10. The answer is 5 | 18. 51. Large intestinal impactions are only mildly to moderately painful, whereas flatulent colics are very painful and may appear to be surgical in nature. Both colic types may be feed-related—an impaction of the large intestine may be related to improper digestion of feedstuffs, whereas a flatulent colic is often associated with grazing 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 antiflatulent 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 3
Diseases of the Bovine Gastrointestinal Tract
Timothy H. Ogilvie

15. BOVINE FORESTOMACH AND ABOMASUM

A. Indigestion. The primary clinical signs for this condition are anorexia and ruminal changes characterized by hypomotility or occasionally hypermotility.

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 rumen 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
Lactic acidosis (ruminal acidosis, acute grain overload, acute rumen impaction, rumen overload, α-lactic acidosis, grain engorgement, toxic indigestion, acid indigestion)

2. Patient profile and history

(a) Any rumen is susceptible to lactic acidosis. Dairy and beef cattle seem to be most commonly affected possibly because of their representative numbers or husbandry and intensive production practices.

(b) Patient history includes access to highly fermentable feed. The offending substrate (vinegar 1–2 L orally) if the rumen pH is more than 7.

(c) Prevention. For prevention of this condition, the client should be instructed to minimize sudden dietary changes and avoid damaged, spoiled, or contaminated feed.

3. Clinical findings are variable and depend on the amount of feed consumed, feed composition, feed particle size, and previous adaptation of the animal to the ration. Clinical syndromes may vary from acute and severe to mild and similar to simple indigestion. A chronic form of lactic acidosis may also occur.

(a) Acute severe cases

(i) Clinical signs appear 12–36 hours after the feed is consumed. Symptoms include anorexia, lethargy, depression, muscle tremors, and ataxia.

(ii) The animal is dehydrated (8%–12%), resulting in loss of skin turgor and a dull, sunken eye. There is severe ruminal distention, rumen stasis, and fluid splashy rumen contents. The animal may exhibit bruxism and grunting.

(iii) Body temperature initially increases and then falls. There is tachycardia and a rapid, shallow respiratory pattern. Dairy cattle have a severe drop in milk production.

(iv) The feces may contain undigested feed material (e.g., grain).

(v) The animal continues to deteriorate, becoming recumbent, comatose, and finally dying.

(b) Subacute cases. The pattern of clinical findings is similar to the acute cases but less intense.

(c) Chronic cases

(i) Repeated episodes of subacute rumen acidosis may be associated with herd problems of subclinical disease (e.g., laminitis, low-fat milk syndrome, liver abscesses, chronic rumenitis, chronic indigestion).

(ii) Individual episodes of indigestion appear mild and similar to simple indigestion or subacute indigestion. Animals appear bright and alert with transient anorexia and decreased effective rumen motility. The rumen may be slightly distended. Feces are grey and porridge-like. Dairy cattle experience decreased milk production.

4. Etiological and pathogenesis

(a) Normally, there is a balance of cellulolytic and carbohydrate-using bacteria within the rumen. The ingestion of excess carbohydrates or sugars promotes growth of lactic acid-producing and -using bacteria within the rumen. Although lactate is used rapidly, the amount that accumulates plus the rapid fermentation of carbohydrates and the accumulation of volatile fatty acids (VFAs) drop the rumen pH. This decrease in pH kills rumen protozoa and microbes, including the initial lactate users. These organisms are replaced first by Streptococcus bovis and then by lactobacilli and gram-positive rods. The pH continues to fall with the further lactic acid production of D- and l-lactic acid.

(b) Finely ground feeds increase the surface area exposed to bacteria for fermentation. These feeds also decrease the amount of saliva secreted by the animal, which lessens the amount of buffer flowing into the rumen. Although both D- and l-lactic acid are absorbed through the rumen wall, only l-lactic acid can be metabolized by the rumen, leaving the D-lactic acid to be eliminated. Therefore, d-lactic acid builds up and creates the systemic acidosis.

(c) Feed fermentation, decomposition of the feed into very fine particles, and the lactic acid all increase the rumen osmolality. The accumulation of VFAs produces extracellular water flows into the rumen, producing rumen distension, diarrhea, and dehydration. These conditions lead to hypovolemia, circulatory collapse, metabolic acidosis, and death.

(d) If the animal survives the initial bout of lactic acidosis, the high rumen acidity, hypertonicity, and corrosive nature of lactic acid produce a chemical rumenitis. This condition allows rumen-associated bacteria and fungi to invade the rumen wall and hepatic portal system.

(e) Sequelae include rumen wall necrosis, hepatic abscesses, and peritonitis. The release of toxins may produce laminitis, abdominal disorders, and cardiac, renal, and hepatic damage.

(f) Laboratory aids. The diagnosis often is determined based on clinical findings supplemented by a history of overeating or sudden dietary change. A sample of rumen fluid should be retrieved and analyzed.}

(c) Laboratory aids

1. Support laboratory is rarely necessary in acute cases where there is a reliable history. The most useful laboratory aid is an evaluation of rumen content for subjective findings, pH, and protozoal activity.

2. The rumen fluid, retrieved by orogastric intubation is milky grey and watery with an acid smell. The pH is variable, depending on time and diet, but it is diagnostic if less than 5.0. A wet mount shows no live protozoa. A Cram stain shows Streptococci with a predominant population of gram-positive rods and other mixed, mainly gram-negative, morphologic forms.

3. Hematologic work-up. A leukogram reveals a degenerative left shift. Hemocencentration is evident on a packed cell volume (PCV). A chemistry panel shows an increase in blood urea nitrogen (BUN) and creatinine, hypocalcemia, hyperglycemia, and hyperlipemia.}

4. Urinalysis shows decreased volume, increased specific gravity, acidity, and glucosuria (often a diagnostic indicator in sheep). If available, a blood gas analysis confirms a metabolic acidosis.

f. Differential diagnosis. Lactic acidosis may appear similar to many other septic or toxic conditions including:

1. Septic mastitis or metritis
2. Acute diffuse peritonitis
3. Parturient paresis

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(4) Poisoning by lead, salt, arsenic, or nitrate
(5) Enzootic enterotoxemia

Therapeutic plan. It is often difficult to determine therapeutic intensity with acute lactic acidosis, particularly early on when the full range of clinical signs has not been manifested. Some guidelines for treatment follow.

There has been access to carbohydrates but the animal(s) has not shown clinical signs.
(a) Prevent further access to feed, and offer free choice, good quality hay.
(b) Exercise the animal(s) hourly for 12-24 hours to encourage movement of ingesta through the digestive tract.
(c) Administer 1 g/kg of magnesium oxide, magnesium hydroxide, or sodium bicarbonate orally.

The animal(s) show anorexia and depression within 6-8 hours of feed consumption.
(a) Surgery. Perform a rumenotomy if the value of the animal warrants the procedure and if the case is still a good surgical risk. Tachycardia (greater than 140 beats/minute), dehydration, a subnormal body temperature, and severe depression indicate a poor prognosis for recovery. Couple surgery with fluid therapy for metabolic acidosis (e.g., Ringer’s Lactate with sodium bicarbonate), and supply a rumen transfaunaition. Other supportive therapies include calcium, thiamine, and nonsteroidal anti-inflammatory drugs (NSAIDs).
(b) Recommend that the animal be slaughtered if economics do not dictate surgery and the animal will pass an anemotest and post-slaughter inspection.
(c) Other treatments. Other less predictable or efficacious treatments include rumen lavage via rumen trocar, systemic or oral antibiotics to limit ruminal bacterial growth, and oral iodine-based disinfectants to kill rumen microbes. Because of the potential for a large number of animals to develop lactic acidosis simultaneously, the use of a large-diameter stomach tube (Kingman tube) to lavage the rumen may be both economic and beneficial.

Animals with chronic lactic acidosis. Individual animal treatment is often unsuccessful and the animal(s) is frequently euthanized. This is most commonly reflective of a herd problem and should be addressed in a preventive manner.

Prevention
(1) Management strategies. Recommendations should revolve around ration and feeding management. Keep feed quality and practices consistent. Avoid abrupt changes and gradually adapt the rumen to concentrates. When introducing new concentrates or carbohydrate forms, prefeed animals with hay. Prevent accidental access to feeds by maintaining good animal holding facilities.
(2) Feed content. Maintain a minimum crude fiber content of 14% of total digestible nutrients (TDN) for fattening cattle and 18%-22% for dairy cattle.

Vagal indigestion (chronic or vaga indigestion, Hoflund’s syndrome). Vagal indigestion refers to a group of conditions that cause forestomach outflow problems. One possible cause of these problems is vagal nerve dysfunction, although this dysfunction has not been proven to be necessary or sufficient to cause the condition. The classification of forestomach disturbances encompassed by vagal indigestion differs from acute lactic acidosis, although this has not been proven to be necessary or sufficient to cause the condition. The classification subtypes are necessary for diagnosis, therapy, and prognosis.

Patient profile and history. Although any ruminant may be affected, this type of indigestion is most common in adult dairy cattle. History may include mild but repeated bouts of transient indigestion with signs of anorexia, decreased milk production, mild bloat, weight loss, abdominal distention, and decreased amounts of manure. An episode of traumatic reticuloperitonitis (TRP) may be reported as an historical event.

Clinical findings. A distended abdomen is often a cardinal sign. The distention may be bilateral with gas distending the upper left flank, while fluid distends the ventral right quadrant. The rumen is often hypermotile with frequent but weak contractions. The animal is often in poor condition. Vital signs are usually normal but occasionally, a Bradycardia is evident (40-60 bpm).

Etiology and pathogenesis
(1) Vagal indigestion is most often described as a sequela to traumatic reticulo-peritonitis, although the syndrome is also associated with tumors (e.g., lymphosarcoma of the abomasum) and abomasal torsion.
(2) If adhesive lesions around the reticulum either interfere with vagal nerve function or the mechanics of reticular movement, then reticular muscular activity and excitation waves are compromised, resulting in ruminal gas retention or free gas bloat. In this case, there are no other abnormalities, and the animal will improve transiently if the gas is removed via orogastric tube and removed fully if a rumen fistula is installed.
(3) If failure of omasal transport occurs, ingesta from the ruminoreticulum is not properly conveyed to the abomasum, and long (2–4 cm) fiber may be found in the manure. Fluid builds up in the rumen, and although acid-base status remains normal, the animal becomes mildly dehydrated. The prognosis for a return to function is guarded. The lesion causing the condition is either a functional or physical obstruction of omasal outflow. Examples of physical obstructions include ingested foreign bodies (baling twine, plastic bags, pellets) or space-occupying lesions (tumor).
(4) Abomasal impaction may be another manifestation of the syndrome and results from obstruction at the pylorus with conditions such as lymphosarcoma. This impaction may also occur secondary to abomasal torsions, where it is postulated that abomasal stretching and visceral compromise impairs gastric motility. The abomasum becomes distended with hard ingesta. A hypochloremic, metabolic alkalosis results due to reflux and pooling of gastric chloride ion in the rumen. This “chloride trap” makes it unavailable for reabsorption in the proximal small intestine.
(5) Primary abomasal impaction may occur in beef cattle on a coarse roughage diet with limited access to water and in calves suffering from abomasal tricho- bezoars or phytobezoars. These conditions are not usually considered as part of the vagal indigestion syndrome.
(6) The final manifestation of the syndrome is chronic indigestion associated with advanced pregnancy. The gravid uterus may occupy enough abdominal space that it interferes with forestomach outflow. It may also exacerbate reticular adhesions or abomasal outflow restrictions, resulting in ruminal distention. The prognosis with this condition is often fair to good following parturition.

Diagnostic plan
(1) The set of clinical signs, chronicity, and continued lack of response to any treatment for simple indigestion is usually sufficient to diagnose vagal indigestion. The presence of a metabolic alkalosis often implicates the abomasum in the syndrome but this is not invariably.
(2) Differentiation of the causes may depend on findings on exploratory laparotomy; however, mixed conditions do occur and lesions are not invariably present. The poor response to interventional therapy with many of these cases often makes a morphologic diagnosis unnecessary.

Differential diagnoses. It is often necessary to rule out simple indigestion and many other causes of decreased fecal output and abdominal distention (e.g., abomasal impaction, hydrops). Vagal indigestion is often a diagnosis of exclusion.

Therapeutic plan
(1) Surgical exploration is often used in valuable animals as an aid to diagnosis and therapy. In cases of free gas bloat, the creation of a rumen fistula often enables a return to normal function while the underlying lesion heals. Indwelling rumen trocars have been used to relieve the bloat but are less reliable than surgery. The prognosis for vagal indigestion involving omasal or abomasal dysfunction is relatively poor, so slaughter should be considered as an option in these cases.
(2) In cases where exploratory surgery has been performed, further surgical inter-vention depends on findings. Foreign bodies (in the abomasum or fore-
stomach) should be removed, abscesses adherent to the reticulum can be drained into the reticulum, and softened agents (e.g., mineral oil) can be instilled into the abomasum by an orogastric tube directed through the omasal-abomasal orifice intraruminally.

(3) Supportive therapies include:
(a) Rumen transfaunation (repeated treatments may be necessary)
(b) Supportive feeding either by rumen fistula or an indwelling nasogastric tube that has been placed into the abomasum at surgery
(c) Intravenous fluids
(e) Calcium salts intravenously or subcutaneously
(f) Potassium chloride at 30–60 g orally twice a day
(g) Exercise

Prevention. There is little that can be done to prevent vagal indigestion. However, if chronic reticular adhesions indicate that traumatic reticuloperitonitis may be occurring in a herd, it would be good to administer magnets to breeding-age heifers if chronic reticular adhesions indicate that traumatic reticuloperitonitis may be occurring should be implemented by decreasing access to indigestible substances and is most often seen in pregnant cows.

4. Abomasal impaction
a. Patient profile. Primary abomasal impaction occurs most commonly in beef cattle and is most often seen in pregnant cows.

b. Clinical findings. Animals present with anorexia, scant feces, variable dehydration, and free gas bloat.

g. Laboratory tests show metabolic alkalosis, hypochloremia, hypokalemia, and decreased arterial pH.

(3) Free gas bloat is associated with legume consumption.
(a) It is thought that the fine, leafy structure of certain varieties of legumes coupled with tender growth (early or late season) allows for more rapid bacterial degradation and intraruminal particle suspension. Gas produced is released from the legume leaves forming monomolecular foams that trap gas bubbles. These foams have great surface tension and are highly stable.

(b) The result is that small gas bubbles do not coalesce, the cardia or the fore-stomach cannot be cleared of this foam, and the animal is unable to eructate.

(c) A stable froth can also be formed in feedlot animals consuming a primarily finely ground grain diet. In this case, however, a mucoprotein slime stabilizes the foam. This foam is stable at a low pH created by lactate and VFA production. Salivation is decreased because of the fine grind of the diet, which also lessens intraruminal buffeting.

(2) Free gas bloat may have a variety of causes. The most common cause is intraesophageal obstruction with solid objects, such as apples or potatoes. Exoesophageal masses may also cause the build up of intraruminal gases. Absences caused by perivascular injections, Hypoderma lineatum reactions, and cervical neoplasia can all constrict the esophagus. Certain postures or diseases also can produce a functional free gas bloat. Examples include milk fever and tetanus. Moderate free gas bloat also may be a finding in vagal indigestion (see I A 3).

d. Differential diagnoses. If the diagnosis is not one of bloat, other causes for abdominal distention must be considered further define the type of frothy bloat; a feedlot bloat should have a pH of less than 5.5.

e. Differential diagnoses. If the diagnosis is not one of bloat, other causes for abdominal distention to rule out include acutes, acute diffuse peritonitis, and hydroptos.

f. Therapeutic plan
(1) Acute bloat is one of the true medical emergencies in bovine practice. Animals can die rapidly and many may succumb before the owner is aware of the problem, particularly if cattle have been turned out to new pasture for an unobserved length of time. In the case of free gas bloat, ileal ruminal pressures build up very rapidly.

(2) With frothy bloat, pass an orogastric tube and administer an oil to reduce the surface tension of the foam and allow the gas bubbles to coalesce. Either mineral oil at 1 L/100 kg orally or diocetyl sodium sulfosuccinate (DSS) in peanut oil at 17–66 mg/kg orally may be used (150–600 g on a 450 kg animal). The treatment should be satisfactory and sufficient if the animal is still standing and not showing evidence of respiratory or cardiac failure. If the animal is in a deteriorating clinical condition, an emergency rumenotomy is warranted.

(3) With acute free gas bloat due to an intraesophageal mass, the orogastric tube may force the obstruction into the reticulum. Should this not occur, turocularization of the rumen and either an indwelling trocar or fistula is placed to allow continued escape of gas while the mass softens and hopefully moves along. Post-surgical efforts can be used (22,000 IU/kg twice daily intramuscularly) should be used. Many masses block the esophagus just posterior to the pharynx. These can often be retrieved manually or with the aid of a wire loop to snare the object.
g. Prognosis: (1) The prognosis for most cases of frothy bloat is favorable if intervention is rapid. A simple indigestion may occur secondary to treatment, and the animal should be fed good quality hay as a major component of its diet for 1-2 days. Animals that have undergone emergency rumenostomies may develop cellulitis or peritonitis, but these occurrences are infrequent. (2) The prognosis for acute free gas bloat is excellent if the offending object can be removed. If the object must be left in place to be swallowed, then complications include sequelae to trocarization or rumen fistulation as well as secondary esophageal stricture development.

b. Prevention: (1) The prevention of frothy bloat may take many forms and several combinations including: (a) Feeding lucerne is fed, select cultivars of bloat-producing forages that are less likely to ferment quickly. (b) Feed dry roughage to animals before turning them out on legrum pastures. (c) Allow only 20 minutes initially of grazing on lush legrum pastures. (d) Feed antimicrobials (poloxalene, diclofenac, 1-2 g/kg 60 kg once daily; ionophore such as monensin at 1 mg/kg once daily or lasalocid at 1.32 mg/kg once daily in nonlactating cattle). (e) Provide for slower introduction or less heavy feeding of concentrates if feedlot bloat becomes a problem. (2) The prevention of free gas bloat due to esophageal choke becomes a matter of cutting up feed objects in smaller sizes, slowing down greedy eaters by feeding hay first upon initial introduction, or introduction of the material gradually.

2. Chronic free gas bloat (see I A 3)

C. Hyperresonance

1. Left displacement of the abomasum (abomasal displacement, twisted stomach)

a. Patient profile and history: (1) Left displacement of the abomasum (LDA) is most common in middle-aged, high producing dairy cows but may be seen in other situations and classes of cattle. This condition is rare in other ruminants but may occur in ruminating calves. It is a sporadic disease of individual animals, but the prevalence may be relatively high in some herds. (2) Cattle are often in early lactation on a high concentrate, low roughage diet. Total mixed rations with short fibre lengths and silage-based rations seem to predispose cattle to the condition. There is often a concurrent disease, such as mastitis, mastitis, or hypocalcemia.

b. Clinical findings: (1) Vital signs are normal to slightly elevated unless there is concurrent disease. (2) Appearance. The animal may have a slabb-sided appearance or the last two ribs on the left may be sprung with a hollow left paralumbar fossa. In some cases, there may be a slight filling of the left paralumbar fossa directly behind the last rib. (3) Feces. There are decreased amounts of pasty manure or small amounts of diarrheaea. (4) Rumen contractions are normal to slightly decreased. There may be tinkling sounds heard while auscultating the rumen. The animal exhibits total to moderate anorexia and may be ketotic. A "ping" is evident on simultaneous auscultation and percussion of the abdomen usually along a line drawn from the left iliac coxae to the left elbow. (5) On palpation of the left paralumbar fossa, the rumen is not palpable, but the dilated abomasum occasionally may be felt. Rectal examination may be relatively normal except for a smaller than normal, medially deviated rumen. (6) Calves with LDA exhibit quite an extensive left flank distention, with gas and fluid sounds on auscultation.

c. Etiology and pathogenesis: (1) The abomasum is firmly attached at its cranial end to the omasum, but the fundus and pylorus are relatively freely movable, being held only by the greater and lesser omentum. The abomasum continually generates gases (CO₂, methane, nitrogen) and secretes HCl and enzymes, which are passed into the duodenum by abomasal contractions (1-2/min). (2) High concentrate, low roughage diets are thought to cause increased VFA production in the rumen, leading to an increase in VFA accumulation in the abomasum, increased gas production in the rumen, and accumulation in the abomasum. This diet also may cause decreased stimulation to ruminination, decreased salivaion, and an increased rate of passage of ingesta, all resulting in decreased abomasal motility and increased gas production. (3) Other factors that are postulated to reduce abomasal tone or motility are hypocalcemia, concurrent diseases through inflammatory mediators (endoxin and interleukin-1), and lack of exercise. With reduced abomasal motility (atony) or increased gas accumulation, the abomasum distends and may rise dorsally out of place on the left or right side of the animal. (4) During late gestation, it is thought that the gravid uterus pushes the rumen cranially and dorsally, and the abomasum pushes to the left. After parturition, the abomasum moves further left into the void, creating the displacement. This may be more likely in deep-bodied, large cows; thus, a breed or genetic predisposition has been postulated. (5) Abomasal displacements in calves tend to occur at a time when the calf is changing from a monogastric to a ruminant. The pathogenesis is unknown and it is a rare event compared with cattle.

d. Diagnostic plan: It is a relatively straightforward diagnosis in animals that fit the subjective and clinical picture. The diagnosis becomes more suspect in nontraditional animals (calves) or when the ping is faint, recurring, or faint.

e. Laboratory tests: (1) Clinical pathology tests are nondiagnostic for the condition but can be helpful, particularly in cases when a ping is not readily auscultable. There may be mild hypocalcemia, hypoglycaemia, hypokalaemia, ketonemia, ketolactia, and ketonuria. (2) Liptak test. Because other distended viscera produce a ping and may mimic LDA, it is sometimes of value to aspirate fluid percutaneously from just below the most caudal location of the ping. This is known as the Liptak test. Analysis of the fluid retrieved aids in identifying the viscous. If the pH of the fluid is less than 5 and has no evidence of protozoa, it is most likely from the abomasum. If the fluid has a pH of more than 6, it is most likely from the rumen.

f. Differential diagnoses. If LDA is indeed the diagnosis but no ping is auscultable, the condition may be confused with ketosis or simple indigestion. Other conditions that may produce a left-sided abdominal ping and mimic LDA are rumen atony with a gas cap and pneumoperitoneum.

g. Therapeutic plan: (1) Medical (nonsurgical) management of the condition is unreliable and usually unsuccessful. However, steps may be taken to treat concurrent disease (e.g., mastitis).

2. Physical intervention: Rolling the cow to replace LDA may be undertaken as a temporary treatment in cases where economics do not favor surgery or in cases where the owner wishes to gain some time before salvage. The procedure involves casting the animal into right lateral recumbency, bringing her to dorsal recumbency, rocking her gently back and forth while kneading the ventral abdomen, rolling the cow to her left side, and allowing her to rise. This seems to reposition the abomasum, but a majority of cases recur.

3. Surgical intervention provides for a good to excellent prognosis for a return to economic potential. The following techniques have similar success rates, and choice of technique is a matter of preference or familiarity: (a) Left flank abomasotomy (b) Right flank omentotomy (c) Right paramedian abomasotomy.
(d) Right paramedian percutaneous toggle pin (bar suture) fixation

h. Prevention
(1) Management strategies. Many clients tend to live with the problem of LDA as a trade-off because they are satisfied with herd production levels, happy with their feeding management [e.g., total mixed ration (TMR), corn silage], or content with their herd genetics (i.e., large, high-producing cows). The best advice if a client wishes to decrease the incidence of the disease is to incorporate more long-stemmed fiber in the ration, provide exercise for stabled cows, feed animals more frequently with the ration divided, and limit postpartum diseases (e.g., milk fever, metritis).
(2) For the individual cow with LDA, most farmers are able to diagnose the condition themselves after seeing a few cases. However, these clients may need to be reminded that prompt correction of the condition returns the cow to a more economic production unit faster.

2. Right displacement of the abomasum (RDA)
   a. Patient profile and history (see I C 1 a)
   b. Clinical findings are similar to LDAs, but the ping is on the right side. In the case of a large distention of the abomasum, the displaced viscus may be felt per rectum.
   c. Etiology and pathogenesis (see I C 1 c)
   d. Diagnostic plan. The diagnosis is based on clinical findings. An aspirate of fluid from within the viscus (e.g., a Liptak test) may be beneficial for diagnosis.
   e. Differential diagnoses. Other conditions that produce a right-sided ping include:
   (1) Colonic gas
   (2) Gas in the rectum
   (3) Cecal dilation
   (4) Cecal torsion
   (5) Abomasal torsion
   (6) Pneumoperitoneum
   (7) Physiometra
   f. Therapeutic plan
   (1) Medical management may be attempted with smooth muscle stimulants, but surgery is the treatment of choice. Some sources think that surgery should not be delayed because the RDA might progress to an abomasal torsion/volvulus. Rollling the cow is contraindicated in RDA cases because an abomasal torsion/volvulus may result.
   (2) Surgery is either a right flank omentopexy or a right paramedian abomasopexy.

3. Right abomasal volvulus (right torsion of the abomasum, abomasal torsion)
   a. Patient profile and history. Subjective findings are similar th LDA and RDA, although in many cases, an abomasal volvulus may occur in animals other than the early postpartum cow. Sudden agalactia and anorexia are indications.
   b. Clinical findings
   (1) Appearance. The animal is markedly depressed and may show bruxism or grunting. There may be some evidence of pain or discomfort, such as treading, but this condition is not as painful as other intestinal accidents.
   (2) A high-pitched ping is evident over a large area on the right side from the eighth rib to the paralumbar fossa. Fluid may be succussed within the distended viscus. The cow will be dehydrated as evidenced by a skin tent and sunken T-cells. Tachycardia is evident (usually 100 bpm), and the pulse is weak.
   (3) There is complete rumen stasis possibly with a mild bloat on the left from a gas cap. Feces are scant. There may be only mucus in the rectum, and occasionally, with a concurrent abomasal ulcer, there may be melena.
   (4) Rectal examination. The volvulus often can be palpated rectally in the right abdomen. RDA can progress to a volvulus by either a clockwise or counterclockwise twist (when viewing the animal from the side). A torsion can occur around the long axis of the abomasum. Various combinations can be found. If twisted long enough or to a large enough degree, the abomasum becomes G- 
gested, hemorrhagic, and infarcted due to occlusion of gastric circulation. As the organ becomes devitalized, the cow goes into shock and dies.
(5) Acid–base changes accompany an abomasal volvulus.
   (a) In the early condition, abomasal outflow of HCl is compromised, and reflux occurs into the rumen. The rumen effectively traps H+ and Cl– ions. K+ ions are lost in the urine in deference to H+ ion retention, creating in total a hypochloremic, hypokalemic, metabolic alkalosis.
   (b) In the advanced condition, a metabolic acidosis may be found related to the state of shock of the animal.
(6) Occasionally, the omasum is involved in the abomasal volvulus (omal–abomasal volvulus), which increases the gravity of the prognosis.
   c. Etiology and pathogenesis. Dilation is thought to precede torsion of the volvulus and is hypothesized to be caused similarly to LDA and RDA. Mechanical events may cause torsion in the volvulus of the distended abomasum.
   d. Diagnostic plan. Diagnosis of the condition is based on clinical findings and laboratory tests. A Liptak test is helpful in determining the affected organ.
   e. Laboratory tests
   (1) Laboratory findings that support the diagnosis include:
   (a) An aspirate of a low pH fluid from the viscus
   (b) A toxic leukogram to varying degrees
   (c) A metabolic alkalosis (in late stages, a metabolic acidosis)
   (d) Evidence of dehydration and prerenal uremia [PCV, total protein (TP), BUN]
   (e) A paradoxical aciduria as K+ ions are excreted in the face of whole-body, H+ ion deficiency
   (2) As the plasma anion gap increases, the prognosis for recovery decreases.
   f. Differential diagnoses. It is necessary to rule out other causes of abdominal pings that result in an animal’s sick appearance. Conditions to be ruled out include:
   (1) Cecal volvulus/torsion
   (2) Mesenteric root torsion
   (3) Small intestinal obstructions
   (4) Acute diffuse peritonitis
   g. Therapeutic plan
   (1) Immediate surgical intervention coupled with fluid therapy is indicated.
   (a) Surgical approaches are a right flank omentopexy or a right paramedian abomasopexy.
   (b) Intravenous fluids include isotonic sodium chloride and dextrose for volume expansion (likely 20–80 L), followed by a slow drip of potassium chloride (1 mg/kg/hour), sodium chloride, and dextrose. Balanced electrolyte solutions are indicated for shock and metabolic acidosis.
   (c) Other supportive treatments, depending on the case, include antibiotics, corticosteroids, and NSAIDs.
   (2) Salvage instead of surgery may be indicated if dictated by economics and if the animal will pass antemortem and post-mortem inspection. The systemic involvement of the animal often dictates against this.
   h. Prognosis. The prognosis for an animal depends on the degree of abomasal compromise determined at surgery. In some cases that recover, abomasal torsion is associated with the subsequent development of vagal indigestion.

4. Cecal dilation or cecal volvulus
   a. Patient profile and history
   (1) Cecal dilation and cecal volvulus are conditions that occur most commonly in mature dairy cattle in the first 2 months postpartum. These conditions are more common in the winter months and show no breed predisposition.
   (2) The farmer reports that the cow is partially to completely anorexic with a drop in milk production. The herd ration is often a high grain diet rich in carbohydrates such as corn silage or high-moisture corn. It may be fed as a total mixed ration (TMR). The clinical signs usually are reported moderately
progressive and not sudden in onset unless a dilation has converted into a torsion, in which case signs deteriorate rapidly.

b. Clinical findings
(1) Cecal dilation. With a cecal dilation, the vital signs are usually normal. The animal may tread, indicating mild pain. Feces may be somewhat loose and decreased in amount. The right paralumbar fossa may be slightly distended, and a ping can be auscultated in the right flank caudal to the site for RDA. Rumen motility is decreased in rate and strength. Rectal examination confirms the diagnosis, as the apex of the cecum will be felt at the pelvic inlet.

(2) Cecal volvulus. A cecal volvulus offers more dramatic clinical signs than a cecal dilation. The pulse is elevated, and there is complete agalactia, rumen atony, and absence of manure. The cow exhibits abdominal pain by shifting hind-end weight, kicking at the abdomen, and frequent lying down and rising. The right paralumbar fossa may be distended. A ping can be elicited over a larger area than with a simple cecal dilation, and fluid, splashing sounds may be heard. Rectal examination reveals a distended cecal body (the apex has been twisted and directed cranially).

c. Etiology and pathogenesis
(1) The pathogenesis of cecal distention is thought to be similar to abomasal displacements. It is believed that cecal organisms metabolize carbohydrates that have escaped upper gastrointestinal microbial degradation. VFAs, methane gas, and CO₂ are all produced. The VFAs inhibit cecal motility, and the gases accumulate, producing distention. Cecal dilations are thought to precede torsions.

(2) When distended, the cecum may then rotate clockwise or counterclockwise (when viewed from the right). The amount of rotation determines the degree of vascular embarrassment of the organ and the extent of resulting clinical signs.

d. Diagnostic plan. The diagnosis is usually made on the basis of clinical signs and rectal findings.
e. Laboratory tests
(1) Laboratory results usually are normal for simple cecal dilation. Animals with cecal volvulus often have a hypochloremic, hypokalemic, metabolic alkalosis. Other laboratory values reflect varying degrees of vascular compromise and shock.

(2) To differentiate cecal volvulus from abomasal volvulus, it may be helpful to perform a Liptak test. The pH is higher (more than 6) with cecal fluid than with abomasal fluid.
f. Differential diagnoses
(1) For cecal dilations, rule out conditions such as abomasal displacement, ketosis, simple indigestion, and colonic gas.

(2) Cecal volvulus may appear similar to abomasal volvulus, traumatic reticuloperitonitis, mesenteric root torsion, and gastrointestinal accidents (e.g., intussusceptions).
g. Therapeutic plan
(1) Simple cecal dilations are treated conservatively.

(a) Diet and exercise. The roughage component of the diet should be increased and forced exercise employed to increase gastrointestinal motility. Calcium salts are used occasionally to treat low-grade hypocalcemia and increase digestive tract motility.

(b) Surgery. Cecal dilation can be a chronic or recurrent condition. Surgery may be used with some success in these cases.

(2) Cecal volvulus requires surgical intervention. The cecum is approached through the right flank. The cecum is decompressed, and ingesta is removed from both it and the adjacent colon. Recovery is usually good with uncomplicated cases. If part or all of the cecum is devitalized, a partial or complete resection may be necessary. Fluid replacement is also recommended to correct the metabolic alkalosis (sodium chloride, potassium chloride, and dextrose).

h. Prognosis. Depends upon amount of devitalization.

d. Diagnostic plan. Clinical findings and the demonstration of positive fecal occult blood provide the diagnosis. Fecal blood is best discovered by the use of tablets which test for occult blood.
e. Therapeutic plan
(1) In the case of adult ruminants, the primary disease and erosions are self-limiting.

(2) In younger animals, treatment usually is not attempted; however, the owner should pay attention to providing a diet composed of more easily digestible roughage.

(3) Laxatives, such as mineral oil, may be used in the case of concomitant foreign bodies (e.g., hairballs). Alternatively, surgery may be necessary.
f. Prevention. The erosions and ulcers are self-limiting when the primary disorder has been corrected.

2. Type II
a. Patient profile. These ulcers occur in adult cattle usually within 2 months of parturition and are commonly associated with a concurrent abomasal disease (e.g., LDA).

b. Clinical findings. The animal may die suddenly or present with dark, tarry stools (melena). The cow will be anemic, as evidenced by pale mucous membranes, tachycardia, and weakness.

c. Etiology and pathogenesis. The pathogenesis of bleeding ulcers is speculated to be similar to type I ulcers; however, the ulceration involves a major blood vessel, resulting in blood loss. Lymphosarcoma of the abomasal wall is also a primary cause for erosion and invasion of major abomasal blood vessels.

D. Bleeding ulcers and erosions. Blood in the feces of ruminants may be frank (visible and either red or black) or occult (unseen). Frank red blood is indicative of lower intestinal origin, whereas dark blood is from higher in the gastrointestinal tract and has been digested by proteolytic enzymes. Occult blood is from minor bleeding of insufficient amount to cause a change in color of the feces.

1. Type I
   a. Patient profile and history. Individual animals suffering from a primary disease process may develop abomasal erosions and ulcers. Dairy cows and weaning age calves seem to be most susceptible. The owner usually notices only the primary disease process (a septic process such as acute mastitis, or an abomasal condition such as LDA or RDA). In calves, the history may be one of poor growth and appetite disturbances.

   b. Clinical findings
      (1) There may be no clinical signs of abomasal erosions or ulcers. The lesions may only be evident at slaughter of a normal animal or necropsy of an animal dying from a primary disease process.

      (2) Alternatively, the clinical signs of a primary disease may be accompanied by signs of abomasal erosion or ulceration. These signs include:

         (a) Dark, fluid feces with mild, chronic abdominal pain
         (b) Periodic bruxism
         (c) A capricious appetite
         (d) Intermittent fecal occult blood

   c. Etiology and pathogenesis. The ulcers in an individual are usually multiple and small. Possible factors contributing to the development of ulcers include:

      (1) Hyperacidity. Gastric acid secretion may be stimulated by histamines, high VFA levels, calcium, or stress.

      (2) Corticosteroids, which lower mucosal cell resistance

      (3) Direct trauma from fiber (straw, low-quality roughage) or foreign bodies (hairballs in calves)

      (4) Abomasal distention, resulting in the tearing of mucosa and exacerbation of the lesion by exposure to HCl

      (5) Pasture grazing and associations with rainfall, fertilization, and forage growth

      (6) Unknown factors, such as endotoxins and mediators substances (e.g., interleukins)

   d. Diagnostic plan. Clinical findings and the demonstration of positive fecal occult blood provide the diagnosis. Fecal blood is best discovered by the use of tablets which test for occult blood.

   e. Therapeutic plan
      (1) In the case of adult ruminants, the primary disease and erosions are self-limiting.

      (2) In younger animals, treatment usually is not attempted; however, the owner should pay attention to providing a diet composed of more easily digestible roughage.

      (3) Laxatives, such as mineral oil, may be used in the case of concomitant foreign bodies (e.g., hairballs). Alternatively, surgery may be necessary.

   f. Prevention. The erosions and ulcers are self-limiting when the primary disorder has been corrected.
Chapter 3

1. Traumatic reticuloperitonitis (TRP, hardware disease, traumatic gastritis, traumatic reticulitis)
   a. Patient profile and history. In North America, the condition is most common in adult dairy cattle and is sporadic in nature. The owner describes a cow that experienced an abrupt drop in milk production and exhibits partial to complete anorexia.
   b. Clinical findings
      (1) Acute local peritonitis. Animals in the early stages of TRP are reluctant to stand or walk downhill. A short groan or grunt may also occur spontaneously when an animal stands or walks downhill.
      (2) Acute local peritonitis. Animals in the early stages of TRP are reluctant to move and appear “tucked-up.” There may be extension of the neck and an arching of the back. The cow may stand with its elbows abducted and hind feet in the gutter to alleviate the abdominal pain. The tricep muscles often tremble.

2. Anterior abdominal pain
   a. Vital signs. Cows may have a shallow, catchy respiratory pattern and a decreased abdominal component to the cycle. The temperature and pulse rate are usually elevated (39.5 °C–40.5 °C and 80–90 bpm, respectively). Usually, the cow is dull and depressed.
   b. On gastrointestinal examination, the clinician finds a decrease in rumen motility or complete atony. The feces of affected animals are scant, firm, dry, and mucus covered.
   c. Rectal examination reveals a small, firm-feeling rumen. The animal may grunt with pain as the viscera are pressed forward.
   d. Manipulative tests may be of value in localizing the pain. Cows normally ventroflex when pinched over the withers. Cows affected by TRP will not ventroflex and may grunt with resentment of the procedure. Percussion over the xiphisternum with a closed fist, lifting under the thorax with a board held by a person on either side of the animal or pressure applied by a knee while the ventral thorax may all elicit the same grunt.
   e. Laboratory tests
      (1) Acute local peritonitis. Animals in the early stages of TRP are reluctant to move and appear “tucked-up.” There may be extension of the neck and an arching of the back. The cow may stand with its elbows abducted and hind feet in the gutter to alleviate the abdominal pain. The tricep muscles often tremble.
      (2) Acute local peritonitis. Animals in the early stages of TRP are reluctant to move and appear “tucked-up.” There may be extension of the neck and an arching of the back. The cow may stand with its elbows abducted and hind feet in the gutter to alleviate the abdominal pain. The tricep muscles often tremble.

3. Sequelae to reticular penetration by the foreign body include:
      (a) Resolution of the infection with or without medical intervention. The foreign body is deposited in the reticulum and penetrates the reticular wall, usually in an anterior direction. Ruminal fluid and bacteria follow the track of the foreign body and enter the peritoneal cavity, producing an acute, localized peritonitis.
      (b) Acute diffuse peritonitis. The infection cannot be controlled by the previous inflammatory processes, and a fatal infection ensues.
      (c) Traumatic hepatitis or splenitis. Movement of a foreign body may occur in a direction other than directly cranial, resulting in the involvement of associated abdominal tissues. Most commonly the spleen or liver.
      (d) Pericarditis, pleuritis, or pneumonia resulting from perforation of the diaphragm.
      (e) Peritonitis. A result of fibrous tissue production may be the formation of reticular adhesions and mechanical interruption of rumen motility (see 1 A 3).
      (f) Fatal hemorrhage due to great vessel perforation (rare)
      (g) Diaphragmatic hernia caused by the weakening of the diaphragm (rare)

4. Diagnostic plan
   (1) The condition is most often diagnosed through the combination of a thorough physical examination and laboratory tests. Ancillary aids to diagnosis may include the use of radiology, a metal detector, or a compass to determine if a magnet is present in the reticulum.
   (2) An abdominocentesis is warranted if the diagnosis is equivocal or requires confirmation. It should be performed 3–4 cm to the right of the midline and 5–7 cm cranial to the foramen of the subcutaneous abdominal vein. Other reported sites for abdominocentesis include a point 10 cm to the right of the umbilicus, on the midline just caudal to the umbilicus, or cranial to the udder under the right flank fold.
   (3) An exploratory rumenotomy and/or laparotomy may be both diagnostic and therapeutic.

5. Laboratory tests
1. Adult cattle
   a. Patient profile and history. Most colics are caused by either a malposition of a gastrointestinal tract component or other causes of acute obstruction to outflow. Surgery is the treatment of choice.
   b. Clinical findings
      (1) Appearance and vital signs. The animal exhibits a painful abdomen by treading, kicking at the abdomen, and frequently lying down and rising. The pulse and respiratory rates are elevated. Auscultation of the abdomen reveals absence of both ruminal and intestinal motility. With abomasal or upper intestinal obstruction, the abdomen is slightly distended, and splashing sounds can be elicited by succussion in the lower right quadrant.
      (2) On simultaneous auscultation and percussion, pings may be evident over distended viscera having a fluid/gas interface. The pings may differ in intensity, location, pitch, and area of auscultation, depending on the source of the distention.
      (3) Rectal examination, the rectum is usually empty except for a thick, tenacious, dark red mucus. Insertion of the arm may cause pain or vigorous straining. The examiner may be able to palpate distended or displaced bowel or tight mesenteric bands.
      (4) The animal may be dehydrated or in shock. The more proximal the lesion in the gastrointestinal tract, the more rapid the deterioration of the patient.
   c. Etiology and pathogenesis
      (1) The pathogenesis of abomasal torsion/volvulus and cecal torsion/volvulus seems to follow the same course of events as simple dilatations. However, twisting of the dilated viscus on either its horizontal or vertical axis results in digestive outflow obstruction and venous vascular occlusion.
      (2) The risk factors or causes of other intestinal accidents are less clearly understood and less easily studied because of their sporadic nature.
      (3) Intestinal accidents
         (a) Indications include persistent abdominal pain, elevated heart and respiratory rates, abdominal distention, absence of feces in the rectum, and the presence of a gas-filled viscus or distended loops of intestine.

II. BOVINE INTESTINES
A. Gastrointestinal pain (colic)
   2. Perforating abomasal ulcers with localized peritonitis. Abomasal ulcers may perforate the serosa, producing an acute local peritonitis. These may be referred to as type III ulcers (instead of bleeding ulcers).
   a. Patient profile and history. Abomasal ulcers in mature ruminants most commonly occur in association with concurrent diseases (e.g., LDA), recent parturition, heavy lactation, and grain or pasture grass feeding.
   b. Clinical findings. Clinical signs resemble those of TRP (see I E 1 b). However, the pain is localized to the right side of the xiphoid. Careful palpation and ballottement is required to determine this distinction. Bleeding into the gastrointestinal tract rarely seems to accompany a perforated abomasal ulcer.
   c. Etiology and pathogenesis (see I D 2 c).
   d. Laboratory tests. Laboratory data are similar to the acute local peritonitis of TRP (see I E 1 e). There may be an associated hypochromic metabolic acidosis if ulcers are concurrent with LDA.
   e. Differential diagnoses. Consider other causes of acute peritonitis on a list of differential diagnoses (e.g., TRP). Also consider conditions that may appear to mimic anterior-abdominal pain, such as:
      (1) Liver abscess
      (2) Pericarditis
      (3) Pleuritis
      (4) Endocarditis

Chapter 3

2. 

1. Acute diarrhea in adult cattle
   a. Bovine virus diarrhea (BVD)
      (1) Patient profile and history. The majority of animals affected are young cattle (6 months to 2 years of age) often housed in an intensive management environment (e.g., beef feedlot, dairy freestall housing). There is a variable incidence of clinical disease (usually low). Several animals in the herd may show clinical signs.

   (b) Intestinal accidents that involve the abomasum or intestinal tract and produce a common subset of clinical findings include:
      (i) Right abomasal volvulus
      (ii) Small intestinal intussusception
      (iii) Mesenteric torsion
      (iv) Intestinal incarceration
      (v) Cecal dilatation/volvulus
      (vi) Stricture of the colon
      (vii) Colonic obstruction (e.g., enterolith)
      (viii) Colonic constriction (e.g., fat necrosis)

   d. Diagnostic plan. Perform a thorough physical examination. The presumptive diagnosis is confirmed by surgery.

   e. Laboratory tests. Laboratory information is seldom available in time to be of value in the prognosis.
      (1) Hematologic work-up. Shock and dehydration is supported by an increased PCV, increased TP, and a high BUN (perrenal uremia). High duodenal obstruction or abomasal volvulus produces a hypochloremic, hypokalemic, metabolic alkalosis. Leukopenia and/or neutropenia is found with devitalization of infarcted intestine.
      (2) Abdominocentesis findings may be variable but often show increased red blood cells, increased leukocytes, and bacteria indicative of devitalized bowel.

   f. Therapeutic plan. If the animal is to be successfully treated, a decision to perform surgery should be made early and the patient vigorously supported by fluid therapy.

2. Calves
   a. Patient profile and history. Most frequently, abdominal pain (colic) in calves is caused by conditions of gastrointestinal origin.

   b. Clinical findings are variable depending on the site of involvement, duration of the condition, and degree of vascular compromise of the viscus. Common signs of colic include frequent lying down and rising, kicking of the abdomen, bruxism, and straining.

   c. Etiology and pathogenesis. Acute abdominal pain is of sporadic incidence and often associated with congenital problems of the gastrointestinal tract of young calves. Some conditions may be hereditary.

   d. Diagnostic plan. A thorough clinical examination is necessary to determine the most likely source of the pain. Ancillary tests include radiography and contrast studies, gastric intubation, rectal probe (for anal or colonic atresia), and ultrasonography.

   e. Differential diagnoses
      (1) Causes to be ruled out include:
         (a) Intestinal atresia
         (b) Atresia coli
         (c) Atresia ani
         (d) Abomasal dilatation
         (e) Abomasal torsion
      (2) Other causes of abdominal distention must be ruled out including bladder rupture and intestinal fluid pooling with neonatal calf diarrhea.

   f. Therapeutic plan. Generally, therapies include surgery and fluid therapy for responsive conditions.

B. Diarrhea

1. Acute diarrhea in adult cattle
   a. Bovine virus diarrhea (BVD)
      (1) Patient profile and history. The majority of animals affected are young cattle (6 months to 2 years of age) often housed in an intensive management environment (e.g., beef feedlot, dairy freestall housing). There is a variable incidence of clinical disease (usually low). Several animals in the herd may show clinical signs.

      (2) Clinical findings. The clinical findings may range from inapparent to acute, severe, enteric disease.
         (a) Clinically inapparent disease is widespread. Serologic evaluation may reveal 60%-80% of animals exposed and seropositive.
         (b) Mild enteric disease may be seen in a large number of animals and presents as mild transient diarrhea, inappetence, depression, and fever. Cattle recover in a few days.
         (c) Acute enteric disease presents as fever, profuse watery diarrhea, dysentery, and tenesmus. Discrete oral erosions are present (or may develop within 7 days of diarrhea), and the animal may salivate excessively. Peracute cases may die without evidence of diarrhea. This has also been described as a hemorrhagic syndrome in cattle.
         (d) Clinical signs referable to other body systems include mucopurulent nasal discharge, lacrimation, corneal edema and central corneal opacity; neoneatal calf diarrhea.

      (2) Hematologic work-up. Shock and dehydration is supported by an increased PCV, increased TP, and a high BUN (perrenal uremia). High duodenal obstruction or abomasal volvulus produces a hypochloremic, hypokalemic, metabolic alkalosis. Leukopenia and/or neutropenia is found with devitalization of infarcted intestine.

      (3) Abdominocentesis findings may be variable but often show increased red blood cells, increased leukocytes, and bacteria indicative of devitalized bowel.

      (4) Therapeutic plan. If the animal is to be successfully treated, a decision to perform surgery should be made early and the patient vigorously supported by fluid therapy.

   b. Clinical findings. The clinical findings may range from inapparent to acute, severe, enteric disease.
      (a) Clinically inapparent disease is widespread. Serologic evaluation may reveal 60%-80% of animals exposed and seropositive.
      (b) Mild enteric disease may be seen in a large number of animals and presents as mild transient diarrhea, inappetence, depression, and fever. Cattle recover in a few days.
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      (2) Hematologic work-up. Shock and dehydration is supported by an increased PCV, increased TP, and a high BUN (perrenal uremia). High duodenal obstruction or abomasal volvulus produces a hypochloremic, hypokalemic, metabolic alkalosis. Leukopenia and/or neutropenia is found with devitalization of infarcted intestine.

      (3) Abdominocentesis findings may be variable but often show increased red blood cells, increased leukocytes, and bacteria indicative of devitalized bowel.

      (4) Therapeutic plan. If the animal is to be successfully treated, a decision to perform surgery should be made early and the patient vigorously supported by fluid therapy.

   (b) The pathogenesis of acute, fatal, enteric disease may involve one of two processes.
      (j) Infection by a particularly virulent biotype (BVD Type 2)
      (j) Infection by a particularly virulent biotype (BVD Type 2)

   (ii) If the fetus is infected by a noncytopathic virus in a BVD-negative dam before 125 days gestation, the calf is immunotolerant to the BVD virus with persistent viral infection and shedding. Subsequent exposure of this animal to a cytopathic virus (likely a mutant of the noncytopathic biotype) results in an acute and fatal infection (mucosal disease).

   (3) Differential diagnoses. Clinical findings that are helpful in the definitive diagnosis include diarrhea, shallow oral erosions, central corneal opacity, and interdigital erosions.

   (5) Laboratory tests. In individual animals, evidence of leukopenia, seroconversion, virus isolation, and polymerase chain reaction (PCR) are helpful for diagnosis. Note that animals that are immunotolerant and suffering from mucosal disease may not seroconvert. Herd serology may aid in diagnosis, as will necropsy of affected animals.

   (6) Differential diagnoses. Conditions to be ruled out include bovine malignant catarrh, rinderpest, and salmonellosis.

   (7) Therapeutic plan
      (a) Mild cases. Therapy is not necessary in cases of mild disease and of little help with severe enteric disease. Treatment is of no value in mucosal disease.
      (b) Severe cases. When therapy is attempted in severe cases, treatment consists of fluid therapy, oral astringents, and systemic antibiotics to control secondary infections.

   (8) Prevention
      (a) Isolation of clinically ill animals from naive animals may limit the spread in the case of a herd outbreak.
      (b) Vaccination may be practiced with either killed or modified live viruses. Modified live virus vaccines have been limited to nonpregnant animals in their use because of possible induced abortions. It is unsure if this is related to the vaccine virus, vaccine virus reversion, or vaccine
contamination. Beef calves should be vaccinated between 6 and 8 months of age and dairy cattle vaccinated annually.

b. Bovine malignant catarrh (BMC, malignant catarrhal fever)

(1) Patient profile and history. The condition is usually sporadic and found in single animals, although occasionally herd outbreaks occur. In North America, the history invariably includes contact with sheep (usually at lambing time). In Africa, history includes contact with wildebeest.

(2) Clinical findings
a. High fever with tachycardia accompanies profuse diarrhea and dysentery. Other signs include anorexia, agalactia, mucopurulent nasal discharge, dyspnea, and lymphadenopathy.

b. Ocular changes include a peripheral corneoscleral opacity that spreads centripetally, ocular discharge, eyelid edema, and blepharospasm.

c. Neurological signs may be seen including incoordination, nystagmus, muscle tremors, head pressing, paralysis, and convulsions.

d. Chronic disease produces eczematous lesions around the perineum, prepuce, axillae, and horns. Sloughing of the skin from the teats and vulva is found in acute cases.

(e) Labeling. This variety of clinical pictures has resulted in labeling the disease by its presenting form (i.e., alimentary form, head and eye form, neurological form, skin form).

(3) Etiology and pathogenesis

a. BMC is a generalized infection of the primitive mesenchyme. Necrosis and proliferation of vascular adventitia (vasculitis) yields epithelial erosions, keratoconjunctivitis, and encephalitis. Lymph node enlargement is caused by atypical proliferation of T lymphocytes.

b. Clinical disease occurs only in cattle, deer, and buffalo. Sheep and goats in North America and wildebeest in Africa transmit the virus, although neither of these hosts develop clinical signs. Alcelaphine BMC virus (BHV-3) is the wildebeest-associated agent. A yet unidentified herpesvirus (sheep-associated herpesvirus) is the North American agent. The sporadic nature of the condition makes studying the disease or determining etiologies difficult.

(4) Diagnostic plan. Diagnosis of the condition is aided by the association with hosts, sporadic nature of the condition, and uniqueness of the clinical findings.

(5) Laboratory tests

a. Hematologic work-up. Animals exhibit an early leukopenia followed by a leukocytosis. There are inflammatory changes of the joint fluid and cerebrospinal fluid.

b. Serological conversion occurs with the African disease but has not been demonstrated with the North American disease. Virus isolation remains difficult.

c. Postmortem examination remains the definitive diagnostic tool.

(6) Therapeutic plan. Therapy is unrewarding and animals will die in spite of supportive care.

c. Rinderpest

(1) Patient profile and history. This disease is confined to the Middle East, Asia, and tropical Africa where it is enzootic. It affects all ruminants and emerges in outbreaks.

(2) Clinical findings

a. Acute cases. A high fever precedes by a few days other clinical signs such as anorexia, agalactia, and laceration. Oral and nasal laceration is soon followed by the development of coalescing necrotic ulcers. Other signs follow including hemoglobin and purulent laceration, skin lesions, diarrhea, dysentery, and dehydration. In an immunologically naive population, many cattle die acutely, most succumb within 7–10 days and few survive.

b. Subacute and chronic forms of the disease occur in more resistant populations and survival rates are higher.

(3) Etiology and pathogenesis. Rinderpest virus is a paramyxovirus spread by animal excretions. Close animal contact is necessary because the virus does not survive for long periods outside of the host. The virus has a high affinity for lymphoid tissue and alimentary mucosa, where it replicates and produces the focal necrotic stomatitis and enteritis. A strong antibody response is generated.

(4) Diagnostic plan. Diagnosis is based on clinical findings, history, and postmortem lesions.

(5) Laboratory tests

a. Leukopenia and lymphopenia occur in affected animals. Postmortem findings are highly suggestive in populations at risk.

b. Viral antigen may be detected in several excretions and will confirm the diagnosis. Serology is of less value because of the acuteness of the condition in many cases.

d. Differential diagnoses. Other diseases that cause oral lesions and diarrhea must be ruled out. It is equally important to differentiate this disease from other that produce similar oral lesions without diarrhea (e.g., foot and mouth disease).

(7) Therapeutic plan. There is no known treatment for this virus.

(8) Prevention. Vaccination programs are successful, and many have lead to the eradication of the disease. Total eradication requires regulatory measures ensuring vaccination of herds and limitations on movements of nomadic populations.

d. Salmonellosis

(1) Patient profile and history. This disease occurs worldwide and is seen in all ages, species, and breeds of animals. There is often a history of stress (e.g., recent shipment, parturition).

(2) Clinical findings

a. Vitals signs and diarrhea. There is an initial fever followed by a subnormal temperature. The heart and respiratory rates are elevated. There is a severe, watery, hemorrhagic diarrhea with mucus, fibrin casts, and blood clots. There is frequently evidence of abdominal pain (groaning, kicking at the flanks), and pregnant animals may abort.

b. The disease has been described as one of three syndromes: septicemia, acute enteritis, and chronic enteritis. The main presenting syndrome in cattle is the acute enteritis.

(3) Etiology and pathogenesis

a. The most common species isolated worldwide is Salmonella typhimurium, although a variety of Salmonella species can cause disease. S. dublin has a more patchy distribution but is the most common isolate in Europe.

b. The most common source of infection is environmental and feed contamination. Any domestic or wild species of animal or bird can act as a source of infection.

c. S. typhimurium causes sporadic, occasionally fatal disease. Infected adults are carriers for short periods of time so that the disease incidence usually subsides when the source of infection is removed.

d. S. dublin is particularly well adapted to cattle, which may act as a reservoir for outbreaks. Continued excretion of the organism may occur for years after exposure.

e. Route of infection. After oral inoculation with the bacteria, salmonella invades the intestinal wall and progresses to localize in the mesenteric lymph nodes. Development of disease then depends on the immune status and age of host, virulence of the organism, and stresses on the animal. In susceptible animals exposed to a virulent species, septicemia and bacteremia occur. A carrier state may develop in survivors. Survivors develop in survivors. Diarrhea occurs due to enteritis and the elaboration of an enterotoxin, which causes an increased secretion of sodium, chloride, and water into the gut lumen.

(4) Diagnostic plan. Clinical findings together with laboratory test results usually
are sufficient for diagnosis; however, fecal culture results may be negative even with diarrhea. (5) Laboratory tests. (a) Clinical pathologic findings reflect a profound toxic state and an inflammatory condition. Anemia from blood loss may be seen. (b) Fecal culture is the best diagnostic test but may need to be repeated several times for success. Organisms may not be present in the feces for up to 2 weeks after commencement of diarrhea because of the dilution effect of the diarrhea. Culturing a rectal mucosal biopsy increases the likelihood of successfully isolating the organism in an affected animal. (6) Differential diagnoses. Other conditions that appear similar to enteric salmonellosis include winter dysentery, coccidiosis, general toxemia, and arsenic or superphosphate fertilizer toxicities. (7) Therapeutic plan (a) Early, aggressive treatment with broad-spectrum or selective antibiotics is necessary for successful therapy. Extralabel use of these antibiotics requires restrictions on meat and milk use from treated animals. Examples include: (i) Trimethoprim-sulfua: 25 mg/kg intramuscularly twice per day (ii) Centaminic: 2 mg/kg intramuscularly three times per day (iii) Amikacin: 7 mg/kg intramuscularly three times per day (b) Oral or intravenous fluids are necessary in amounts calculated for replacement and ongoing losses. Oral astringents and protectants (e.g., bismuth subsalicylate) and parenteral nonsteroidal anti-inflammatory agents (e.g., flunixin meglumine) may be employed. (8) Prevention (a) Recovered animals (whether from treatment or naturally) may excrete the organism and expose herdmates for significant lengths of time. Treatment and isolation procedures must take this into account. (b) Management strategies. Seek to remove sources of contaminated food, litter, and water. Salmonella organisms may infect humans, so hygienic precautions should be taken. e. Winter dysentery (1) Patient profile and history. This disease commonly occurs in young adult, housed dairy cattle in the winter months. Diarrhea is most severe in lactating and pregnant animals and is rare in bulls and steers. Diarrhea is often related to a feed change, a change in housing, or sudden, significant temperature shifts. (2) Clinical findings (a) Herd. This disorder causes an outbreak of diarrhea involving the majority of animals in the herd. There is an accompanying drop in milk production. The condition may persist for up to 2 weeks. (b) In individual animals, the TPR is usually normal. There is a nasolacrimal discharge and a cough, both of which precede the diarrhea. The diarrhea is projectile and hemorrhagic and lasts from a few hours to a week. The animal may demonstrate abdominal pain, have increased intestinal sounds, and become dehydrated and weak. (3) Etiology. The cause is considered to be a bovine coronavirus. (4) Diagnostic plan. The clinical picture and subjective findings along with response to therapy are enough to establish a diagnosis. (5) Laboratory tests. Individual animals have a mildly elevated hematocrit and total plasma protein value. Direct electron microscopy and an enzyme-linked immunosorbent assay (ELISA) may be applied to the feces to demonstrate the virus. Acute and convalescent serum samples reveal seroconversion to coro.

f. Arsenic toxicosis (1) Patient profile and history. Recent exposure to arsenicals (e.g., ectoparasite sprays, arsenic-based herbicides, arsenic-based wood preservatives) may be deduced from the history. The owner may report sudden death of an individual or group of animals. (2) Clinical finding. (a) Animals may experience acute, subacute, or chronic signs referable to arsenic poisoning. With the acute toxicosis, cattle experience abdominal pain, diarrhea, dehydration, regurgitation, muscular tremors, convulsions, and death within 4–6 hours of showing clinical signs. (b) Other signs may involve the central nervous system (CNS; see Chapter 11). (3) Etiology and pathogenesis. Ingested inorganic arsenic causes inactivation of sulfhydryl groups in tissue enzymes. Tissues that are most susceptible are the alimentary tract, liver, kidney, spleen, and lung. In the gastrointestinal tract, this condition causes extensive capillary damage, hemorrhage, necrosis, and sloughing of the intestinal mucosa. (4) Diagnostic plan. Clinical findings and history are important, but diagnosis relies on laboratory confirmation. (5) Laboratory tests. Urine and hair samples are suitable antemortem specimens for arsenic analysis. Postmortem confirmation is best supported by liver arsenic levels. (6) Therapeutic plan (a) An attempt should be made to absorb the enteric arsenic with activated charcoal at 1–4 g/kg orally. Cattle are also treated with sodium thiosulfate at 15–30 g in 200 ml H2O intravenously followed by 30–60 g orally, four times daily. Treatment should be continued until recovery occurs. (b) British antilewisite (BAL), also called dimercaprol, ml, may be used but is less effective against inorganic salts than the organic forms. Intravenous fluid therapy is warranted in dehydrated animals. (7) Prevention. Limit exposure to arsenicals. g. Toxemia. Toxemia, such as peracute coliform mastitis or toxic metritis in cattle, often are accompanied by diarrhea. The pathogenesis may be an effect of endotoxemia or by stress-related etiologies.

2. Subacute to chronic diarrhea in young and adult cattle a. Chronic parasitism. This condition is common, but diarrhea due to parasitism is relatively infrequent Chronic wasting is the obvious sign, and of the parasites affecting the bovine abomasum and small intestine (e.g., Ostertagia, Cooperia, Trichostrongylus, Nematocidus, Ostertagia) infestation produces the diarrhea. (1) Patient profile and history. Several young (6 months to 2 years) animals often are affected in a herd. There is a history of persistent diarrhea and weight loss. (2) Clinical findings. The TPR is usually normal. There is diarrhea without odor, mucus, or blood. Emaciation, dependent edema, and poor growth also may be signs of infection. (3) Etiology and pathogenesis (a) Type I. Infective (third-stage) larvae are ingested, molt to fourth-stage larvae, and penetrate the abomasal glands, causing hyperplasia of the mucus-secreting cells. This nodular hyperplasia may be discrete or confluent, presenting as a "moroccan-leather" appearance on necropsy. Mucosal layer destruction results in protein leakage. (b) Type II. Fourth-stage larvae enter the mucosal glands and remain there, causing little or no damage (a pretype condition). Type II disease occurs when larvae emerge, producing marked cellular hyperplasia. This emergence occurs at various times of the year in different countries. Abomasal pH rises due to a loss of parietal cells and failure of the conversion of pep.

3. Toxemia. Toxemia, such as peracute coliform mastitis or toxic metritis in cattle, often are accompanied by diarrhea. The pathogenesis may be an effect of endotoxemia or by stress-related etiologies.

4. Diagnostic plan. Clinical findings, environmental findings, and investigation of management practices may indicate a parasite burden.
Laboratory tests. Fecal egg counts are diagnostic, however, they may need to be repeated in the case of hypobiotic larvae.

Therapeutic plan. Deworm in the event of any parasite load. Ivermectin and levamisole continue to be the drugs of choice (ivermectin for the hypobiotic stage).

Prevention. Reduce pasture or drylot contamination through management of stocking densities, pasture rotation, and routine anthelmintic treatment.

b. Chronic BVD

(1) Patient profile and history. The age range of affected cattle is usually 6 months to 2 years. There may be a history of a previous outbreak of BVD in the herd with the recovery of most animals. A few animals may have remained stunted with intermittent diarrhea, lameness, or both. There may be a history of recurrent bacterial infections (e.g., pneumonia).

(2) Clinical findings. Animals appear stunted with a rough, dry haircoat. Oral examination reveals the occasional erosion with blunted oral papillae. Crusty or erosive dermatitis is present at the commissures of the mouth, medial canthus of the eye, around the perineum, scrotum, coronary band, bulbs of the heels, and in the interdigital cleft. The animal may be lame. Diarrhea is intermittent or continuous.

(3) Etiology and pathogenesis. Cattle previously exposed to a noncytopathic strain of BVD virus in utero (before 125 days gestation) are immunotolerant to the BVD virus and are incapable of mounting a humoral antibody response when subsequently challenged with the virus. They are chronically infected with the virus, continue to excrete antigen, but remain negative by serum neutralization tests.

(4) Diagnostic plan. Diagnosis may be based on clinical findings, virus isolation or PCR for viral nucleic acid, and necropsy findings.

(5) Therapeutic plan. Therapy is useless and salvage should be considered as a realistic option.

(6) Prevention. Recommendations for control are as presented in I I B 1 a (8).

c. Coccidiosis

(1) Patient profile and history. Subjective findings with this disease are similar to other enteric diseases predisposing to parasitism (e.g., overcrowded housing conditions, management practices that encourage fecal–oral spread of organisms). Groups of young animals are affected in a seasonal pattern depending on region of the country.

(2) Clinical findings. The cardinal signs are diarrhea (with mucus and blood) and tenesmus. Anemia also may be a finding, and some cattle may have nervous signs (see Chapter 11). Cattle often are unthrifty in appearance. Clinical syndromes may vary from peracute cases to inapparent infections.

(3) Etiology and pathogenesis

(a) Causative organisms are Eimeria zuernii and E. bovis in the large intestine and E. ellipsoidalis in the small intestine.

(b) Infestation occurs through the ingestion of sporulated oocysts from the environment. Oocysts are resistant to most environmental conditions. Multiple-species infections are quite common, and disease seems to be more prevalent in stressed or undernourished animals.

(c) Route of infection. Sporozoites are released from the ingested oocysts and invade the endothelial cells of the small intestine. asexual schizonts develop, mature, and release merozoites through rupture of the endothelial cells. This cycle repeats itself in the large intestine followed by the sexual life cycle of macrogametocyte and microgametocyte production. These stages of the life cycle also produce intestinal destruction. Fertilization of the gametocytes produces oocysts, which are shed coincident with the development of diarrhea and dysentery. The prepatent period (and development of diarrhea) may be 15-20 days.

(d) Laboratory tests. Oocysts are seen in the feces 2-4 days after the onset of dysentery. Therefore, clinical signs may be present without demonstrable oocysts. Coccidia may be found in the feces of normal calves, so laboratory data must correlate to clinical findings. Direct fecal smears and fecal floatations are the most common laboratory tests used.

(5) Therapeutic plan

(a) Individual treatment. Coccidiosis is a self-limiting disease but causes death in severely affected animals. Clinical signs subside when the multiplication stages have passed. Most coccidiosids suppress early first-stage schizonts; therefore, treatment of clinical disease with coccidiosids is of limited value. Other supportive care for the individual animal (e.g., fluid therapy) may be warranted under certain circumstances.

(b) Herd treatment. Mass medication, decreasing stock densities, and removing feed and water from ground level are all treatment strategies used in a herd outbreak. Medications include sulfadimethoxine at 2.72 mg/kg orally daily for 3-5 days and amprolium at 10 mg/kg orally for 5 days.

(c) Prevention. Avoid overcrowding, overcrowding, and feeding from ground level. Coccidiosids may be efficacious and necessary under conditions that do not allow for management alterations. Control medications include sulfadimethoxine, amprolium, decoquinate, monensin, and lasalocid.

d. Chronic salmonellosis. Salmonellosis in adult cattle usually presents as an acute, explosive, mucoshemorrhagic diarrhea (see I I B 1 a). However, when salmonellosis has become enzootic, chronic diarrhea and unthriveness may be observed.

Johnes' disease (paratuberculosis)

(1) Patient profile and history. This disease occurs in adult dairy cows and beef cattle older than 2-3 years of age. There is a breed disposition, with Short-horn, Angus, and Channel Island dairy breeds being over-represented. This may be an effect of historical numbers possibly related to breed popularity, similar to the way Holsteins are affected now.

(2) Clinical findings. Early clinical disease presents as intermittent or continuous watery or "pea-soup" diarrhea associated with stress (e.g., parturition). Vital signs are normal, and appetite is good to excellent. Emaciation becomes progressive, and milk production decreases. Terminal signs include profound emaciation, profuse watery diarrhea, and dependent edema.

(3) Etiology and pathogenesis

(a) The disease is caused by Mycobacterium paratuberculosis. The infection is contracted principally by the ingestion of feedstuffs or water contaminated by animals that are fecal shedders. Nursing calves may ingest the organism off the dam's udder. Calves born to clinically affected cattle may be infected in utero.

(b) Age of infection. Animals are most susceptible to infection as calves (less than 30 days of age), and most animals with the disease are infected before 4 months of age. However, clinical disease develops 2-5 years later.

(c) Age of shedding. Animals may become fecal shedders 15-18 months before the development of clinical signs. Some animals are shedders without ever becoming clinically affected. The organism may be persistent in suitable soils.

(d) Route of infection. Following oral ingestion, M. paratuberculosis localizes in the small intestine and associated lymph nodes. The bacteria multiply, and the animal either becomes resistant, a shedder, or a clinical case. Animals that are infected older than 6 months of age apparently are able to mount an effective immune response and develop resistance.

(e) Disease progression. In those animals in which the organisms multiply unchecked, there develops a granulomatous enterocolitis initially around the ileocecal valve. The enterocolitis spreads, and a malabsorption syndrome results with a protein-losing enteropathy. There is a compensatory increase in protein synthesis by the liver, but eventually hypoproteinemia occurs with signs of edema.

(f) Disease progression. In those animals in which the organisms multiply unchecked, there develops a granulomatous enterocolitis initially around the ileocecal valve. The enterocolitis spreads, and a malabsorption syndrome results with a protein-losing enteropathy. There is a compensatory increase in protein synthesis by the liver, but eventually hypoproteinemia occurs with signs of edema.

(g) Disease progression. In those animals in which the organisms multiply unchecked, there develops a granulomatous enterocolitis initially around the ileocecal valve. The enterocolitis spreads, and a malabsorption syndrome results with a protein-losing enteropathy. There is a compensatory increase in protein synthesis by the liver, but eventually hypoproteinemia occurs with signs of edema.

(h) Disease progression. In those animals in which the organisms multiply unchecked, there develops a granulomatous enterocolitis initially around the ileocecal valve. The enterocolitis spreads, and a malabsorption syndrome results with a protein-losing enteropathy. There is a compensatory increase in protein synthesis by the liver, but eventually hypoproteinemia occurs with signs of edema.

(i) Disease progression. In those animals in which the organisms multiply unchecked, there develops a granulomatous enterocolitis initially around the ileocecal valve. The enterocolitis spreads, and a malabsorption syndrome results with a protein-losing enteropathy. There is a compensatory increase in protein synthesis by the liver, but eventually hypoproteinemia occurs with signs of edema.

(j) Disease progression. In those animals in which the organisms multiply unchecked, there develops a granulomatous enterocolitis initially around the ileocecal valve. The enterocolitis spreads, and a malabsorption syndrome results with a protein-losing enteropathy. There is a compensatory increase in protein synthesis by the liver, but eventually hypoproteinemia occurs with signs of edema.
(a) Fecal culture is the most reliable and frequently used diagnostic test for infection in individual cattle. DNA probes and fecal culture by radiometric technique are also available and considered valuable diagnostic tools.

(b) Serological tests offer some value in determining herd status of infection but often must be carried out as a series of tests. Problems with sensitivity and specificity are common. These tests include complement fixation test (CFT), agar gel immunodiffusion test (ACID), and ELISA (the least costly and most accurate serological test).

(c) Other tests. Tests for cell-mediated immunity vary in their reliability. The intradermal and intravenous infections tests are available. Lymphocyte immunostimulation tests are also available, of which some are highly reliable.

(6) Therapeutic plan. Therapy is as yet unwarranted because response to treatment with medications (e.g., streptomycin, isoniazid, clofazamine) is only transient.

(7) Prevention
(a) Methods of herd management. The disease may be kept in check on a herd basis by test and slaughter methods. Removal of reactor or culture-positive animals and their offspring is necessary. This needs to be combined with improved hygiene practices, which decrease the fecal–oral spread of the organism. Calves must be removed from the dams at birth and reared separately. The herd should be maintained as a closed herd or replacement animals purchased from known Johnes-free herds.

(b) Problems with herd management. The disease may be effectively eliminated from a herd, but the management changes required are intensive, rigorous, and expensive. Because of the inaccuracies in diagnostic tests, there is as yet no test and slaughter program that effectively eliminates all carriers. Therefore, the only effective method of elimination is one of re-population of a new environment with unexposed or known negative animals.

(c) Vaccination. If local legislation permits, vaccination of calves may provide protection against clinical disease and reduce the rate of spread of infection. A major complication of a vaccination program is that vaccines are positive to the tuberculin test for bovine tuberculosis.

f. Bovine leukemia (enzootic bovine leukosis). Bovine leukemia is of major significance, but the gastrointestinal effects are of limited importance to the overall clinical picture and health management of the disease.

(1) Patient profile and history. The disease is seen most commonly in adult dairy cows. History related to gastrointestinal signs includes intermittent diarrhea. Several procedures help limit the spread of infection within herds including feeding colostrum to newborn calves from BLV-free animals.

(ii) Housing calves in individual hutches
(iii) Control of insect vectors
(iv) Disinfection of blood-bearing fomites to individual use (e.g., rectal sleeves, needles)
(v) Disinfection of veterinary instruments
(vi) Embryo transfer

(3) Etiology and pathogenesis
(a) The pathogenesis of the various forms is covered under the appropriate systems. Gastrointestinal signs are a result of tumor growth in the abomasal wall. Ulceration and gastrointestinal bleeding follows.

(b) Etiology. Enzootic bovine leukemia is caused by the bovine leukemia virus (BLV), a type-C retrovirus. Infection with the virus is common, but the development of solid tissue tumors is less common, depending on host genetic and environmental factors. Persistent lymphosarcoma and bovine lymphosarcoma are manifestations of the endemic form of bovine leuko-

s. The usual incubation period between infection and development of clinical signs is 4–5 years.

(c) Horizontal transmission via blood or contaminated instruments, which transmit infected lymphocytes, is thought to be the most common method of infection. Therefore, insect bites, surgical instruments, rectal sleeves, and contaminated needles have all been implicated in viral transmission.

(d) Vertical transmission is possible through contaminated semen and transplacental exposure to the virus during gestation. There is a familial tendency in the development of disease, indicating the possibility of a genetic predisposition.

(4) Diagnostic plan. The diagnosis of bovine lymphosarcoma as causative for gastrointestinal disease relies on definitive tests, necropsy or exploratory laparatomy with abomasotomy. Clinical findings of diarrhea with palpably enlarged lymph nodes or other multicentric expressions increase the level of suspicion.

(5) Laboratory tests. Laboratory tests that support a diagnosis include abdominal lymph node form—superficial, palpably enlarged lymph nodes

Cardiac form—muffled heart sounds due to hydropericardium, dyspnea due to hydrothorax, jugular vein engorgement, brisket edema, and bottle law.

(d) Nervous form—posterior paralysis
(e) Respiratory form—upper respiratory noise and dyspnea
(f) Ocular form—exophthalmos

(3) Etiology and pathogenesis
(a) The pathogenesis of the various forms is covered under the appropriate systems. Gastrointestinal signs are a result of tumor growth in the abomasal wall. Ulceration and gastrointestinal bleeding follows.

(b) Etiology. Enzootic bovine leukemia is caused by the bovine leukemia virus (BLV), a type-C retrovirus. Infection with the virus is common, but the development of solid tissue tumors is less common, depending on host genetic and environmental factors. Persistent lymphosarcoma and bovine lymphosarcoma are manifestations of the endemic form of bovine leukemia.
(5) Therapeutic plan. There is no approved treatment for this disorder, although the condition may resolve if the animals are taken off an offending pasture. However, salvage has shown experimental efficacy. Salvage is recommended.

h. Primary copper deficiency

(1) Patient profile and history. The condition usually occurs in young adult cattle (1-3 years) that graze on sandy or peat soils. The condition presents as a herd problem. There is no breed predisposition.

(2) Clinical findings. In clinically evident cases, animals are consistently poor in appearance with persistent diarrhea. Occasional degeneration is evident among the rumen. Lameness may be evident as is anemia, decreased milk production, or occasionally sudden death. CNS signs (e.g., incoordination, coma occur in lambs (see Chapter 11).

(3) Etiology and pathogenesis. Copper may be inadequate in the diet because of soil deficiency or unavailability. Lack of copper limits the cytochrome oxidase system through decreased production of ceruloplasmin. Failure of these enzyme systems result in the many manifestations of copper deficiency.

(4) Diagnostic plan. The diagnosis may be supported by laboratory data (generated from herd-level sampling) and response to treatment.

(5) Laboratory tests. Plasma and tissue (liver, hair) copper levels are low, but interpretation may be difficult because of wide variations in values for individual animals. Anemia is evident on a CBC. The diet may be analyzed for copper levels.

(6) Therapeutic plan

(a) Oral dosing of 4 g of copper sulfate for young animals and 8-10 g of copper sulfate for mature cattle weekly for 3-5 weeks is recommended. Controlled-release boluses are also available, but absorbed amounts may be less than desirable. Copper oxide fragmets for oral dosing may be the most reliable and efficacious method of supplementation.

(b) Injectable copper treatments may provide advantages over oral forms in terms of ease in administration and rapidity of results.

(c) Prevention. Treatment must be followed by oral maintenance. Oral dosing weekly with 5 g of dietary supplementation in the mineral mix is recommended. Salt licks containing 2% copper sulfate should be adequate. Dietary modification to ensure 10 parts per million (ppm) copper as measured in the dry matter of the final ration is also adequate. Top dressing of the pasture with copper sulfate at a rate of 10 kg/ha may be employed.

i. Secondary copper deficiency (molybdenum toxicity)

(1) Pathogenesis. E. coli adheres to the intestinal epithelium via pili, which usually possess the K-99 antigen. When adherent, ETEC produces a heat stable toxin (ST). This result in a secretory diarrhea (loss of fluid and electrolytes), mediated by cyclic guanosine monophosphate (cGMP). The ST leaves the glucose (glycine Na+ transport system intact but interferes with the Ca++-mediated Na+ Cl- co-transport system.

(b) In addition to the bacteria, contributing factors to the development of diarrhea include:

(i) Intensive management conditions (e.g., overcrowding, communal feeding)

(ii) Synergism with other diarrhea-producing agents (e.g., rotavirus)

(iii) Ingestion of insufficient quantities of or substantial quality of colostomy.

(4) Diagnostic plan. Clinical findings must be supported by a laboratory diagnosis to allow appropriate recommendations to be made regarding herd prevention.

(5) Laboratory tests

(a) Fecal or intestinal culture reveal ETEC often based on polyvalent antibody testing. ELISA test kits for demonstration of the K99 antigen are also available.

(b) For the individual calf, laboratory findings show varying degrees of acidemia, hypoglycemia, and hypokalemia. Clinical pathologic findings are reflective of hemococoncentration (e.g., elevated BUN and hematocrit). Total protein levels may be normal if hypoproteinemia is coincident with dehydration.

(6) Therapeutic plan

(a) Antibiotics. Diarrhea caused by ETEC is often self-limiting without antibiotic therapy since supportive care is instituted early. However, antibiotics often are used with success in the field (e.g., sulbactam–ampicillin, trimethoprim–sulfas, and water is dragged along the osmotic gradient). Part of this rationale is because of the difficulty of accurately differentiating between septicemic colibacillosis and diarrhea due to ETEC.

(b) Supportive care for the individual calf is an absolute necessity. Oral replacement solutions are efficacious early in the course of the disease. Later, it becomes necessary to replace lost fluids and electrolytes intravenously.

(i) Oral replacement solutions work on the principle of an intact glucose absorption mechanism in the gut. Sodium is absorbed via coupling (e.g., sodium bicarbonate), and an energy source (e.g., glucose). The oral agents should be used in calves in small quantities frequently, based on replacement needs and ongoing losses. It is not necessary to take calves completely off milk, but decrease quantities and alternate with electrolyte feedings. Do not feed oral electrolytes and milk simultaneously as this may interfere with milk clot formation in the background.

(ii) In calves more than 8% dehydrated, intravenous replacement and oral therapy are necessary. Isotonic NaHCO3 (1.3%) is often administered in conjunction with balanced electrolyte solutions (0.85% sodium chloride) and isotonic dextrose (5%).

(c) Other therapies include nursing care to keep the calf warm and dry and intestinal protectants (e.g., kaolin–pectin combinations, bismuth...
Subsalicylate). There is no evidence that anticholinergics or oral antibiotics influence the course or magnitude of the diarrhea.

7. Prevention. Total prevention of this condition is usually an unrealistic goal. A control program is built on the principles of reduction of exposure of neonates through hygienic and management practices, provision of adequate colostrum, and vaccination of the dam or calf. Many of these practices are difficult to fully achieve and require creative modifications, depending on the numbers of animals and the population at risk (i.e., dairy, beef, or veal calves).

b. Rotavirus diarrhea

1. Patient profile. Rotavirus causes diarrhea in all breeds of calves from 5 to 7 days up to 3 weeks of age. This virus often is found in mixed infections with ETEC and cryptosporidia.

2. Clinical findings. Clinical findings in pure rotavirus infections are diarrhea, dehydration, and anorexia. The condition may last for a few days, and recovery is usually uneventful. Combined infections with other pathogens (e.g., Cryptosporidium) result in a clinical picture of undifferentiated neonatal diarrhea indistinguishable from ETEC or combined enteric pathogens.

3. Etiology and pathogenesis
(a) The condition is caused by one of several strains of RNA rotavirus. The virus attacks absorptive cells at the tips of the villi of the small intestine. Loss of these mature epithelial cells results in malabsorption (lactase washout), osmotic diarrhea, dehydration, electrolyte loss, and acidois.
(b) Intestinal regeneration and epithelial cell function return to normal within approximately 7 days, although normal growth rates for the calf may take 10–21 days to return.

4. Diagnostic plan. Accurate diagnosis depends on laboratory confirmation.

5. Laboratory tests. The virus may be isolated from fresh feces or intestine. Tests that should be performed include electronmicroscopy, immunofluorescence, latex agglutination, and ELISA.

6. Therapeutic plan. The treatment is as for ETEC or undifferentiated neonatal diarrhea [see II B 3 a (6)].

7. Prevention
(a) Management strategies. The principles of control are the same as for undifferentiated neonatal diarrhea—limit exposure to the organism, ensure colostral intake, and increase specific antibody levels by vaccination of the calf or dam.
(b) Vaccination of calves has given less than satisfactory results in field studies. For vaccination of the dam to provide protection to the calf, it must occur at a time of colostrum production, and continued feeding of milk or colostrum with high antibody titers during the times calves are susceptible to infection is necessary. This may require management changes in veal or dairy operations.

Coronavirus diarrhea

1. Patient profile. Coronavirus causes diarrhea in beef or dairy calves under a variety of management practices. The age range of infected calves is generally 5–20 days.

2. Clinical findings. Clinical findings are similar to other cases of neonatal diarrhea, with the exception that flecks of frank blood may be seen in the feces of coronavirus-infected calves.

3. Etiology and pathogenesis. Coronavirus replicates in and damages the villus epithelium of both the small and large intestines. The crypt cells are also damaged, which results in a longer rejuvenation time for cell repair and replacement. Loss of epithelial cells results in malabsorption, malacidosis, and diarrhea.

4. Diagnostic plan. Diagnosis is based on confirmatory laboratory tests as for rotavirus infection [see II B 3 b (4), (5)].

5. Therapeutic plan and prevention. Therapy and prevention are as for rotavirus infection, with the added caveat that calves with coronavirus diarrhea take longer to recover because of crypt cell destruction. Convalescence may be prolonged and weight loss significant. Attention to nutritional supplementation may be necessary.

d. Cryptosporidiosis diarrhea

1. Patient profile and history. Neonatal dairy or beef calves are affected.

2. Clinical findings
(a) The clinical signs are indistinguishable from other causes of neonatal diarrhea. Calves are usually 1–3 weeks of age. Tenesmus may be a feature of the condition as is weight loss and the persistence of the diarrhea.
(b) Appearance. Severe dehydration, weakness, and recumency are not characteristic of uncomplicated cases of cryptosporidiosis.

3. Etiology and pathogenesis
(a) The causative agent is the protozoan parasite Cryptosporidium parvum. Infective oocysts are ingested and develop through six stages within the lower small intestine, large intestine, and cecum. The parasite produces villus atrophy, impairment of digestion, and absorption with a resultant mild diarrhea in uncomplicated cases. The organism is frequently seen in combination with other agents that cause neonatal diarrhea. This increases the severity of the diarrhea and the clinical effect on the calf.
(b) The prepatent period is 2–7 days, and sporulated oocysts may be passed for 3–12 days in the feces.

4. Diagnostic plan. Diagnosis depends on laboratory confirmation.

5. Laboratory tests. Fecal oocysts may be detected by fecal floatation, direct staining of fecal smears, immunofluorescence, or ELISA.

6. Therapeutic plan
(a) Medical therapy. Diarrhea with uncomplicated cases of cryptosporidiosis is self-limiting. However, there is no recommended or specific treatment for cryptosporidiosis. On an experimental basis, the anticoccidial agent halofuginone has been used with some success at 60–125 mg/kg orally for 7 days.
(b) Supportive therapy. Other treatments are supportive and similar to treatments of all diarrheas (e.g., warmth, oral or intravenous fluids, milk feeding in small quantities several times daily, oral protectants).

7. Prevention
(a) The only control at present is to limit exposure of calves to the organism. Procedures used include:

(i) Segregation of infective calves
(ii) Separation of feeding utensils
(iii) Manure removal
(iv) Disinfection of the environment with 5% ammonia or chlorine dioxide-based disinfectants

(b) Clients should be warned that C. parvum is a zoonotic agent and causes diarrhea in humans. The condition has serious implications in immunocompromised individuals.

e. Giardiasis

1. Patient history and etiology. Giardia [e.g., G. duodenalis] has been recovered in feces of diarrheic calves. Its etiological significance is yet to be proven because infection occurs experimentally, but clinical signs do not develop.

2. Clinical findings and therapeutic plan. A syndrome is described of Giardia-associated, chronic, pasty diarrhea lasting for 2–6 weeks. Growth is depressed. Fenbendazole at 11 mg/kg has shown efficacy against this parasite.

3. Giardia species cause disease in humans, so a zoonotic potential exists when calves are excreting the organism.

f. Salmonellosis

1. Patient profile and history. Salmonella usually affects calves older than 10–14 days of age.

2. Clinical findings. Three syndromes have been described in calves 10 days to 3 months of age. These syndromes are:

(a) Peracute—sudden death, neurological, or gastrointestinal signs (abdominal pain, diarrhea)
(b) Acute—fever, anorexia, depression, diarrhea, and dehydration. Diarrhea progresses from watery to mucus/epithelial casts to hemorrhagic feces.

(c) Chronic—loose feces with poor growth rates and ill-thrift.

(3) Etiology and pathogenesis. Salmonellae dublin and S. typhimurium are the most common Salmonella isolates. The pathogenesis of the diarrhea is similar to that described for salmonellosis in the adult (see II B 1 d (3)). A bacteremic form also may exist.

(4) Diagnostic plan. Diagnosis is supported by laboratory findings and farm history.

(5) Laboratory tests. Necropsy and culture findings of feces or intestinal contents confirm the diagnosis. Repeated culture attempts may be necessary to isolate the organism from feces.

(6) Therapeutic plan and prevention. Therapy and prevention are as for the adult (see II B 1 d (7), (8)).

g. Clostridial diarrhea

(1) Patient profile and history. This type of diarrhea may be reported as an outbreak in rapidly growing, vigorous, nursing calves. It is more commonly reported as a disease of older calves or sheep.

(2) Clinical findings. Sudden death may be the most common presentation. Less acute disease may present as abdominal pain, distention, and hemorrhagic enteritis.

(3) Etiology and pathogenesis

(a) *Clostridium* perfringens normally is found in the intestinal tract and may proliferate at times of abrupt feed changes or overfeeding of carbohydrates. Enterotoxins (alpha, beta, epsilon, and iota) produced by the various organism types produce the clinical signs.

(b) *C* perfringens type A produces a hemorrhagic enteritis in Europe.

(c) *C* perfringens type B causes diarrhea in many types of neonates in Europe, South Africa, and the Middle East.

(d) *C* perfringens type C causes hemorrhagic enteritis of calves in North America.

(e) *C* perfringens type E causes necrotic hemorrhagic enteritis but is rare.

(4) Diagnostic plan. A tentative diagnosis may be made based on subjective and clinical findings but requires laboratory confirmation.

(5) Laboratory tests. Necropsy and/or mouse inoculation with intestinal contents and neutralization studies with specific antitoxin confirm the diagnosis.

(6) Therapeutic plan. Therapy with antibiotics (penicillin) and hydration support should also contain 10%-20% fat, which is an energy source and limits diarrhea.

(i) Infectious bovine rhinotracheitis may cause diarrhea in young calves as part of a systemic infection (see Chapter 6 II B 6 a).

(j) Bovine viral diarrhea is discussed in II B 1 a.

(k) Prolonged antibiotic therapy. Prolonged oral antibiotic therapy may predispose calves to intestinal overgrowth of pseudomonas, proteus, or fungi. Intractable diarrhea may result.
4. What is the most likely diagnosis?
(1) Bovine virus diarrhea (BVD)
(2) Salmonellosis
(3) Winter dysentery
(4) Arsenic poisoning
(5) Rotavirus diarrhea

5. What is the appropriate next step?
(1) Treat all affected animals with potentiated sulfonamides, flunixin meglumine, and oral or intravenous fluids.
(2) Treat all affected animals with broad-spectrum antibiotics and intestinal protectants.
(3) Treat all affected animals with oral charcoal and systemic British antilewisite (BAL).
(4) Run serologic tests to confirm the diagnosis.
(5) Wait for the disease to run its course. Supportive care (e.g., fluid therapy and astringents) is indicated for dehydrated animals.
The answers are 1 [II B 3]. Included on a list of differential diagnoses for diarrhea in a calf younger than 1 week of age would be enterotoxigenic Escherichia coli (ETEC) diarrhea, coronaviruses diarrhea, and rotavirus diarrhea. Salmonellosis, clostridiosis, and coccidiosis are usually a cause of diarrhea in older calves. Primary disaccharidase deficiency has not been reported in calves. Giardia lamblia, although recoverable from the feces of some calves, is unproven as a cause of neonatal calf diarrhea. Transmissible gastroenteritis (TG) is a disease of swine.

2. The answer is 1 [I E 1]. Traumatic reticuloperitonitis (TRP, hardware disease, traumatic gastritis, traumatic reticulitis) is most common in mature dairy cows. Clinical signs include an abrupt drop in milk production (to less than 50% of normal) and odd postures (e.g., the animal may stand with its hind feet in a gutter in an attempt to relieve diaphragmatic reticulitis). A right, not a left, paramedian approach is recommended to collect peritoneal fluid for laboratory analysis. Conservative therapy entails confinement and administration of antibiotics; rumenotomy may be necessary if conservative management fails and economics warrant surgical intervention.

3. The answer is 4 [I A 3]. The clinical findings suggest vagal indigestion. The history may include mild but repeated bouts of transient indigestion with signs of anorexia, decreased milk production, mild bloating, weight loss, abdominal distention, and decreased amounts of manure. The animal may have experienced an episode of traumatic reticuloperitonitis (TRP) in the past. Chronic bovine virus diarrhea (BVD) is seen in young animals with diarrhea, a stunted growth pattern, and lameness. Simple indigestion resolves within a few days. Acute local peritonitis (while perhaps the initial cause of this chronic disease) would have resolved. Intractable diarrhea is the most evident complaint and finding in animals with Johne's disease.

4-5. The answers are: 4-3, 5-5 [II B 1 el. The most likely diagnosis is winter dysentery. The subjective findings (e.g., housed cattle, month) support a diagnosis of winter dysentery. The cows present as essentially normal, except for an explosive outbreak of diarrhea. These findings eliminate salmonellosis and arsenic poisoning from the list of differential diagnoses. Rotavirus has not been demonstrated as an etiologic agent for diarrhea in adult ruminants. A virulent virus type responsible for bovine virus diarrhea (BVD) would cause similar, but more severe, clinical findings (e.g., severe dehydration).

A bovine coronavirus is the etiologic agent of winter dysentery. Treatment consists of supportive care and waiting for the disease to run its course. Antibiotics are unnecessary and ineffective. British antilewisite (BAL) is a treatment for arsenic toxicity. Serologic studies would be of no value for diagnosis.

6. The answer is 4 [I A 2]. In a convulsing animal in shock as a result of grain overload, surgery is not economically warranted because these animals have a poor prognosis for recovery. Lactic acidosis resulting from grain overload occurs when gram-positive rods overgrow the normal rumen flora. Absorption of both D- and L-lactate occurs, but only L-lactate causes acidemia. The severity of the clinical signs depends on the amount and particle size of the grain ingested; finely ground feeds are associated with more severe clinical signs. Administration of oral magnesium oxide to sheep not showing signs of shock and dehydration is one appropriate therapy.

7. The answer is 1 [II B 2 b]. Calves that have been exposed to the bovine virus diarrhea (BVD) virus in utero are not born with mucosal disease; rather, they may develop a fatal mucosal disease sometime later after being exposed to a cytopathic form of the virus. Affected calves are immunotolerant to the virus but are unable to mount a humoral antibody response against it. Affected calves may appear normal at birth, or they may suffer from Ill thrift. Affected calves are chronic shedders of the BVD virus.

8. The answer is 1 [II B 1 b]. Bovine malignant catarrh (BMC) occurs sporadically and is caused by a wildebeest-associated virus (in Africa) or a sheep-associated virus (in North America). The virus is not known to be spread cow-to-cow. Disease outbreaks are often associated with ewes lambing near cattle. BMC causes vasculitis, atypical proliferation of lymphocytes, panosteitis, and lymph node enlargement and is frequently accompanied by nervous system signs.

9. The answer is 4 [I C 1]. High dietary concentrate: roughage ratios increase the production of volatile fatty acids (VFAs). VFAs are known to decrease intestinal motility; it is hypothesized that this leads to gas build-up within the abomasum and eventual displacement of the left abomasum. It is thought that abomasal tone is also negatively affected by low serum calcium levels, circulating toxins, and lack of exercise in deep-bodied cows.