given intravenously or intramuscularly:

- Flunixin meglumine: 1.1 mg/kg every 12 hours
- Xylazine: 0.1–1.0 mg/kg as necessary
- Butorphanol: 0.02–0.05 mg/kg as necessary
- Detomidine: 0.005–0.03 mg/kg as necessary
- Pentazocine: 0.3 mg/kg as necessary

(2) Laxatives

(a) Laxatives and wetting agents aid in softening the mass. The following substances are all oral medications:

- Mineral oil: 2–4 L every 12 hours
- Dioctyl sodium sulfosuccinate (DSS): 10–20 mg/kg
- Bran mashes
- Intravenous or oral fluids also may be employed to soften intestinal masses. Doses are empirical.

(3) Surgery may be necessary if the condition persists, worsens, or if clinical signs become repetitive.

h. Prevention

(1) Revisits to the patient or a client’s attention to clinical signs are necessary to judge the response to therapy. If repeated doses of analgesics are necessary to control the pain or if the pain increases in intensity or duration, the diagnosis of a primary medically responsive large intestinal impaction must be reassessed. A decision for surgery must be made early for the maximum probability of success.

(2) Clients need to consider management changes in order to address the risk factors (e.g., feed types, feeding techniques, access to water, dental management, proper parasite control).

3. Distention colic (spasmodic or gas colic)

a. Patient profile and history. This is a commonly diagnosed colic with similar subjective findings to other medical colics. Horses that crib (windsock) often seem predisposed to distention colic.

b. Clinical findings. As with other medical colics, there might be slight increases in TPR. On abdominal auscultation, there may be increased peristaltic activity, particularly between bouts of pain. Abdominal percussion may reveal tympanic sounds of intestinal gas. There will be minimal reflux on nasogastric intubation. Often during a rectal examination, bowel distended with gas is felt.

c. Etiology and pathogenesis

(1) Simple distention colics result from intestinal spasm or ileus. The intestines distend with fluid and gas cranial to the site(s) of spasm, causing visceral pain. Peristalsis may increase in the distended segments due to local myoelectrical stimulation.

(2) The initial cause of the intestinal spasm or ileus may be related to the same risk factors associated with the development of simple obstruction colics (i.e., parasite migration, feed changes, management deficiencies).

(3) As a special case of distention colics, horses that crib and swallow air cause gastric distention and pain.

d. Diagnostic plan. The clinical findings should be compared with the degree of pain and response to therapy. Simple distention colics may appear similar to early cases of obstruction colics, strangulating obstruction colics, and nonstrangulating infarctions, which are more serious and may require surgical intervention.

e. Laboratory tests. Laboratory values are not outside of normal ranges for this condition.

f. Differential diagnoses. The following categories of colics and specific conditions are surgical in nature but early in their course may appear similar to a simple intestinal distention.

(1) Nonstrangulating obstructions

(a) Foreign bodies

(b) Ascarid impactions (young animals)

(c) Meconium impaction (foals)

(d) Muscular hypertrophy of the ileum

(e) Pseudoulcinated lipomas

(f) Abscesses

(g) Adhesions

(h) Neoplasms

(2) Strangulating obstructions

(a) Small intestinal volvulus
4. Proximal enteritis (antecedent enteritis, duodenitis) is an idiopathic syndrome characterized by chronic and transmural leakage of fluid into the gut. Horses with this condition exhibit a moderate colic but marked depression. There are diminished gut sounds and copious gastric reflux.

- Patient profile and history: This disorder is seen primarily in adult horses. Proximal enteritis is not uncommon in occurrence but is similar in appearance to a small intestinal obstruction, which presents as a surgical colic. There may be a history of recent grain ingestion or heavy grain feeding.

- Clinical findings: This colic is usually mild, but affected horses are extremely depressed. 
  1. A fever is often evident (38.5°C-40.0°C), and the heart rate is increased (60-120 beats/min). An elevated respiratory rate is caused by pain.
  2. The horse may be dehydrated, with resultant signs of fluid volume depletion (e.g., dry and injected mucous membranes, increased capillary refill time, decreased skin elasticity).
  3. Gastric reflux is invariably present with severe illness being untreated. The reflux is green-yellow with an alkaline pH (6–7) indicating small intestinal origin.
  4. Peristaltic sounds are weak, and rectal palpation reveals slightly distended small intestines. This distension does not increase over time. The course of the disease is 7–10 days.

- Etiology and pathogenesis
  1. Etiology. Suspected causes include pancreatitis, ileitis, and gram-negative enteritis (e.g., salmonellosis).
  2. Pathogenesis. The gastric reflux produces the pain associated with the condition. There is an associated toxemia with varying signs of shock, coagulopathy, anemia, and renal dysfunction. Postmortem findings demonstrate inflammation and degeneration of duodenal intestinal mucosa. A fibrinopurulent exudate is present on the serosal surface. Lesions are less commonly found in the jejunum or the pylorus.

- Diagnostic plan. The determination of a clinical diagnosis is challenging. A horse with mild to moderate pain, severe depression, and fever is more likely to have proximal enteritis than small intestinal obstruction. There is, however, gastric reflux in both instances. A small intestinal obstruction causes a progressive deterioration in clinical signs, whereas proximal enteritis has a steadier course and pain that is relieved by nasogastric intubation.

- Laboratory tests
  1. The packed cell volume (PCV) and total serum protein (TSP) are elevated, which indicates dehydration.
  2. There is hypokalemia due to potassium sequestration in the small intestine.
  3. There may be a hypochloremic, metabolic alkalosis due to HCO₃⁻ pooling in the stomach, but serum electrolytes are usually within normal ranges.
  4. The complete blood cell count (CBC) may show a white blood cell picture indicative of an infective or inflammatory response.
  5. Results of an abdominocentesis are non-diagnostic. There may be an increase in protein content of the abdominal fluid, but cellular elements are usually normal.

- Differential diagnosis. Rule out small intestinal obstructions of surgical nature (e.g., strangulating obstructions). Also, consider primary causes of enterocolitis (e.g., salmonellosis).

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5. Flatulent colic (tympany, bloating, wind colic)

a. Patient profile and history. Adult horses are affected with this type of colic under conditions of recent travel or management changes. Horses might include any of the following:
   1. Overeating on high-fiber, fermentable feed.
   2. Cold water engagement.
   3. Feeding poor-quality or overheated hay.
   4. Moldy hay or grain feed.
   5. Behavioral abnormalities (e.g., cribbing, greedy eating).
   6. Medication administration, such as atropine or broad spectrum antibiotics.

b. Clinical findings.
   1. Animals appear in distress and TPR is elevated out of proportion to other clinical signs.
   2. Abdominal distention may be evident, but gas is contained in the colon or cecum. Cecal tympany sounds filling the right paralumbar fossa, whereas hollow properties.
   3. Gastric distention is not evident; externally. Simultaneous auscultation and percussion may reveal the location of the distended viscus.
   4. Pain and signs of colic accompany the visceral distention. Often, the distended gut is palpable per rectum (tender to palpation) in the stomach. Passage of a nasogastric tube often relieves any gastric distention.
   5. Etiology and pathogenesis
      1. Flatus or tympanic colic results from excessive gas accumulation in the intestinal tract. The overdistention of the visera stimulates pain receptors, causing mild to severe colics. The severe forms mimic surgical colics.
      2. Gaseous distention usually is caused by increased fermentation and gas production, air accumulation (e.g., as with croupous, or obstructed gastrointestinal tract motility, causing gas buildup.
      3. Gas may accumulate anywhere along the gastrointestinal tract, resulting in some variation in the clinical findings.

- Diagnostic plan. This condition is diagnosed on clinical findings. It is important to make the distinction of the degree of pain relative to other findings that are present (e.g., shock). This helpful differentiates the condition as a medical colic.

- Laboratory tests. Laboratory findings are normal on hematology and abdominocentesis.

- Therapeutic plan
   1. Employ nasogastric intubation to relieve stomach distention if this is the source of the colic. Treat with sedatives and analgesics (e.g., xylazine, butorphanol, pentazocine, detomidine). Administer mineral oil for its anti-fermentative properties.
   2. If medical therapy is ineffective, trocarization of the cecum or colon may provide relief. This is a relatively radical therapy but may provide temporary relief until surgical intervention can work to alleviate the problem. Trocarization must be carried out in an aseptic manner.
      1. Determine the site of trocarization by simultaneous auscultation and percussion.
      2. Block the site with local anesthetic, then use a number 15 scalpel blade to pierce the skin.
      3. A 14-cm trocar is used to penetrate the abdominal wall and tympanum of the bowel. Hold the trocar in place until gas is no longer free flowing.
II. DIARRHEA

The large intestine of the horse has tremendous water absorptive capabilities. Diarrhea (acute or chronic) results from large intestinal pathology or pathological changes to the small intestine that cause an overwhelming amount of fluid and ingesta to be presented to the large intestine.

A. Acute diarrhea in adult horses

1. Salmonellosis

a. Patient profile and history. Salmonellosis is usually a sporadic disease in single animals unless in a referral center setting or in a barn or stall with frequent animal movement on and off premises. Salmonellosis infection may vary with forms of the acute diarrheaic form is most often seen in weanlings and young performance horses that are stressed following transport, shows, or surgery.

b. Clinical findings. Clinical salmonellosis has a spectrum of clinical expression. The enteric form in the adult may be asymptomatic, mild, acute severe, or chronic.

   (1) Mild infections are associated with fever, anorexia, depression, and the production of poorly formed feces (cow-pie).

   (2) With acute severe infections, fever and depression is seen during the first 24-48 hours. Simultaneously with this is mild to severe abdominal pain. At this time, the condition can be confounded with a surgical colic. Diarrhea begins sometime after the initial signs but may take 2-4 days to develop. Diarrhea is projectile, foul smelling, and persistent. Expression of diarrhea often is accompanied by improvement in the other clinical signs. Horses usually continue to eat, but in the case of anorexic animals, the prognosis for survival is poor.

   (3) The diarrhea may persist for 3-4 weeks, at which time horses will have experienced significant weight loss. Ventral edema caused by hypoproteinemia also may be a finding. Laminitis is a frequent sequela to salmonellosis.

   (4) A peracute form of enteric salmonellosis may occur; affected horses die within 6-12 hours.

c. Etiology and pathogenesis

   (1) Etiology. Salmonella typhimurium is the isolate most commonly associated with equine diarrhea (60% of cases). The organism adheres to and invades the mucosa of the intestine. The development of enteritis is then dependent on factors such as the age of host, immune status, other stressors, and virulence of the organism strain.

   (2) Pathogenesis

      (a) Diarrhea and enteritis result from the effects of the bacteria and host inflammatory mediators (prostaglandins). There is an increased secretion of chloride, sodium, and water into the intestinal lumen via an increase in mucosal cell cyclic adenosine monophosphate (cAMP) content.

      (b) The characteristic fever and leukopenia are caused by the release of lipopolysaccharide endotoxin from the bacterial cell wall. White blood cells pool at the site of the infection, and protein leakage occurs across permeable intestinal vessel walls.

d. Diagnostic plan. The diagnosis is based on clinical findings supported by laboratory confirmation.

e. Laboratory tests

   (1) Hematologic findings are a neutropenia with a left shift and varying degrees of cellular morphologic changes (toxicity). The albumin fraction of the TSP is low, although the total protein may be elevated or normal due to dehydration; The PCV is elevated due to dehydration, and the horse will have a metabolic acidosis with electrolyte losses through the feces.

2. Equine monocytic ehrlichiosis (acute equine diarrhea syndrome, Potomac horse fever)

a. Patient profile and history

   (1) First described in the United States Northeast, this disease is now evident throughout North America and has been recorded in Europe. It is seasonal in occurrence, with summer being the most common time of incidence. Any age group of animal may be affected, but the disease peaks in adult animals at age 12 years. It is most often found in Thoroughbred horses on pasture. Females are more at risk than males, and it is usually sporadic with single horses on any given farm.

   (2) The owner may report a mild depression and anorexia followed by diarrhea.

b. Clinical findings

   (1) Cardinal signs are anorexia, fever (39.5°C), injected mucous membranes, and depression. A profuse, watery diarrhea commences 24-48 hours after the onset of fever and lasts up to 10 days in the majority of animals. Mild abdominal pain with decreased borborygmi is evident.

   (2) Laminitis may be a sequela in 25% of cases. Occasional horses may show injected mucous membranes, severe abdominal distention, and abdominal pain. Frequently, death ensues before diarrhea develops. Abortion may occur in pregnant mares.
c. Etiology and pathogenesis. *Eubacterium histicum* is the etiologic agent. The organism has a predilection for mononuclear cells and is hypothesized to be spread by an arthropod vector.

d. Diagnostic plan. Indirect fluorescent antibody (IFA) may be performed on serum collected at 1-2 week intervals. Serum should be separated promptly and submitted cool but not frozen because freezing lowers the antibody titre. A latex agglutination test has also been developed for diagnosis.

e. Therapeutic plan. Supportive care is essential; as with any acute enteritis. Tetracycline at 6.6 mg/kg administered intravenously once per day (if given 24 hours after the onset of fever) for 5 days results in a dramatic response. Diarrhea does not develop. Treatment with tetracyclines after the onset of diarrhea does not alter the course of the disease.

f. Prevention. Treatment is costly and often futile when full clinical signs develop. A vaccine, is now available that seems to protect approximately 75% of horses.

3. *Clostridium* X

a. Patient profile and history. This is a sporadic disease associated with a history of recent stress in adult horses.

b. Clinical findings. A short febrile period is followed by a normal to subnormal body temperature. There is marked tachypnea, hyperpnea, and depression. There is a rapidly developing, intense dehydration and occasional abdominal pain. The horse may die before diarrhea is evident.

c. Etiology and pathogenesis. The causative agent is believed to be *Clostridium perfringens* type A. Clinical signs result from an enterotoxemia.

d. Diagnostic plan. The condition usually is diagnosed post mortem.

e. Therapeutic plan. Intensive therapy with massive quantities of isotonic saline and added bicarbonate is required to combat the dehydration and metabolic acidosis. Supplemental potassium therapy also may be necessary. Plasma transfusions may be warranted if hypoproteinemia is present. Flunixin meglumine and heparin also may be employed. Antibacterial therapy may include penicillin – aminglycoside or trimethoprim – sulfadiazine combinations.

f. Prevention. Little can be recommended to the client to prevent or treat this highly fatal, sporadic disease.

4. Antibiotic-associated enteritis

a. Patient profile and history. There are anecdotal reports of enteritis in horses following antibiotic administration. Tetracycline is the antibiotic most often incriminated, but lincomycin, tylosin, and high doses of penicillin and erythromycin also have been associated with the disease. There also have been reports of diarrhea after the use of trimethoprim – sulfadiazine.

b. Clinical findings. The frequency of enteritis associated with most of these drugs is low enough that they continue to be used when indicated. Typical signs of acute enteritis develop. Signs may subside rapidly when the antibiotics are discontinued.

c. Etiology and pathogenesis. Antibiotics may upset the normal gut flora, allowing overgrowth by *nonpathogenic* or *pathogenic* bacteria. Pathogenic bacteria (e.g., salmonellae, *clostridia*), when established, may have rapidly fatal consequences. Occasionally, chronic diarrhea has been seen in association with *Salmonella* isolated from the feces.

d. Therapeutic plan. Discontinue the antibiotic use, and treat as other acute diarrheas.

5. Intestinal *Clostridium*

a. Patient profile and history. The disease affects horses that are most commonly over 1 year of age. The disease is sporadic and may be accompanied by the history of recent, severe stress. Although reported as a distinct condition, intestinal *Clostridiosis* may be similar or identical to *Clostridium X*.

b. Clinical findings. The disease is of peracute onset with profound depression, tachycardia, dehydration, and diarrhea, which is profuse and malodorous. Shock is evidenced by a rapid heart rate and cardiovascular compromise. Affected animals die within 24 hours of the onset of clinical signs.

c. Etiology and pathogenesis. *C. perfringens* type A is the etiologic agent.

d. Laboratory tests. *C. perfringens* counts may be performed on the feces. Laboratory findings are consistent with dehydration and circulatory collapse.

e. Therapeutic plan. Massive fluid therapy is essential for any hope of success. Antibiotics are of little value, but penicillins may be employed as a logical choice for antibacterial therapy.

6. Gastrointestinal ulceration

a. Patient profile and history. Foals and young animals are extremely susceptible to NSAIDs. However, older horses occasionally are affected if the manufacturer's recommended dosages are grossly exceeded.

b. Clinical findings. Diarrhea is an occasional clinical finding, but more commonly the condition is associated with recurrent abdominal pain, anorexia, and weight loss. Oral ulceration with excessive salivation may be evident. Dependent edema may be a finding.

c. Etiology and pathogenesis. NSAIDs (e.g., phenylbutazone) produce toxic side effects if used in excess or in dehydrated horses. Organ systems most commonly affected include the gastrointestinal tract, kidneys, and hematopoietic system. Toxicity of the gastrointestinal tract results from depletion of protective prostaglandins (such as PGE). These prostaglandins normally decrease gastric acid secretion and increase the protective layer of gastric mucosa. The agents also may produce vasodilatation, resulting in devitalization and ulceration of mucosa along the entire intestinal tract. Oral ulceration is caused by the local irritative effect of the drug.

d. Diagnostic plan. Diagnosis is based most often on clinical findings and a history of long-standing or excessive use of phenylbutazone. The diagnosis may be supported by endoscopy of the stomach or double-contrast gastric radiography in foals and ponies. These techniques reveal ulceration of the glandular portion of the stomach.

e. Laboratory tests. Laboratory tests may be helpful by revealing a hypoproteinemia and hypoalbuminemia from protein leakage across a reduced and devitalized gastrointestinal mucosa. Occult blood may be found in the feces, accompanied by a lowered hematocrit.

f. Therapeutic plan

(1) Discontinue all NSAIDs, and administer 1-2 g/100 kg of sulfacetaflate orally four times daily and 6 mg/kg cimetidine orally, intravenously, or intramuscularly 2-3 times per day. Ranitidine may be substituted for cimetidine at 1-3 mg/kg orally twice daily or at 65 mg/kg intravenously twice daily. Plasma transaminases may be warranted in cases of severe hypoproteinemia.

(2) Intravenous feeding or nasogastric intubation and alimentation may be necessary.

g. Prevention. Phenylbutazone should be used with caution in ponies, younger horses, and dehydrated animals. Foals that are heavily parasitized or malnourished are extremely prone to toxic side effects.

7. Fungal enteritis

a. Patient profile and history. Fungal enteritides are sporadic in occurrence.

b. Clinical findings. The condition is indistinguishable from other acute diarrheas. Cases usually present with severe toxemia, profound dehydration, and severe, profuse, watery diarrhea.

c. Etiology and pathogenesis. Fungal overgrowth of the gastrointestinal tract and lungs may occur due to immunocompromise or secondary to excessive antibiotic use.

d. Diagnostic plan. There is little help for diagnosis. Fecal fungal elements occasionally may be found.

e. Therapeutic plan. There is no known treatment.
a. Patient profile and history. Foals often develop diarrhea between 6 and 14 days of age. This may correspond with the dam's first postpartum estrus.

b. Clinical findings. The foal presents with soft to watery feces, but all other signs are usually within normal limits. There may be mild dehydration, but foals are generally alert with normal appetites. The condition is most often self-limiting in 2-3 days but may precede other diarrheas in the same foal.

c. Etiology and pathogenesis. The etiology of foal heat diarrhea is unknown but may be associated with a changeover of cell type as the intestinal mucosa of the neonate matures. Other postulated but less likely causes include hormonal or nutritional alterations in the mare's milk, coprophagia, Strongyloides westeri infestation, and alterations in intestinal microbiological flora.

d. Diagnostic plan. The diagnosis usually is based on clinical findings without the need for laboratory support.

e. Therapeutic plan. Therapy is dictated by the severity of the diarrhea. Uncomplicated cases may be treated with simple attention to nursing care, such as washing the perineum and applying petroleum jelly. If diarrhea persists beyond 3 days, treatment with 1-2 mg/kg bismuth subsalicylate four times per day orally and oral fluid replacement with commercial calcium formulations should be considered.

2. Nutritional diarrheas

a. Clinical findings. Diarrhea may range from soft feces to very watery stool. Other clinical findings are usually normal, and the foal may have a normal appetite and appetite.

b. Etiology and pathogenesis. Diarrhea may develop secondarily to the following situations:

(1) Ingestion of excessive amounts of milk. This may occur with foals that are greedy eaters or when the mare and foal are reunited after a period of separation. Normally, a milk clot forms in the stomach within minutes of ingestion, and the whey advances to the small intestine in gradual amounts as the clot contracts. Overingestion can result in excessive amounts of whey entering the duodenum, overwhelming absorptive capabilities and creating an osmotic drive towards fluid accumulation in the gut.

(2) Abnormal nursing. Foals that ingest milk too rapidly or are fed by nasogastric tube experience decreased salivary secretion, which adversely affects milk digestion and clot digestion.

(3) Sudden dietary changes

(4) Ingestion of fibrous material. Grain, forage, mare’s feces, or other fibrous material require digestion in the immature large intestine of the foal. This promotes indigestion and diarrhea.

(5) Carbohydrate intolerance. Young foals may have primary or secondary carbohydrate intolerance. Primary milk intolerance is relatively rare. Secondary carbohydrate intolerance results from an enteric infection, which causes an increase in mucosal cell turnover. More immature cells make up the absorptive cell component of the gut mucosa, decreasing the disaccharidase and absorptive activities in the mucosal brush border.

c. Diagnostic plan. The diagnosis is most often based on clinical findings and history. Diarrhea is usually self-limiting but may be unresponsive in the case of primary carbohydrate intolerance.

d. Laboratory tests. Laboratory findings are unrewarding. In the case of an unresponsive milk intolerance, a lactose tolerance test may be performed. Lactose is administered per os, and corresponding blood glucose levels are determined.

e. Therapeutic plan

(1) Nursing care may be the only therapy necessary in the case of short-lived diarrheas. Lactose intolerance presents a special case, in that continued exposure to a milk diet exacerbates the problem.

(2) Elimination of milk and dietary replacement with hand feeding of a commercially soy-based milk supplement may be necessary until the foal can be weaned back on whole milk. A commercial calf diarrhea oral replacement solution may aid in the recovery.

(3) A commercially available lactase enzyme preparation may be added to milk before feeding to partially digest lactose into its constituent monosaccharides.

3. Antibiotic-related diarrheas

a. Patient profile and history. Oral antibiotics, such as aminoglycosides, may kill normal gut flora and predispose foals to diarrhea. Systemic antibiotics with an enteric-binding pattern (oxytetracycline, lincomycin, erythromycin) also have been shown to induce diarrhea.

b. Clinical findings. Diarrhea may range from soft feces to very watery stool. The foal's appearance may range from systemically normal to significantly dehydrated with circulatory collapse.

c. Diagnostic plan. The diagnosis is based on clinical signs, history, and response to therapy.

d. Therapeutic plan. Stop antibiotic therapy. Oral administration of a slurry of fresh feces from an older horse may be beneficial in restoration of gut flora but is not proven for efficacy.

4. Mechanical irritation

a. Patient profile and history. Young or older foals may be affected by consuming inordinate amounts of sand or dirt. Sand has an abrasive effect on the intestinal mucosa, resulting in enteritis and diarrhea. Also, physical impaction may result from the accumulation of sediment.

b. Diagnostic plan. Demonstration of sand in the feces aids in the diagnosis. Sand can be seen by mixing feces with water in a rectal sleeve, then identifying and quantifying the gritty sediment.

c. Therapeutic plan. Repeated therapy is necessary. Mineral oil may be used if an impaction is suspected. However, for elimination of sand over time, an agent producing fecal bulk is preferable. Bran, in the case of older foals, or psyllium hydrophylia mucilloid is recommended at 0.5 kg/454 kg four times per day orally for 5-7 days or weeks.

d. Prevention. Foals or horses that actively eat soil are difficult to manage. Feeding elevated mangers and ensuring adequate pasture cover may be effective in preventing further cases.

5. Diarrhea caused by Strongyloides westeri

a. Patient profile and history

(1) Intestinal infestations occur when the foal ingests infective larvae in the dam's milk. The greatest number of larvae are shed 2-3 weeks postpartum. The prepatent period is 8-14 days.

(2) It is speculated that diarrhea is associated with larval burdens. The larvae may cause enteric mucosal damage and suppression of disaccharidase or polypeptidase production. However, foals with high fecal egg counts may have no evidence of diarrhea. Conversely, diarrhea may occur in foals with very low fecal egg counts. Thus, causation is speculative.

b. Diagnostic plan. The diagnosis may be strengthened by the presence of S. westeri larvae in the mare's milk or the characteristic embryonated eggs in the foal's feces.

c. Therapeutic plan. Various anthelmintics are effective against the adult parasites, including ivermectin at 200 pg/kg, thiabendazole at 50 mg/kg, cambendazole at 20 mg/kg, and oxibendazole at 10 mg/kg. The daily administration of cambendazole at 50 mg/kg to postpartum mares eliminates infective larvae in the milk for the duration of therapy.

6. Bacterial enteric disease

a. Patient profile and history. The incidence of bacterial-induced diarrheas in foals is much lower than in other domestic species. Generally, if bacteria cause the diarrhea, there is a concomitant systemic disease. These systemic conditions (e.g., salmonellosis, Actinobacillus equuli infection) are covered in Chapter 18.

b. Etiology and pathogenesis

(1) Enteric colibacillosis caused by Escherichia coli has been documented but is
Chapter 8. Viral enteritis
d. Therapeutic plan. Treatment of bacterial enteric disease is as for neonatal calves.

c. Diagnostic plan. The virus is shed in greatest quantity early in the infection. Diagnosis depends on the demonstration of virus in the feces through electronmicroscopy, complement-fixation, or latex agglutination.

e. Laboratory tests. Fecal flotation, or immunofluorescence.

b. Clinical findings. The clinical findings with intestinal parasite infestation include diarrhea, poor weight gain, unthrifty appearance, colic, depression, inappetence, and occasional elevations in body temperature.

c. Etiology and pathogenesis

3. Cryptosporidiosis
a. Patient profile and history. Cryptosporidiosis is ingested as a sporulated oocyst and matures through six major developmental stages. Some maturation events occur within the cells of the distal small intestine, cecum, and colon. Villous atrophy with malabsorption and diarrhea result.

b. Clinical findings. The disease occurs in foals less often than in calves but has been documented as a cause of diarrhea in foals 5 days to 6 weeks of age.

c. Laboratory tests. Fecal oocysts can be detected by the staining of fecal smears, fecal flotation, or immunofluorescence.

d. Therapeutic plan. There is no known treatment for cryptosporidiosis, and animals are cared for symptomatically, with oral or intravenous fluids if necessary.

e. Prevention. The transmission of infective oocysts is through a fecal-oral route. Therefore, hygiene and management changes may be warranted if a number of foals are affected. The disease is a zoosis and is of particular concern in immunocompromised people. Proper personal hygiene is imperative when handling infective cases.

7. Viral enteritis
a. Patient profile and history. Rotavirus is a definitive cause of diarrhea in foals. Clinical signs occur in foals under 3 months of age. Diarrhea can occur in individual animals or in farm outbreaks. Rotavirus in foals produces a profuse watery diarrhea, and foals may become dehydrated, depressed, and anorexic. The diarrhea may last for days or weeks.

b. Diagnosis and recommendations for therapy and control are the same as for the

c. Veterinary practices.

a. Patient profile and history. Salmonella species, most commonly S. typhimurium, produce an acute diarrhea or septicaemia in any age horse.

b. Diagnosis and recommendations for therapy and control are the same as for the

c. Veterinary practices.

1. Parasite burdens
a. Patient profile and history. Intestinal parasites produce diarrhea and other clinical signs relative to the parasite. Intestinal parasites are ubiquitous and, under conditions of poor management techniques, produce serious problems.

b. Clinical findings. The clinical findings with intestinal parasite infestation include diarrhea, poor weight gain, unthrifty appearance, colic, depression, inappetence, and occasional elevations in body temperature.

c. Etiology and pathogenesis

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Chapter 2

III. WEIGHT LOSS

A. Granulomatous enteritis (lymphocytic-plasmacytic enteritis)
   1. Patient profile and history. This condition is relatively uncommon, sporadic in occurrence, and found in adult horses. There is no demonstrable breed or sex predilection.
   2. Clinical findings. Diarrhea is infrequent, but thinness of the animal is evident. The horse may have ventral edema. Rectal examination may reveal a roughened, friable rectal mucosa, thick-walled intestines, and prominent mesenteric lymph nodes or abdominal masses.
   3. Etiology and pathogenesis
      a. This condition appears to be an immunologic phenomenon that is poorly understood. It may present as a spectrum of inflammatory bowel disease in the horse.
      b. The bowel disease results in a malabsorption syndrome.
      c. Agents postulated to be involved in this immunemediated condition include dietary constituents, cell wall components, infectious agents (e.g., mycobacteria), and internal parasites (e.g., Strongylus vulgaris larvae, cyathostomes).
      d. Granulomatous changes are most evident in the mucosa of the small intestine. Histological examination reveals infiltration of the lamina propria by mononuclear cells. Absorptive capacities are compromised and protein leakage occurs into the lumen.
   4. Diagnostic plan. The diagnosis is based on history, clinical findings, laboratory data, and the unresponsiveness of the condition.
   5. Laboratory tests. Neoplastic lymphocytes may be found in a peripheral blood smear,_A bone marrow aspirate or abdominocentesis may show neoplastic lymphocytes. There will be hypoproteinemia and hypoalbuminemia. Liver enzymes may be elevated with hepatic involvement.
   6. Therapeutic plan. There is no treatment for this condition.

B. Gastric ulceration gastritis (see III A 6)

C. Parasitism

1. Patient profile and history. Parasitism is one of the most common causes of weight loss and chronic diarrhea in horses. It should not be overlooked, particularly in younger animals. Parasitism is most clinically significant in the young, weak, or stressed animal. Management factors, such as overcrowding, inadequate nutrition, and neglecting pasture rotation, or parasite prophylaxis all greatly impact the internal parasite burden. It is most commonly a disease of populations of horses. Poor thrift and recurrent abdominal pain (episodes of colic) may be historical findings in individual animals.
   2. Clinical findings. The animal may present as thin with a poor haircoat. Diarrhea of varying degrees is a common finding. If a rectal examination can be performed, the anterior mesenteric artery may feel roughened or exhibit fremitus. Inappetence, anemia, and a low-grade fever also may be present to varying degrees.
   3. Etiology and pathogenesis
      a. Large strongyles cause intestinal ischemia through the migration of larval forms within the walls of blood vessels supplying portions of the large intestine. Intestinal damage may also be caused directly, as larvae mature within the walls of the large intestine and cecum and emerge into the lumen. Ulceration and erosion of the cecum and colon also result from feeding of adult strongyles.
      b. Cyathostomes (small strongles) cause less damage, except under the specific conditions of simultaneous maturation of many hypobiotic larvae. In this case, significant intestinal damage occurs with resultant diarrhea and rapid weight loss.
   4. Diagnostic plan. The diagnosis is most often made on clinical signs and environmental and management history. Other horses on the premises often show evidence of harboring a parasite burden. For individual animals where verminous arteritis is considered, ultrasonography may be an aid to diagnosis.
   5. Laboratory tests
      a. Fecal egg counts are the best diagnostic but may be negative if clinical signs are caused primarily by larval forms of the parasite(s). Egg counts are also affected by host immunity, species of parasite, and history of treatment.
      b. Clinical pathology findings of some (e.g., eosinophilic) animals include eosinophilia, an increase in B-globulins, and an abdominalcoentesis consistent with chronic abscession (macrophages with ingested bacteria, eosinophilia, increased protein content, increased leukocytes).
   6. Therapeutic plan. There are many effective, broad spectrum anthelmintics that may be used for large strongyle or cyathostome infestation.
      a. The most common and efficacious treatments are:
         (1) Ivermectin paste—0.2 mg/kg
         (2) Oxibendazole—10 mg/kg
         (3) Benzimidazoles plus piperazine
      b. For migrating larval forms of the strongles producing verminous arteritis, the following treatments may be employed:
         (1) Ivermectin at 0.2 mg/kg and oxendazole at 10 mg/kg in single doses
Chapter 2

1. What is the most common cause of colic in newborn foals?
   (1) Meconium impaction
   (2) Atresia ani
   (3) Atresia coli
   (4) Ascarid impaction
   (5) Gastric ulceration

2. Which one of the following statements regarding distention colics (also known as spasmodic colics) is true?
   (1) They are accompanied by very high pulse rates.
   (2) They are not at all similar in presentation to obstruction colics.
   (3) They produce large quantities of gastric reflux.
   (4) They cause reflex intestinal atony
   (5) They are seen with greater frequency in horses that swallow air.

3. Which statement regarding proximal enteritis is true?
   (1) Causes signs of severe colic in affected foals.
   (2) May be seen in horses with a recent history of grain engorgement.
   (3) Is best treated by observation.
   (4) Causes sequestration of large amounts of fluid in the large intestine.
   (5) Is seen primarily in juvenile horses (yearlings).

4. In horses, the diarrheic form of salmonellosis and equine monocytic ehrlichiosis differ in what way?
   (1) Fever and depression is exhibited with equine monocytic ehrlichiosis but not with salmonellosis.
   (2) Fluid replacement therapy is not necessary with equine monocytic ehrlichiosis.
   (3) Laminitis is a frequent sequela to salmonellosis but not to equine monocytic ehrlichiosis.
   (4) Salmonella organisms invade the intestinal mucosa, whereas Ehrlichia risticii invade mononuclear cells.
   (5) Salmonella are readily isolated from the feces of horses with salmonellosis, whereas Ehrlichia risticii cannot be recovered.

5. Which statement regarding viral diarrheas in foals is true?
   (1) Do not produce changes to enteric cell morphology.
   (2) Can be prevented by vaccination.
   (3) Have been eradicated via infection by rotationally.
   (4) Are acute but very short-lived.
   (5) Are diagnosed by analysis of paired serum samples.
6. Abdominocentesis is often performed in horses with colic. Which one of the following statements is correct?

(1) Peritoneal fluid with normal cell counts and low protein levels will be retrieved in cases of early, small intestinal obstruction.

(2) The collection of peritoneal fluid contaminated with ingesta confirms the presence of a ruptured viscus.

(3) The retrieval of frank blood on abdominocentesis indicates gastric ulceration.

(4) Peritoneal fluid can be obtained from most normal horses but should have a low total white blood cell (WBC) count and high protein level.

(5) Malodorous peritoneal fluid confirms the presence of acute diffuse nonseptic peritonitis.

8. All of the following statements concerning proximal enteritis of horses are true EXCEPT:

(1) colic signs may be mild to moderate but the patient is often depressed and dehydrated.

(2) repeated or continuous gastric decompensation is therapeutic.

(3) it is a condition usually found in foals or weanlings.

(4) this condition is very similar in presentation to a strangulating small intestinal obstruction.

(5) laminitis may be a complication of proximal enteritis.

9. Which one of the following agents is NOT thought to cause acute diarrhea in adult horses?

(1) Enterotoxigenic Escherichia coli

(2) E. coli

(3) Clostridium perfringens type A

(4) Salmonella typhimurium

(5) Clostridium difficile

7. Which one of the following sets of clinical signs would be compatible with a medically manageable equine impaction colic?

(1) A pulse of 56 beats/min, a capillary refill time of 3 seconds, and a rectal temperature of 36.7°C

(2) A capillary refill time of 2 seconds, a respiratory rate of 16 breaths/min, and a negative retrieval of fluids on gastric intubation

(3) Peritoneal surfaces that feel granular on palpation, a respiratory rate of 36 breaths/min, and a negative retrieval of fluid on gastric intubation

(4) Increased borborygmi on abdominal auscultation, a capillary refill time of 5 seconds, and warm feet with an easily palpable digital pulse

(5) Anorexia, a rigid, splinted abdomen, and a pulse of 76 beats/min

10. All of the following statements comparing large intestinal impaction colics to flatulent colics in horses are true EXCEPT:

(1) both colic types may be feed related.

(2) both colic types may be relieved by treatment with mineral oil.

(3) clinical pathology findings are usually normal with both colic types.

(4) both colic types are seen more commonly in mature horses.

(5) both colic types are usually severely painful.

1. The answer is 1 [B 1 a]. Meconium impaction is much more common than the congenital atresias in foals. Ascariid impaction and gastric ulceration may occur relatively frequently but in older foals.

2. The answer is 5 [B 3 a]. Air swallowing results in pain due to gastric or small intestinal distension. This is not the only or most common cause of distention colic, but it will produce colic signs more frequently in horses with this vice. Distention colics do not result in major changes to vital signs or cause fluid reflux to accumulate in the stomach. Distention of intestinal segments usually result in a reflex hyperperistalsis in adjacent portions of the bowel. Distention colics may appear very similar to early obstruction colics or other more serious colics in early stages.

3. The answer is 2 [B 4 a]. Grain engorgement or heavy grain feeding in adult horses is associated with the development of proximal enteritis. Colic signs are minimal with this condition but depression is significant. Treatment must be aggressive and consist of gastric decompression, large volumes of intravenous fluids, analgesics, and antibiotics.

4. The answer is 4 [A 1, 2]. These organisms have predilection for different tissues even though resultant clinical signs may be similar. Both diseases cause horses to exhibit fever, depression, and often a subsequent laminitis. Both require aggressive fluid replacement therapy. Salmonella species often cannot be easily isolated from the feces of affected horses because of the dilution nature of the clinical diarrhea and the invasive nature of the organism.

5. The answer is 3 [B 7]. Rotavirus causes diarrhea in foals, whereas coronavirus has been demonstrated in the feces of diarrheic foals and adenovirus involved with the diarrhea seen in foals with combined immunodeficiency. Viral diarrheas may be short or protracted in duration and have not proven to be preventable through vaccination. Diagnosis is usually via examination of the feces.

6. The answer is 1 [A 21 (f) e]. Table 2-3; Table 2-4. Peritoneal fluid findings on abdominocentesis are most consistent with equine impaction colic, which is most commonly treated medically. The condition presents with normal vital signs and little reflux on nasogastric intubation. The impaction may be palpable per rectum. Generally, increased respiratory rates and capillary refill times are associated with surgical colic.

7. The answer is 1 [A 2, B 21. A pulse of 56 beats/min, a capillary refill time of 5 seconds, and a rectal temperature of 36.7°C would be the clinical findings most consistent with equine impaction colic, which is most commonly treated medically. The condition presents with normal vital signs and little reflux on nasogastric intubation. The impaction may be palpable per rectum. Generally, increased respiratory rates and capillary refill times are associated with surgical colic.

8. The answer is 3 [B 41. Proximal enteritis (anterior enteritis, duodenitis) is most common in adult horses, not foals or weanlings. The condition may be confused with a strangulating small intestinal obstruction. Continuous or periodic gastric decompression relieves the colicky signs and depression becomes the major finding. Dehydration is a reflection of gastric pooling of fluid and decreased fluid intake. Laminitis, a common complication of proximal enteritis, is believed to occur secondary to an endotoxemic state.

9. The answer is 1 [B 8 b (1)]. Enterotoxigenic Escherichia coli causes neonatal diarrhea in many species and has been isolated from the feces of foals with diarrhea; however, its clinical significance is unproven in foals and it has not been shown to be cause of acute diarrhea in adult horses. Escherichia risticii, Clostridium perfringens type A, Salmonella typhimurium, and Clostridium difficile have been associated with diarrhea in adult horses.
on succulent green feeds. Mineral oil can be beneficial in the treatment of both types of colics (i.e., it may coat an impaction, allowing for easier passage and it may act as an antiflammable in the case of flatulent colics). Clinical pathology findings are usually normal for both conditions and both conditions are most common in adult horses.

Chapter 2
Diseases of the Bovine Gastrointestinal Tract
Timothy H. Ogilvie

1. Simple indigestion
   a. Patient profile and history
      (1) Simple indigestion is a common disease in dairy cattle and less common in feedlot cattle and other ruminants. The condition is sporadic, usually affecting individual cows, but groups can be affected. This type of indigestion occurs more frequently in older cows, greedy eaters, or cows in advanced pregnancy.
      (2) There may be reported changes in the feeding program (i.e., quality, quantity, frequency) or other management changes. The owner might report that the cow is off feed and down in milk production. Feed refusal may have been progressive in that grain may have been refused first, followed by silage and hay.
   b. Clinical findings. The cow may be partially to completely anorexic. Vital signs [temperature, pulse, and respiration (TPR)] are normal to slightly elevated. The animal has a normal to mildly depressed attitude. Rumen motility may be normal but usually is slightly decreased in frequency and vigor. Feces may be normal in consistency or firm, and fecal output usually is reduced. Occasionally, the rumen is hypermotile, resulting in feces that are looser than normal.
   c. Etiology and pathogenesis
      (1) The condition is caused by a change in rumen fermentation resulting from a shift in feed quality, quantity, or presentation. Some predisposing factors in the development of the condition include:
         (a) Sudden changes of feed
         (b) Poor feed quality (e.g., moldy, spoiled)
         (c) Animal fatigue or stress (e.g., shipping)
         (d) Prolonged antibiotic therapy
         (e) Insufficient water
      (2) Any of the predisposing factors might change the ruminal environment necessary for fermentation and microbial degradation of feedstuffs. The rumen environment is composed of a mixture of proteins, carbohydrates, and fluid. Bacteria and protozoa act on these substances within an environment with a pH and temperature that is regulated by secretion and motility.
   d. Diagnostic plan. The history, lack of specific findings other than minor gastrointestinal changes, and a knowledge of the farm husbandry usually is sufficient to make the diagnosis. It is often a diagnosis by exclusion of other diseases, and the animal’s recovery within 24–36 hours confirms the diagnosis.
   e. Laboratory tests. Laboratory tests ordinarily are not requested because all values usually appear normal. Occasionally, the cow may exhibit a "stress" leukogram. The rumen pH may be slightly alkaline (6–7) and show somewhat decreased protozoal activity. A mild metabolic alkalosis also may be present.
   f. Differential diagnoses. Other conditions to be considered would have clinical signs in addition to mild indigestion. These conditions include:
      (1) Traumatic reticuloperitonitis
      (2) Abomasal displacements
      (3) Vagal indigestion
      (4) Primary ketosis
      (5) Lactic acidosis
   g. Therapeutic plan