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Diseases of the Gastrointestinal System

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Infectious and noninfectious diseases of the gastrointestinal tract (GIT) are very common in small ruminants. A complete history and thorough physical examination are of utmost importance in the characterization of gastrointestinal disease in small ruminants, with attention made during the physical examination to body condition score (BCS), abdominal contour, manure characteristics, and motility of the reticulorumen. Combined use of auscultation, percussion, and ballottement over the entire abdomen should be carried out, especially given preclusion of rectal palpation in sheep, goats, and most cervids. Even with a complete physical examination, localizing the exact nature of GIT disease can be difficult. The physical examination can be augmented using ancillary tests, including the assessment of clinicopathological parameters including rumen fluid evaluation, as well as the use of imaging modalities. If indicated, an exploratory laparotomy can serve both as a diagnostic and therapeutic tool.

Diagnostic Procedures

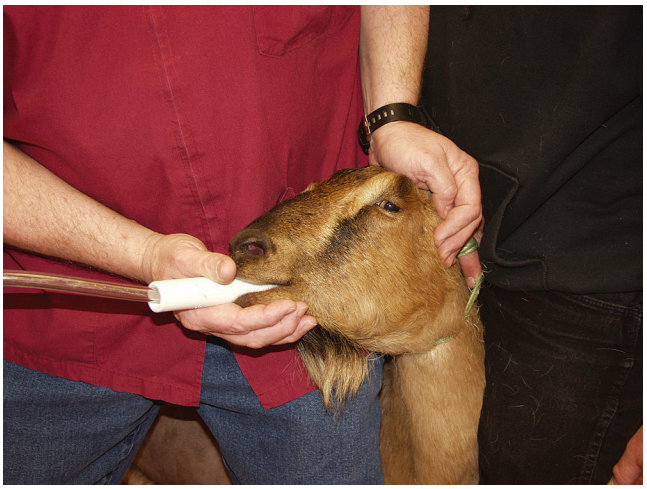
Basic Laboratory Studies

Basic clinicopathological analyses include a complete blood cell count (CBC), serum biochemistry, and urinalysis. These tests, along with diagnostic imaging, help define the differential list generated from the physical examination. Furthermore, they can be helpful in determining the severity of disease, prognosis, and response to therapy with serial evaluation. Rarely, a specific disease is identified based on clinicopathological tests. Usefulness of the CBC includes the evaluation of the erythrogram and leukogram to characterize the severity of anemia, dehydration, and inflammatory response. Interpretation of the packed cell volume (PCV) should be done in conjunction with total protein concentration as well as the estimation of dehydration on physical examination. An anemic or dehydrated hypoproteinemic animal may have a normal PCV and total protein. Both the CBC and serum biochemistry can be helpful in determining the presence and severity of an inflammatory disease process. Changes in the total and differential white blood cell counts indicate acute or chronic inflammation, ranging from neutropenia and a degenerative left shift to a mature neutrophilia. Increases in globulins or fibrinogen suggest a chronic inflammatory disease. Low protein levels, especially albumin, need to be further evaluated for the potential of chronic blood loss due to gastrointestinal parasitism, infiltrative bowel disease, liver dysfunction, third-space compartmentalization, or protein loss through the kidneys. Liver disease should be suspected if liver enzymes or bilirubin levels are increased. However, liver enzyme concentrations can be normal in the presence of chronic liver disease. Also, albumin levels rarely drop in ruminants with liver disease as they do in other species. Liver function tests may be performed, including measurement of bile acids and blood ammonia concentrations. Point-of-care (POC) meters for the stall-side measurement of blood glucose, L-lactate, and ketone concentrations have been evaluated in cattle and small ruminants.[1–4] Urine strips for the detection of ketone bodies are also useful in monitoring ewes and does at risk for pregnancy toxemia. Changes in electrolytes are common with gastrointestinal diseases, especially in anorexic animals or those with profound diarrhea. Electrolyte measurements are also useful in the formulation of treatment plans. Although abomasal disease is rare in small ruminants, a metabolic alkalosis with hypochloremia and hypokalemia may be observed. Gastrointestinal stasis can result in hypokalemia, hypochloremia, and a mild hypocalcemia. In the case of surgical intestinal obstructions, small ruminants can develop severe metabolic acidosis due to ischemic necrosis of tissues and shock. The major biochemical changes commonly associated with diarrhea are metabolic acidosis, with the loss of sodium and bicarbonate in diarrheic feces, and the presence of azotemia and hypoproteinemia. Renal disease should be ruled out in these cases.

Normal ranges for clinicopathologic laboratory values are available in Appendix II, Tables 1 to 5 and are published elsewhere. However, familiarity should be made with normal values of both a CBC and serum biochemistry as established by the laboratory commonly used for analyses in their practice.

Rumen Fluid Analysis

Rumen fluid analysis is useful in characterizing the health of the forestomach and aids in differentiation of diseases, including types of vagal indigestion, ruminal acidosis, and potential intoxications. Collection of rumen fluid can be via orogastric or nasogastric intubation or percutaneous rumenocentesis. Regardless of method chosen, proper restraint of the animal and suitable equipment (Figure 5.1) should be used to avoid trauma to the esophagus or abdominal viscera and damage of equipment (e.g., chewed tubes). To perform percutaneous rumenocentesis, a 16- to 18-gauge, 3-inch (7.6-cm) needle is inserted into the rumen fluid below the fiber mat. The site for rumenocentesis can be estimated by ballottement or percussion of the left flank (approximately 5–10 cm



• **Fig. 5.1** Passage of an orogastric tube through a mouth speculum made from a polyvinylchloride (PVC) pipe. To avoid oral and esophageal trauma, the animal should be well restrained, and the tube should be lubricated and passed slowly down the esophagus.

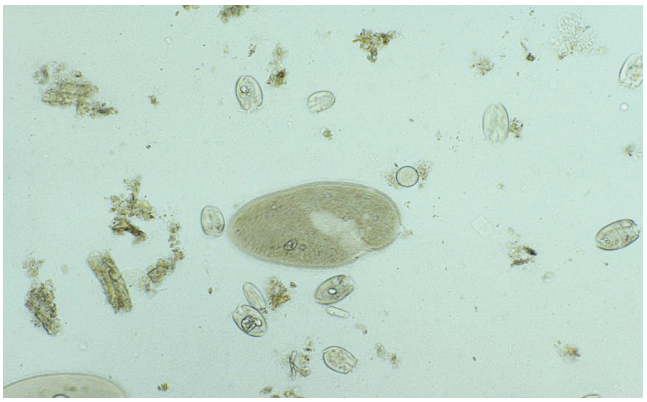
caudal to the last rib along an imaginary line drawn at the level of the patella)[5] or, alternatively, at a ventral location caudal to the xiphoid and to the left of midline.[6] Use of sedation and/or a local block with 2% lidocaine may be necessary in fractious or patients likely to struggle. The site is clipped and aseptically prepared. The needle is introduced forcefully, in one swift motion. Once the rumen is entered, fluid is aspirated with a syringe. If the needle becomes obstructed with ingesta, a small amount of air or fluid should be forced backed through the needle. Rumenocentesis carries the advantage of avoiding salivary contamination of the sample, which can occur during orogastric intubation, as well as possibly being less stressful to the animal. The procedure does carry a slight risk of peritonitis, which is minimized through proper restraint. Rumenocentesis is contraindicated in pregnant females.[5,6]

Once collected, rumen fluid is analyzed for color, odor, pH, motility and types of protozoal species present, methylene blue reduction (MBR) time, Gram-staining characteristics, and chloride concentration. Normal rumen fluid characteristics are listed in Table 5.1. The pH of rumen fluid can be measured on pH strips with 0.5 increments or the use of sophisticated handheld meters. The sample pH will be falsely increased with salivary contamination.[7] Microscopic examination of a drop of fresh, warm fluid under a cover slip examined at 40× to 100× allows visualization of protozoa species, with no special staining required (Figure 5.2). Routine Gram staining is performed on a dried, fixed slide.[5] The MBR reflects the activity of bacterial fermentation in the rumen. It is performed by mixing 1 mL of 0.03% methylene blue with 20 mL of rumen fluid at normal body temperature and measuring the time required to return to the appearance of a control tube.[5] Rumen chloride concentrations can be determined from the supernatant of a centrifuged sample. Rumen chloride concentrations are minimally impacted by saliva contamination and a time lag between sample collection and analysis.[5] Normal rumen fluid is aromatic, olive to brownish-green, and has a pH between 6.5 to 7.5 depending on the diet fed. Microorganisms include a mixed population of large and small protozoal species with active motility and a predominance of gram-negative rods. Normal rumen chloride concentration is less than 30 mEq/L and MBR should be less than 6 minutes.[6] Changes observed in anorexic ruminants include thinner, darker

TABLE 5.1 Normal Rumen Fluid Characteristics of Sheep and Goats.

Characteristic	Normal Finding
Color	Green
Odor	Aromatic
pH[a]	6.5–7.5
Protozoa[b]	Mixed sizes and species rapidly moving
Methylene blue reduction time[c]	3–6 minutes
Gram stain	Gram-negative rods predominate
Rumen chloride	Less than 25–30 mEq/L

[a]Use pH paper with at least 0.5-unit gradations.
[b]Place a drop of fluid on a warm slide and cover with a coverslip. Examine under 100× magnification.
[c]Mix one part 0.03% methylene blue to 20 parts rumen fluid. Measure time for blue color to clear to match a control tube of fluid.
Data from Nordlund KV, Garrett EF: Rumenocentesis: a technique for collecting rumen fluid for diagnosis of subacute rumen acidosis in dairy herds. *Bovine Pract.* 28:109, 1994; Keefe GP, Ogilvie TH: Comparison of oro-ruminal probe and rumenocentesis for prediction of rumen pH in dairy cattle, Proceedings of the 30th Annual American Association of Bovine Practice Convention, 1997, 168; Smith MC, Sherman DM: *Goat medicine,* 2nd ed., Ames, Iowa: Wiley-Blackwell, 2009



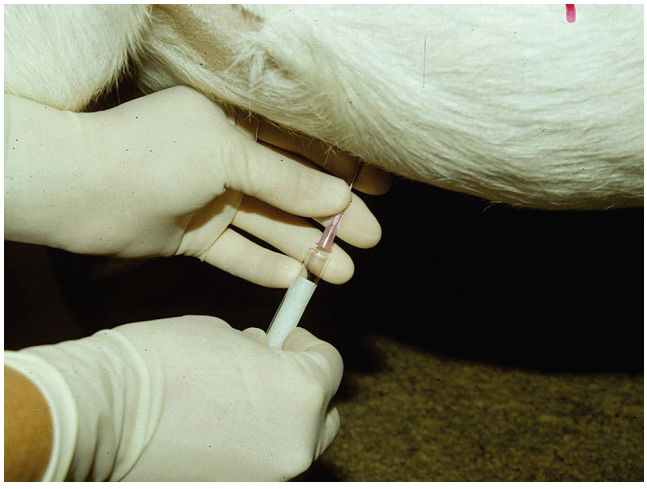
• **Fig. 5.2** Fluid obtained by rumenocentesis should be examined for both bacteria and protozoa. A drop of rumen fluid is placed on a microscopic slide and viewed under a coverslip. At low power (40×), normal rumen fluid will be observed to contain 35 to 40 organisms per field from several populations of protozoa, as seen here. Both low numbers and loss of motility signal a need for medical intervention or transfaunation.

fluid, with an increase in pH (7–7.5), and a reduction in the species and motility of protozoa present. In acute ruminal acidosis, the fluid is fetid, yellow to grey, with a low pH (< 5.2), and dead or no protozoa are present with a predominance of gram-positive rods (*Lactobacillus* species).[5,8] The MBR is prolonged with any type of indigestion/digestive disorder in which inactivity of the microflora is present. Increased rumen chloride concentrates indicate an abomasal or proximal small intestinal obstruction, either functional or mechanical, as a result of the internal reflux of hydrochloric acid from the abomasum into the reticulorumen.[9]

Abdominocentesis

Abdominocentesis is useful in the assessment of abdominal disease in ruminants and aids in the differentiation and diagnosis of

ascites, peritonitis, strangulating intestinal lesions, enteritis, uroperitoneum, and abdominal neoplasia. There is normally a small amount of transudative fluid present in the peritoneal space. Characterization of peritoneal fluid as a transudate (low protein concentration and cell count), modified transudate (normal or mild increase in cell count with increased protein concentration), or exudate (increased protein concentration and cell count) is important from a pathophysiological standpoint and allows refinement of possible differentials for the abdominal disease present. Characteristics of peritoneal fluid from healthy sheep and goats are similar to cattle: transparent, colorless to slightly yellow, $<$ 5 g/dL protein, and less than 5000 to 10,000 cells/mL.[10,11] Peritoneal fluid protein concentration can be measured using a refractometer. Other biochemical analyses may include determination of creatinine (e.g., to diagnose uroperitoneum), L-lactate, D-dimer, and glucose concentrations.[12,13] Cytologic examination is needed to characterize the types of cells present, the morphology of those cells, and to assess the presence of phagocytized bacteria. Typically, the cell population is made up of large mononuclear cells, lymphocytes, and non-degenerative neutrophils. Lymphocytes comprise $<$ 20% of cells present, and a few mast cells or plasma cells may be seen.[10] Both the absolute number and proportion of cell types present need to be considered. Changes in peritoneal fluid cytology following exploratory laparotomy, rumenotomy, and enterotomy are reported in goats.[14–16] Two methods can be used. The first technique involves tapping the cranial abdomen at its lowest point cranial to the umbilicus and slightly to the right of midline (Figure 5.3). This technique is useful in conditions with a significant amount of free fluid such as uroperitoneum. When using the cranial abdominal site, one needs to avoid the prepuce in males and the mammary veins in females.[6] The second technique is a four-quadrant approach, as ruminants are very proficient at walling off inflammatory and infectious foci (e.g., peritonitis), which can hinder successful fluid collection. The two cranial sites are slightly caudal to the xiphoid and medial to the milk veins on both sides. The two caudal sites are slightly cranial to the mammary gland and to the left and right of midline.[5] For either technique, manual restraint with sedation is recommended; the use of real-time ultrasonography may help locate fluid pockets. Importantly, amniocentesis or allantocentesis can occur at these sites during gestation and caution is warranted.



• **Fig. 5.3** Ventral and caudal sites for performing abdominocentesis. The needle indicates the ventral site. The caudal site is the clipped area below the flank.

An 18- to 20-gauge needle or teat cannula can be used for fluid collection. The site should be clipped and prepped using sterile technique and local anesthesia provided when a teat cannula is used. Fluid should be collected in an ethylenediaminetetraacetic acid (EDTA) tube for cytological analysis and a sterile red top tube or suitable inoculation vial for aerobic and anaerobic culture. Abdominal fluid can be difficult to obtain because of the small amounts normally present in both small ruminants. It is important to minimize the ratio of EDTA to fluid in the sample because EDTA can falsely increase the protein levels. Using EDTA tubes made for small animals, filling tubes to at least one-quarter full, or shaking excess EDTA out of large tubes resolves this problem. Air-dried, unstained slides should be prepared and shipped with EDTA tubes for samples shipped to an external laboratory for analysis.[10]

Radiography

Radiography of the abdomen can be performed in small ruminants using small animal techniques. In adult small ruminants, the rumen normally fills the entire abdomen. In cattle, radiography is a useful tool in demonstrating reticular metallic foreign bodies and changes suggestive of traumatic reticuloperitonitis.[17,18] Radiography of the abdomen may also demonstrate the displacement, distortion, distention, or superimposition of abdominal structures, as well as the presence of soft tissue opacities, gas-fluid interfaces, or abnormal gas inclusions.[19] Contrast techniques are useful for diagnosis of atresia of the rectum or colon. Unlike in small animals, contrast techniques are not possible for characterizing small intestinal problems in small ruminants because the rumen dilutes and slows passage of the contrast media.

Ultrasonography

Ultrasonography is well suited for examination of the ruminant GIT and other abdominal viscera. Ultrasonography allows the characterization of contour, dimensions, content, and motility patterns of the forestomach and intestines, as well as the presence of masses, intraluminal and free abdominal fluid, and lesions within the parenchyma of abdominal viscera. Ultrasonography also can be used to guide fluid and tissue sampling for abdominocentesis and biopsy of organs or masses, respectively.[20] Normal parameters for the forestomach compartments, small and large intestines, liver, and spleen have been described in small ruminants.[21,22] Imaging is best achieved using a linear or convex transducer with a frequency of 3.5 to 5.0 MHz. The reticulum is imaged in the cranioventral abdomen, bilaterally, as a crescent-shaped structure immediately adjacent to the diaphragm. Goats demonstrate monophasic, biphasic, and triphasic reticular contractions.[23] The rumen is visualized in the 8th through 12th intercostal spaces (ICS) and flank on the left, and from the 12th ICS and flank on the right. The rumen wall appears as a thick echoic line, and the ability to differentiate the gas cap, fiber mat, and fluid layer is variable. Rumen motility is discerned indirectly by changes seen in layering of the ruminal content. The dorsal and ventral rumen sacs are most easily distinguished caudally by the presence of the longitudinal groove.[24] The omasum is found on the right side from the 6th to 11th ICS (mainly in the 8th and 9th ICS), appears as a crescent-shaped echoic line medial to the liver, and moves passively with respiration due to its proximity to the diaphragm. Due to the gaseous nature of omasal content, the omasal leaves and omasal wall furthest from the transducer cannot

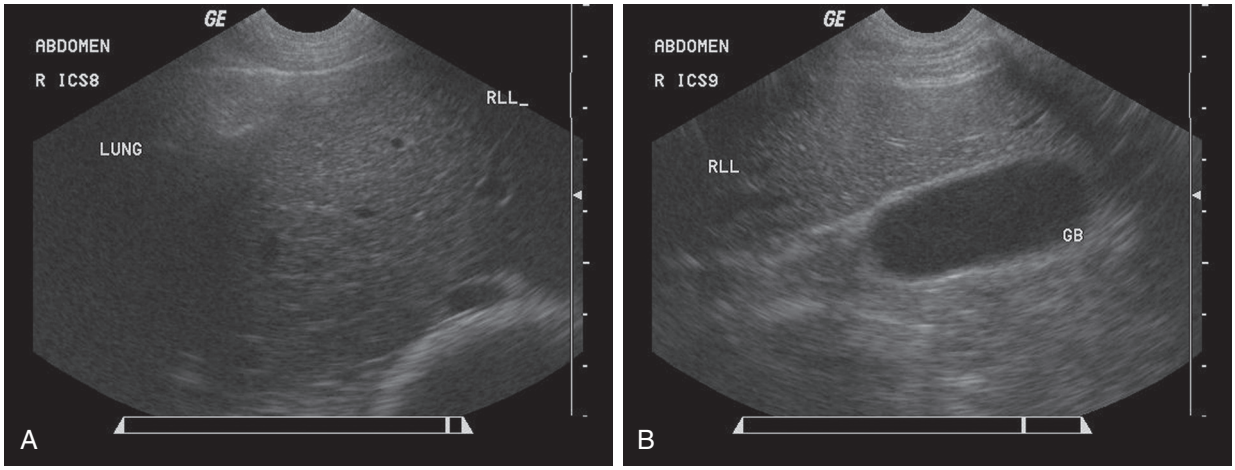
be visualized.[25] The abomasum is visualized along the ventral midline and to the left and right paramedian areas as a heterogeneous, moderately echoic structure with echogenic stippling. Visualization of abomasal folds as prominent echoic bands is possible in approximately two-thirds of goats.[26] Examination of the small intestine takes place from the 8th to 12th ICS and the flank on the right side, from dorsal to ventral midline. Similarly, the large intestine (i.e., spiral colon and cecum) is visualized in the right flank. The descending duodenum can be differentiated based on proximity to abdominal wall and location between two serosal layers of greater omentum, whereas the jejunum and ileum cannot be differentiated from each other. Normal luminal diameters and wall thickness of the small intestine are described in normal goats.[27] The spiral colon and cecum are visible in the caudal right flank. The spiral colon often located medial to the small intestine, is garland-like in appearance, and visualization of only the wall closest to the transducer is possible due to intraluminal gas, which is also true of the cecum.[27] Ultrasonography of the liver for position, parenchymal and surface appearance, as well as visualization of the caudal vena cava, portal vein, and gall bladder are evaluated on the right side between the seventh and ninth ICS (largest visible extent of liver) and variably between the fifth to sixth and the 10th to 12th ICS. The parenchymal pattern of the normal liver consists of numerous fine, homogeneous echoes (Figure 5.4). On cross-section, the caudal vena cava is triangular in shape and is visualized in approximately 75% of goats in the 11th and 12th ICS. The portal vein always has a more ventral position and is closer to the liver surface compared with the caudal vena cava. It is circular to oval in cross section, with stellate ramifications into the liver parenchyma, and typically is visualized in all ICS in which liver is visible. The gall bladder is variable in shape and size, depending on amount of bile present and is visualized in most goats from the 9th to 10th ICS.[28] The spleen is visualized on the left side from the 11th and 12th ICS, situated between the rumen and abdominal wall. The parenchymal pattern consists of numerous, homogeneous, weak echogenic shadows.[29] Description of ultrasonography of the urinary and female and male genital tracts can be found in Chapters 12 and 8, respectively.[30]

Other Imaging Modalities

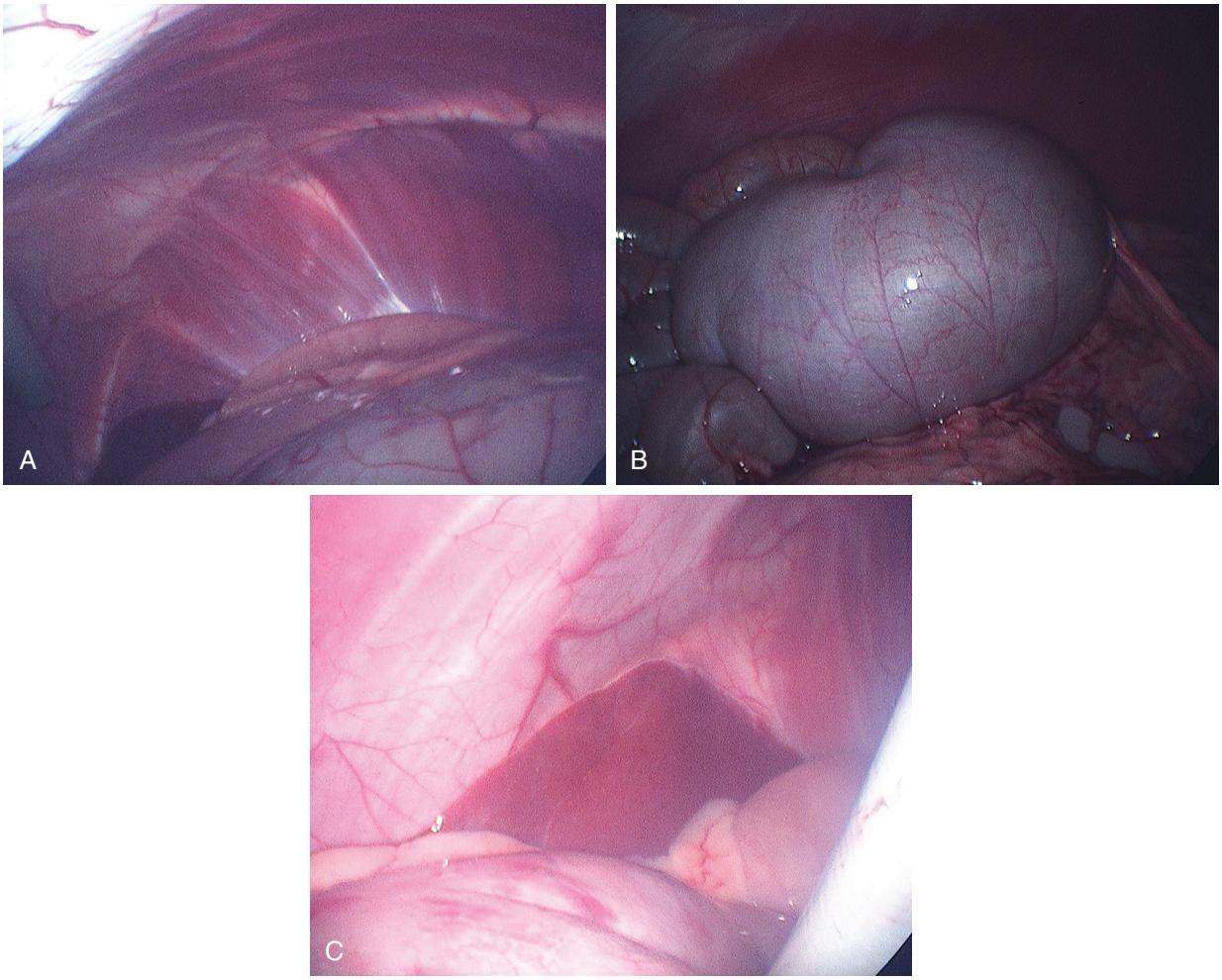
Although limited in its availability to referral centers, characterization of the thorax and abdomen in goats using computed tomography (CT) has been described.[31–34] Use of CT and magnetic resonance imaging (MRI) is of considerable expense and requires general anesthesia in most cases. However, given the use of sheep and goats as animal models for human medicine, as well as the growing popularity of goats as companion animals, the application of these imaging modalities will likely continue to increase.

Laparoscopy

Laparoscopy is more commonly used as a reproductive tool, but it also can be used diagnostically as an alternative to exploratory laparotomy in small ruminants.[17–21] General anesthesia is recommended to allow more inflation of the abdominal cavity and thus a more thorough examination, but laparoscopy can be done with sedation and local anesthesia at portal incision sites. The technique for laparoscopic exploration of the abdomen used for cattle and llamas can be modified for use in sheep, goats, and cervids.[18–21] Laparoscopic evaluation of the abdominal cavity is usually done via a ventral approach with the animal secured in dorsal recumbency. The abdominal cavity can be inflated with CO_2 by a needle, teat cannula, or after placement of a laparoscopic cannula. A time-saving method is to use suture to make a "bite" through the skin and into the external rectus sheath which can be used to tense the body wall. A stab incision can then be made in the skin and external rectus sheath before introducing a guarded trocar



• **Fig. 5.4** **A.** Ultrasound image of the right abdomen obtained from the right eighth intercostal space in a 3-year-old La Mancha cross doe, showing the right liver lobe with the characteristic hepatic and portal veins represented by the small, tubular anechoic structures within the liver parenchyma. The ventral border of the lung is seen on the left side of the image. This ultrasound scan was obtained using a 7-MHz microconvex transducer. Dorsal is to the left of the image. **B.** Ultrasound image of the right abdomen obtained from the right ninth intercostal space of the same animal as in **A**, demonstrating normal right liver lobe and gallbladder. The gallbladder appears as an anechoic, fluid-filled structure directly adjacent to the right liver lobe. This ultrasound scan was obtained using a 7-MHz microconvex transducer. Dorsal is to the left of the image. (Courtesy Dr. Karine Pader, Purdue University.)



• **Fig. 5.5** A laparoscopic examination (performed using a 10-mm-diameter direct vision scope) of the abdomen in a 2-year-old Pygmy buck. **A.** The muscle fibers of the diaphragm are evident cranially in the center of this photograph. A small part of the liver is in the lower left of the image. **B.** The larger organ in the center of this photograph is the cecum. It normally appears darker in comparison with other portions of the intestine and contains ingesta of a doughy consistency. **C.** This photograph shows part of the liver on the right body wall.

into the abdominal cavity while tensing the abdominal wall with the previously placed suture. The laparoscope can then be placed through the trocar and the abdomen inflated while visualized through the scope. The clinician places the cannula in the inguinal area as described for laparoscopic insemination (see Chapter 8). This technique allows a more efficient use of time and minimizes the likelihood that the omentum will be "ballooned". Laparoscopic placement into the right side allows visualization of most of the abdominal organs (Figure 5.5 A, B, C). Obviously, the clinician should avoid the rumen when introducing the laparoscope into the abdomen. This procedure may be enhanced by lowering the head or rear of the animal, allowing better visualization of the entire abdomen. Visualization of the abdominal cavity and the ability to manipulate organs will be greatly improved by fasting the animal 24 to 48 h or at least decreasing the bulk in the diet. Respiration must be monitored closely, and assisted ventilation should be available during this procedure because inflation of the abdomen and lowering of the head can put pressure on the diaphragm.

Exploratory Laparotomy

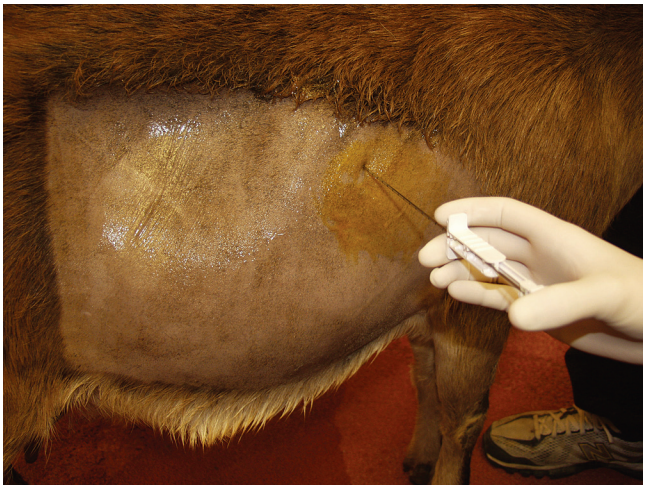
Exploratory laparotomy can be a valuable diagnostic tool in evaluating gastrointestinal diseases when other tests indicate abdominal disease. It is often indicated in the ruminants presenting for acute abdomen. In some cases, therapeutic surgical procedures can be performed at the same time. The technique of exploratory laparotomy used in cattle can be adopted for small ruminants with the understanding that these animals are more likely to lie down during surgery. Therefore, standing surgery is the exception, with most performed with the patient in lateral or dorsal recumbency.

For this procedure, small ruminants should be heavily sedated or placed under general anesthesia. Use of a high-volume lumbosacral epidural can augment sedation and minimize the use of inhalant anesthetics needed. The use of perioperative antimicrobials and nonsteroidal antiinflammatories (NSAIDs) should be based on the clinical status of the animal, diagnostic findings found at surgery, as well as the environment in which the surgery

takes place. Antimicrobial agents are not necessary for elective exploratory surgery performed aseptically, in a hospital setting, and without complications. However, antimicrobials are indicated under field conditions if infection is present, and if the forestomach or intestinal tract is opened (i.e., clean-contaminated surgery). A combination of ceftiofur (1.1–2.2 mg/kg intravenous/intramuscular/subcutaneous [IV/IM/SC] twice a day) and procaine penicillin G (22,000 IU/kg IM twice a day) or potassium penicillin (22,000 IU/kg, IV, every 6 h) can be administered until clinicopathological tests and bacterial culture results indicate an absence of infection. Use of NSAIDs (e.g., flunixin meglumine, 1.1 to 2.2 mg/kg, IV, every 12–24 h) for pain control and potential antiendotoxemia effects should be utilized when indicated. Other medications for control of pain may be required in some small ruminants, such as the short-term use of opioids (see Chapter 18). Postoperative care should include fluid therapy and rumen support (e.g., transfaunation, B-vitamins, highly palatable diet) in depressed and anorectic patients.

Liver Biopsy

Liver biopsy in sheep, goats, and cervids is performed using a similar technique used in cattle, but access to the liver in small ruminants is more limited. Therefore, whenever possible, ultrasound guidance is recommended. The biopsy is performed in the standing animal that is well restrained and sedation used as necessary. The recommended biopsy site is the ninth intercostal space slightly above an imaginary line drawn from the point of the elbow to the craniodorsal angle of the paralumbar fossa (Figure 5.6).[6] Other techniques, including laparoscopic liver biopsy are described.[35–39] The site should be surgically prepared and a local anesthetic (2% lidocaine) infused subcutaneously. A small scalpel blade is used to make a stab incision through the skin. A 14-gauge, 11.5-cm liver biopsy instrument (e.g., Tru-Cut biopsy needle) is inserted through the incision and the intercostal muscles and into the liver. The biopsy instrument is directed toward the opposite elbow, but the use of real-time ultrasonography greatly aids in determining the direction and depth needed (2 to 4 cm).[5] Perforation of the gall bladder as well as large vessels along the caudal border of the rib should be avoided. Samples can be submitted for culture (in a sterile plastic or glass tube), histopathologic study (in formalin, at a 10:1 ratio of formalin to tissue), or mineral analysis (in a trace element or plastic tube). The laboratory should be contacted for appropriateness of sample containers and specific instructions. When performing a liver biopsy for mineral analysis, the clinician should rinse the biopsy site with distilled and deionized water after sterile preparation to minimize sample contamination. Samples for mineral analysis should not be placed in formalin. Closure of the skin incision can be accomplished by suture or stapling, or if it is small enough, the wound can be left alone to heal by second intention. Fly repellent should be applied to the site as needed. Use of antimicrobials is at the discretion of the veterinarian, and should be considered in regions where *Clostridium novyi* or *Clostridium haemolyticum* are prevalent.[39] Vaccination status for clostridial diseases should be up-to-date, and if there is any doubt, a toxoid vaccine given before or at the time of biopsy.



• **Fig. 5.6** Liver biopsy: after the skin is clipped, anesthetized, and aseptically prepared, the surgeon makes a stab incision in the skin and introduces a 14-gauge biopsy needle.

Diseases of the Forestomachs

Bloat

Bloat is less common in sheep compared with cattle, despite sheep having more selective eating habits (e.g., leaves over stems) and a tendency to select legumes over grasses which would promote the occurrence of bloat. Sheep are more tolerant than cattle to nonscabrous diets, as well as more tolerant of increases in intraruminal pressure. Dramatic increases in intraruminal pressure in sheep results in rapid changes in the frequency and type of ruminal contraction patterns responsible for eructation, promoting evacuation of intraruminal gas.[40] Goats are less commonly affected than sheep, and deer are remarkably resistant to bloat.

Bloat results as a failure in eructation of gases produced by microbial fermentation in the rumen. Most commonly, eructation fails because the gas remains trapped as tiny bubbles throughout the rumen ingesta (i.e., frothy bloat).[41] Other variations of this type of bloat include foamy or slime bloat, dependent on the class of animal and diet fed (e.g., high concentrate diets).[42,43] Another type of bloat is the build-up of free gas in the rumen with concurrent failure to eructate (e.g., free-gas bloat).[44] An example is the presence of an intraesophageal foreign body (e.g., potato, sugar beet) impairing eructation.

Pathogenesis. Acute, frothy bloat commonly occurs in animals grazing pastures of bloat-provoking forages. These include alfalfa, clovers (red, white, alsike, ladino, and sweet clover), and certain cereal grains, in contrast to forages relatively resistant to bloat such as sainfoin, birds-foot trefoil, and crown vetch.[45] Field conditions allowing rapid vegetative growth and ingestion of legumes during the vegetative (i.e., prebud) stage pose particular bloat risk, but bloat can occur on dry forage and at full bloom. Bloat-promoting forages are prone to rapid cell disruption and degradation with the release of soluble proteins and other constituents. The highly soluble plant protein ribulose-1,5-bisphosphate carboxylase/oxygenase is especially important in contributing to the formation of stable foam in the rumen.[46,47] This entrapment of gas within the rumen contents is the primary cause of frothy bloat. The stable foam prevents the coalescence of gas bubbles as well as the clearance of the cardia, impairing eructation. Frothy bloat can also occur on high-grain diets with limited roughage content. Plant factors that suppress bloat include condensed tannins, higher stem to leaf ratios, and plant lipids.

Reports of frothy or free-gas bloat are rare in farmed deer. A low incidence of frothy bloat likely reflects differences in forage

browsing preferences, feedstuffs, pastures typically utilized on farmed deer operations, and an inherent ability of the rumen flora and physiology to consume and tolerate high tanniferous forages. For example, deer have a high concentration of proline-rich tannin-precipitating protein in their saliva, as well as alterations in rumen microflora that accommodate high tannin-containing diets. By forming insoluble complexes with plant proteins, tannins limit the production of stable foam in the rumen and prevent bloat in cattle, small ruminants, and deer species.[48,49]

The occurrence of slime (frothy) bloat under feedlot conditions (i.e., high grain diets) has a similar pathogenesis with the formation of a stable foam in the rumen, preventing eructation. When large amounts of corn or other cereal grains (e.g., barley) are fed, an excess release of mucopolysaccharides and other constituents from rumen bacteria and protozoa occurs. This alters the rumen fluid viscosity and allows a stable slime (or foam) to form.[42,43] Compounding the issue is a reduction in the amount of saliva contributing to the rumen liquor due to reduced rumination on diets lacking adequate fiber. Saliva normally acts as a buffer and limits the formation of a stable foam. Also, episodes of ruminal acidosis, commonly encountered on high-grain diets, can contribute to alterations in rumen motility patterns and the development of free-gas bloat.[43]

In the case of free-gas bloat, failure to eructate has a variety of causes. Physical obstruction of the esophagus with intraluminal foreign bodies (e.g., feedstuffs, masses) or extraluminal compression of the esophagus (e.g., enlarged mediastinal lymph nodes, thymoma) can result in bloat, ranging from mild to severe ruminal tympany, depending in the completeness of obstruction. Diseases of the rumen wall can result in mechanical disruption of normal motility patterns, impairing eructation. Systemic diseases, damage to the nerves innervating the esophagus and forestomach, electrolyte imbalances, endotoxemia, and pain can impair eructation.[50–52] Use of alpha2-agonists (e.g., xylazine) can impair reticulorumen motility, thereby altering eructation.[53,54]

Clinical Signs. Clinical signs of frothy bloat and free-gas bloat due to physical obstruction of the esophagus can be severe and life-threatening compared with bloat due to rumen wall or systemic diseases. Ruminal tympany is observed in the left paralumbar fossa which can extend above dorsal mid-line. The animal may appear anxious and demonstrate a tense abdominal wall and signs of colic. Changes in abdominal contour may be subtle and difficult to fully appreciate in heavily fleeced animals. The rumen may be either hypomotile or hypermotile. Due to compression of the diaphragm and lungs, respiratory distress with flaring of nostrils, open mouth breathing, and an altered stance are common. Death can be rapid if ruminal tympany is left untreated.[40,50]

Diagnosis and Treatment. Bloat is a medical emergency, necessitating decompression of the rumen and stabilizing the animal before a thorough workup is performed. If the animal is not in immediate danger of dying, an orogastric tube can be passed. Most cases of free-gas bloat are relieved with passage of the tube. A thorough history and complete physical examination are then indicated to find the cause of the free-gas bloat. If the bloat is not relieved with passage of an orogastric tube, the tube should be removed and examined for evidence of froth. Frothy bloat can be treated with poloxalene (44 mg/kg) or dioctyl sodium sulfosuccinate (DSS) (28 mL [1 oz]) delivered by orogastric tube. If frothy bloat is due to high-concentrate feeding, the pH is less than 5.5 and may be treated with mineral oil (100 mL, PO) and/or poloxalene. In emergency situations, other surfactants and detergents may be attempted, including peanut oil (20 to 50 mg/kg), vegetable oil (100 to 200 mL), and hand soap (10 mL).

If the animal is in severe respiratory distress, the clinician should insert a trocar or large needle into the rumen at the paralumbar fossa. If gas does not escape, or froth is seen coming out of the trocar, an emergency rumenotomy is indicated. With occurrence of bloat in multiple animals of a pastured group, the entire group should be removed from the pasture and reintroduced slowly after gradual acclimation. If only one or two cases of bloat are encountered, the healthy animals can remain on the offending pasture, but grazing should be limited to ensure gradual acclimation.

Prevention. Prevention of frothy bloat includes pasture management and use of antifoaming agents. Cultivated pastures should be seeded to grass-legume mixtures, with fertilizing and grazing management maintaining 50% or less of bloating legumes on pasture, depending on the incidence of bloat, as this percentage may need to be decreased to < 25 to 30%.[41] Use of nonbloating legumes in grass mixes may be used depending on geographic location as well as nutritive and carrying capacity of pastures required. Under intensive grazing conditions, management of exposure (e.g., creep, swath grazing with 24–48 h of wilting) and use of legume varieties engineered to possess less bloat risk can be utilized (e.g., AC Grazeland).[55,56] Grazing legumes with high leaf-tannin concentrations (e.g., arrowleaf clover, kudzu) usually are safer because tannins form insoluble complexes with legume proteins, which help break down stable foam in the rumen.[41] Monitoring of pasture conditions and recognizing weather events that impact the incidence of bloat should be viewed with respect to effects on plant growth. Examples are the avoidance of grazing during the presence of heavy dew (morning and evening), recent heavy rains, and frost. Important is the recognition that alfalfa still poses a bloat risk even after a killing frost and the observation of the predominant forage present on pastures in order to assess the bloat risk (e.g., regrowth of alfalfa faster than grasses in the fall or the selective grazing habits of the herd).[45]

Limiting access to offending pastures and feedstuffs, with slow introduction over the course of 2 to 3 weeks, should be carried out. Prior to introduction to offending pastures or feedstuffs (and when intermittently housed off pastures, e.g., overnight), sheep or goats should be fed to satiety with a coarse roughage. Offering supplemental roughage (e.g., grass or cereal hays) while on bloat-provoking pastures can be attempted but ensuring intake can be problematic and economically cost prohibitive.

Natural and synthetic surfactants are effective in preventing bloat when administered at the recommended levels. Use of poloxalene is widely used in cattle and appears to be efficacious in sheep, although the level required in sheep may be higher per unit of bodyweight (BW) compared with cattle and more variable in its control.[57] Inclusion of poloxalene in concentrate feed mixes as either a top-dressing or as pelleted premix can be fed twice daily at a rate of 2 to 4 g/100 kg BW. Water-soluble formulations delivered in metered-water sources have been found efficacious in grazing sheep and are available in other countries.[56,58] Inclusion in mineral supplements (e.g., salt molasses blocks, liquid molasses lick feeders) is another alternative. However, ensuring adequate intake of poloxalene from water sources and mineral blocks can be variable and should be monitored under extensive grazing conditions, with blocks most useful in small pastures. The efficacy and economic validity of these uses in small ruminants have not been critically evaluated under field conditions. Caution is warranted with respect to copper levels in minerals intended for cattle use when used in small ruminants. Use of poloxalene-containing

products should be continued for 1 to 2 weeks prior to moving animals onto bloat-promoting pastures.

Oils (e.g., soybean, corn, peanut, olive) and emulsified tallow also exhibit good bloat control. However, disadvantages include rapid degradation in the rumen, requiring large doses. Mineral oil is effective but is problematic due to its laxative effect and impairment of vitamin A metabolism in the rumen.

Free-gas bloat from concentrate feeds can be controlled by slow introduction to these feeds to allow for rumen adaptation, proper balancing of a ration, type of grain and its processing, and bunk management, as well as the inclusion of ionophores in the diet.[43] Monensin (15 mg/head/day in sheep and 1 mg/kg/day in goats) and lasalocid (0.5 to 1 mg/kg/day in sheep and goats) both decrease the formation of free ruminal gas.[59] By enhancing propionic acid formation, these drugs not only reduce the amount of methane produced in the rumen but also improve the efficiency of nutrient assimilation from feedstuffs.[43]

Bloat in lambs and kids can have the same causes as in adults but also can be caused by improper milk feeding. Overfeeding, feeding of large infrequent meals, and feeding spoiled or cold milk have all been associated with bloat in lambs and kids. Rapid overdistention of the abomasum and improper chemical or physical composition of milk replacers both will inhibit rumen motility, leading to bloat. Even though the feeding of cold milk has been associated with bloat, the practice can be used effectively in orphan feeding programs. Lambs and kids tend to limit their intake of cold milk after they have become accustomed to a free-choice feeding system that delivers refrigerated milk. Milk usually is placed in the rumen when animals are tube-fed; this may result in milk spoilage.

Simple Indigestion

Simple indigestion is a mild form of upset of reticulorumen function caused most often by a change in feed. This can be the sudden addition of grain or other concentrates to the diet, or alteration of the energy provided, such as a change in grain processing. Changes in pasture and hay or ingestion of toxic plants or moldy hay or grain can also cause simple indigestion. Clinical signs include reduced feed intake to anorexia, diarrhea, and bloat, which are mild in their characteristics and short-lived, often resolving within 1 to 2 days. Minimal to no changes in rumen fluid characteristics may be observed depending on the cause. Most mild cases of simple indigestion resolve without therapy. Appropriate steps should be taken if the cause can be identified.

Rumen Acidosis

Ingestion of rapidly fermentable sugars and starches, such as corn and small cereal grains (e.g., barley, wheat, oats) as well as bread, candy, apples, and fruits can result in dramatic changes in ruminal fermentation and the development of ruminal acidosis. The common name for this condition is "grain overload". The type of grain processing (e.g., flaking, rolling) reduces the size of the feed particles and allows more rapid fermentation by rumen bacteria. Rumen acidosis commonly follows excessive consumption of offending feedstuffs (accidental or inappropriate ration formulation), abrupt changes in the diet not allowing for adaptation of the rumen microflora, inconsistent delivery of ration, or mixing errors. The severity of the disease depends on the composition of the feed, particle size, amount of feed consumed, and the period of adaptation to the diet.

Pathogenesis. Under normal conditions, a low concentration of lactate is found in the rumen and is rapidly metabolized by lactate utilizers such as *Selenomonas ruminantium* and *Megasphaera elsdenii*. The introduction of high-concentrate diets with rapidly fermentable sugars and starches leads to unbalanced ruminal fermentation and the accumulation of lactic acid.[60] Initially, the excess fermentable carbohydrates cause a general increase in the growth rate of all bacteria with a resultant increase in volatile fatty acid (VFA) production, which lowers ruminal pH. Bacterial species tolerant of lower ruminal pH, specifically *Streptococcus bovis*, outpace other bacterial species, resulting in increased lactate production. An increase in lactate concentration further decreases the rumen pH, and eventually it falls to a level where death of protozoa and gram-negative bacterial spp. occurs. In addition, the growth of *S. bovis* is inhibited and only very acid tolerant lactate-producing *Lactobacillus* spp. predominate. The rumen pH can decrease to 5, and in severe cases to less than 4.0. Lactate production (L- and D-isoforms) continues to increase.[61,62] The osmolality of the rumen fluid increases which pulls fluid from the systemic circulation and interstitium into the rumen. Stasis of reticulorumen motility, mucosal damage, and absorption of lactic acids, inflammatory mediators, as well as bacteria and endotoxins into the peripheral circulation results.[63–65] Clinically, dehydration, hypovolemic shock, acute inflammatory response, and metabolic acidosis result.[63] Depending on the severity of metabolic derangement, thiamine deficiency and the development of polioencephalomalacia (PEM) can occur.[66] Sequelae of severe ruminal acidosis or recurrent bouts of subacute ruminal acidosis may include laminitis, mycotic ruminitis, and occasionally liver abscessation; although, the latter is far more common in cattle than in small ruminants.[65,67–69]

Clinical Signs. Clinical manifestations vary with the amount and type of feed ingested and the time since ingestion. Clinical signs first appear 12 to 36 h after ingestion of the offending feed, ranging from anorexia, depression, reduced rumen motility, nasal discharge, and diarrhea. The presence of weakness, ataxia, or recumbency can develop in animals suffering from circulatory shock and severe metabolic derangements. Dehydration usually is severe, and evidence of toxemia is present (e.g., tachycardia, altered body temperature, infected mucous membranes, and scleral vessels). Rumen stasis, ventral abdominal distension, and a fluid-filled rumen are found on abdominal auscultation and percussion. Signs of abdominal pain, such as bruxism, stretching, and kicking at belly may be observed. Osmotic diarrhea commonly occurs, which can worsen the severity of dehydration. Diarrhea can range from pasty feces to soupy, watery diarrhea with the presence of whole grain/corn. Neurological deficits such as blindness, ataxia, head pressing, opisthotonus, seizures, and other abnormalities can develop due to thiamine deficiency PEM, as well as other metabolic derangements and endotoxemia.

Diagnosis. Examination of rumen fluid should be carried out in suspected cases of ruminal acidosis. Rumen fluid is milky colored, foul smelling with acidic odor, and has a reduced pH (below 5.5). Protozoa numbers and types are markedly reduced with poor to absent motility. Methylene blue reduction time is markedly prolonged (> 9 minutes) in most cases. The normal 60:40 ratio of gram-negative to gram-positive bacteria is altered with a predominance of Gram-positive rods (*Lactobacillus* spp.). Rumen lactic acid concentration is increased.[62] Clinicopathologic laboratory data include hemoconcentration (increased PCV and total protein), prerenal azotemia, and metabolic acidosis, characterized by a low blood pH, low blood bicarbonate concentration, and a negative base excess.[62,70] Renal compensation results in acidic

urine production after 12 h, along with an increase in urine-specific gravity.[70] Dehydration and impaired tissue perfusion, as well as an overwhelming delivery of inflammatory mediators and endotoxins to the liver can result in increased liver and muscle enzymes, depending on the severity of the disease. Changes in the leukogram and acute-phase proteins reflect an acute inflammatory response, ranging from normal to a degenerative left shift as well as increases in haptoglobin, ceruloplasmin, and reduced albumin concentration.[63] The transketolase test performed on peripheral blood can be used to determine the active thiamine status of the animal.[66] Increases in cerebrospinal fluid (CSF) leukocyte counts and total protein have been reported in sheep.[71]

Treatment. Treatment is aimed at correcting dehydration, metabolic acidosis, toxemia, and shock as well as the removal or neutralization of the offending feedstuff. Use of IV isotonic crystalloids supplemented with bicarbonate should be administered. Ideally, bicarbonate supplementation would be based on serum biochemistry analysis but can be empirically based on estimated base deficit. In certain instances, calcium may be indicated and can be added to the IV fluids (as calcium gluconate). The clinician should avoid mixing calcium salts and sodium bicarbonate. Administration of parenteral NSAIDs to alleviate pain and potentiate toxemia are indicated (e.g., flunixin meglumine 1.1 to 2.2 mg/kg, IV). Use of parenteral antibiotics is indicated in most cases, given a high likelihood of bacterial translocation and bacteremia. The systemic antimicrobial of choice is penicillin (procaine penicillin G, 22,000 IU/kg, IM, q12h, or potassium penicillin 22,000 IU/kg, IV, q6h) due to anaerobes being the most likely offending organisms. Use of oral fluids to restore hydration is contraindicated and counterproductive, as fluid absorption is impaired, and administration can worsen rumen distention and abdominal discomfort. Administration of agents to neutralize the rumen pH, such as magnesium hydroxide and magnesium oxide (1 g/kg, PO) can be sufficient in mild cases. However, if much of the feed is still in the rumen, these two alkalinizing agents will only work temporarily. The use of oral antibiotics is likely counterproductive, as their administration negatively impacts the regrowth of the healthy rumen microflora. Oral antibiotics are contraindicated as they have poor bioavailability (e.g., neomycin). If available, the animal should be transfaunated daily with rumen fluid from a healthy donor until rumen motility and appetite are restored. More effective, is the prompt removal of the offending feedstuff in order to curtail fermentation. Ruminal lavage is likely futile in most small ruminants, given the size limitation of orogastric tubes to allow sufficient bore diameter without becoming blocked with feed. Rumenotomy is indicated in severe cases of ruminal acidosis to remove the offending feed.

After the rumen pH is corrected, transfaunation of the sheep or goat with ½ to 1 L of rumen fluid from a donor animal (cow or small ruminant) is beneficial. Thiamine supplementation (vitamin B1, 10 mg/kg, SC, q6–8h) is indicated until rumen function is restored and is of utmost importance in animals demonstrating clinical signs suggestive of PEM. Supportive care should also include provision of grass hay and water when rumen motility returns in order to prevent excessive ruminal distention. With aggressive treatment, the prognosis for short-term survival is good. Delays in seeking medical treatment can result in poor outcomes and death in severe cases. Sequelae to ruminal acidosis (previously discussed), can significantly impact long-term survival and production.

Prevention. Prevention must involve addressing inappropriate management practices that put animals as risk for the development of ruminal acidosis, especially in classes of sheep and goats being fed high-grain rations (e.g., club lambs, feedlot lambs, dairy goats). A balanced diet with adequate forage and fiber should be formulated, properly mixed, and consistently delivered, along with adequate feeder/bunk space. The crude fiber content should constitute a minimum of 20% of the diet's total digestible nutrients (TDN). For example, if the TDN is 75%, the minimum acceptable crude fiber is 15%. Crude fiber levels lower than this can be fed for short periods if the rumen is properly adapted, but problems may nevertheless occur. In sheep and goats unaccustomed to high-concentrate rations, gradual introduction to increasing rates of inclusion should take place over several weeks, to allow for adaptation of the rumen microflora. In addition to a well-formulated ration, inclusion of rumen modifiers such as buffers, yeasts, and direct-fed microbials may also be utilized. Rumen buffers may improve milk production, increase feed intake, and increase rate of gain. Sodium bicarbonate probably is the most commonly used buffer; it can be offered on a free-choice basis or included in the diet as 1% of dry matter intake. Calcium carbonate or limestone (both of which have low rumen solubility) and magnesium oxide (which has poor palatability) also can be included in the feed. Magnesium oxide should be limited to 0.5 to 0.8% of the dry matter intake.

In the United States, direct-fed microbials refer to a source of live (viable), naturally occurring microorganisms which are used for supplementing microbes and modulation of the rumen microbiota, with the goals of maintaining a stable rumen pH, decreasing lactic acid, optimizing VFA production, and improving nutrient digestibility. Single or mixed bacterial cultures, as well as different species of yeasts can be found in commercial products, including *Lactobacillus acidophilus*, *Propionibacterium freudenreichii*, *Megasphaera elsdenii*, and *Saccharomyces cervisiae*.[72] Use of a yeast-based culture (*S. cerevisiae*) demonstrated a positive effect in the treatment and prevention of ruminal acidosis and potential sequelae in sheep under experimental conditions.[73,74] However, much research is needed as to the efficacy and proper use of pre- and probiotics in small ruminants.[75]

Reticulitis, Rumenitis, and Parakeratosis

Pathogenesis. Reticulitis and ruminitis can result from chemical or mechanical damage to the mucosal lining of the reticulorumen. The most common cause of chemical damage in sheep and goats is rumen acidosis. Rumenitis associated with a high carbohydrate supplemental feed has also been reported in white-tailed deer.[76] However, ingestion of caustic toxins also can damage the mucosa. Mechanical damage can occur from ingested foreign bodies or rumen bezoars. In cattle, viruses such as the agents of bovine virus diarrhea and infectious bovine rhinotracheitis can infect the rumen wall. Similar viruses have yet to be identified in sheep and goats.

After the mucosa has been damaged, secondary infection by bacteria or fungi can occur. Previous treatment with oral antibiotics may predispose animals to development of fungal infections of the rumen wall, especially if the mucosa is already damaged. Actinobacillosis, actinomycosis, and tuberculosis rarely affect the rumen wall. Tumors of the rumen wall also have been reported. Not all of these causes of reticulitis and ruminitis have been reported in sheep, goats, and cervids, but all are potential problems.

Clinical Signs. The clinical manifestations of these diseases are vague. Anorexia and forestomach hypomotility may be the only clinical signs.

Diagnosis. Confirming a diagnosis of these diseases also may prove difficult. Samples of rumen fluid may show only changes

• **Fig. 5.7** An 8-year-old female white-tailed deer's rumen showing enlarged ruminal papillae. The pen-raised doe had signs of chronic acidosis/ruminitis prior to death. She was in a pen with other does being offered an ad lib grain/carbohydrate feed with minimal forage. The rumen had enlarged, hardened, and fused together papillae, and diffuse, severe ruminal hyperkeratosis. (Courtesy of Dr. Kelley Steury, ALVDL, Auburn, AL.)

associated with anorexia (alkaline pH, decreased numbers and motility of protozoa, prolonged MBR time; see Table 5.1 for normal values). Occasionally, fungal organisms may be seen on Romanowski (Diff-Quick)-stained slides of rumen fluid. In such cases, a diagnosis of fungal ruminitis should be made. An exploratory laparotomy and rumenotomy may be required to identify foreign bodies or masses. Rumen parakeratosis is characterized by dark, thickened, and clumped rumen papillae. It is seen mainly in feedlot lambs that consume finely ground or pelleted rations. The parakeratotic rumen papillae are fragile and vulnerable to damage which can increase the risk for development of rumenitis (Figure 5.7).

Treatment and Prevention. Treatment depends on the inciting cause. Dietary changes should be made to decrease energy density and increase fiber intake. Mild ruminitis may subside with time and supportive care (e.g., transfaunation, fluid support, high-quality feed). Fungal rumenitis can be treated with oral thiabendazole, 25–44 mg/kg, when available. Severe changes may lead to scarring and permanent impairment of rumen function.

Diseases of the Reticulorumen

Traumatic Reticuloperitonitis

Traumatic reticuloperitonitis (hardware disease) is an uncommonly reported condition in small ruminants, unlike cattle in which hardware disease is a primary cause of vagal indigestion. The selective grazing and browsing habits of sheep and goats, respectively, likely limit the intake of sharp, metallic objects such as wire, nails, and needles. Goats appear to be affected more commonly than sheep.[77] Penetration of a foreign body through the reticular wall can result in reticulitis, localized or diffuse peritonitis, abscessation and adhesion formation, as well as the development of pleuritis, pericarditis, or myocarditis if the foreign body penetrates the diaphragm into the thoracic cavity. Clinical signs include depression, anorexia, poor body condition, reluctance to ambulate, altered rumen motility (e.g., bloat, vagal indigestion), and abdominal pain.[78] Involvement of the pleura or pericardium can present with signs of respiratory distress and heart failure.[79] Abscessation and draining tracts of the thorax and forelimbs may be present.[77] A thorough workup is required to determine the cause of vagal indigestion and the potential internal sites involved. This may include the use of radiographs and ultrasound, as these imaging modalities will help determine the extent of infection and the most suitable approach to treatment. An exploratory laparotomy can be both diagnostic and therapeutic. Physical examination and diagnostics will direct the surgical approach used (e.g., left versus right flank), as well as the need for a rumenotomy. Most animals will require stabilization with fluid therapy as well as long-term antibiotics and supportive care. Reticuloperitonitis carries a guarded to poor prognosis.[78,80]

Rumen Impaction

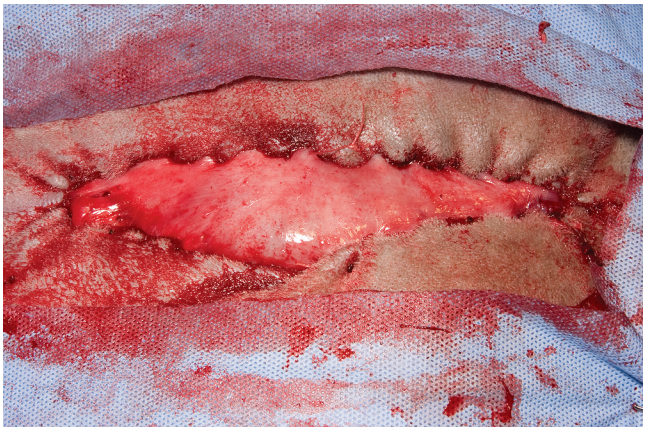
Rumen impaction as the result of feeding inappropriate forages or feedstuffs (e.g., high fiber diets with low digestibility; *Ficus esquiroliana*), sand ingestion, or consumption of indigestible foreign material (e.g., plastic) can lead to the disruption of normal reticulorumen motility and function, as well as partial or complete blockage of the omasal orifice.[81,82] Malnutrition and unbalanced dietary habits results in pica and ingestion of indigestible foreign materials and is of growing concern worldwide. Goats reared in suburban and urban environments are particularly at risk.[83] Clinical manifestations are non-specific, such as depression, weakness, anorexia, and ruminal atony. A firm rumen can usually be palpated in the left flank. Signs of vagal ingestion such as ruminal tympany and scant dry feces may be present. Prolongation of methylene blue reduction time reflects poor anaerobic fermentation in the rumen.[82] Oral fluids containing magnesium sulfate (60 g), mineral oil, or DSS administered daily for a week may resolve fibrous and sand impactions, but a rumenotomy is required in severe cases and for impactions involving indigestible foreign materials. Prevention includes use of feed troughs and racks to elevate feedstuffs off the ground to minimize sand intake as well as ensuring a properly formulated diet, including the provision of a loose mineral source.[84]

Rumenotomy. Exploratory celiotomy is both a diagnostic and therapeutic intervention in ruminants with vagal indigestion. The decision to perform a standing left or the right flank, or use of other positions in recumbency, will be dictated by physical examination, clinicopathological tests, and the temperament and stability of the patient. A standing or recumbent left flank celiotomy and rumenotomy is suited for type II vagal indigestion (e.g., hardware disease, perireticular abscess). A right-sided approach (right flank, right paramedian, or right paracostal) for exploratory celiotomy is suitable for type III and IV vagal indigestion (e.g., abomasal impaction, pyloric obstruction). Many small ruminants may become recumbent during a standing flank approach. Recumbency can be facilitated by using a lumbosacral epidural anesthesia and sedation or by inhalant general anesthesia in very fractious animals. In nonemergent situations, the rumen fill should be minimized by withholding feed for 24 h. However, many cases present as an emergency. Perioperative antibiotics should be administered and be efficacious against anaerobes. Examples commonly used include procaine penicillin, ampicillin, and oxytetracycline. Use of nonsteroidal anti-inflammatories (e.g., flunixin meglumine, meloxicam) is indicated perioperatively. IV fluids should be used to correct dehydration and cardiovascular shock, both concurrent with and following surgery, as needed.

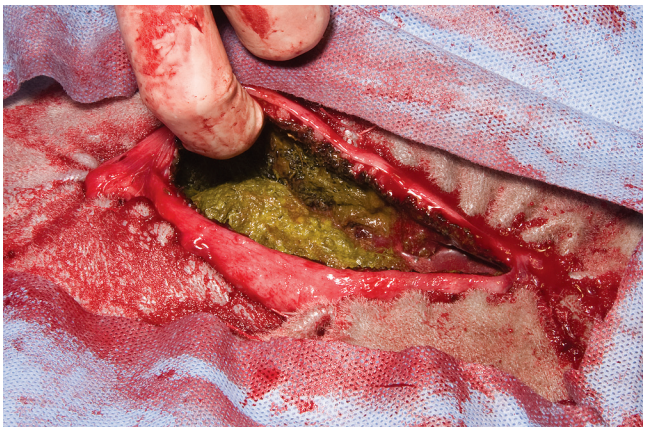
A brief description of a left flank celiotomy and rumenotomy is described herein.[78,85–87] In-depth review of other surgical and

laparoscopic celiotomy approaches can be found elsewhere. The surgical site encompasses an area including the last two to three intercostal spaces cranially to the paralumbar fossa, extending caudally to the tuber coxae, and from dorsal midline to the lower abdomen. The surgical site is clipped and aseptically prepared.

A routine vertical incision is made through the skin and abdominal muscles in the middle of the left paralumbar fossa. Because the abdominal wall is relatively thin, and the rumen may be very distended, the surgeon should take care not to enter the rumen or bowel. To allow exploration and potential evaluation of the rumen, the body wall incision must be of adequate size to allow the surgeon's hand and forearm to comfortably enter the rumen without undue tension on the rumen wall. Once secured and incised, the rumen incision will be smaller than the body wall incision and should be considered. Thorough exploration of the abdomen should take place before the rumenotomy is performed (and is absolutely contraindicated after the rumenotomy is performed). Attention should be paid as to the presence of adhesions and perireticular abscesses while palpating the diaphragm and reticulum. After abdominal exploration, the rumen is secured to the skin by creating a watertight seal with continuous suture. The watertight seal is critical in preventing abdominal contamination. A monofilament (or coated) type of suture on a cutting needle should be used in a Cushing's pattern. It is important to exteriorize a generous part of the dorsal rumen sac to facilitate the creation of the rumenotomy without disrupting the rumen-to-skin seal. To prevent or minimize leakage, the rumen suture bites should be through the seromuscular layer but should not penetrate the mucosa, which could lead to leakage at closure. The suture line is started dorsally at the 12 o'clock position and continued ventrally until the 6 o'clock position is reached. A similar suture line is started on the other side dorsally and continued ventrally, overlapping with the initial suture line to prevent gapping and abdominal contamination at the 6 o'clock position. Two separate suture lines are used to limit the circumferential decrease in lumen size created by one suture line pulled tightly. Once the rumen is sutured to the skin, the rumen-skin suture line is carefully checked for a good seal (Figure 5.8). The rumenotomy incision is then made in the center of the exposed, secured rumen (Figure 5.9). The rumenotomy incision should be large enough to allow entry of the surgeon's hand into the rumen, but care must be taken to ensure inadvertent incision of the skin-rumen seal does not take place (e.g., 3-cm ventral to 12 o'clock position and extending to 3-cm dorsal to the 6 o'clock position). Once the rumen has been secured and opened, no other modifications should be made to the rumenotomy. Depending on the character of the rumen contents, the rumen can be evacuated by hand or by creating a siphon with a large-bore stomach tube. The surgeon then explores the reticulum and rumen in its entirety, ensuring palpation of the ruminoreticular fold, esophageal orifice, and omasal orifice. All foreign bodies should be removed, regardless of whether they are penetrating or nonpenetrating. To facilitate finding ferromagnetic foreign bodies, a magnet can be held in the surgeon's hand while sweeping the reticular wall. An ultrasound probe (5-MHz sector) within a rectal sleeve can also be taken into the rumen to help identify adhesions and abscesses. Advancement of a hand through the reticulo-omasal canal is not typically feasible in small ruminants but may cautiously be attempted in large sheep and goats. Confirmed perireticular abscesses tightly adherent to the reticulum can be opened into the reticulum using sharp incision.

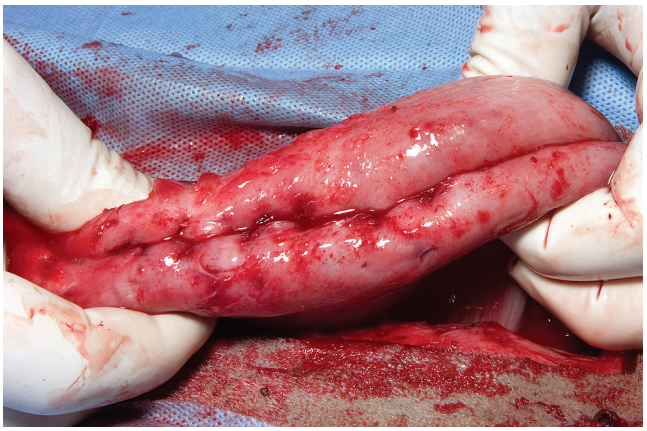


• **Fig. 5.8** Rumenotomy: the rumen is secured to the skin with a watertight seal, ready for the rumenotomy incision.



• **Fig. 5.9** Rumenotomy: rumen contents are visible through the rumenotomy incision.

Closure of the rumen is performed in two layers. Absorbable suture in a simple continuous pattern is used to close the rumen lumen for the first layer. Surgeon preference may dictate the use of a double-layer inverting pattern as described by Niehaus.[87] Once the initial layer of rumen closure is complete, the site is copiously lavaged with sterile saline. All soiled materials (e.g., gloves, gowns, drapes) are then removed and replaced, and sterile instruments used in the second part of closure. The second layer of the rumen closure is with absorbable suture in an inverting pattern (e.g., Cushing, Lembert). Suture of this second layer should start at the 12 o'clock position of the rumen incision, and retention sutures securing the rumen to the skin are removed as needed to free enough rumen for closure. When the second layer closure (Figure 5.10) is complete, the rumen is cleaned with moist sponges before being returned to the abdominal cavity. Again, it is emphasized that exploration of the abdominal cavity at this point is associated with an increased incidence of septic peritonitis and is contraindicated. The muscle and skin are inspected for gross contamination and cleansed with moist gauzes as needed. Routine closure of the muscle layers and skin are performed, based on surgeon's preference. Given that some contamination will occur during the procedure, incisional infections should be anticipated. The ventral aspect of the skin closure should include two to three interrupted sutures to allow drainage, if necessary, postoperatively.



• **Fig. 5.10** Rumenotomy: the final inverted closure of the rumenotomy incision.

The sheep or goat should be observed closely by the clinician for signs of complications, including the major complication of peritonitis as well as incisional infection, abscessation, or dehiscence, and hernia formation. Given the nature of the procedure (i.e., clean-contaminated surgery), incisional infections can occur, and ventral drainage may need to be established by removing the most ventral two to three interrupted sutures of the skin closure. The skin sutures can be removed in 10 to 14 days after surgery. Antibiotic therapy (e.g., procaine penicillin at 22,000 IU/kg, IM, q12h or potassium penicillin 22,000 IU/kg, IV, q6h) should be continued for at least 5 days. The need for prolonged antibiotic use in uncomplicated cases may be of questionable value in cattle, but this has not been critically evaluated in small ruminants.[87,88] However, medical management of concurrent diseases may include long-term antibiotic therapy (e.g., treatment of local or generalized peritonitis). Postoperative antiinflammatory medication and pain control are indicated. Reestablishing rumen flora and rumen motility using rumen transfaunate as well as maintaining the patient's hydration with oral or IV fluids should be performed.

Diseases of the Abomasum

Abomasitis and Abomasal Ulcers

Several clostridial species are implicated as a cause of abomasitis in small ruminants. Sheep are more commonly reported compared with goats. Most cases present as sudden death, with the occasional observation of animals with severe abdominal pain, depression, and prostration early in the course of disease. Death can be within hours. Braxy is a necrotizing and hemorrhagic abomasitis of sheep caused by *Clostridium septicum*. Overgrowth of the clostridial species is proposed to be associated with recent frosts, snowfalls, or the feeding of frozen feedstuffs resulting in hemorrhagic, necrotic abomasitis with fatal enterotoxemia. Braxy is reported in Europe, Africa, and the Middle East and infrequently in the United States. Braxy-like lesions caused by *C. septicum* have been demonstrated in sheep under experimental conditions as well as naturally occurring disease in lambs, with evidence of suppurative abomasitis with extensive edema, emphysema, and necrosis of the abomasal wall on histopathology.[89,90]

Similar abomasal disease characterized by hemorrhage, necrosis, and potential ulcers is reported in pre-ruminant lambs caused by *Clostridium sordellii, Clostridium fallax,* and *Sarcina ventriculi*. A high incidence is observed in 3- to 10-week-old lambs and kids.[91–95] The presence of severe gaseous distention of the abomasum was often observed in cases with *Sarcina*-like bacteria, whereas hemorrhage and ulcer were more often observed with clostridial species.[92] *Sarcina* species are anaerobic, Gram-positive bacteria that occur in cubical packets, able to grow at very low pH conditions, and ferment sugars with significant gas production. The ability to tolerate low pH and the presence of sufficient fermentable carbohydrates in milk or milk-replacer fed lambs and kids may allow this bacterial species to overgrow in the abomasum.[95] Predisposing factors for abomasal bloat and hemorrhage in young lambs and kids may include free-choice milk replacer feeding regimens at inappropriate temperature and frequency as well as iron deficiency.[95–97] Iron deficiency may promote the ingestion of soil-containing clostridial and *Sarcina* species.

Abomasitis and abomasal ulcers in adult small ruminants, apart from clostridial diseases, are poorly documented and likely uncommon. Implicated risk factors include abrupt feed changes or inappropriately formulated rations (e.g., course feed, pelleted feed) with resultant rumenitis and ruminal acidosis. Heavy-metal intoxication can produce severe abomasitis and ulceration. Small ruminants with systemic illnesses, such as pasteurellosis or pregnancy toxemia, may develop abomasal ulcerations.[98] The potential role of a mineral deficiency (e.g., copper) has not been proven. Phycomycotic ulceration of the abomasum has been reported in sheep. Fungi species involved likely represent secondary invasion of disrupted and damaged mucosa due to an underlying digestive disturbance.[99] Abomasal ulcers have also been reported in white-tailed deer with ulceration occurring at the abomasal-pylorus and at the abomasal-duodenal junction. All deer reported had other concurrent disease.[100]

Clinical Signs and Diagnosis. The diagnosis of these conditions due to clostridial diseases is by postmortem examination. Overgrowth of clostridial species occurs quickly after death necessitating timely collection of samples for anaerobic culture and molecular diagnostics.

Abomasitis and abomasal ulcers may present with vague clinical signs or be asymptomatic. If abdominal pain is the main presenting sign, other causes of acute abdomen need to be ruled out. Bleeding abomasal ulcers may present with melena. No definitive antemortem diagnostic tests are available. Fecal occult blood test results can be negative in ulcerative disease, as well as confounded by the presence of gastrointestinal parasites.[101]

Treatment. Treatment in suspected antemortem cases of clostridial abomasitis is unsuccessful. Use of therapies for the treatment of stomach ulcers in monogastric animals can prove ineffective in ruminants, due to the rumen and the delay and dilution of medications before they reach the abomasum. Most research on oral antacid therapy in ruminants has been carried out in milk-fed calves, which presents suitable therapies in preweaned lambs and kids. These include coating agents (e.g., sucralfate), acid neutralizers (e.g., aluminum and magnesium hydroxide), histamine type-2 receptor antagonists (e.g., cimetidine, ranitidine), and proton pump inhibitors (e.g., omeprazole).[102–104] Use of intravenous formulations of H2-antagonists or proton-pump inhibitors (e.g., pantoprazole) may be attempted in sheep and goats but likely are cost prohibitive.[105,106] Diet formulations contributing to underlying mucosal damage should be addressed. Buffers can be added to the feed.

Prevention. Vaccinating against clostridial diseases may decrease the occurrence of abomasal hemorrhage. Ideally, ewes and does are vaccinated prior to parturition to maximize specific

immunoglobulins in colostrum consumed by the neonate. Lambs or kids on farms where such disease has been a problem can be vaccinated with multivalent bacterins against clostridial infections during the first week of life.

Abomasal Impaction

Abomasal impaction can occur with the feeding of poor-quality roughage, but it can also be seen with foreign body obstruction of the pylorus. An example of the latter is the presence of abomasal phytobezoars as described in South African goats and sheep.[107] Goats appear to be more commonly affected than sheep, and Boer goats are more commonly affected than Angora goats. Pregnant animals may be more prone to impaction due to poor-quality roughage, whereas younger goats (6–12 months of age) appear to be at particular risk for the development of abomasal phytobezoars. In the South African condition, the composition of abomasal phytobezoars is made up of pappus hairs of certain Karoo bushes.[108]

Clinical Signs and Diagnosis. The onset of disease is insidious and affected animals usually are anorexic, depressed, and are in poor body condition. Distention of the ventral abdomen is characteristic, and in some cases the firm abomasum can be palpated through the abdominal wall on the right side. With deep abdominal palpation, the presence of phytobezoars may be appreciable. Sudden death is possible in the case of acute pyloric or intestinal obstruction with subsequent rupture of the intestinal or gastric wall. Clinicopathologic evaluation may be normal, or mild hypochloremic metabolic alkalosis may be present with increased rumen chloride concentration.

Treatment. Treatment of abomasal impaction due to poor quality forage most often requires dietary changes and oral administration of mineral oil. Abomasotomy can be attempted, although it has rarely been reported in small ruminants. For this procedure, the animal is positioned in dorsal recumbency and placed under general anesthesia. The abomasum can best be visualized through a right paramedian incision. The prognosis is poor for both conservative medical management and surgical intervention. The possibility of an underlying abomasal emptying defect should be considered. In the case of abomasal phytobezoars, the offending feedstuff should be removed from the diet. Surgical removal is the only treatment option.[107]

Prevention. Dietary manipulation to improve feed or forage quality is the best means of prevention.

Abomasal Emptying Defect

Abomasal emptying defect is a disease of predominantly Suffolk sheep, 2 to 6 years of age, which manifests as chronic, progressive weight loss and anorexia with abdominal distention. The duration ranges from several days to months. The disease is frequently reported in the post-lambing period (within 30 days), although it appears variable in onset relative to parturition and is reported in rams.[109,110] Although Suffolk is the predominant breed, cases in two Hampshire, one Dorset, and one Texel sheep are reported.[111–113] It is considered sporadic in occurrence, but herd outbreaks have been documented.[114] The underlying cause is unknown. Based on histological changes observed in the celiacomesenteric ganglion of affected sheep, a proposed pathophysiologic mechanism is alteration of autonomic innervation. Observed lesions, affecting only sympathetic nerves, suggest exposure to an excitotoxin resulting in an acquired dysautonomia.[114] Unlike abomasal impaction, this disease is due to increases in concentrate feeding associated with diet changes occurring at the time of lambing. The clinical signs are chronic weight loss, abdominal distention, and anorexia. Clinicopathologic laboratory findings reflect dehydration and metabolic alkalosis with hypochloremia. Rumen chloride concentration is increased due to abomasal reflux. Liver enzymes are increased in most cases, reflecting an increase in intra-abdominal pressure and impairment of the liver's vasculature which results in congestion, ischemia, and leakage of enzymes from damaged hepatocytes.[115] At necropsy, the abomasum is markedly distended, and the contents may be liquid or dry in character. Attempted treatment has included the use of laxatives, cathartics, motility modifiers (e.g., metoclopramide, neostigmine), and abomasotomy, with poor short- and long-term outcomes reported. The disorder carries a poor to grave prognosis.[109]

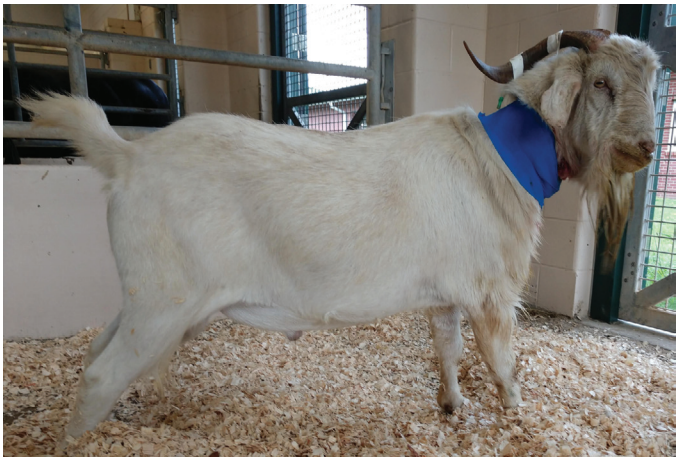
Azalea, Laurel, and Rhododendron Toxicity

Many plants of the Ericaceae family, including rhododendron, azalea, and laurel species contain diterpene grayanotoxins (also known as andromedotoxin). Ornamental and naturally occurring varieties are found in North America. All parts of the plant are potentially toxic. In cattle, *Rhododendron* has a toxic dose of 0.2% BW, while *Kalmia* has a toxic dose of 0.4% BW.[116] Toxic doses of 0.1% and 0.2 to 0.6% in goats and sheep, respectively, are reported.[117] The Japanese Pieris *(Pieris japonica)* is another broadleaf evergreen shrub that contains grayanotoxins and has resulted in small ruminant poisonings.[118–120] Grayanotoxin exerts its effects by binding to voltage-gated sodium channels of cells, especially neurons. Binding of the toxin modifies the configuration and prevents the inactivation of the sodium channel, thus rendering the neuron in a prolonged, depolarized (activated) state.[116] Cattle, sheep, and goats may present within hours of plant ingestion with evidence of GIT irritation (e.g., salivation, vomiting), cardiac arrhythmias, and neurological symptoms. Collapse and sudden death can occur in severe cases. Aspiration pneumonia of rumen contents is a significant sequela and common cause of death.

Clinical Signs. History may include the inadvertent feeding of plant clippings or access to stands of toxic plants, especially if alternative forage sources are scarce. Clinical signs include salivation, bruxism, vomiting, diarrhea, and colic. Other signs may include nasal discharge (with attempts to vomit), epiphora, and ataxia. Onset of clinical signs can be within 4 to 16 h of ingestion and occur with ingestion of only a few leaves. As severe intoxications progress, depression, collapse (bradycardia and hypotension), opisthotonus, and coma can occur.[117,121,123] Intoxicated sheep and goats are at a significant risk for aspiration pneumonia which can result in death.

Diagnosis. The diagnosis of this condition usually is based on clinical signs coupled with a history of ingestion of one of the offending plants. Identification of plant parts in ingesta is a diagnostic tool, as well as the identification of grayanotoxins in feces, urine, vomitus, and rumen contents using liquid chromatography-mass spectrometry analysis.[117]

Treatment. Intoxicated animals may recover in 1 to 2 days without any therapy if the offending plants are removed from the diet and ingestion was minimal. However, the administration of activated charcoal (2 to 9 g/kg orally), atropine (0.05 to 0.2 mg/kg, IV), antiarrhythmic drugs, thiamine (10 mg/kg, SC, q6–8h), laxatives, and IV fluids may be indicated. The risk of aspiration pneumonia is high in affected animals and should be treated with



• **Fig. 5.11** Azalea Toxicity. A 3-year-old Kiko buck that presented for projectile vomiting (note beard stained from ruminal contents) and colic (note stretched out appearance due to abdominal pain).

appropriate antibiotics (e.g., procaine penicillin, oxytetracycline). The risk of aspirating orally administered medications should be heavily weighted against their use, especially when vomiting is frequent. IV lipid emulsion therapy has been used for *Pieris* ingestion in goats with recovery from severe clinical signs reported to occur within hours of administration[118] (Figure 5.11).

Prevention. Mountainous or hilly areas should be fenced to prevent animal access to toxic plants. Alternate forage sources should be offered during times when grazing or browsing is scarce to limit the intake of poisonous weeds. Feeding shrubbery clippings is discouraged.

Diseases of the Intestines

Diarrhea in Lambs and Kids: Overview

Diarrhea in lambs and kids is a complex, multifactorial disease involving the animal's susceptibilities, the environment, nutrition, infectious agents, and management. Decades of research have been devoted to the study of the pathophysiology of infectious diarrhea in calves; the pathophysiologic picture in lambs and kids is quite similar. Despite improvements in management practices and prevention and treatment strategies, diarrhea is still the most common and costly disease affecting neonatal ruminants.[124–128]

Some general preventive measures (e.g., improved sanitation) will decrease the risk of diarrheal disease from any cause. By contrast, specific control measures such as vaccination require the definition of a specific cause of diarrhea. Table 5.2 lists the agents most likely to cause diarrhea in lambs and kids, tissues or other samples required for diagnosis, and commonly used test methods. The color and consistency of the feces and any gross lesions can appear similar with numerous diseases. Laboratory identification of infectious agents and tissue histopathologic examination are therefore key to establishing a diagnosis (see Chapters 16 and 20). Because autolysis and secondary bacterial invasion of the gut begin within minutes of death, necropsy samples taken immediately from euthanized lambs and kids yield the most reliable diagnostic material. Mixed infections with two or more pathogens are common, and clinically important farm-specific pathogens change from year to year.[126–130] In some cases, an underlying nutritional deficiency or excess may be present, concurrently with infective disease. The clinician should therefore take a variety of samples to ensure identification of all pathogens and predisposing factors involved; continued reevaluation of the causes of diarrhea is

TABLE 5.2 Diagnostic Samples and Testing Methods Required for Differentiation of the Most Common Causes of Infectious Diarrhea in Lambs and Kids.

Causative Agent	Sample Required	Test Method[a]
Escherichia coli	2–3 g feces Formalin-fixed small intestine	Culture and serotyping for K99 and F41, PCR Histopathologic examination
Rotavirus	2–3 g feces or colonic contents Formalin-fixed small and large intestine Frozen small and large intestine	EM, ELISA, VI, CF tests, PCR assay Histopathologic examination VI, FA test, IP assay
Cryptosporidia	2–3 g feces Air-dried fecal smear Formalin-fixed small and large intestine	FA test, fecal flotation, PCR Acid-fast stain Histopathologic examination
Salmonella	2–3 g feces Formalin-fixed small and large intestine Frozen small and large intestine and mesenteric lymph nodes	Culture, PCR assay Histopathologic examination Culture
Giardia	Wet mount of feces Feces	Iodine staining ELISA, FA test, PCR
Clostridium perfringens	Frozen small intestinal contents and abomasum, small and large intestine Formalin-fixed abomasum and small and large intestine	Culture, toxin identification Histopathologic examination
Coccidia	2–3 g feces Formalin-fixed small and large intestine	Fecal flotation Histopathologic examination

[a]*EM,* Electron microscopy; *ELISA,* enzyme-linked immunosorbent assay; *VI,* virus isolation; *CF,* complement fixation; *PCR,* polymerase chain reaction; *FA,* fluorescent antibody; *IP,* immunoperoxidase
Data from Rings DM, Rings MB: Managing *Cryptosporidium* and *Giardia* infections in domestic ruminants. *Vet. Med.* 91:1125, 1996; Cohen ND, et al: Comparison of polymerase chain reaction and microbiological culture for detection of salmonella in equine feces and environmental samples. *Am. J. Vet. Res.*57:780, 1996; Drolet R, Fairbrother JM, Vaillancourt D: Attaching and effacing *Escherichia coli* in a goat with diarrhea. *Can. Vet. J.* 35:122, 1994.

crucial. Evaluation of material from multiple cases, with a focus on those in the acute phases, is important. Although examination of antemortem fecal samples can be diagnostic, laboratory testing of tissue samples may yield better results.

Treatment and preventive measures for specific diarrheal diseases are the focus of the remainder of this section, which is followed by sections on general supportive treatment and control measures for all infectious diarrheal diseases.

Causes of Diarrhea in Neonatal Lambs and Kids

Four major pathogens cause diarrhea in lambs and kids during the first month of life: enterotoxigenic *Escherichia coli* (ETEC), rotavirus, *Cryptosporidium* species, and *Salmonella* species. The relative prevalence of these infectious agents varies greatly among studies. This variability probably results from differences in location, season, and diagnostic techniques and the occurrence of mixed infections. Other, less common causes of diarrhea in neonates are *Giardia* infections and nutritional diarrhea.

Enterotoxigenic Escherichia Coli

Pathogenesis. ETEC employs two virulence factors to cause disease. The first is the ability to attach and colonize the intestinal villi, which is accomplished by means of fimbriae or pili. The most important fimbriae in lambs are K99 and F41.[131] The fimbrial antigens can be recognized from samples sent for analysis in most diagnostic laboratories and are important in identifying this agent as a cause of diarrhea. After the organism attaches to the villi, it produces the second virulence factor, enterotoxin. Enterotoxin interferes with the normal physiology of the gut, with resultant diarrhea.[131] Calves have an age-associated resistance that probably is related to the blocking of fimbrial attachment to the gut, so ETEC diarrheal disease occurs mainly in calves younger than 1 week of age.[132,133] The mode of infection is fecal-oral.

Clinical Signs. ETEC diarrhea is seen in lambs and kids younger than 10 days of age but is most common at 1 to 4 days, so age-related resistance also may be a factor in newborns of these species.[126,130] It usually manifests as an outbreak in lambs and kids between 12 and 48 h of age. Because ETEC causes a secretory-type diarrhea, bicarbonate loss in the diarrhea leads to severe acidosis, with lambs and kids quickly becoming dehydrated and recumbent. However, many infected animals die before developing diarrhea. Affected neonates are depressed, stop nursing, and may show excessive salivation. Fluid sequestration in the abomasum produces a splashing sound on movement. This condition is associated with high mortality if animals are not treated promptly.[130,131]

Diagnosis. Fecal culture and serotyping for the K99 and F41 fimbrial antigens constitute the basis for diagnosis. Because many nonpathogenic *E. coli* bacteria are normal gut inhabitants, growth of this organism on cultures usually is an insignificant finding.[131] Occasionally, the bacteria do not express the fimbrial antigens in culture, so ETEC cannot be ruled out if the culture is negative for K99 and F41.[134] Histopathologic evidence of colonization of the small intestine can support a diagnosis.

Treatment. Supportive care consisting of fluid therapy with either oral, IV, or SC administration of a polyionic solution is the mainstay of therapy. The use of oral antimicrobial agents is controversial. Although antibiotics may kill the ETEC, they also may interfere with normal gut flora. If fluid support is provided, the diarrhea usually subsides without antibiotic treatment. Nevertheless, oral neomycin (10 to 22 mg/kg twice daily) or trimethoprim-sulfa (30 mg/kg PO) and systemic ampicillin (10 to 20 mg/kg IM twice a day) or amoxicillin (10 to 20 mg/kg IM three times a day) may be beneficial. NSAIDs are indicated to decrease inflammation of the gut and provide some analgesia. The use of flunixin meglumine (1–2 mg/kg IM) has been shown to decrease fecal output in ETEC infections in calves[135] and appears to be beneficial in lambs.

Prevention. Vaccination of ewes and does with bovine ETEC vaccine before they give birth is recommended to increase passive immunity in the neonate.[126,127,131] Monoclonal and polyclonal antibody products for calves may be beneficial during an outbreak if administered to lambs or kids within the first 12 h of life. The use of neomycin (10 to 12 mg/kg PO twice daily) in lambs that appear clinically normal may help stop the progression of an outbreak. Shearing ewes prepartum to minimize fecal ingestion by neonates and ensuring that newborns ingest adequate colostrum both will help decrease the incidence of this disease. Making sure that ewes and does have a 2.5 to 3.5 BCS at parturition and are fed adequate diets in the final 2 months of gestation will increase the chance of adequate colostrum manufacture by the dam.

Rotavirus

Pathogenesis. Lambs and kids are infected with group B rotaviruses whereas most other animals, including cervids such as roe deer, and human beings are infected with group A rotaviruses.[136] Rotaviruses infect villus tip cells of the small intestine, which results in villus atrophy and malabsorptive diarrhea.[137]

Clinical Signs. Rotavirus generally causes diarrhea in lambs and kids 2 to 14 days of age, but older animals also can be affected. Young animals can become very depressed and dehydrated.[126,136,138,139]

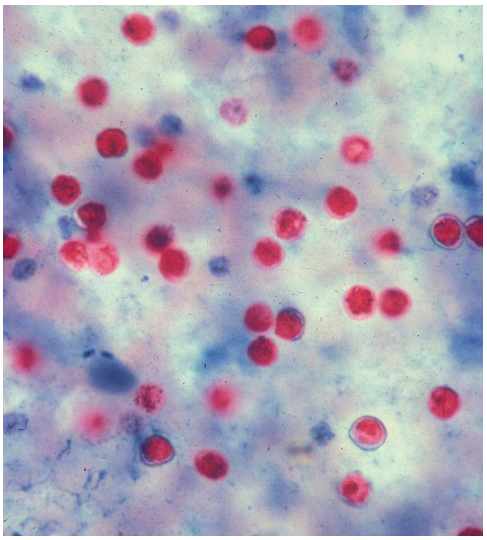
Diagnosis. Detection of the organism by electron microscopy of fecal or colonic samples or by immunologic techniques applied to feces or tissue sections is the basis of diagnosis.[136,139] Because these organisms are sloughed with the villus tip cells they infect, and viral antigens are complexed with the animal's antibodies, tissue samples from acutely infected animals are of highest diagnostic value.[140] Rotavirus has been detected in animals without diarrhea, so other causes of diarrhea should be investigated as well.[127,129]

Treatment and Prevention. Rotavirus diarrhea is treated with supportive care. Prevention by vaccination of small ruminants with bovine rotavirus vaccines before they give birth is recommended to increase passive immunity in neonates.[126,127]

Cryptosporidium Species

Pathogenesis. *Cryptosporidium parvum* is a protozoan that can cause a malabsorptive diarrhea similar to that seen with rotavirus infection. Unlike other protozoal agents, such as the one that causes coccidiosis, cryptosporidia do not require fecal excretion for sporulation to infective stages.[141] They sporulate in the gut, whereupon approximately 20% become immediately infectious to other villus tip cells without leaving the intestines. This method of autoinfection can result in severe disease that may be sustained for long periods. Because some of the oocysts also are immediately infectious when they are shed in feces, spread of infection may be rapid.

Clinical Signs. Cryptosporidia can cause diarrhea in small ruminants at 5 to 10 days of age.[127,142,143] Affected animals often are active, alert, and nursing. The diarrheal stools usually are very liquid and yellow. Diarrhea can range from mild and self-limiting to severe, especially with mixed infections.[127,129,142,144] Relapses are quite common, and this organism usually occurs as a component of mixed infections.



• **Fig. 5.12** Red-staining *Cryptosporidium* on a blue-green background in a fecal smear prepared with an acid-fast stain. This protozoal parasite induces villus atrophy and decreased digestion.

Diagnosis. Acid-fast staining of air-dried fecal smears is a quick and easy method of diagnosis. Examination under 40× to 100× magnification reveals round protozoa that have taken up the red color of the carbol fuchsin portions of the stain on a green background (Figure 5.12). Although cryptosporidial infection can be diagnosed by fecal flotation testing, the very small size (4 to 6 μm) of these organisms makes this method difficult and subject to false-negative results.[145,146] Both immunologic and polymerase chain reaction (PCR) techniques have been developed to improve detection limits.[145,147] Cryptosporidia also can be identified on histopathologic examination. Cryptosporidiosis is a zoonotic disease, and people can become infected from handling infected animals or feces.[141]

Prevention. No consistently effective treatment for cryptosporidiosis in ruminants has been identified. However, proper hydration and electrolyte balance should be maintained, along with other supportive care. Prevention through decreased exposure of neonates to organisms in the environment is critical, especially exposure of neonates at birth.[148] On farms endemic with coccidiosis or during an outbreak, improved hygiene may be of benefit (e.g., precolostral intake udder wash, feeding only low-heat-pasteurized colostrums, isolation of all exposed animals). Anecdotal reports suggest that decoquinate and monensin sodium may be useful in control of cryptosporidiosis. Decoquinate (2.5 mg/kg PO) fed to does and kids may be useful in decreasing morbidity and mortality associated with cryptosporidiosis in goat kids.[149] Treatment in all affected animals also should include fluid-electrolyte therapy.

During an outbreak, affected animals should be isolated from the rest of the flock. No new animals should be added to a pen in which the disease has been diagnosed. Keepers should depopulate pens in which the disease has been diagnosed and attempt to clean the environment. Cryptosporidiosis can be particularly difficult to control because of the organism's persistence in the environment and resistance to most chemical disinfectants. Ammonia (5–10%) and formalin (10%) seem to be the most effective agents, but, due to the potential for toxic effects, caution is indicated with the use of either chemical.[142,150] Feeders should be constructed to minimize fecal contamination. Early results are favorable for vaccine development in cattle, and vaccination may prove to be the best control method in the future.[151] Cryptosporidiosis is potentially a zoonotic disease; clinicians and keepers should therefore exercise great caution when handling affected animals, and well-planned biosecurity programs should be instituted (see Chapter 19).

Salmonella Species

Pathogenesis. The bacterial genus *Salmonella* has thousands of serotypes, all of which can potentially cause diarrhea in animals. Salmonellae can cause diarrhea in small ruminants of any age.[126,127] These microbes produce enterotoxins, are invasive, and cause severe inflammatory disease and necrosis of the lining of the small and large intestines.

Clinical Signs. Affected animals younger than 1 week of age are more likely to die acutely before onset of clinical signs, whereas animals older than 1 week are more likely to have diarrhea.[127,130,152] An acute onset of fever, depression, tenesmus, and shock occasionally is observed. *Salmonella*-induced diarrheal stool is more likely to contain blood.[127] Enteric salmonellosis also is a zoonotic disease that warrants implementation of protective measures.

Diagnosis. A diagnosis of *Salmonella* diarrhea is based on culture of the organism in feces or tissues and characteristic changes on histopathologic examination of the small and large intestine.[153] More sensitive PCR techniques for identifying *Salmonella* species in feces are being developed.[154] The diarrheal feces occasionally may contain fibrin, but many animals die before this development is observed. The clinician may note leukopenia or leukocytosis in the CBC results.

Treatment. Therapy for *Salmonella*-induced diarrhea involves supportive care and possibly parenteral antimicrobial therapy. The use of antimicrobial agents is controversial and probably does not influence the gastrointestinal infection. Nevertheless, because *Salmonella* is an invasive organism, parenteral use of antimicrobial agents may be beneficial in preventing septicemia. Antimicrobial susceptibility patterns are difficult to predict for *Salmonella* species, so antimicrobial therapy should be based on culture and sensitivity results. Ceftiofur sodium (1.1–2.2 mg/kg IM twice daily) or trimethoprim-sulfadiazine (15 mg/kg SC once a day) can be administered until antimicrobial sensitivity results are available.

Prevention. Latent carriers of *Salmonella* can potentially shed organisms to other animals, particularly when they are stressed.[127] Newly introduced animals should be isolated for 1 month, and fecal culture should be considered.[127] Bleach (sodium hypochlorite) and chlorhexidine are effective disinfectants to apply to the premises and animal handling/feeding equipment during an outbreak. Identification of carrier animals by fecal culture is recommended for herd problems. Vaccine efficacy is questionable, and to date its effects have not been thoroughly evaluated in sheep and goats.[155]

Giardia. *Giardia*-induced diarrhea is more commonly seen in, but not limited to, 2- to 4-week-old lambs, kids, and fawns.[127,156] The diarrhea usually is transient, but infected animals can continue to shed cysts for many weeks, even when they appear to be clinically normal.[145,157,158] Therefore, simply finding the pathogen in feces does not mean that it is the cause of the diarrhea, especially in older animals. *Giardia* can be found in herds without any history of neonatal diarrhea, so finding *Giardia* in herds in which newborn animals are experiencing diarrhea is of questionable relevance.[159] However, these animals may be a source of infection for others and possibly humans.[145,156] Identification of the organism

on iodine-stained wet mounts of feces or tissue is the classic method of diagnosing giardiasis, but more sensitive immunologic techniques are now available.[145,156] Infected animals can be treated effectively with fenbendazole (5–10 mg/kg twice daily for 3 days or once daily for 5 days).[145] Giardiasis has historically been treated with oral metronidazole (50 mg/kg once a day for 5 days). However, use of this drug class in food animals is currently illegal in the United States. Giardiasis is potentially a zoonotic condition.

Nutritional Diarrhea. Infectious agents are not the only cause of diarrhea in neonates. Nutritional problems can result in diarrhea, but cases related to nutrition are underreported in the literature because the resulting diarrhea usually is mild and subsides without treatment. Nutritional diarrhea is most common in orphaned animals and usually is a result of improper management practices such as use of poor-quality milk replacers, mixing errors, or infrequent feeding of large amounts (see Chapter 2). Diarrhea resulting from consumption of lush pasture or high-energy rations also is commonly seen and usually is self-limiting. The incidence of this form of gastric upset can be minimized by a slow introduction (over 2–3 weeks) to energy-dense diets.

In calves with infectious diarrhea that develop maldigestion or malabsorption, secondary nutritional diarrhea may result from an inability to digest carbohydrates (lactose, xylose).[160,161] This digestive defect has been reported in goats and also is probably a cause of diarrhea in lambs.[162] Diarrhea resulting from primary lactose deficiency also has been reported in calves.[163] Calves on poor-quality milk replacers can develop an overgrowth of normal enteric *E. coli,* resulting in diarrhea.[164] If lactose intolerance is suspected, decreasing the amount of lactose fed and using commercially available lactose enzymes may alleviate clinical problems.

Causes of Diarrhea in Older Lambs, Kids, and Fawns

The most common cause of diarrhea in older lambs and kids is nematode infestation. Other major causes of diarrhea in older small ruminants are *Clostridium perfringens* infection and coccidiosis. Coccidiosis is covered in Chapter 6. *Giardia* has been reported to cause weight loss without diarrhea in 2- to 3-month-old lambs.[165]

Clostridium Perfringens. *C. perfringens* types A, B, C, and D all can cause diarrhea in lambs and kids, but type D is the most common etiologic agent in the United States.[127,130,166,167]

Pathogenesis. Clostridial diarrhea occurs in peracute, acute, and chronic forms and commonly is called *enterotoxemia* or *overeating disease.* In type C infection, a beta toxin can cause acute hemorrhagic enteritis. Type C infection is seen mostly in lambs or kids younger than 3 weeks of age. An epsilon toxin is responsible for pathologic findings in type D infections. Enterotoxemia usually is seen in rapidly growing feedlot lambs on high-concentrate rations. It also is associated with other feeding changes, including changes in type of pasture. However, it occasionally has been reported in the absence of any dietary changes, particularly in goats.[127,130,168] This disease commonly occurs in the fastest-growing and most well-conditioned animals. Even vaccinated herds (again, more usually goats) can be affected, so it should not be ruled out despite confirmation of previous vaccination.[127]

Clinical Signs. The *peracute* form of clostridial infection is characterized by the rapid onset of severe depression, abdominal pain, profuse and bloody diarrhea, and neurologic signs. Death occurs within hours of onset of clinical manifestations. Sudden death may occur without diarrhea. Sudden death following the onset of neurologic signs is more common in sheep, whereas goats are more likely to show signs of diarrhea before death.[127] Similar but less severe signs are seen in the *acute* form of the disease. The *chronic* form occurs more commonly in goats.[127,167]

Diagnosis. Antemortem diagnosis is based on clinical signs. At necropsy, *C. perfringens* can be cultured from intestinal tissue samples. The significance of a positive culture can be difficult to interpret, however, because these organisms can be a normal component of the gut flora but subsequently proliferate after death. This is true especially of type A, for which a role in actual disease is controversial.[169] Histopathologic examination of sections of the gut can be helpful. Identification of the toxins (namely, the epsilon toxin) in intestinal contents is required for a definitive diagnosis.[127,130] Because the toxin degrades within several hours of death, its absence does not preclude enterotoxemia as a diagnosis[166] (Figure 5.13).

Treatment. Treatment is rarely effective but consists mainly of aggressive supportive care. *C. perfringens* type D antitoxins (15–20 mL SC) can be administered to animals during an outbreak of enterotoxemia if clinical signs are noted. The antitoxin may be more effectively used as a preventive early in an outbreak of the disease. During an outbreak, any animals that have not been vaccinated should be given the antitoxin and vaccinated with the toxoid simultaneously; those previously vaccinated should receive a booster vaccination.

Prevention. Routine vaccination should start at 4 to 6 weeks of age and be followed by a booster 3 to 4 weeks later. In settings in which the disease has become endemic, lambs or kids can be vaccinated and given antitoxin during the first week of life. Yearly vaccination, preferably a few weeks before the ewes and dams give birth, increases colostral immunity in neonates and improves prevention programs. Goats may not respond as well to vaccination as sheep do, so biannual, triannual, or quarterly vaccination is recommended, especially in herds in disease-endemic areas.[127,162] Vaccination with only *C. perfringens* type C and type D vaccines and tetanus toxoid is superior to the use of more polyvalent clostridial vaccines.[127] Reducing the energy density of the diet and avoiding sudden dietary changes or alterations in the feeding routine are

• **Fig. 5.13** A field necropsy of a 10-week-old intact, male Boer cross kid with watery diarrhea demonstrated necro-hemorrhagic enteritis. Laboratory diagnostics demonstrated *Clostridium perfringens* as the cause, and histologic examination of tissues was consistent with this diagnosis. The kid had no history of vaccination. (Courtesy of Dr. Kelley Steury, ALVDL, Auburn, AL.)

crucial to prevention. Control of internal parasites, particularly tapeworms, may further reduce the incidence of these disorders.

Miscellaneous Causes of Diarrhea in Kids, Lambs, and Fawns

Adenovirus, caprine herpesvirus, coronavirus, *Campylobacter jejuni, Escherichia fergusonii, Yersinia* species, and *Strongyloides papillosus* can cause diarrhea in lambs, kids, and fawns of various ages.[125,127,129,170] An adenovirus-induced hemorrhagic enteropathy has been seen in a captive, black-tailed deer herd.[171] Enterohemorrhagic *E. coli* (EHEC) and enteropathogenic *E. coli* (EPEC) also have been isolated from feces of both diarrheic and normal lambs and kids.[172–176] These *E. coli* serotypes are K99- and F41-negative. Culture and serotyping of these organisms from feces and tissue samples with typical histopathologic lesions are diagnostic. Although ETEC disease is not zoonotic, EHEC and EPEC can potentially affect humans and cause foodborne illness.

Treatment of Lambs and Kids With Diarrhea

Although specific therapies are available for some causes of diarrhea, many animals need to be treated for dehydration and metabolic acidosis regardless of the inciting cause. Animals with only mild diarrhea, especially mild nutritional diarrhea, may not require therapy unless they become dehydrated. If kids or lambs become less than 8% dehydrated and are only mildly depressed but still willing to nurse, they can be treated with oral electrolytes designed for calves. Fluids can be administered by bottle or by feeding tube (~ 18- to 24-inch, 3/8-inch diameter, catheter tip) if the animal will not nurse. The keeper or the clinician should carefully adjust the amount of fluids for lambs and kids (250–500 mL [8–16 oz], as opposed to 4 L in a calf). Because most electrolyte solutions designed for calves contain glucose, they should be refrigerated after they have been mixed and any leftovers discarded within 24 h. IV fluids may be needed to treat more severe dehydration. If the lamb or kid is too weak to stand, IV fluids are indicated. Isotonic fluids containing electrolytes should be given to replenish losses. Glucose can be added to make a 1 to 2.5% solution. Sodium bicarbonate also may be administered, especially if the dehydration is severe. A rule of thumb is to give one-fourth of the calculated fluid needed as isotonic bicarbonate (1.3%). Extra potassium (10–20 mEq/L) can be added to fluids, because most animals are severely dehydrated from diarrhea and depleted in potassium, even though their blood potassium levels may be elevated. If extra potassium is added, acidosis must be corrected concurrently. After correcting the dehydration, the keeper or the clinician can offer oral electrolyte-enriched fluids to replace ongoing losses caused by continued diarrhea (see also Chapter 3).

Removing milk or milk replacer from the diet is not recommended. Young animals need nutrients, and even high-energy, glucose-containing electrolyte solutions are no substitute for milk. Animals should continue to receive milk replacer in normal amounts or be allowed to nurse; oral electrolytes also can be given if necessary. Animals being hand-fed should be offered small amounts frequently to help minimize problems. Electrolytes should never be mixed with milk but should instead be given in separate feedings. If lactase deficiency is suspected, lactase drops or capsules (available in health food stores) can be added to milk or milk replacer.[162]

NSAIDs (e.g., flunixin meglumine, 1.1–2.2 mg/kg IV, or ketoprofen, 3 mg/kg IV) are beneficial, especially if toxemia is involved, as in ETEC, enterotoxemia, and salmonellosis. Antimicrobial agents should be reserved for proven outbreaks of salmonellosis and for animals with other causes of diarrhea that do not respond to fluid therapy and NSAIDs; these drugs should be administered only parenterally. Oral coating agents and antacids are popular, but such agents have not been shown to be beneficial, and their use is not therapeutically logical in light of the pathogenesis of these diseases. The therapeutic use of probiotics is questionable, but anecdotal reports suggest they may be beneficial in reestablishing the normal flora of the small intestine. Our own rule of thumb is that nothing should be given orally except milk, oral electrolytes, and possibly probiotics.

General Control Measures for Infectious Diarrhea

Ensuring adequate intake of high-quality colostrum and minimizing stress are important for prevention of all neonatal diseases. A normal lamb or kid will stand and nurse within 45 minutes to 1 h of birth. The ingestion of colostrum within 2 to 3 h is essential in preventing hypothermia and hypoglycemia and decreasing the incidence of various diseases. Lambs or kids born as twins or triplets, weak or injured neonates, those born during severe weather, those born from a dam with dystocia, and those delivered by cesarean section all are candidates for colostrum supplementation. Supplemental colostrum should be good-quality colostrum from females that have tested negative for Johne's disease, ovine progressive pneumonia (OPP), and caprine arthritis encephalitis (CAE). Mixing colostrum from several cows decreases the incidence of the "cow colostrum–associated" hemolytic disease sometimes seen in lambs. If the lamb or kid is unable to nurse, it should be tube fed 50 mL/kg of colostrum. The veterinarian or animal handler can sit comfortably holding the lamb or kid in sternal recumbency in the lap. A 12 to 14 French soft feeding tube is then lubricated, inserted into the side of the mouth, and passed slowly to the depth of the thoracic inlet. If the tube is placed in the trachea, the lamb or kid will show signs of discomfort and may shake and cough. The tube may be palpated on the left side of the throat. After correct placement of the tube, colostrum can be administered by gravity flow.

Antepartum shearing of the dam may decrease the likelihood of ingestion of feces by lambs. Good sanitation in lambing and kidding areas is paramount in management programs that stress prevention. The presence of organic matter interferes with the effectiveness of many disinfectants, so removal and proper disposal of feces, carcasses, and placentas are essential. When disposing of waste material containing either *Cryptosporidium* or *Giardia,* the keeper should be careful to avoid contaminating water sources. Infected animals should be isolated to prevent spread of the infection throughout the flock or herd. In general, infected animals should remain in the environment where the infection was first diagnosed, because it is already contaminated. Removing pregnant ewes or dams to a clean area before lambing or kidding helps minimize the continued spread of disease. If possible, lambs and kids already born but not showing clinical signs should be removed to a third area. If "safe" pastures are maintained for internal nematode control, they are ideal for use in an emergency situation to control these diseases (see also Chapter 6). Although some animals may appear normal, they may be incubating and possibly shedding the infective agents of disease. If such animals are moved with

pregnant females, they can be a source of contamination in a clean area. If healthy lambs and kids cannot be moved to a third, relatively safe area, they should be left with the clinically infected animals because they have already been exposed.

Diarrhea in Adult Sheep and Goats

The list of considerations in the differential diagnosis for acute and chronic diarrhea in small ruminants is extensive.[1,2] The most common cause of diarrhea in adult sheep and goats is parasitism; another major cause is Johne's disease. Parasitism is discussed in Chapter 6. Other causes of acute diarrhea include rumen acidosis, peritonitis, endotoxemia, and ingestion of toxins. The list of toxins that cause diarrhea also is very long, and often diarrhea is not the primary clinical sign. Some of the more commonly encountered toxins that produce diarrhea are arsenic, salt in toxic amounts, levamisole, copper, oak, selenium, and pyrrolizidine alkaloids.[1] *Salmonella* infection and chronic enterotoxemia can cause diarrhea in adult animals. Coccidiosis can occur in adults under severe stress or in animals that possess limited immunity because of lack of exposure. Hepatic and renal disease and copper deficiency sometimes are accompanied by chronic diarrhea, but weight loss is a more common sign in adults.

Johne's Disease. Johne's disease, also called *paratuberculosis*, is a chronic wasting and diarrheal disease caused by the bacterium *Mycobacterium avium* subspecies *paratuberculosis.* All ruminants, including cervids, are thought to be susceptible to infection. While varying from each herd/flock, the prevalence of Johne's in most ruminants, including cervids, increases when animal density is higher.[3] Transmission of the organism is primarily by the fecal-oral route. Young animals are more susceptible to infection than adults. It can be transmitted through milk and the placenta.

Pathogenesis. Bacterial shedding in feces and milk and transplacental transmission are more common in animals showing clinical signs.[4–6] Therefore, the offspring of infected animals, and especially the offspring of animals showing clinical signs, are more likely to acquire the infection than other members of the flock/herd. After an animal is exposed, it will either clear the organism or acquire a chronic, persistent infection. The infection most commonly is isolated to the ileal regions of the small intestine where it causes granulomatous thickening of the intestinal wall and subsequent malabsorptive diarrhea. Infected animals may be asymptomatic for years.

Clinical Signs. Morbidity rates are low (approximately 5%), but for every infected animal with clinical signs, several are in the subclinical state and may be a source of both horizontal and vertical transmission.[4] Both sheep and goats appear to remain asymptomatic until they reach 2 to 7 years of age. The most consistent clinical sign in sheep, goats, and cervids is chronic weight loss. Chronic diarrhea occurs in approximately 20% of cases.[4] Signs may appear with or be exacerbated by stress, especially after parturition.[4,5] Hypoproteinemia and chronic mild anemia are the only consistent findings from clinicopathologic laboratory tests. Submandibular edema may develop as a consequence of low protein levels in infected animals, and because parasitism is ubiquitous, an accurate diagnosis may be difficult.

Diagnosis. Diagnosis is by culture of the organism from feces. Such testing unfortunately takes between 8 and 14 weeks but can identify 40 to 60% of clinically infected goats. Feces of noninfected sheep and goats within heavily infected herds can yield a positive culture from oral-fecal pass-through of the organism. Sheep strains of Johne's disease and some goat variant strains seem to be more difficult to culture in media used to identify cattle strains of the disease. Therefore, fecal culture in sheep and goats appears to be of limited benefit in a clinical setting.[5,6] A relatively inexpensive and easily performed method of identifying approximately 50% of all clinically infected animals is acid-fast staining of fecal smears.[4,5] A PCR fecal assay also is available, but its sensitivity is lower than that of fecal culture. Good diagnostic results can be obtained with serologic testing for antibodies (e.g., agar gel immunodiffusion [AGID] test, enzyme-linked immunospecific assay [ELISA], complement fixation test) in animals showing clinical signs. The specificity of all of the serologic tests is greater than 95% in sheep and goats with signs of clinical disease, although the sensitivity is not as high.[5–8] Therefore, a positive serologic test result in an animal showing clinical signs indicates that the animal has Johne's disease. However, the disease cannot be ruled out with a negative test result. Identification of subclinically infected animals using serologic tests is more problematic. A sensitivity of approximately 50% is all that should be expected. With the ELISA and complement fixation test, cross-reactivity with *Corynebacterium pseudotuberculosis* may occur, thereby limiting the value of such testing in flocks with caseous lymphadenitis infections.[5,9] ELISA performed on milk samples from goats had reduced sensitivity but increased specificity (less cross-reaction) compared with serum ELISA.[10] Sheep and goats appear to respond differently with regard to the formation of antibodies. In sheep, antibodies tend to develop in the later stages of the disease, whereas antibodies may be detected much earlier in the goat. Necropsy diagnosis is based on the finding of thickened, corrugated intestines, especially in the area of the ileum. Acid-fast staining of impression smears (taken from the ileum and ileocecal lymph nodes) can help yield a quick diagnosis. The staining of numerous clumps of acid-fast rods is highly suggestive of Johne's disease.

Prevention. Johne's disease has no effective treatment, so prevention and control are imperative. However, preventing the introduction of Johne's disease into a herd can be difficult. Because animals with subclinical infection may not shed the organism or shedding may occur in only small quantities, fecal culture is helpful only if a positive culture is obtained. The sensitivity of serologic tests of animals with subclinical disease is low and variable among flocks.[5,6] Negative test results in subclinically infected animals are common. However, the specificity of serologic tests remains high, so a positive test result is a valid reason to not purchase an animal.[5] Because Johne's disease also occurs in cattle, supplemental colostrum supplies should come only from dairy herds free of Johne's disease.

After Johne's disease is diagnosed in a herd, several control measures should be implemented. Sanitation is important, because the organism is highly resistant in the environment (i.e., capable of surviving longer than 1 year under most conditions).[6] Reduced stocking rates, frequent cleaning of pens, and use of automatic waterers will decrease fecal transmission. Keepers and herdsmen should cull the offspring of infected animals. Culling animals based on the results of flock/herd -wide AGID testing or ELISA and fecal culture is recommended. Animals should be tested at least once a year. More frequent testing as resources allow will speed the identification of infected animals. A vaccine for cattle is available only in some locales, and clinicians or keepers may require official permission for its (extra-label) use in sheep and goats. Vaccination for Johne's disease in cattle does not eliminate infection but can decrease herd prevalence, delay the onset of clinical signs, and decrease cross transmission by infective bacterial shedding in the feces.

Intestinal Obstruction

Any cause of intestinal obstruction that occurs in other ruminants may occur in sheep, goats, and cervids. Obstructive diseases of the intestinal tract may be divided into two general categories — strangulating and nonstrangulating (simple and functional). Examples of strangulating lesions include intussusception and torsion of the mesenteric root, whereas simple, nonstrangulating obstructions include enteroliths and phytobezoars. Functional, nonstrangulating lesions are most commonly associated with inflammation or infection, often presenting as intestinal ileus.[187] Most of these diseases produce abdominal pain and occasionally abdominal distention. Diagnosis is based on physical examination but distinguishing true obstruction from functional obstruction can be difficult. Abdominal radiographs and ultrasonography can help differentiate among differential diagnoses with further support given with the use of clinicopathological analyses (e.g., rumen fluid analysis, abdominal fluid analysis). On occasion, a "target-shape" lesion of an intussusception may be found via ultrasonography.[188] However, exploratory surgery may be required to obtain a definitive diagnosis and should be considered a reasonable diagnostic and therapeutic intervention in a small ruminant presenting for an acute abdomen. Clinical signs suggestive of obstructive intestinal diseases include vocalization, kicking at the abdomen, frequent lying bouts, and even recumbency with severe pain. The heart rate is increased due to hypovolemia and pain. Changes in the abdominal contour may become apparent with the development of low, bilateral abdominal distention, depending on the amount of fluid buildup in the intestinal tract proximal to the obstruction. Manure may be scant or absent with changes in appearance such as the presence of melena or mucus. The initial colic episode may be followed by chronic low-grade pain and signs suggestive of peritonitis.[187]

Intussusception

Intussusception is the telescoping of one segment of intestine into an adjacent segment. Any segment of the intestine can be affected, but the ileum and ileocecal junction are the most common areas involved. The condition is more commonly reported in young animals but can occur in mature animals. In this condition, one segment of the intestine telescopes into an adjacent segment (intussusceptum and intussuscipiens, respectively), resulting in narrowing of the intestinal lumen and blood supply compromise. The initiating cause is not always known, but suspected predisposing factors include segmental motility differences caused by enteritis (e.g., coccidiosis), intestinal parasitism (e.g., *Oesophagostomum* infestation), and intestinal masses.[189–193] Clinical signs and diagnosis are as described earlier. Treatment requires surgical correction, as well as fluid therapy and supportive therapy.

Torsion of the Root of the Mesentery and Cecal Volvulus

Torsion of the root of the mesentery and cecal volvulus can occur sporadically in small ruminants. Clinical signs include extreme abdominal pain, rapidly progressive abdominal distention, and circulatory collapse. Immediate surgical correction and circulatory support are needed.[187]

Foreign Body Obstruction

Ingested foreign bodies or bezoars can obstruct portions of the intestines.[194–198] The signs are similar to those of obstruction caused by other small intestinal accidents and depend on which part of the intestine is blocked. In some cases, the obstructing body can be seen with use of radiography or ultrasonography. Surgical removal is required for treatment.

Intestinal Atresia

Intestinal atresia can affect singly, or in combination, the segments of the small intestine, large colon, rectum, or anus, and are reported in common food animal species.[199–205] Apart from atresia anovaginalis (i.e., presence of a rectovaginal fistula), all cases are lethal if not surgically corrected. The clinical presentation of affected lambs and kids is progressive abdominal distention with failure to produce feces, as well as signs of abdominal pain, inappetence, weakness, and dehydration. In the case of atresia ani, lack of an anus is apparent, and bulging of the perineum may be observed when the animal strains or with abdominal palpation. A thorough physical examination is important for the discernment of other congenital abnormalities present.[205,206] Radiography (including contrast fistulogram) and ultrasonography of the abdomen, pelvis, and perineum may help classify the type of anorectal malformation present. Definitive diagnosis of atresia conditions of the colon and small intestine may require an exploratory celiotomy. The presence of other congenital abnormalities should be thoroughly evaluated on physical examination.

Surgical repair techniques are described for atresia ani and atresia coli in ruminants; however, based on economical and prognostic considerations, surgical correction of atresia ani is far more likely to be a viable option compared with atresia coli in small ruminants. A description of the surgical correction of atresia ani is reviewed later, whereas the reader is referred elsewhere for the detailed descriptions of creating a permanent colostomy and end-to-end anastomoses for the treatment of other intestinal atresia conditions.[207–209] Although not necessarily lethal, surgical correction of rectovaginal fistulas (as well as rectourethral and rectovesicular fistulas) should also be advocated, as the condition predisposes to urinary tract infections and can result in dilation of the rectum oral to the fistula, resulting in constipation and abdominal pain. Three basic classifications of intestinal atresia classification are described in animals. Type 1, membrane atresia, is caused by a membranous diaphragm occluding the lumen of the intestine. Type 2, cord atresia, is characterized by a fibrous band or muscular cord-like remnant of the gut connecting the two blind ends of oral and aboral intestinal segments. Type 3, blind end atresia, is caused by an absence of a segment of the intestine with unconnected ends and a corresponding gap in the mesentery. The etiopathogenesis of intestinal atresia conditions is likely multifactorial, including reported heritability in lambs. Affected animals should not be used for breeding purposes, ideally with neutering of the animal concurrently at the time of surgical correction of the atretic condition.[207]

Preparation of the animal for surgery should include stabilization including the administration of intravenous fluids where appropriate. Surgical correction of atresia ani is best achieved in animals in which a bulge in the perineal skin where the anus should be is appreciable. The procedure is performed using epidural anesthesia with or without light sedation, depending on the tractability of the animal. General anesthesia may be more appropriate in cases where extensive tissue dissection is anticipated. The animal is positioned in sternal recumbency with the perineal area clipped and aseptically prepared. A 1- to 1.5-cm diameter circular incision is made through the skin and subcutaneous tissues at the location of the bulge (or where the anus would

normally be located). Blunt dissection is performed cranially to identify the rectal pouch. The pouch is gently retracted caudally with tissue forceps, and the rectum is sutured to the subcutaneous tissue with four to six interrupted sutures. The rectal pouch is incised, and the rectal mucosa sutured to the skin in a simple interrupted pattern or in a simple continuous pattern performed in quadrants. In females with rectovaginal fistulas, these should be located and transected prior to suturing the rectum to the perineal skin.[207,210]

In most cases, the absence of functional anal sphincter musculature is apparent during surgery and fecal incontinence is commonly encountered postoperatively. Postoperative care should include antibiotics and the use of laxatives (e.g., mineral oil) as indicated. Cases of atresia ani amenable to surgical correction carry a relatively good prognosis given the animal is not severely debilitated at the time of initial presentation.

Intestinal Ileus

Ileus of the small intestine is a functional, nonstrangulating obstruction most often secondary to abdominal pain, inflammation, or infection, resulting in the absence of intestinal motility. The animal's failure to pass ingesta leads to signs like those of other obstructive lesions discussed earlier. The cause of ileus may be unclear, but the condition is often secondary to systemic disease or as a complication of previous surgery (i.e., postoperative ileus). The same factors that can result in rumen stasis (e.g., abdominal inflammation, pain) may result in intestinal stasis and ileus. Treatment includes fluid therapy to address electrolyte derangements and dehydration, as well as the administration of NSAIDs for control of pain and inflammation. Administration of a lidocaine continuous-rate infusion, as commonly used in horses, may be considered with the caveat of potential lidocaine toxicity.[211,212] Use of prokinetic drugs is poorly described in small ruminants and should be used with caution, especially if the underlying cause is unknown (i.e., contraindicated in true obstructive disease).[213] If signs persist, however, surgical exploration is indicated.

Peritonitis

Pathogenesis

Anatomically, the peritoneum is divided into two continuous parts, the parietal and visceral peritoneum. The parietal peritoneum lines the diaphragm, abdominal wall, and pelvic cavity, whereas the visceral peritoneum encloses the intra-peritoneal organs and forms the omentum and mesentery. Normally, a small amount of fluid lies between the parietal and visceral peritoneum, which is transparent and straw-like in color with a total protein concentration less than 3 g/dL and a total nucleated cell count of less than 5000 cells/μL.[214–216] Inflammation of the peritoneum can be infectious or noninfectious (traumatic, chemical, or neoplastic) in etiology. Classifications of peritonitis include: cause (primary or secondary); onset and duration (peracute, acute, or chronic); location (localized or diffuse); and whether bacteria are present (i.e., septic or aseptic). Septic peritonitis is most common in ruminants. Common causes of septic peritonitis include rupture of gastrointestinal viscera (e.g., intestine secondary to obstruction; rupture of the abomasum secondary to abomasitis or abomasal ulcers), leakage of bacteria and intraluminal contents from ischemic or compromised viscera (e.g., ischemic intestine, uterine tears, urolithiasis), iatrogenic (e.g., trocarization of the rumen for bloat), and complications associated with surgical manipulation and entry into abdominal viscera (e.g., leakage at resection and anastomosis site).[217]

Clinical Signs

Signs of peritonitis are often nonspecific and are dependent on the stage, extent, and severity of the underlying condition. Clinical signs include altered body temperature, depression, anorexia, dehydration, and reduced gastrointestinal motility, including abdominal distention, reduced fecal output, and colic. The presence of fever is variable, and the systemic effects of dehydration, bacteremia, and endotoxemia typically manifest as tachycardia, tachypnea, and injection of mucous membranes. Peracute cases may present as sudden death, whereas chronic cases may include weight loss, poor body condition, intermittent diarrhea, or lack of fecal production with changes in abdominal contour.

Diagnosis

Abdominocentesis is important for the definitive diagnosis of peritonitis. Fluid should be collected in an EDTA tube for cytologic analysis, protein concentration, and Gram staining; in plain sterile tubes for aerobic and anaerobic culture; and in a lithium heparin tube if biochemical analysis is needed. The gross appearance of abdominal fluid can be suggestive of peritonitis, including an increase in the amount of fluid, and changes in the color, transparency, viscosity, and odor (e.g., cloudy, turbid, or red-tinged, to thick and purulent in character). Increases in total white blood cell counts, the percentage of neutrophils, and total protein concentration are observed.[218–221] In septic peritonitis, degenerative changes to neutrophils may be appreciable and, on occasion, intracellular bacteria are observed on cytologic examination.[215] Aerobic and anaerobic culture of abdominal fluid, including antimicrobial sensitivity testing, is indicated for proper treatment. A Gram- stain may aid identification of bacteria and assist in the choice of antimicrobial therapy before culture results are known (or in the absence of a positive culture). Failure to identify or culture bacteria should not rule out a diagnosis of septic peritonitis. Septic peritonitis usually involves a mixed bacterial population depending on the source of peritoneal contamination. Common bacterial isolates from exudative peritonitis include the Enterobacteriaceae, obligate anaerobic bacteria, and gram-positive organisms. Rumen bacteria are typically gram-negative anaerobes, whereas *E. coli* and other enteric species are common if the intestine is the source of infection.[218]

Use of ultrasonography may be useful in detecting increased amounts and changes in peritoneal fluid, as well as locating fluid pockets for abdominocentesis.[222] Hematologic and biochemical parameters reflect changes expectant of a systemic inflammatory response or sepsis (e.g., an inflammatory leukogram and, in severe cases, a degenerative left shift). Exploratory laparotomy may be required to identify the presence and source of abdominal infection.

Treatment

Treatment includes supportive, antibiotic, and surgical therapies. Supportive therapy includes crystalloid fluid administration to correct shock and electrolyte imbalances. Other supportive measures include NSAID medications (e.g., flunixin meglumine) for their pain control and anti-endotoxemia effects, as well as transfaunation. Systemic broad-spectrum antibiotic therapy is indicated with appropriate changes made when culture and susceptibility results are available. Surgical therapy includes peritoneal debridement, irrigation, and

drainage. This may entail surgical correction of leaking abdominal viscera and physical removal of gross contamination. Use of abdominal drains can be problematic in ruminants, often readily becoming clogged with fibrin. The prognosis is guarded, especially if an intestinal rupture has occurred.[217]

Rectal Prolapse

Clinical Signs, Classification, and Pathogenesis

The typical presentation of a rectal prolapse is a mucosal mass (types I, II, and III) or tube (type IV) protruding beyond the anus with variability in the extent of edema, bruising, inflammation, and necrosis present. Type I rectal prolapse involves only the rectal mucosa and submucosa which can be symmetrical or asymmetrical in its protrusion from the anus. Type II rectal prolapse is a full thickness prolapse of all or part of the rectal ampulla. Type III rectal prolapse is a continuation of a type II rectal prolapse, with the addition of a variable amount of small colon intussuscepted into the rectum. Type IV rectal prolapse involves the intussusception of the peritoneal rectum and variable length of the small colon through the anus.[223] Types I to III palpate as a continuous protrusion from the mucocutaneous junction of the anus, whereas type IV is tube-like in appearance and forms a palpable trench inside the rectum on manual palpation. Rectal prolapse is more common in sheep than in goats and is often associated with short docked tails in lambs.[224] Producers should be encouraged to dock tails at the level of the attachment of the caudal tail fold rather than close to the body, as the latter is associated with an increased incidence of rectal prolapse. Other causes of rectal prolapse include excessive straining associated with diarrhea (e.g., coccidiosis, salmonellosis), chronic coughing, and tenesmus associated with dystocia or urolithiasis.[225] Over-conditioning (i.e., fat animals), grazing lush pastures or feeding of legumes (e.g., alfalfa, clover), and use of growth implants are also implicated as risk factors for the condition.[226] Regardless of the cause, eversion and exposure of the rectal mucosa results in irritation and inflammation, which causes further straining, which results in a vicious cycle of more and more tissue becoming prolapsed. Venous drainage of the prolapsed tissue may become compromised, further contributing to the swelling. Exposed tissue becomes edematous, inflamed, and eventually necrotic.

Treatment

Management of rectal prolapse includes the immediate resolution of the prolapse as well as addressing predisposing risk factors or causes for increased straining (e.g., treatment of coccidiosis). The type of rectal prolapse and severity of damage to the exposed tissue plays an important role in the type of treatment method selected. In general, the rectum recovers relatively well from injury and an attempt to salvage the prolapsed tissue should be made whenever possible, albeit within reason, as deep necrosis or extensive trauma to the tissue may necessitate surgical resection. A caudal epidural using 2% lidocaine should be performed to facilitate examination and cleansing of the prolapsed tissue while eliminating straining and providing adequate anesthesia for placement of a purse-string suture and surgical procedures, if necessary. Depending on the tractability of the animal, sedation may be required. In sheep with extremely short-docked tails, a lumbosacral epidural may be easier to perform and more likely successful in providing complete anesthesia of the perineum. In very mild, early type I cases, frequent topical application of hemorrhoidal ointment and replacement of the everted tissue may be successful and preclude the use of a purse-string suture. Another quick and inexpensive treatment option for mild cases is the injection of counterirritants (e.g., Lugol's iodine) in the retroperitoneal, perirectal space, either alone or in conjunction with a purse-string suturing.[227] The solution is injected using an 18-gauge needle, deeply (5 cm) within the soft tissues around the anus at the 12, 3, and 9 o'clock positions. Injection at the 6 o'clock position is avoided to prevent swelling and obstruction of the urethra.[228] Although the earlier suggestions are quick and inexpensive, resolution of a rectal prolapse often requires one of the following treatment options. These include: (1) replacement of the prolapse and placement of a purse-string suture, (2) amputation using a prolapse ring, (3) submucosal resection, or (4) resection and anastomosis.[223,225,229] In the case of type IV prolapses, an exploratory celiotomy with resection of affected tissue and an end-to-end anastomosis may be indicated. With all treatment options, restricted feeding for 24 to 48 h and the administration of mineral oil (or other appropriate laxatives) is recommended. Elimination of risk factors, such as the feeding of dusty hay, as well as the treatment of diarrhea or pneumonia should be carried out following replacement of the prolapse, as these conditions lead to increased abdominal pressures due to coughing or straining. In animals where correction of the rectal prolapse is cost-prohibitive, immediate harvest or euthanasia is recommended. Tetanus prophylaxis should be provided. Antibiotics used should be effective against anaerobes (e.g., penicillin), and their use is indicated when extensive necrosis and tissue damage is present (even if the prolapse is successfully replaced) or when surgical techniques are performed.

The use of an epidural anesthetic (e.g., 2% lidocaine, 0.5 mL/45 kg of BW) to decrease straining and ease pain associated with the procedure is required, regardless of treatment option used. The reader is referred to Chapter 18 for description of both caudal and lumbosacral epidural procedures. Combining xylazine (0.01–0.03 mg/kg) with 2% lidocaine may provide longer analgesia and reduced straining than that obtained by lidocaine anesthesia alone. In animals with irretractable straining, use of an alcohol epidural with either isopropyl alcohol or ethanol may be required to prevent straining for extended periods. This type of anesthesia is permanent and is not without risk, as its use causes demyelination of both sensory and motor nerves.[228] Potential complications include sciatic nerve damage, injection site necrosis, and the inability to pass feces. Therefore, it should be reserved for salvage purposes in animals intended for harvest. Because of the potential sciatic nerve damage, the clinician should perform a test injection of 2% lidocaine before using alcohol, to ensure the epidural is effective in eliminating straining with no apparent ataxia or muscle weakness in the hindlimbs. Following the test injection, a mixture of equal parts lidocaine and alcohol is used at the site previously injected.[228]

Replacement and Purse-String Suture. This technique is indicated for the treatment of salvageable rectal prolapses. Following epidural anesthesia, the prolapsed tissue is thoroughly evaluated, and the perineum and prolapsed tissue cleansed with a mild antiseptic. Edema can be reduced with the temporary topical application of hyperosmolar solution, such as granulated sugar or hypertonic saline. The prolapsed tissue is coated with lubricant (e.g., petroleum jelly) and gently manipulated back into its normal position. Placement of a purse-string suture is accomplished using appropriately sized nonabsorbable suture on a cutting

needle, with tissue bites situated at the mucocutaneous junction of the anal sphincter. To minimize fecal contamination and allow easy adjustment, the bow-knot should be situated either dorsal or lateral to the anus. To facilitate tightening of the purse-string suture, an appropriately sized syringe case, tube, or the clinician's finger is placed in the rectum during suture tightening which is subsequently removed once the purse-string suture is secured. The purse-string suture is tightened sufficiently to prevent recurrence of the prolapse while allowing the passage of feces. Removal of the purse-string ideally should be within 1 week of placement to minimize suture-tract infection. If possible, topical application of petroleum jelly and hemorrhoidal gel daily will reduce inflammation and edema, facilitating earlier removal of the purse-string suture.[225,230]

Rectal Amputation Using a Prolapse Ring. Placement of a prolapse ring should be considered a salvage procedure. This procedure is often used under field conditions where surgical procedures are not economically or logistically possible. The prolapse ring is inserted into the rectum and an elastrator band placed. If a ligature is used in place of an elastrator band, it should be tightened sufficiently to allow purchase on the prolapse ring. Both the elastrator band or ligature serve to induce vascular compromise and necrosis of the aboral, prolapsed tissue eventually causing it to slough. A fibrotic band forms cranial to the elastrator band and mucosa subsequently grows across the area. Complications include failure due to premature dislodgement of the prolapse ring, rectal stricture, peritonitis, and abscess formation.[229]

Submucosal Resection. Submucosal resection involves the removal of necrotic or traumatized mucosa while salvaging healthy, underlying tissue. Advantages of submucosal resection compared with full-thickness resection (amputation) and anastomosis include: faster healing times, less postoperative straining, minimal constriction of lumen diameter, salvage of healthy tissue, the maintenance of the main blood supply thereby minimizing postoperative hemorrhage, and by not exposing the serosa, a reduction in the likelihood of peritonitis and perirectal abscess formation. However, submucosal resection requires more surgical time.

After surgical preparation of the perineum and prolapsed tissue, a piece of flexible tubing of appropriate diameter is inserted into the lumen of the prolapsed tissue and is fixed in place using two 18-gauge, 15-cm (6-inch) spinal needles placed at 90 degrees to each other in a cross-pinning fashion. Alternatively, stylets from similar in length catheters can be used if appropriate spinal needles are not available. The needles are placed through the external anal sphincter and approximately 2 to 4 mm cranial to the prolapse in healthy tissue in order to maintain the prolapse during dissection. Two complete circumferential incisions are made through the mucosa (of healthy tissue) on either side of the tissue to be removed. A longitudinal incision at the same tissue depth is made to connect these circumferential incisions. Deep dissection of the necrotic mucosa and submucosa is carried out, essentially elevating a strip of tissue to be removed but leaving a deep layer of healthy tissue. Hemorrhage is controlled by ligature of individual vessels. The mucosa is aligned with four simple interrupted sutures placed equidistant around the circumference of the prolapse, in order to prevent twisting of the closure. The four quadrants are then apposed separately in a simple continuous suture pattern, using 2-0 to 3-0 monofilament absorbable suture material on a taper needle. The spinal needles are removed, and the tissue replaced into the rectum.[225,229,231] A purse-string suture is placed as previously described to prevent prolapse of the surgical site. Postoperative management is as described earlier for all treatment options. The use of a nonsteroidal antiinflammatory postoperatively should be considered for control of pain and inflammation.

Resection and Anastomosis. Resection and anastomosis may be indicated in types III and IV prolapses if the prolapsed tissues are devitalized or the amount of tissue precludes the ability of reduction. The procedure can be performed as for submucosal resection, including the pinning technique for stabilizing the tissue during dissection. In contrast to the submucosal resection, full-thickness circumferential incisions are made through the inner and outer walls of the intussusceptum (in healthy tissue) with removal of all necrotic tissue. All mesenteric vessels within the prolapse are identified and ligated during resection. The proximal and distal edges are apposed with a monofilament absorbable suture (e.g., 1–2 polydioxanone suture (PDS)) using full-thickness, interrupted horizontal mattress sutures circumferentially. The mucosal edges are then apposed with a simple continuous pattern using 2-0 monofilament absorbable suture (e.g., PDS), divided into interrupted quadrants.[225] Alternatively, a stairstep amputation can be performed to maximize the length of the inner mucosal and submucosal layers, which facilitates easier adaptation of mucosal apposition of the respective segments.[230] Following resection and anastomosis, the cross-pins are removed and a routine purse-string suture is placed. Aftercare is as for the other techniques described. Antibiotics should be administered for 7 to 10 days postoperatively and the animal closely monitored for signs of peritonitis and sepsis. Potential complications include stricture formation, dehiscence of surgical site resulting in peritonitis, or evisceration of intestines, adhesion formation, and abscessation of the perirectal tissues.

Prevention

Preventative measures should address management practices that predispose to rectal prolapse. Importantly in sheep, this includes advising producers on the appropriate length of tail docking. Environmental factors should be addressed, including removal of dusty feeds and improvements in ventilation and air quality in intensively housed small ruminants to minimize coughing. Prevention and treatment of disease conditions, including diarrhea, pneumonia, and urinary calculi should be instituted. Other conditions such as over-stocked or unhygienic living conditions should be addressed. The producer should consider culling animals with a history of rectal prolapse from the breeding flock or herd. Sound nutrition and feeding practices, with frequent monitoring of BCS to avoid over-conditioned (excessively fat) animals will also aid in the prevention of rectal prolapse.

Diseases of the Liver

Liver Abscess

Formation of liver abscesses usually is the result of chronic rumenitis in cattle, but these lesions are rare in sheep and goats. They may occur in feedlot lambs and kids and other animals fed rations high in grain. In lambs and kids, septicemia or extension of an umbilical vein infection can lead to formation of liver abscesses.[232] In most cases, however, liver abscess is an incidental finding. Weight loss, anorexia, depression, and decreased production (e.g., growth, milk) may be noted in affected animals.

In adults, *C. pseudotuberculosis* is the most common pathogen. *Actinomyces pyogenes* and *Fusobacterium necrophorum* also are cultured from abscesses.[232,233] Liver enzymes may or may not be

elevated. Diagnostic ultrasonography of the liver may help detect abscesses, especially if they are numerous and widespread. However, no specific treatment or control measure is available. Many of the preventive protocols used for feeder cattle can be applied to the control of abscesses in sheep and goats. Such strategies include slowly introducing concentrates into the diet, offering long-stemmed hay on a free-choice basis, and including rumen buffers (alkalizing agents) and antimicrobial agents in the feed.

Pregnancy Toxemia and Fatty Liver Syndrome

Pathogenesis. Fatty liver occurs in conjunction with pregnancy toxemia in ewes and does during the last month of gestation.[234,235] It is most common in both thin or obese ewes or does with a single large fetus, twins, or triplets.[235,234–236] During late gestation, particularly in obese females, the abdominal space is filled with accumulated fat and an ever-expanding uterus. Because of the lack of rumen space, these animals have difficulty consuming enough feedstuffs to satisfy energy requirements. In most management systems, late gestation occurs during the winter months when less pasture is available and poorer quality feedstuffs are offered. Energy requirements for ewes and does carrying twins or triplets are greatly increased during the final 2 months of gestation, because 70 to 80% of fetal growth occurs during this time. Ewes with twins require 180% more energy, and those with triplets need 200 to 250% more dietary energy. Glucose maintenance in ewes pregnant with twins is significantly more prone to disturbance resulting in hypoglycemia than in ewes bearing singletons.[235] Pregnancy toxemia also occurs in association with anorexia caused by other diseases (e.g., foot rot, OPP, CAE) or sudden stresses (e.g., feed or weather changes, predator attacks, hauling). A period of anorexia or lack of sufficient energy intake will result in a negative energy balance. Affected animals begin to mobilize body stores of fat and transport them to the liver. In the liver, fat is catabolized to glycerol and free fatty acids (FFAs). FFAs can be used in the citric acid cycle (Krebs cycle) as an energy source, but not in the direct formation of glucose. Anorexic animals have less ruminal substrate available for production of the glucose precursor, propionic acid. However, oxaloacetate, which is an integral part of the citric acid cycle, is removed from the cycle and converted into glucose. Depletion of oxaloacetate inhibits the normal function of the citric acid cycle, thereby inhibiting the use of FFAs. As the pool of FFAs increases, they are converted to ketone bodies or repackaged into lipoproteins. Because ruminants are not efficient at transporting lipoproteins out of the liver and back to the adipose stores, the lipoproteins overwhelm the liver's ability to handle fats, leading to a massive buildup, and resulting in a fatty liver. Because less substrate is available for glucose formation, more oxaloacetate is "cannibalized" from the citric acid cycle, further inhibiting the body's ability to use FFAs. This impairment in turn results in the continued accumulation of ketones. Hypoglycemia, hyperketonemia, and, potentially, uremia and death can occur.

Clinical Signs. Animals suffering from fatty liver or pregnancy toxemia become anorexic and depressed or dull, with altered behavior patterns, and may lag behind others in the group or become recumbent. Some are constipated, grind their teeth, have a ketone smell to the breath, demonstrate labored breathing or frequent urination, and suffer from dystocia. Neurologic signs include blindness, circling, incoordination, "star-gazing," tremors, and convulsions.[237–239] Death can occur if the condition is left untreated. In the case of fetal death in utero, maternal septicemia-endotoxemia and death are common sequelae.

Diagnosis. Diagnosis is based on clinical signs, the presence of multiple fetuses, and typical clinicopathologic findings.[234] CBC results may be normal or show an eosinophilia, neutropenia, and lymphocytosis. Affected animals may or may not be hypoglycemic, but ketoacidosis, hypocalcemia, and hypokalemia are common.[236–239] Liver enzymes usually are within normal limits but occasionally may be increased. Azotemia, both from dehydration and secondary to renal disease, is a common finding, and a fatal uremia may occur. Blood concentrations of β-hydroxybutyric acid greater than 7 mmol/L are consistent with pregnancy toxemia. Urinalysis will be positive for both ketones and protein.[234] Urine is collected from sheep by holding the nares and from does by frightening them and then allowing them a perceived escape, whereupon they stop, squat, and void.

Although not commonly performed, liver biopsy can help determine the extent of fatty infiltration. Serum protein pattern changes may become an available tool in the diagnosis of this condition in the future.[236] This syndrome must be differentiated from hypocalcemia, hypomagnesemia, PEM, encephalitis, lead toxicity, and cerebral abscesses.

Treatment. Very early cases (before onset of recumbency) may be treated with oral or intravenous glucose. A balanced electrolyte solution with extra calcium (25 mL of 23% calcium borogluconate/L), potassium (10–20 mEq/L), and 5% dextrose is needed.[234] In some cases, sodium bicarbonate is valuable in treating acidosis (see Chapter 3). Energy intake must be increased, and propylene glycol can be administered (15–30 mL every 12 h) as a glucose precursor. Rumen transfaunation and supplementation with vitamin B complex (including vitamin B_{12}, biotin, niacin, and thiamine) also are recommended.

After affected females become recumbent, treatment must be very aggressive. Flunixin meglumine (2.5 mg/kg once daily) appears to improve survivability, but should be used in conjunction with other therapies.[234] Flunixin meglumine can be given daily in depressed anorexic animals, and its use appears to improve feed intake.[234] Researchers using recombinant bovine somatotropin showed a response, but it was not significant in comparison with that in control animals.[240] Removal of the fetuses is crucial in these cases. Chemically inducing parturition (by administering 2.5–10 mg of prostaglandin $F_2\alpha$ or 0.75 μg/45 kg of cloprostenol in does and 15–20 mg of dexamethasone in ewes) and giving the ewe or doe medical support (fluids, B vitamins, glucose) while waiting is a useful protocol in some cases. Unfortunately, during the time before parturition, endotoxemia from dead fetuses further compromises the female's wellbeing. For this reason, we recommend immediate cesarean section in depressed moribund animals (see Chapter 8). The owner should be forewarned of the poor prognosis for animals already in a moribund state. Fluid support during and after surgery is crucial.

Regardless of the therapeutic plan, the animal should be offered a palatable, energy-rich, highly digestible feedstuff. The keeper and the clinician should take care to minimize the risk of a confounding disease during convalescence (e.g., lactic acidosis, PEM).

Prevention. Fatty liver and pregnancy toxemia can be prevented through proper management and nutrition. Maintaining animals in proper body condition throughout the year and making sure energy and protein levels are adequate in late gestation (see Chapter 2) are two key preventive measures.[234,237,238] The owner or manager should be taught to assess body condition in individual animals, avoid extremes in body condition, and maintain emergency stores of feed in case of severe weather or natural disasters. In over-conditioned females, the keeper should be encouraged to restrict institution of

weight loss programs to early gestation (if at all) and to avoid abrupt feeding changes, while promoting exercise (e.g., by increasing walking distances from mineral access to shelter). The requirement for energy may be one and a half to two times maintenance for dams with single fetuses and two to three times maintenance for those with multiple fetuses. Prevention of concurrent disease, which may further increase energy demands or cause anorexia (e.g., intestinal parasitism, foot rot), is crucial. The keeper should take care to increase the grain portion of the diet slowly, and ensure the consistent availability of fresh, clean water, as anorexia from rumen upset can lead to pregnancy toxemia. Ewes should be offered 0.5 to 1 kg of a cereal grain (corn, oats, barley, or a combination) every day during the final months of gestation; does can be offered 1/2 to 1 kg of grain. Keepers should maintain ewes and does at a BCS of 2.5 to 3 (see Chapter 2) throughout gestation and evaluate the animals' energy intake every 2 to 4 weeks.

Ultrasonography can help identify females with multiple fetuses. These animals should be separated into groups and fed accordingly.[234] Ultrasonographic determination of fetal numbers is best accomplished between 35 and 90 days after breeding (see Chapter 8). Determination of fetal number may be enhanced with use of proper technique: shearing the hair or fiber in front of the udder; applying a coupling substance to the skin (e.g., alcohol, oil, lubricating gel); and interrogating (viewing) as much of the abdomen as possible while systematically moving from one side of the posterior abdomen to the other, to obtain an appreciation of the abdominal structures including any fetuses present.

Animal keepers and clinicians should ensure that ewes are healthy and free of chronic diseases (e.g., OPP, CAE, foot rot, chronic parasitism) and that a good-quality trace mineral salt mixture is available on a free-choice basis. The addition of lasalocid (0.5–1 mg/kg/day) or monensin (1 mg/kg/day) to the feed or mineral mixture will enhance the formation of the glucose precursor propionic acid and improve the efficiency of feed use. Monensin should be used with caution, however, because associated toxicity has been reported; the agent should be composed of no more than 30 ppm of the complete diet. The inclusion of niacin (1 g/head/day) in a feed supplement or mineral mixture will help prevent pregnancy toxemia. Supplementation with lasalocid, monensin, or niacin should begin 2 to 4 weeks before the animals give birth.

Shearing in the last trimester also is recommended in ewes.[238] Many sheep producers routinely clip the wool around the vulva. If complete body shearing is performed, the incidence of fatty liver or pregnancy toxemia may be decreased, by several mechanisms: sheared sheep require less energy to walk and graze. Sheared ewes also tend to shiver on cold days, exercising the enzyme systems that promote the more efficient use of FFAs as energy substrate. These ewes tend to seek shelter during cold weather, which may decrease lamb losses resulting from hypothermia. Obviously, if ewes are to be shorn, keepers should make adequate shelter available.

Keepers should avoid hauling or moving females during late gestation. Proper predator control measures should be maintained. Good hoof care programs should be in place on farms or ranches where grazing is the predominant form of nutrient intake. Sheep and goats should have their teeth checked to ensure good dentition before the breeding season. Animals with poor teeth should be culled.

Measuring serum β-hydroxybutyric acid concentrations is useful in assessing energy status in ewes. Values of 0.8 to 1.6 mmol/L suggest a negative energy balance. Keepers should take steps to correct the problem by feeding better-quality, more digestible feedstuffs.

White Liver Disease

White liver disease is a form of fatty liver disease reported only in Angora and Angora-cross goats and sheep. It is associated with cobalt deficiency.[241–245]

Pathogenesis. Cobalt is needed by rumen microflora to produce cyanocobalamin, or vitamin B_{12}, which is a coenzyme for methylmalonyl-coenzyme A (CoA) mutase. This enzyme is in turn needed to convert propionate to glucose through the Krebs cycle. Cobalt deficiency leads to the accumulation of methylmalonyl-CoA, or methylmalonic acid, which is converted to branched-chain fatty acids that accumulate in the liver. Diets high in grain, which is fermented to propionate, coupled with deficient or marginal cobalt intake, may predispose to this condition. White liver disease has not been reported in the United States, but ill thrift from cobalt deficiency has been observed. It is therefore possible that the disease goes unrecognized in some cases.[242–245]

Clinical Signs. Signs most commonly are seen in young animals and include ill thrift, anorexia, and diarrhea; sheep may exhibit photosensitivity. Clinical laboratory findings include a macrocytic-normochromic anemia and hypoproteinemia.[232,242,245]

Diagnosis. Abnormal serum or liver concentrations of vitamin B_{12} or liver cobalt levels are the basis for diagnosis. Liver cobalt concentrations of 0.08 ± 0.02 ppm determined on a dry matter basis were reported in goats with white liver disease, compared with 0.53 ± 0.11 ppm in control animals.[242,243]

Treatment and Prevention. Sheep can be treated with oral cobalt (1 mg/head/day) or vitamin B_{12} injections. The condition usually can be prevented by including cobalt in the ration by feeding a good-quality trace mineral salt; however, in areas in which cobalt is extremely deficient or absent from all feedstuffs, the oral administration of cobalt-containing "bullets" along with supplementation with a cobalt-containing salt-mineral mixture, may be required.[244]

Copper Toxicosis

Pathogenesis. Copper toxicosis is more common in sheep than in goats.[232,237,239] Goats appear to excrete copper more efficiently than sheep and are more cow-like in their ability to resist toxicosis, but nevertheless are susceptible.[232,237,246–248] The use of copper oxide wire particles to treat internal parasitism has been suggested as a cause of copper toxicity in goats. Toxicity results from chronic accumulation in the liver from the ingestion of excess copper in relation to molybdenum or sulfate in the diet. In sheep, a copper-to-molybdenum ratio greater than 10:1 leads to the accumulation of excess copper. The most common sources of excess copper in sheep and goats are trace mineral mixtures and feeds formulated for cattle or horses. Clinical signs often are absent during the chronic accumulation phase. Onset of acute disease is related to the sudden release of copper from the liver in large amounts. Stress usually precipitates this acute phase. Acute release of copper and subsequent high blood copper concentrations cause an acute hemolytic crisis, resulting in anemia, hemoglobinuria, and acute renal failure. Existing hepatic disease (such as that caused by liver flukes) may predispose animals to this condition. Some breeds (e.g., Merino sheep) seem to be prone to copper absorption and storage problems, whereas others (e.g., pygmy goats) tend to be more resistant and prone to deficiency (see Chapter 2).

Clinical Signs. Anorexia, depression, diarrhea, and weakness all are signs of copper toxicity. In many instances, affected animals are found dead with hemolysis and icterus. Abdominal pain and diarrhea sometimes are present. Port wine-colored urine is evidence of hemoglobinuria. Hemoglobinemia produces icterus of the mucosal membranes and fever.

Diagnosis. Findings on clinicopathologic examination include anemia, hemoglobinemia, hyperbilirubinemia, increased liver enzymes, and azotemia. Urinalysis reveals hemoglobinuria and isosthenuria. The combination of azotemia and isosthenuria indicates acute renal failure. Definitive diagnosis of acute disease requires measurement of copper concentrations in serum. Normal blood copper concentrations are approximately 50 to 200 μg/dL in sheep and goats.[249] These concentrations increase ten- to twentyfold with an acute hemolytic crisis.[237] On necropsy, kidney copper concentrations are the most diagnostic tissue, because liver concentrations may be normal after release into the bloodstream. Generally, kidney concentrations greater than 100 ppm and liver concentrations greater than 350 ppm on a dry matter basis are diagnostic. If tissue copper is reported in wet weight, the conversion to dry tissue weight can be estimated by multiplying the tissue concentration by a factor of 3.5.

Treatment. Treatment of acutely affected animals often is futile. Appropriate management consists of supportive therapy for the acute renal failure and anemia and attempts to lower liver copper stores. Fluid therapy for the acute renal failure (see Chapter 3) is of clinical benefit, and a blood transfusion may be needed if the PCV drops precipitously. Ammonium tetrathiomolybdate (1.7 mg/kg IV or 3.4 mg/kg SC on alternate days for three treatments) is the most economical agent for treatment in acute cases. In valuable animals, oral D-penicillamine (26–50 mg/kg twice daily or 52 mg/kg once daily for 6 days) increases urinary copper excretion. Trientine is used in human beings but has shown variable results in sheep. Treatment of the remainder of the flock should include the oral administration of ammonium molybdate (50–500 mg/head/day) and sodium thiosulfate (300–1000 mg/head/day) for 3 weeks. Stress should be minimized, so keepers and clinicians should delay routine maintenance procedures such as deworming and hoof trimming until after treatment. When applicable, spraying a combination of ammonium molybdate and sodium sulfate onto harvested forages low or deficient in copper to approximate the required therapeutic amount will decrease the stress required in daily oral dosing of chemicals. Allowing free access to grazing of forages high in sulfur (greater than 0.5% sulfur), if available, for all surviving ambulatory animals also may help to minimize death losses in a flock or herd. Overzealous attempts to clear excessive hepatic copper stores may potentially lead to deficiency, excessively stress the animal, and can be costly, thus should be avoided. The offending source of copper should be eliminated. Caution should be taken in such cases to remove ionophores from the diet, because these agents may contribute to copper absorption.[250]

Prevention. Avoiding high dietary copper (more than 10 ppm), a high copper-to-molybdenum ratio (greater than 10:1) in the feed, use of copper-containing foot baths, and other sources of copper is crucial. Including supplemental molybdenum in the diet to lower the copper-to-molybdenum ratio to 6:1 to 8:1 is beneficial. Addition of up to 2 to 6 ppm of molybdenum may be required in many instances.

Often too much emphasis is placed on the trace mineral component of the diet. The clinician should be aware that even if no copper is added to the trace mineral mixture and the element does not appear on the product label, the mineral mixture may nevertheless contain copper. Many components of mineral mixes are contaminated with copper (zinc sulfate may contain 400 ppm of copper, dicalcium phosphate may contain more than 30 ppm of copper). Therefore, the clinician needs to perform a dietary analysis to find and correct the problem.

Toxic Hepatitis

Pathogenesis. The liver is vulnerable to toxic insult because one of its major functions is detoxification. The most common plants that are gastrointestinal and liver toxins are shown in Table 5.3. Clinical signs will depend on the offending agent. Acute, severe toxicity is more common with chemical toxicosis, whereas plant toxins usually cause chronic disease. A thorough history is important, and in many cases, inspection of the animals' environment is required.

Clinical Signs. The clinical signs of toxic hepatitis can be subtle and nonspecific. Animals may exhibit only anorexia and depression. Icterus is more common with hemolytic diseases and is not always seen with liver disease. Photosensitivity is a common clinical feature in ruminants, and hepatoencephalopathy also can occur.

Diagnosis. Clinicopathologic data are more helpful in diagnosing acute toxicity. Serum aspartate aminotransferase (AST) and lactic acid dehydrogenase (LDH) levels can increase with hepatocellular necrosis, but such changes are not liver-specific, so muscle injury and disease must be ruled out. These enzymes also increase if serum is not separated from a blood clot in a timely fashion.[232] Increased levels of alkaline phosphatase (AP) and gamma-glutamyl transferase (GGT) indicate biliary stasis. AP concentrations also are not liver-specific, but increased serum levels of GGT are very specific for liver disease. GGT also increases in some hepatocellular diseases, so testing for normal concentrations is important.[249] Unfortunately, levels of all of these enzymes can be normal with liver disease, especially if it is chronic. Hyperbilirubinemia, hypoglycemia, low blood urea nitrogen (BUN), and hypoalbuminemia are not always evident, as is classically taught. If hepatoencephalopathy is suspected, blood ammonia concentrations may be elevated. Blood ammonia analysis may be impracticable in the field, because the blood should be kept on ice and the test should be performed within 30 minutes of collection. To enhance the accuracy of blood ammonia analysis, the clinician should collect blood from a normal control animal for comparison. Ammonia concentrations three times those in the control animal are diagnostic.[251] Liver biopsy remains the most valuable tool for diagnosing liver disease. Although clotting dysfunction may occur in liver disease, it is an uncommon complication in ruminants, and risk of bleeding should not discourage the clinician from performing a liver biopsy.

Treatment. If the intoxication is caught in the acute stage, activated charcoal (500 g in the adult animal) can be given. Supportive care, especially fluid support with dextrose solutions, is the mainstay of therapy. Low-protein diets may suppress ammonia production temporarily, but they can be detrimental over time, depending on the production status of the animal. Animals exhibiting photosensitivity should be housed indoors if possible, and broad-spectrum (systemic or topical) antibiotics may be necessary to control secondary bacterial dermatitis. Corticosteroids (e.g., dexamethasone 0.1 to 1 mg/kg IV or IM) may be indicated in early cases of photosensitization to decrease inflammation. Neurologic signs can be controlled with phenobarbital (initial dose: 10–20 mg/kg IV diluted in saline and administered over 30 minutes; subsequent doses: 1–9 mg/kg IV diluted in saline, as needed, up to three times daily). Diazepam (Valium) is contraindicated in hepatoencephalopathy because it may worsen deficits.[252]

TABLE 5.3 Plants That may Cause Gastrointestinal or Hepatic Disease.

Plant	Comments	Signs
Cocklebur	Erect annual herbage in sandy soils, flood plains, and overgrazed pastures; seeds are toxic	*Within hours to days of ingestion:* anorexia, vomiting, colic, dyspnea, gastroenteritis, chronic hepatitis, hepatic damage, death
Senecio (groundsel), *Crotalaria,* heliotrope, *Amsinckia* (fiddleneck), *Echium*	Pyrrolizidine alkaloids; excreted in milk and urine and can cross placenta; young more susceptible	Dullness, weakness, weight loss, icterus, fibrosis, hepatocytomegaly on histopathology, bile duct proliferation, photosensitivity, subcutaneous edema, diarrhea
Lantana	Found in sandy, tropical areas; berries, leaves, and hay are toxic	*Chronic toxicity:* slow hepatic failure, icterus, photosensitization, weakness, bloody diarrhea, cholestasis, hepatic failure
Sneezeweed, bitterweed, rubberweed	Grows in overgrazed pastures; all parts of plant are toxic	*Acute toxicity:* gastrointestinal upset, depression, serous nasal discharge, salivation, bloat; *chronic toxicity:* vomiting, hepatic and renal congestion, gastric edema, aspiration pneumonia, pulmonary edema
Cabbage, kale, rape, mustard, wild mushroom	Remove from diet; add iodine to diet (for goiter)	Gastroenteritis, hepatic necrosis, photosensitization, goiter, hemolysis
Horsebrush	Stop grazing, keep animals indoors	Itching, uneasiness, inflamed eyes, blindness, serum discharge from scabs; degenerative changes in liver and elevated liver enzymes
Clover (crimson, red, subterranean burclover)		Photosensitization
St. John's wort	Perennial herb; grows along roadsides and in overgrazed fields; remove from diet and keep animals in shade	Increased respiration, diarrhea, pruritus, dermatitis, death
Blue-green algae	Toxic after a bloom	Vomiting, diarrhea, liver failure, photosensitization; necropsy findings include swollen bloody liver, edema around gallbladder, centrolumbar apoptosis, necrosis
Pokeweed		Vomiting, cramps, diarrhea, weakness, dyspnea, prostration, tremors, convulsions
Gossypol (cottonseed)	Toxicity seen in younger pre-ruminants	Poor performance, convulsions, cardiac toxicity
Rhubarb	Contains oxalic acid	Gastrointestinal toxicity
Oak	Acorns and oak buds are most toxic	Abdominal pain, pseudomembranes in gastrointestinal tract, bloody diarrhea, depression, renal toxicity
Castor bean	Beans most toxic	Gastrointestinal irritation, bloody diarrhea, central nervous system disturbances
Mistletoe	Berries not toxic	Nausea, diarrhea
Other potentially pathogenic plants English ivy *Sesbania* Narcissus Elderberry Spurge Buckwheat Queen Anne's lace Milkweed Parsley, giant hogweed		

Miscellaneous Liver Diseases

Congenital hyperbilirubinemia, or black liver disease, occurs in certain mutant Corriedale sheep.[232] The underlying disorder, very similar to *Dubin-Johnson syndrome* in humans, is a genetically recessive condition characterized by an abnormality in the excretion of conjugated bilirubin and phylloerythrin. Appearance of disease manifestations in animals often is related to consumption of green forage. Clinical signs include anorexia, photodermatitis, and icterus. Liver biopsy in affected animals reveals dark pink to black granules in otherwise normal hepatocytes. The syndrome first manifests itself in lambs around 5 months of age.[253]

A similar condition, termed *Gilbert's syndrome* in people, occurs in Southdown lambs around 6 months of age. It appears to

be a recessive condition characterized by decreased hepatic uptake of phylloerythrin and bilirubin, with concurrent renal failure.[253] Clinical signs include icterus, photodermatitis, and ulceration around the ears and mouth. Liver biopsy reveals normal hepatic tissue. In both of these conditions, affected animals should be kept out of sunlight and fed minimal amounts of green forage. Obviously, these animals should be neutered or culled.

Various tumors of the liver, including fibrosarcoma, lymphosarcoma, and cholangiocellular carcinoma, have been reported in sheep and goats.[252,253] The use of ultrasonography and ultrasound-guided liver biopsy may aid in diagnosis.

Pathological Conditions of the Umbilicus

The umbilicus consists of the urachus, umbilical vein, and paired umbilical arteries. These umbilical remnants normally regress after birth to become a vestigial part of the bladder apex, round (falciform) ligament of the liver, and lateral ligaments of the bladder, respectively. Umbilical masses can be uncomplicated umbilical hernias or involve infection of the umbilical remnants or subcutaneous tissues, with or without concurrent umbilical herniation.[254] Umbilical hernias are a common congenital defect in ruminants. Infection of the umbilicus is a common morbidity in neonates associated with partial or complete failure of passive transfer of immunity due to inadequate colostrum intake.[255] Infection can also be the result of environmental contamination or inappropriate human handling of the umbilical cord, with excessive tension or torsion. Dipping navels with antiseptic solutions shortly after birth is commonplace and proven to reduce umbilical infections under intensive rearing conditions.[256,257] However, the overzealous use of these solutions (i.e., not allowing the umbilical stalk to dry) or the use of caustic agents (strong tincture of iodine) can cause severe inflammation and infection of the umbilical structures. Physical examination of an animal with an umbilical mass should aim to distinguish between an uncomplicated umbilical hernia from infection of the umbilical remnants with or without the presence of an umbilical hernia. Visual inspection and palpation of the mass includes evaluation of its size, shape, temperature, discharge, pain on manipulation, reducibility and the presence of a hernial ring. Deep palpation of the abdomen in a calm animal (or under sedation) may allow discernment of the umbilical structures involved. Ultrasonography can be used to determine the umbilical structures involved (and extent) as well as the characteristics of hernial sac contents (e.g., omentum, abomasum, or small intestine).[258]

Uncomplicated Umbilical Hernia

Uncomplicated umbilical hernias are considered hereditary in cattle and are a common congenital defect in sheep.[259] Although a genetic predisposition has not been identified in goats, all sheep and goat breeding stock should be free of congenital defects.[260] Umbilical hernias can also be the result of infection of the umbilical remnants and body wall. Surgical correction of umbilical hernias should be considered for those having a diameter larger than 2 cm (1 finger in diameter) and persisting beyond 3 to 4 weeks in duration. Immediate surgical intervention is indicated in animals demonstrating signs of colic associated with hernias, regardless of size.

Pinning of Umbilical Hernias. Pinning techniques using umbilical clamps or elastrator bands may be considered for umbilical hernias which are small, nonpainful, display no evidence of infection, and are completely reducible (e.g., do not contain any abdominal viscera following reduction). Pinning is most useful in females and should be used with caution in males due to anatomical considerations and complications associated with urine scalding. The animal is lightly sedated, placed in dorsal recumbency, the skin infiltrated with local anesthetic (e.g., 2% lidocaine), and then followed by reduction of the hernial sac contents back into the abdomen. The skin is then tented away from the body wall and the clamp or elastrator band applied. Large safety pins can be placed in a crossing fashion distal to the elastrator band to keep the band immediately adjacent to the abdominal wall. The ensuing inflammation will cause the hernial ring to adhere to itself and heal within 7 to 14 days. The tissue distal to the elastrator band will undergo ischemic necrosis and will eventually slough. Tetanus prophylaxis should be provided at the time of pinning. The animal should be closely monitored for signs of colic and wound complications.[7]

Umbilical Hernia Surgical Resection. In large (greater than 2 cm), uncomplicated umbilical hernias, surgical resection is the treatment of choice. The procedure can be carried out using sedation and local anesthesia (including the use of a high epidural) or under general anesthesia, with the animal positioned in dorsal recumbency. The surgical site is clipped and aseptically prepared. A fusiform skin incision is made around the umbilicus, with sharp and blunt dissection of the subcutaneous tissues to expose the hernial ring at the external rectus sheath. In males, a semilunar skin incision and reflection of the sheath and prepuce may be required, depending on the hernia size. The abdominal cavity is opened just cranial (or caudal) to the hernial ring on the linea alba to allow digital palpation of intra-abdominal structures, including the presence of adhered viscera or infected umbilical remnants. The hernial sac is then carefully excised at the scarred edge of the hernial ring. Enlarged or infected umbilical remnants and adhesions are excised (see later) before closure of the defect in the abdominal wall. Closure of the hernial ring is performed by simple apposition of the incised edges of the external rectus sheath using an absorbable suture in a simple continuous or simple interrupted pattern. If significant tension on the body wall is present, a tension relieving pattern should be used (e.g., a near-far-far-near suture). The subcutaneous tissue is closed in a simple continuous pattern using an absorbable suture, and the skin is closed based on surgeon preference.[254,261] Alternatively, a closed herniorrhaphy technique, whereby the peritoneum is not incised, may be elected in uncomplicated umbilical hernias. Closure of the body wall is as described earlier. However, an open technique has the benefit of inspecting the umbilical remnants. Postoperative management includes tetanus prophylaxis, antibiotics where indicated (as in the case of infected umbilical remnants), exercise restriction for 1 to 2 weeks, and the monitoring for excessive swelling, discharge, surgical site dehiscence, and evidence of systemic illness (i.e., peritonitis, sepsis).

Umbilical Infections

Infection of the umbilical remnants includes omphalophlebitis, omphaloarteritis, and abscessation or persistent patency of the urachus. Involvement of multiple umbilical structures may occur. A concurrent umbilical hernia may also be present. Bacterial isolates from lambs and kids with omphalitis include *E. coli*, *Trueperella pyogenes*, *Pasteurella* sp., and *Streptococcus dysgalactiae*.[262,263] On physical examination, the umbilicus is enlarged, painful to palpation, and may be actively draining purulent discharge (or urine) or have a scab suggestive of drainage in the past. If an

umbilical hernia is present concurrently, the hernia is typically only partially reducible or nonreducible, and the hernial ring is more difficult to fully discern on palpation compared with uncomplicated hernias. Deep abdominal palpation, effectively facilitated by proper restraint and sedation of the animal, may allow differentiation of the different umbilical structures. For example, the umbilical vein courses craniodorsally towards the liver, whereas the umbilical arteries and urachus course caudodorsally towards the bladder. However, ultrasonography of the ventral abdomen is the ideal method to document which umbilical structures are involved, as well as the presence of cellulitis, abscesses, or free abdominal fluid. Evidence of a patent urachus includes the presence of dermatitis, urine scalding of the ventral abdomen, and urine dribbling. The animal may have a history of poor weight gain, previous or concurrent infectious diseases (e.g., pneumonia, arthritis), and signs of systemic illness such as fever, depression, and anorexia. Use of clinicopathological analyses such as a complete blood cell count, blood culture, or cytology of the peritoneal fluid should be based on physical exam findings suggestive of sepsis or peritonitis.

Treatment

Medical Versus Surgical Management of Umbilical Remnant Infections. On occasion, some cases of omphalophlebitis-omphaloarteritis can be effectively treated medically with prolonged broad-spectrum antibiotic therapy.[7] However, if medical therapy is ineffective, the infected umbilical remnants should be surgically resected. The authors prefer timely surgical removal of the umbilical remnants over prolonged medical therapy, the latter of which may fail and still require surgical intervention.

Surgical Resection of Infected Umbilical Remnants. Anesthesia, positioning, and preparation of the surgical site is as described earlier for umbilical hernia repair. If extensive involvement of the umbilical remnants is present and prolonged or complex resection anticipated, general anesthesia should be considered. The surgical site should be of sufficient size to allow the abdominal incision to be extended cranially or caudally (depending on the umbilical structures involved), including the need to perform marsupialization of the umbilical vein or visualization of the bladder. Draining tracts should be sutured closed prior to surgery to prevent contamination of the abdomen. A fusiform skin incision is made around the infected umbilicus, with sharp and blunt dissection of the subcutaneous tissues to expose the fibrous ring. A small incision is made in the linea alba cranial or caudal (opposite to infected umbilical structure). Initially, digital palpation of the abdomen is performed through this small opening to identify involved structures and the presence of adhesions, followed by further opening of the abdominal wall in an elliptical fashion using scissors.[254,261] The infected umbilical structures are identified and resected as described later. Following resection of the umbilicus, the body wall is closed in three layers, as described earlier for umbilical hernia repair as well as based on surgeon's preference.

Omphalophlebitis

If the infection of the umbilical vein ends distally to the liver, the vein can be removed en bloc, with ligation prior to transection. If the infection extends to and involves the liver, marsupialization of the umbilical vein is needed. Marsupialization of the umbilical vein has been described in the cranial aspect of the midline surgical incision or using a separate incision lateral or cranial to the midline incision.[254,261] The former technique is associated with an increased risk of herniation of the marsupialization site. In the latter approach, the vein is dissected free from surrounding tissue, covered with a finger-tip of a sterile glove (or sutured closed), and exteriorized through a separate right paramedian, circular incision. The vein is sutured to the rectus sheath using multiple interrupted absorbable sutures, under minimal tension. In a similar fashion, a second layer of sutures between the vein and the skin is performed using either delayed absorbable or nonabsorbable suture.[254] The abdominal incision is then closed as described earlier. The venous stump end is then reopened and allowed to drain. Daily flushing with dilute antiseptic solution can be performed but should be done carefully without back pressure. The animal should be maintained on antibiotics until cessation of drainage, and healing of the venous stump is complete (typically more than 14 days). Rarely, a second operation may be required to resect the marsupialized umbilical vein.[254,261]

Patency or Abscessation of the Urachus

The urachus is identified and traced caudally to the urinary bladder. The body wall incision may need to be extended caudally to allow sufficient visualization and exteriorization of the bladder apex. The urachus and bladder apex are packed off from the abdomen using moist lap sponges or towels. Either stay sutures or use of Doyen forceps can be used to facilitate sharp resection of the urachus and tip of the bladder apex. The bladder is closed in two inverted layers in a continuous pattern (e.g., Cushing, Lembert) using 2-0 absorbable suture material, ensuring that the bladder lumen is not penetrated, and a water-tight seal is achieved.[254,261] The abdominal wall, subcutaneous tissue, and skin are closed as described for umbilical hernia repair.

Omphaloarteritis

En bloc resection is the treatment of choice for infection of the umbilical arteries. Visualization of the arteries can be difficult, and care must be taken not to exert excessive traction during manipulation, as this can lead to tearing of the internal iliac artery. The arteries are ligated with absorbable suture material as deep as safely possible, ideally using a three forceps technique for maximum safety.[254] Marsupialization of the umbilical artery is described, but fortunately is rarely necessary.[264]

Prevention. Prevention of umbilical infections is based on sound husbandry principles. This includes ensuring adequate intake of quality colostrum as well as clean lambing/kidding sheds and yards. Depending on the management scenario, dipping of the naval with noncaustic antiseptics may help reduce the incidence of infection.

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