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CHAPTER 40

Disorders of the Digestive System

Christopher Cebra

Disorders of the digestive system are among the leading causes of morbidity and mortality in New World camelids, accounting for approximately one quarter of both fatal and nonfatal illnesses in these species. In spite of our recognition of the importance of these disorders, the number of reports on the diseases of this system is still relatively small, and some important, common disorders remain sketchily described, if at all, in the primary scientific literature. Veterinarians presented with a sick camelid often are forced to extrapolate from experiences with other species to determine whether the disorders are gastrointestinal (GI) in origin, to establish lists of differential diagnoses, and to formulate diagnostic and treatment plans.

Anatomy and Physiology

All species of camelid have demonstrated the ability to thrive in harsh, dry, nutrient-poor environments where domestic ruminants are unable to survive. Their survival is aided by their ability to extract maximal water and nutrients from feedstuffs, which is a function of the gut. Understanding this ability to tolerate poor conditions helps explain some of the functional and physiologic adaptations that have occurred in the camelid digestive system. It also lays the groundwork for recognizing that the camelid gut is different from that of the ruminant.

Lips, Tongue, and Oral Cavity

Llamas and alpacas selectively graze high-quality plants in environments where overall nutrient availability is low. One anatomic feature that contributes to this ability is the labial cleft, or philtrum, which divides the upper lip into halves. Each half can move independently, allowing camelids to fastidiously investigate potential food sources and then to procure choice foods. With its tactile qualities and mobility, this upper lip plays some of the role of the tongue in traditional ruminants. The lower lip is less mobile and less sensitive than the upper lip.

Camelids have a small oral cavity with an elongated soft palate and narrow oropharynx. The length and narrowness of the oral cavity makes visual examination difficult. The anterior oral mucosa is smooth and often has areas of pigmentation, whereas the buccal mucosa is papillated. The tongue further impedes oral examination. Although it does not extend from the mouth nearly to the degree of the bovine tongue, the tip is reasonably motile, and the main muscular portion of the tongue creates a pronounced caudal dome that fills most of the caudal oropharynx.

Dentition

Infant New World camelids may have up to 22 deciduous teeth, of which 16 generally erupt and become functional.[1,2] The deciduous dental pattern per side is as follows:

	INCISORS	CANINES	PREMOLARS
Upper	1 to 2	1	2 to 3
Lower	3	1	1 to 2

Some deciduous teeth, particularly the upper incisors, rarely erupt but may be seen on radiographic examination of the mouth. Deciduous canines also rarely erupt; they are seen in about 5% of males and even fewer females. All deciduous teeth are usually visible at birth in the term neonate, although the incisors may not have completely erupted through the oral mucosa.

Most adult New World camelids have 30 or 32 permanent teeth. Their dental pattern per side is as follows:

	INCISORS	CANINES	PREMOLARS
Upper	1	1	1 to 2
Lower	3	1	1 to 2

	MOLARS
Upper	3
Lower	3

The single upper incisor is caudal to the last lower incisor. It is crescent shaped and pointed, similar to the canines. Adult canines and upper incisors together make up the fighting teeth (Figure 40-1). They are larger in males than in females and also poorly developed or do not erupt in camelids castrated before sexual maturity. Development of the fighting teeth is the only gender-based differences in dental pattern in camelids.

Eruption times for the lower incisors, from front to back, are 2 to 2.5 years, 3 to 3.25 years, and 3 to 6 years.[1] Canines usually erupt at between 2 and 4 years of age but may come in years later and grow more slowly in females or geldings. Premolars usually erupt between 3.5 and 5 years of age, and the molars, from back to front, erupt at 6 to 9 months, 1.5 to 2 years, and 2.75 to 3.75 years of age. The significance of this is that younger adults will frequently have their permanent cheek teeth but still have one or more deciduous incisors (Figure 40-2).

Llama and guanaco incisors are covered in enamel and have a broad, flat cutting surface that works against the upper dental pad.[3] Alpaca and vicuña incisors have a narrower cutting surface with enamel only on the labial surface. The incisors of llamas and guanacos are tapered toward the root, whereas the incisors of vicuñas and alpacas are rectangular,

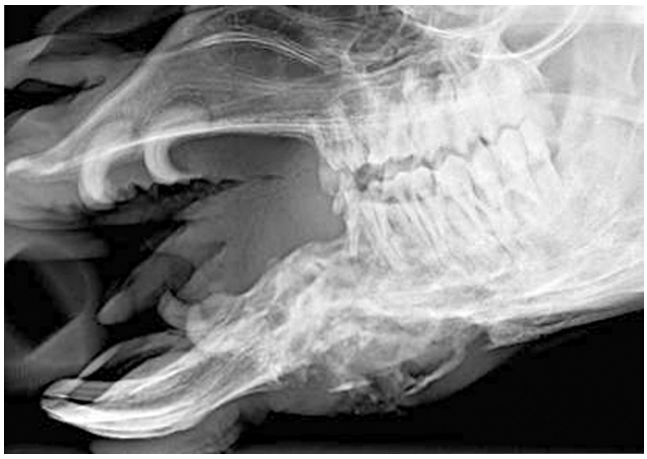

Figure 40-1 Radiographic appearance of the adult camelid mouth, including fighting teeth.

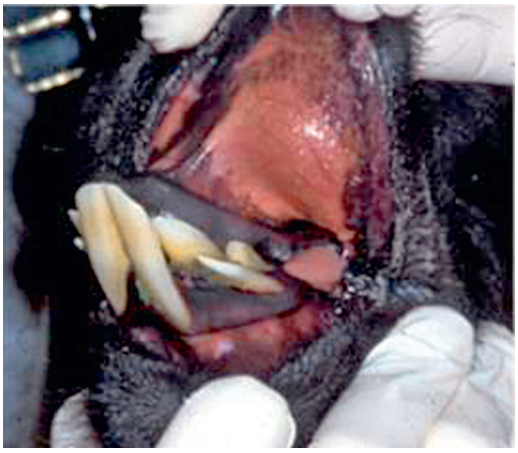

Figure 40-2 Retained deciduous incisors in a camelid. The middle incisors depict malocclusion as well.

have an open root, and erupt continuously. Hybridization has confused this distinction somewhat.

The maxillary premolars have three roots (medial, craniolateral, and caudolateral), whereas the molars have four roots. All the lower cheek teeth have two roots (cranial and caudal), with the caudal root of the last molar consisting of two fused roots. Of the premolars, only the second mandibular one has an infundibulum. All molars have two infundibula. The frequency of patent infundibula appears to be low, but this condition is seen sporadically and may contribute to periapical infections. The cheek teeth have closed roots and do not continue to grow after maturity.

Adult premolars are less than one quarter the size of molars and occasionally are absent or do not erupt. Mandibular cheek teeth are narrower compared with maxillary teeth, and mandibular arcades are closer together. Although camelids chew with a rotary motion, they tend to have very uneven wear on the occlusal surface of their cheek teeth (Figure 40-3). Points or ridges may develop on the lateral maxillary arcades and the medial mandibular arcade and may require filing. In spite of the unevenness, evidence that these points cause trauma or chewing disorders is rare.

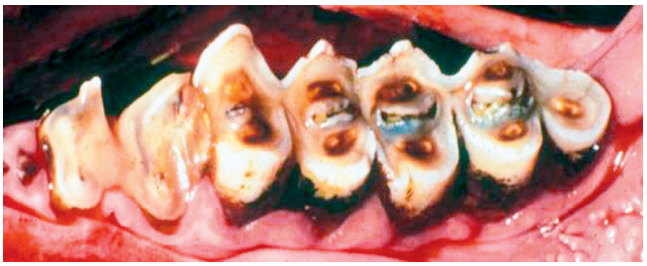

Figure 40-3 An example of sharp molar points, a fairly normal finding in older camelids, albeit sometimes associated with dropping feed and weight loss.

Salivary Glands and Saliva

Two large salivary glands are located along the caudal border of the vertical ramus of each hemimandible: (1) the parotid glands are the largest and most superficial and have a serous secretion, and (2) the ventromedially located mandibular glands have a mixed mucous–serous secretion.[4,5] Numerous mucus-secreting glands are located throughout the oral cavity, including the buccal, palatine, sublingual, and labial regions. Most of these are diffuse aggregates of secretory cells, not distinct tissue masses. Some of these glands, namely, the sublingual glands, also secrete small amounts of serous fluid, but camelids appear to lack a large serous salivary gland in the mouth comparable with the inferior molar gland of sheep.

The parotid duct courses craniad along the lateral aspect of the masseter muscle, roughly following the orientation of the muscle fibers, and dorsal to the facial artery and vein. This differs from ruminants, in which the duct courses with the facial vessels along the ventral aspect of the masseter muscle. The duct moves medial to the buccinator muscle and empties into the mouth through a prominent papilla on the inside of the cheek. In adult llamas, the papilla is located across from a point 1 centimeter (cm) dorsal to the gingival margin between the last upper premolar and the first molar. The mandibular ducts course under the tongue to the sublingual papillae, which are poorly recognizable.

The anatomy and physiology of the salivary glands allow efficient use of saliva. The multitude of mucus-secreting glands lubricate the entire oral cavity against the abrasive action of harsh feedstuffs. In contrast, the alkaline parotid secretion is secreted exclusively into the molar region, where feed and cuds are chewed and stored. This allows selective infusion of the food bolus with large amounts of bicarbonate-rich fluid, which helps buffer gastric fermentation when swallowed.

Several investigations into the content of both parotid and mixed salivary secretions of camelids have been conducted.[5–8] Normal camelid saliva is alkaline (pH = 8.0 to 8.6) with high bicarbonate (100 to 150 milliequivalents per liter [mEq/L]), phosphorus (18 to 34 mEq/L), sodium (148 to 175 mEq/L), and potassium (6.5 to 14.0 mEq/L) concentrations. Salivary chloride concentration is very low (20 to 30 mEq/L). A slight increase in bicarbonate concentration occurs during feeding. Total fasting salivary output is approximately 6 milliliters per kilogram per hour [mL/kg/hr], with each parotid salivary gland secreting about 2 mL/kg/hr.[6,7] Both parotid and total salivary outputs increase by over 50% during eating and cud chewing. On the basis of this information, camelid saliva

appears to have similar biochemical features to ruminant saliva, and probably plays an important role in buffering gastric acid. The rate of saliva production in camelids is also similar to that in roughage-fed ruminants. However, the parotid glands of camelids are proportionally similar in mass to those of ruminants adapted to poorly fermentable diets and approximately one third the size of the glands of ruminants adapted to rich diets.[9] This suggests that camelids have limited ability to increase salivary buffering of fermentation in response to higher rates of forestomach acid production.

Cud-Chewing Behavior

Similar to true ruminants, camelids use cud chewing to break down large feed particles. Initial mastication by camelids is cursory and consists of only enough chewing to mix the food with saliva to form a bolus for swallowing. To further decrease particle size and increase surface area for microbial digestion, cud is propelled from the first gastric compartment back up the esophagus into the mouth, where it is rechewed and mixed with saliva. This process has been studied extensively in the vicuña and probably is similar in other camelids.[10] After regurgitation of a cud, the mandible makes a "figure-of-8" arc during chewing. In approximately 15 seconds, 25 to 30 chews occur, after which the cud is swallowed. The cycle may repeat itself many times. In contrast to ruminants, which tend to retain a cud on one side for several chews, camelids appear to move the cud from side-to-side with each chew.[9] Saliva is expelled primarily from the ipsilateral parotid duct each time, potentially minimizing salivary loss and maximizing the contribution of both glands to the digestive process.

The actual grinding is a concerted lateral-to-medial motion of the working side of the mandible.[11] Adults with full molar occlusion have longer chewing cycles compared with juveniles both before and after the first molar comes into wear.[12]

Esophagus

The esophagus lies deeper in the camelid neck than in other species and is somewhat obscured by thick skin between the trachea and the left transverse processes of the cervical vertebrae. The mucosa and submucosa are similar to those of ruminants, with an abundance of submucosal mucus glands underlying the stratified squamous mucosa. The tunica muscularis consists predominantly of skeletal muscle distributed in an inner longitudinal and an outer circular muscle layer (Figure 40-4). This is opposite to the pattern of fiber orientation in ruminants. The well-developed skeletal musculature of the camelid esophagus is necessary to move a cud up the long, vertical neck. The esophagus receives its sensory and motor innervation from the vagus nerve and its branches.

Gastric Anatomy and Physiology

Adult camelids, similar to adult cattle and other ruminants, rely on bacterial fermentative digestion in the forestomach to extract nutrients from plant material. This requires a warm, moist anaerobic environment with a large capacity and long fiber retention times and the ability to mechanically and enzymatically break down large feed particles. Mechanical breakdown is achieved through cud chewing and gastric contractions.

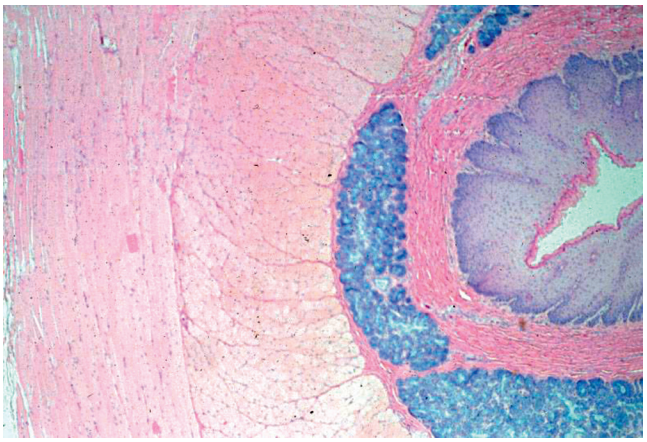

Figure 40-4 Esophageal anatomy of camelids. Note the outer circular and inner longitudinal layers of muscle.

Forestomach enzymes are primarily microbial in origin. Bacterial fermentation of plant carbohydrate results in the production of organic volatile fatty acids (VFAs). Under normal fermentative conditions, acetate, propionate, and butyrate are the major VFAs produced, in a ratio of approximately 4 : 1 : 1 before feeding and approximately 7 : 2 : 1 a few hours after feeding.[13] Absolute VFA concentration after feeding in both New World camelids and Old World camelids is approximately 140 millimoles per liter (mmol/L), which is 30% to 40% higher than in ruminants under similar conditions.[13,14] The production and presence of these VFAs and other organic acids acidify the fluid within the forestomach compartments. Although gastric (forestomach) fluid pH varies with diet and fermentative activity, camelids on a diet consisting predominantly of forage should have a forestomach fluid pH between 6.4 and 6.8; that is, the VFAs exist predominantly in their ionic forms. Carbon dioxide and methane are additional products of fermentation. Further discussion of fermentative function may be found in Chapter 8.

In camelids, fermentative digestion occurs in the first two gastric compartments (C1 and C2) and the cranial four fifths of the third gastric compartment (C3). The first gastric compartment occupies most of the left side of the abdomen. It consists of large cranial and caudal sacs, which are divided ventrally by a prominent transverse pillar and together contain about 83% of gastric volume and about 50% of abdominal volume (Figure 40-5).[15] The delineation between the sacs is easily visible or palpable from either outside of or within the compartment. The transverse pillar forks medially into cranial and caudal portions near the cardia. The small, reniform second compartment (6% of gastric volume) rests atop C3 medial to C1, with its lesser curvature oriented toward C1 and its greater curvature protruding toward the right dorsolateral body wall. The first two compartments and their contents account for approximately 10% of the body weight of an adult alpaca and 15% of an adult llama.[7,16] The canal between C2 and C3 is very narrow and probably represents an important size regulator for particle transport to the lower GI tract. The third compartment (11% of gastric volume and 1.5% to 2.5% of adult body weight) is long and tubiform, with both ends upturned.[7,16] It extends cranioventrally and then caudally from its attachment to C2, occupying the right cranioventral

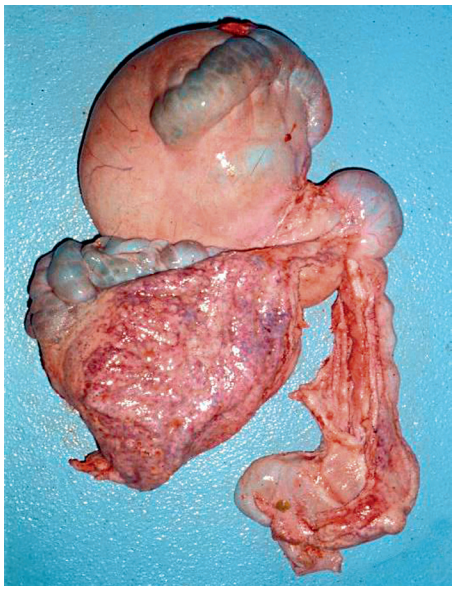

Figure 40-5 Comparison of the sizes of the large cranial (*top left*) and caudal (*bottom left*) sacs of the first gastric compartment, and the smaller, narrower third compartment (*bottom right*). The small second compartment makes up the rounded canal toward the top right. The two large, dark areas on the surface of the first compartment are the saccular regions.

abdomen. Near the umbilicus, its terminal portion curves craniodorsad and may extend 10 to 30 cm forward atop the tubiform portion.

Arterial blood is supplied to the gastric compartments via the celiac artery. Several branches traverse C1, including a large branch which courses down the medial horizontal pillar to supply the caudal saccular region. A single branch supplies C2 and C3, coursing ventrad along the lesser curvature of C2, branching into four branches: one supplies C2, a second provides collateral circulation to the caudal saccular region of C1, and the other two course caudad along the greater and lesser curvatures of C3, respectively.

First and Second Compartments

Stratified squamous epithelium lines most of the superficial mucosal surface of C1 and the lesser curvature and primary septa of C2.[15] The squamous region of C2 may contract together with a muscular band located near the cardia in C1 to form the esophageal groove for transport of milk directly into C3. The squamous epithelium of the forestomach is keratinized in camels, but not in New World camelids.[17,18] No structures are analogous to the frondular papillae that ruminants have extending into the forestomach lumen (Figure 40-6). Instead, camelids have two types of specialized structures that evaginate from the forestomach lumen: (1) the glandular saccules of C1 and (2) the glandular cells of C2. The saccules of C1 are found in two groups, one on each side of the transverse pillar. Each group is arranged in rows. The smaller group is found at the cranioventral aspect of the cranial sac, and the larger group is found immediately caudal to the transverse pillar on the ventral surface of the caudal sac. The rows are divided by muscular primary septa, which are

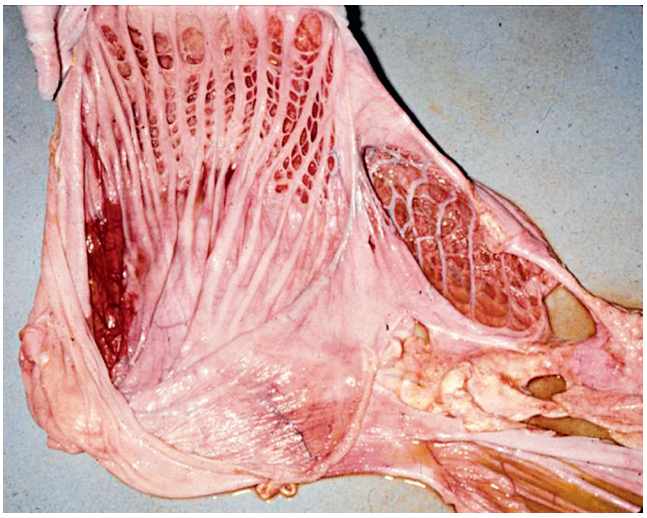

Figure 40-6 The internal surfaces of the first (*left*) and second (*right*) compartments. Note the lack of papillae and the presence of muscular septae dividing the glandular saccules or cells, and the wider opening to the second compartment cells.

much more prominent in the caudal sac, where they are contiguous with and perpendicular to the transverse pillar. The saccules in each row are separated by smaller secondary septa. In the middle rows of each group, two saccules usually are present side-by-side between the primary septa, divided by a small tertiary septum and occasionally a quarternary septum. Each saccule has a very small orifice (about 5 millimeters [mm] in an adult llama) controlled by a muscular diaphragm protecting a wider saccular lumen (about 10 mm). The saccules of the caudal sac alone account for half the surface area of the first compartment.[7] The saccules are visible as bumps along the serosal surfaces of the gastric compartments or as evaginations when viewed from the mucosal side.

The glandular saccules of C1 are located on its ventral aspects. During gastric contractions, fluid is expelled from the fiber mass and passes through the muscular septa and into the saccular lumina. The smooth muscle of the caudal sacs is organized so that the entire saccule can be everted during contraction cycles, expelling its contents into the compartment lumen. This allows constant renewal of the saccular contents. The muscular structure of the cranial saccules and the occurrence of eversion have not been reported.

The glandular cells of C2 are larger with wider openings (see Figure 40-6).[15] Primary septa originate along the squamous region and extend to the greater curvature. Secondary and tertiary septa branch off to subdivide the region into cells but do not form muscular sphincters. The mucosa within these cells can have small papillae, but these are not always present. The serosal surface of C2 is uniform, and the cells are not apparent as individual structures. The C2 cells are weakly muscular and do not evert.

The nonsaccular mucosa and septa are covered with stratified squamous epithelium, and the mucosa within the C1 saccules and C2 cells consists of folds of simple columnar epithelial cells.[18] These cells have microvilli along their luminal membrane and contain numerous mitochondria. They also contain both mucoid and nonmucoid secretory granules. Cellular morphology suggests both absorptive and secretory

functions. Saccular cells may be subdivided into two populations: (1) simple columnar epithelial cells, which resemble those of the small intestinal mucosa and have primarily absorptive function, and (2) a second group that resemble tubular gland cells.[7] It is postulated that this second population of cells, especially those nearest to the compartment lumen, secrete a mucus coat that protects the saccular membrane against mechanical trauma, acid injury, and microbial invasion. Evidence of this mucus coat may be found in live camelids.[17]

In spite of their designation as "glandular" saccules, little evidence exists for substantive secretion beyond this thin mucus coat. Water, electrolytes, and VFAs are absorbed very quickly across the saccular membrane.[7,19] Absorption of these molecules across this membrane is two to three times faster than across the rumen membrane in sheep and goats and results in a generally drier gastric contents than is commonly found in cattle or sheep. Unlike ruminants, camelids appear to absorb water across the forestomach membrane in direct relation to the amounts of sodium and VFAs absorbed. In contrast to sodium and water, camelids appear to absorb less chloride. The submucosa of each saccule is filled with a complex network of capillaries, which supports the importance of the saccules in nutrient absorption.

Both camelid saccules and ruminant papillae increase the surface area for the absorption of nutrients but function very differently. The camelid saccules contain small quantities of gastric fluid in small pockets surrounded by highly absorptive epithelium and constantly renew this fluid through saccular eversion and refilling. The high rate of contractions, the high surface area-to-volume ratio, and long particle retention times of the camelid forestomach may be responsible for the digestive advantage that camelids have over ruminants with regard to poor-quality forage.[14,20,21] The papillae of ruminants also help preserve the ruminal environment through absorption and removal of organic acids from the lumen. This is especially important when VFAs are produced in large quantities, as occurs when a highly fermentable feed is provided. The size of the papillae and surface area increase, over time and with limitations, when VFA production is high, allowing faster removal of VFAs. Thus, the ruminant can adapt itself to higher energy feeds by increasing the rate of acid removal. We do not know if the saccules of camelids are capable of similar adaptation, and our belief is that they are not. Thus, camelids may be less adapted to handle high energy feeds compared with their ruminant counterparts. This is contrary to the popular belief that camelids are resistant to this type of feed-related disease.

How the saccular epithelium facilitates absorption is unknown. Saccular fluid has a higher bicarbonate concentration than forestomach fluid, and this bicarbonate concentration increases with time if the saccules are exposed to chloride-rich or VFA-rich solutions.[7,8] This has led to one theory that bicarbonate is secreted across the saccular membrane into the forestomach in exchange for chloride to buffer the acids produced by fermentation. A similar function has been described for the cells of the gastric cardia of monogastrics, which secrete bicarbonate in exchange for chloride from hydrochloric acid to protect that region from acid damage. The problems with extrapolating this function to camelids are twofold: (1) The gastric saccules are rarely exposed to high chloride concentrations because the chloride concentration in the forestomach fluid of camelids is normally very low (less than 20 mEq/L); and (2) it would be unusual to assume that an animal adapted to a low nutrient environment would have developed a mechanism to protect itself against overingestion of rapidly fermentable nutrients.

According to another theory, the high bicarbonate concentration in the saccule could be an example of ion trapping, rather than active secretion by the columnar epithelium of the saccules.[7,22] By this, it is understood that some of the carbon dioxide and water, both of which are found in abundance within the forestomach fluid, combine to form carbonic acid, which also may exist as separate bicarbonate and hydrogen ions. VFA, although a weak acid, usually exists in its ionized form at typical C1 pH. The ionized form crosses the lipid cellular membrane with difficulty, but if it momentarily captures the hydrogen molecule from carbonic acid, it is removed from the system by absorption across the saccular membrane. This leaves the bicarbonate molecule within the saccular fluid, which explains the high bicarbonate concentration at the site of VFA absorption and perceived secretion into the saccular fluid and would be compatible with our understanding of normal conditions in the camelid stomach.

More recent work suggests a third theory. Some evidence suggests that VFAs are transported across the camelid forestomach wall primarily in their ionized form.[19] This may occur through VFA–bicarbonate ion exchange. Although this could potentially promote gastric buffering during times of high VFA production, it would not suitably buffer lactate, half of which is poorly absorbed.

Neonatal camelids start with a nonfermentative stomach. In spite of that, their fermentative stomach compartments are relatively large compared to their analogs in neonatal ruminants. The full first compartment is about 45% of total gastric weight at birth, and reaches 60% by about 6 weeks of age.[23] The second compartment starts at around 10% of gastric weight and declines slightly into adulthood. A functional microbial population becomes established between the first and second weeks of life, and by 12 weeks, fermentative function is at adult levels.

Forestomach Motility

Two described contraction cycles occur in the forestomach of camelids. The *A-cycle* begins with a strong contraction of the C2-C3 canal, followed by a rapid contraction of C2 and relaxation of the canal.[24–26] Following this, both caudal C1 and the canal contract up to eight times. In guanacos, the C1 contractions move from caudoventral to caudodorsal, followed by a cranial sac contraction.[27] In alpacas, the whole caudal sac appears to contract together.[26] A final weak contraction of the canal ends the cycle. The entire sequence takes about 1 to 2 minutes. Clinical interpretation of multiple contraction cycles during that time results from interpretation of the multiple C1 contractions of a single A-cycle as multiple cycles. The A-cycle is responsible for fluid passage into C3 (during the first C2 contraction only), expressing fluid from the fiber mass, and mixing of the contents of C1. Experimental data from one llama revealed that fluid passage into C3 occurs at a rate of approximately 7 mL/kg/hr, with about 0.14 mL/kg passing through the canal with each A-cycle.[28] The order of contraction

of the various parts of caudal C1 is still being debated, but the overall effect is slow counter-clockwise rotation of the ingesta, when viewed from the left.

One or more B-cycles follow each A-cycle.[25] The *B-cycle* starts with contraction of the cranial compartment of C1 during the last previous canal contraction, followed by a weak contraction of C2, and finally by contraction of the caudal compartment of C1. The B-cycle lasts about 9 seconds and may be repeated several times before the next A-cycle. The B-cycle primarily aids in mixing of digesta, expressing fluid from the fiber mass, and renewing the saccular contents but generally does not stimulate passage of fluid into C3. Rumination and eructation of gas each occur during some B-cycles. If the camelid inspires with a closed glottic prior to the contraction of cranial C1, ingesta is drawn into the esophagus by negative intrathoracic pressure, and propelled to the mouth by an antiperistaltic wave of contraction of the esophageal musculature.[29] Eructation is accomplished by an additional contracture of caudodorsal C1 at the end of the B-cycle. Up to approximately 100 A- and B-cycles may occur in an hour during eating and rumination, but both cycles may be absent for 10 to 20 minutes at a time when the camelid is not ruminating.

Both A- and B-cycles depend on vagal stimulation, and are blocked by atropine, acetylcholine, or epinephrine.[25] The frequency of A-cycles is decreased and B-cycles increased by mechanical distension of cranial C1 (A-cycles only), the C2-C3 canal, and cranial portions of C3. Mechanical distension of distal C3 appears to inhibit A-cycles and stimulate B-cycles, although its effects on A-cycles are inconsistent. The clinical relevance of these effects is that forestomach distension stimulates gastric mixing and rumination but not necessarily gastric outflow, whereas distension of the terminal fermentative stomach (distal C3) alone promotes aborad flow of ingesta. Clinical experiences support that pathologic distension of the gastric compartments inhibits motility as in ruminants, although this effect has not been verified experimentally.

The Third Compartment

The cranial portion of C3 is covered with glandular epithelium similar to that which lines the forestomach saccules.[30] Most of this is organized in longitudinal folds, although a small wedge of reticular-type folds is present near the junction with C2. The caudal 20% of the compartment, as measured along the greater curvature, is lined with gastric glands, which secrete hydrochloric acid and acid proteases. The mucosa in this region is much thicker than the mucinous glandular region, 7 to 10 mm compared with the 3 to 4 mm of the fermentative region.[31] Because the diameter of the lumen decreases caudally and the lesser curvature bends almost 180 degrees on itself toward the fundus, the gastric glandular portion represents only a small percentage (about 20%) of the surface area of C3. Luminal pH drops abruptly from forestomach levels in the fermentative portion to four (or less) in the gastric portion.[9,32] Some researchers consider this area the "true stomach," a separate entity from C3, although most consider that C3 has both fermentative and acid-secreting regions. The rugal folds become much deeper near the pylorus, which is a fibrous band, and the epithelium in this region is dominated by mucus and bicarbonate-secreting pyloric glands.

Water, sodium, and VFAs are absorbed rapidly across the C3 membrane.[28] Approximately 70% of the VFAs, 60% of the sodium, and 30% of the water that enter from C2 are absorbed in C3. Extensive water and chloride are secreted into the terminal gastric portion, but this has not been quantified.

Third Compartment Motility

Circumferential contractions occur along the length of C3. These occur simultaneously and are weak and common (10 per minute) in the cranial portion but have peristaltic organization and are stronger and slower (5 per minute) in the caudal regions.[27] Distension stimulates local progressive motility while inhibiting activity both cranially and caudally.[25] Distension of the cranial region inhibits the passage of fluid from the forestomach into C3, whereas caudal C3 distension promotes it. Motility of the fermentative region is unaffected by C1 and C2 distension but is inhibited by canal distension and vagolytic drugs. Motility of the fundic region of C3 is affected only by local stimuli, not vagal input. Nitric acid appears to play an inhibitory role.[32] Under normal conditions, once fluid and ingesta have reached the fundic portion of C3, they should not reflux into the fermentative portion.[27]

Omentum

The greater omentum attaches diagonally across the caudal saccular region of C1, along the greater curvatures of C2 and C3, and the caudoventral surface of the cranial duodenum. The greater omentum covers the caudomedial aspects of C1 and C2 as it courses dorsad. It then courses abruptly craniad with the caudal flexure of C3. Although a potential space exists within the omental sling, this is occupied by the gastric compartments; the jejunum rests caudal and dorsal to C3. Fat deposits are usually small, and the omentum is often translucent.

The lesser omentum fills the area between the lesser curvature of C3, the duodenal ampulla, the liver, and the porta hepatica, where the hepatic artery and portal vein enter the liver and the pancreatic and bile ducts enter the small intestine. The mesentery begins a short distance caudal to the porta hepatica and attaches the small intestine to the dorsal body wall. The small gap between the dorsal duodenum, the cranial extent of the mesentery, and porta hepatica form the epiploic foramen, which is in contact with the jejunum lying atop the greater omentum by the pylorus.

Small Intestine

The first 10 cm of the duodenum in adult llamas are similar in diameter to terminal C3 (Figure 40-7). This ampulla wraps around the pylorus from the lesser curvature to the greater curvature, turning caudad at the ventral border of the liver. After this, the duodenal diameter decreases abruptly. The most prominent feature of the subsequent duodenum is the M-shaped flexure of its cranial loop, which is intimately attached to the greater curvature of C3 by the greater omentum. This tortuous passage and the approximately 10-fold reduction in diameter from C3 to the small intestine make this a common site for feed or foreign body impaction.

After coursing craniad to receive the hepatic ducts, the duodenum courses caudad and wraps around the root of the

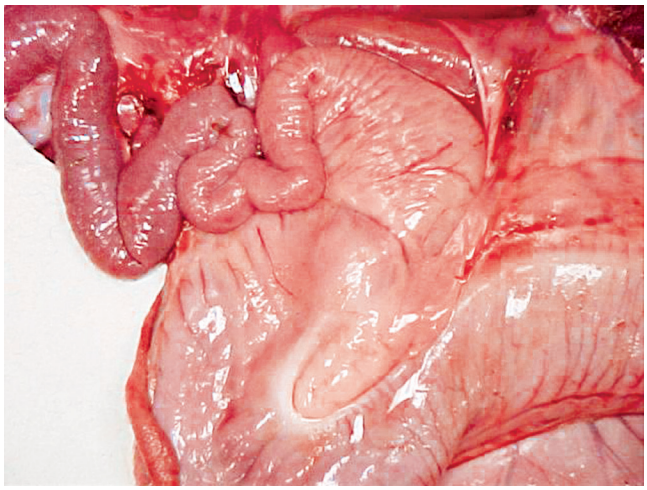

Figure 40-7 The pyloric region of the third compartment and the duodenal ampulla, leading into the narrow cranial duodenum. This area is a common site for bezoar obstruction, especially in alpacas.

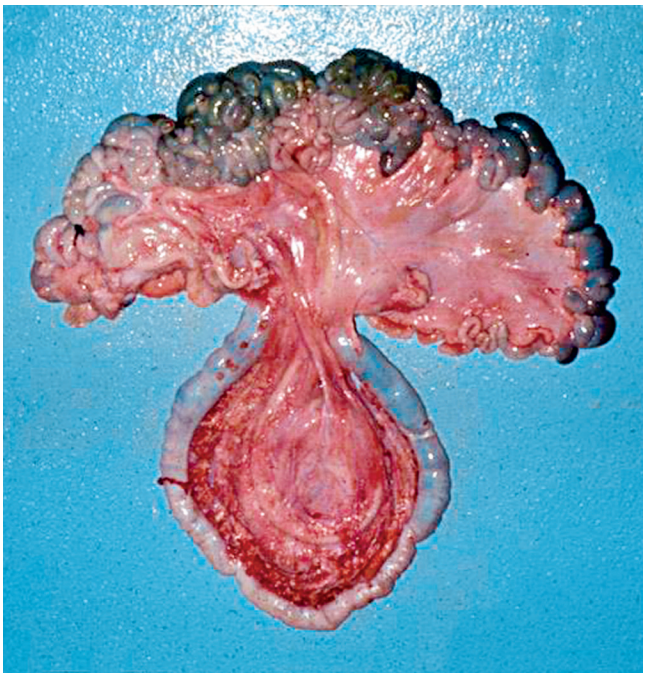

Figure 40-8 The pendulous nature of the long, narrow mesenteric attachment to the ascending colon. This section of bowel is most prone to become entrapped or rotate on its root.

mesentery (right to left around the back), becoming intertwined with the transverse colon at that point. The intertwining of these two structures suggests that camelids undergo the same clockwise rotation of intestinal structures around the mesenteric root during development as has been described in other mammals. The jejunum begins a short distance cranial to the root of the mesentery, and its abundant loops occupy most of the right dorsal abdomen. The proximal two thirds of the jejunum consist of tight coils on a short mesenteric attachment (5–7 cm). These loops are difficult to exteriorize through an incision. The distal one third consists of a longer (30–35 cm) mesenteric flange, which imparts more mobility. Small rents are occasionally found in this mesenteric flange. The ileum is short and enters the cecum near the midpoint of the abdomen. The small intestine usually is flaccid and contains only a small amount of fluid and semisolid feed boluses. Very little is known about the physiology of the camelid small intestine. It is presumed that fluid and electrolyte cycling, digestion, and nutrient absorption occur much as in other mammals. Sugar digestion has been studied in dromedaries, and it has been found that glucose, galactose, and lactose are readily absorbed, whereas maltose and sucrose are not.[33]

Except for the duodenal ampulla, the maximal diameter of the small intestine is around 2 cm in an adult llama and 1 cm in an adult alpaca or juvenile llama.[31] However, distension to this degree should occur only with the intermittent passage of a food bolus. The cranial mesenteric artery courses through the mesentery, sending short, arcuate branches to the jejunum. These branches become progressively longer into the flange until a single long branch courses along the mesenteric border of the ileum.

Cecum, Colon, and Rectum

The camelid cecum is fairly small (about 7 to 10 cm long in adult alpacas and 15 to 20 cm in adult llamas), and usually contains a few handfuls of doughy material. The ileocecal valve is small and tight. The base is usually centrally located in the abdomen, where it is held in place by ligamentous attachments to both the ileum and the colon and a short mesenteric root. Almost the entire length of the cecum is attached by a ligamentous band to the antimesenteric border of the ileum. The beginning of the ascending colon is usually as large as the cecum, and often more prominent. The ascending colon may be followed into its spiral, which usually centers over the ventral abdomen caudal to the gastric compartments, or extends up to the right lateral abdomen. The mesentery of the cecum and ascending colon is an extension of the distal mesenteric flange (Figure 40-8). Hence, it is long and relatively narrow and contains the arterial blood supply to the region.

The large proximal loop of the ascending colon forms a large circle which then enters the first loop of the spiral colon. The spiral colon has five inward-progressing (centripetal) loops and then folds back on itself at the central flexure. The beginning of the five outward-progressing (centrifugal) loops tends to dive into the fat and connective tissue deep to the inward loops such that the centrifugal loops are often difficult to view. The spiral colon is attached loosely to its vascular supply so that the entire structure can easily be exteriorized through a ventral midline or right paralumbar incision. Although the spiral colon as a whole is very movable, the mesentery between individual loops is only 0.5 to 1 cm long. Over the course of the ascending colon, the luminal diameter decreases to approximately 20% of its original width (from 5 cm at the cecocolic junction to 1 cm at the end of the spiral in an adult llama). Water resorption in the spiral colon leads to the production of formed fecal pellets, which can be palpated in the distal centrifugal loops.

The colon aborad to the spiral loop passes very closely to the ileocecocolic junction, to which it is attached by the cecocolic ligament. The transverse colon then reverses direction 4

more times as it courses dorsad, forming another small spiral, and wraps around the mesenteric root (left to right around the front) to intertwine with the duodenum. The descending colon courses caudad into the rectum. These sections of the colon and rectum are held on a very short mesenteric attachment. The cranial portion of the colon is usually filled with doughy feed material which is gradually dehydrated to form fecal pellets. Fecal pellets may be found as far forward as the centrifugal loops of the spiral colon but are more normally found only in the transverse and descending colon and rectum.

Mesentery, Blood Supply, and Innervation

Branches of the cranial mesenteric artery supply arterial blood to the cecum and ascending colon. The cecal artery courses between the cecum and ileum. Two large branches course in either direction along the mesenteric border of the first large loop of the ascending colon, and the main branch arborizes on the mesenteric side into several small branches to supply different sections of the spiral loops. These branches are close enough to provide collateral arterial blood supply to most sections of the spiral. Arterial blood is supplied to the descending colon and rectum through the caudal mesenteric root.

Diagnostic Approaches

Behavior

A normal camelid should appear bright and curious, with ears erect. When in a strange environment, camelids often stand with their heads stuck over a fence or through a gate to survey nearby activity. At rest, they sit in the cushing position, sternal with all four legs tucked under the body, and the head and neck either held erect or stretched out to the front. Whether standing or lying down, they should make some effort to move away from approaching strangers, possibly pinning their ears back or raising their head to sniff or spit.

The normal camelid usually eats or grazes throughout the day and visits the communal dung pile two to four times daily to defecate and urinate. After inspection of the pile, defecation and urination occur quickly and simultaneously. Frequent or prolonged visits, a straining posture, and frequent straining or attempts to pass urine or feces away from the pile are abnormal. Observations of eating, drinking, and voiding behavior frequently must be done from a distance, as camelids often stop these activities if the observer is close. Periodic examination of the dung pile may also be helpful, as fresh feces and urine may be seen, and the occurrence of diarrhea in any camelids in the herd also may be determined. Stance and posture, amount of time spent in elimination behavior, and presence of vomiting or regurgitation should be noted.

Physical Examination and Diagnostic Tests

Thorough physical examination is helpful in determining that a disorder is digestive in nature and in assessing the severity of the disease. The sclera and nonpigmented oral mucosa should be examined for discoloration, moistness, petechiae, and signs of vasomotor dysfunction. The narrow oral aperture, immobile, domed tongue, and large distance between the commissure of the lips and posterior molars make examination of the oral cavity difficult, but persistence and use of a flashlight should allow for inspection of the mucosal papillae, the tongue, and the occlusal surfaces of teeth. Extreme care must be taken to avoid personal injury when examining camelids with erupted fighting teeth or particularly sharp molar ridges. The maxillae and the horizontal rami of the mandible should be palpated for bony masses, and the intermandibular space, retropharyngeal space, and skin over the jaws should be palpated for enlarged salivary glands, lymph nodes, soft tissue tumors, edema, or cellulitis. The oral cavity should be examined carefully with any external mass for displacement of teeth, oral extensions of a mass, or drainage and to make sure that the supposed mass is not a retained cud in the cheek. The neck should be palpated carefully, especially in reference to fluctuance in the esophagus.

General attitude should be assessed for depression, excitability, or weakness. Heart rate, respiratory rate, pulse strength, body condition, and rectal temperature should be determined to evaluate general body condition, with the considerations that heart rates greater than 72 beats per minute (beats/min) in adults should be considered abnormal and that GI disorders more frequently result in a decrease in rectal temperature rather than an increase.

Abdominal organs may be assessed through palpation and auscultation. Lifting the fleece and listening through the inguinal window offers the best auscultation. Forestomach contractions can be heard ventrally on the left and usually occur somewhat more commonly than in cattle. Assessment of abdominal contour offers insights mainly into fluid or gas accumulation. Fluid (ventral) accumulation is far more common in camelids than in ruminants and is commonly missed on cursory physical examination. Organomegaly, usually of the liver or uterus, may mimic the ventral distension seen with ascites or gastric or intestinal distension. Simultaneous auscultation and succussion may help differentiate the possibilities. Seeking for "pings" is rarely fruitful.

The abdomen is also a good place to assess ventral edema and body condition score. Pain assessments are more problematic. The inguinal window is one of the places camelids attack each other, and thus camelids are understandably sensitive to palpation in that area. Their struggling attempts are often overinterpreted as a pain response.

Rectal Examination

Though juvenile llamas and most alpacas may be too small, rectal examination of most adult female llamas is possible with good restraint (preferably in a camelid chute), adequate lubrication and, if necessary, chemical sedation. Adding 20 mL of a local anesthetic agent (2% lidocaine hydrochloride [HCl]) to 100 to 120 mL of lubricant and injecting the mixture into the rectum by plastic dose syringe or insemination pipette may relax the anal sphincters and caudal rectum sufficiently to allow a good rectal examination. However, clinicians with large hands may choose not to perform the examination to avoid the risk of causing injury to the patient.[34]

Rectal examination of the llama is similar to that of the cow; the bladder, reproductive tract, left kidney, and caudal first gastric compartment can usually be palpated. The right abdomen usually has no palpable viscera. The bladder may be distended with urinary tract obstruction or simply because of lack of urination in the stressed camelid, and portions of the

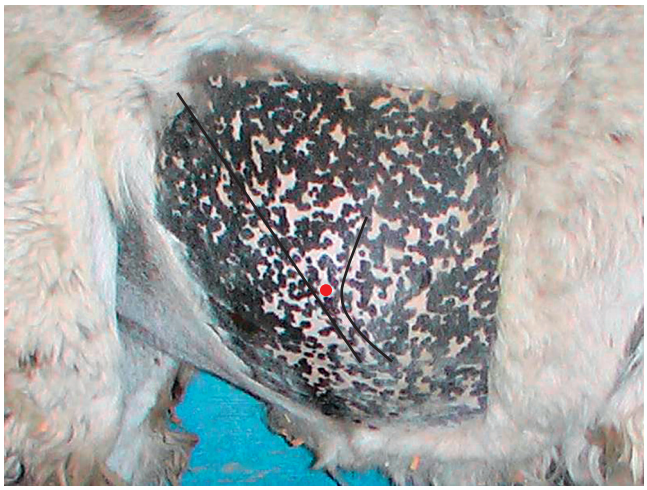

Figure 40-9 The landmarks for right paralumbar abdominocentesis. The black lines represent the caudal margin of the last rib (*curve*) and the linea semilunaris or aponeurosis of the external abdominal oblique muscle (*straight*). The red dot represents the approximate site for centesis.

Figure 40-10 Obtaining peritoneal fluid from the right lateral site in an alpaca.

small and large intestine may be distended with intestinal obstruction. Care must be taken, as rectal injuries and bladder rupture have been reported.[34] For the most part, abdominal ultrasonography has supplanted rectal palpation.

Abdominal Paracentesis

Abdominal fluid is best obtained from a paracostal site on the right flank (Figure 40-9).[35] This site is caudal to the last rib and approximately one half the distance from the ventral to dorsal midline. Alternatively, the operator may choose a site two (alpacas and crias) or three (adult llamas) finger-widths caudal to the caudal-most aspect of the ribs. The patient should be restrained and possibly sedated to restrict movement. The fleece should be clipped and the site aseptically prepared. Two milliliters of lidocaine HCl are infused subcutaneously and intramuscularly, and a stab incision made into the muscle with a #15 scalpel blade. A sterile teat cannula is introduced perpendicular to the skin through the incision into the abdomen, and fluid s collected by gravity flow into a Na-EDTA (sodium–ethylenediaminetetraacetic acid) tube for cytologic analysis and a clot tube for bacteriologic culture (Figure 40-10). Heparinized fluid may be collected for immediate biochemical analysis. This technique is successful in about 95% of camelids, with unintentional gastrocentesis rarely occurring because of the sideways orientation of the cannula. The procedure may be done with the patient standing in the kush position or in left lateral recumbency.

Normal camelid peritoneal fluid is colorless to slightly yellow and clear, and contains up to 2.5 milligrams per deciliter (mg/dL) of protein and 3000 nucleated cells per microliter (μL).[35] A few camelids have higher cell counts and protein concentrations, usually not simultaneously and associated with very low fluid volumes. Camelids have a higher percentage of neutrophils (from 15% up to 98%) among the nucleated cells in abdominal fluid compared with most other domestic herbivores. Most of the remainder are large mononuclear cells. Because of the wide variations in normal, drawing inferences from cell percentages is more difficult than from absolute counts. Typically, we see up to about 2000 neutrophils per microliter and 1000 mononuclear cells per microliter in healthy camelids.

Care must be taken because camelids often become recumbent during the procedure. Although fluid yield is less likely, flank abdominal paracentesis also may be performed with the animal restrained in lateral recumbency. Alternatively, a ventral right paramedian approach may be tried, with the camelid in the standing position or in lateral recumbency. Because of the anatomic variation in the location of the pocket of fluid between C1 and C3, these areas are avoided, and ultrasonographic identification of landmarks is recommended prior to attempting ventral paracentesis.

Forestomach Fluid Analysis

Although the diagnostic value of forestomach fluid analysis is well established in ruminant medicine, its use is rarely reported in scientific papers about camelids.[36] Fluid may be retrieved from the forestomach by passing an orogastric tube into the stomach, applying suction to the tube, and crimping the tube to retrieve the sample held within its distal end.[37] Unless abnormal fluid accumulation is present within the forestomach (GI reflux, forestomach acidosis, recent oral fluid administration), it is rare that fluid is siphoned all the way to the proximal end of the tube. Adequate physical restraint is necessary, and chemical sedation may facilitate the procedure. A length of a semirigid hose, a roll of tape, a wooden block with a hole drilled through it, or a polypropylene tube may be used as a mouth speculum (see Figure 30-1). Regurgitation may occur around the tube, especially when abnormal fluid accumulation is present in the forestomach. In the case of regurgitation, the tube should be pulled immediately and the camelid's head depressed to prevent aspiration.

A percutaneous technique has also been described in llamas.[38] With this technique, a 16-gauge, 7.5-cm needle is advanced dorsocraniomedially from a point approximately

20 cm caudal to the costochondral junction of the last rib on the left side. Once the first gastric compartment is punctured, fluid is aspirated and the needle withdrawn rapidly. This technique avoids salivary contamination but has not been used much in sick camelids, which potentially would have more complications from peritonitis compared with their healthy counterparts.

Forestomach fluid may be analyzed in a similar fashion to rumen fluid. The pH is usually between 6.4 and 6.8 but may be higher in anorexic camelids or with salivary contamination. Color, smell, and consistency also vary with diet. On most forage-based diets, the color is olive green to light brown, the smell is a mixture of acid and fecal odors, and the consistency is more mucoid than liquid because of the small amount of free fluid. Microscopic examination should reveal a mixed population of bacteria, which are predominately gram negative if stained. Multiple sizes of active protozoa should be present.

Determination of the chloride concentration of forestomach fluid has proven to be a reliable test for GI obstruction. However, care must be taken to ensure an appropriate method of analysis. Other anions including VFAs affect the chloride electrodes of many analyzers.[39] Coulometric titration appears to be the most accurate method. Samples taken from healthy as well as sick and nonobstructed camelids almost always has a concentration less than 30 mEq/L.

Sources of chloride in the forestomach include ingested (or orally administered) feeds and medications, swallowed saliva, saccular secretions, and fluid refluxed from the hydrochloric acid–secreting portion of the third compartment or sodium chloride–secreting small intestine. The chloride concentration of camelid saliva and the likelihood that saccular secretion of chloride occurs at a significant rate under physiologic conditions are both quite low. Therefore, if oral administration of chloride-rich (electrolyte) fluids can be eliminated through knowledge of treatment history, the veterinarian can be reasonably certain that high forestomach fluid chloride concentrations are caused by reflux from C3 or the small intestine.

Other biochemical tests such as methylene blue clearance may be useful for assessing viability of microorganisms. The color from 0.03% methylene blue added to C1 fluid in a 1 : 20 ratio should clear in less than 5 minutes.

Blood Interpretation

Techniques for venipuncture are described elsewhere. The clinical pathology tests most useful for determining the severity and nature of gastrointestinal disease include the following:

1. Fresh whole blood for total and differential white blood cell (WBC) counts, total protein and fibrinogen concentrations, red blood cell (RBC) numbers and morphology
2. Serum or plasma for sodium, potassium, and chloride, total protein, albumin and globulin fractions, bicarbonate–total carbon dioxide (HCO_3/TCO_2) reflective of gut function and health, and blood urea nitrogen (BUN) and creatinine, liver enzymes and lactate, which are reflective of more global health issues

Fecal Occult Blood Analysis

Because of the attention given to fatal perforating ulcers of C3, some need exists for a diagnostic test. Unfortunately, fecal occult blood tests have been of questionable value, sometimes yielding positive results in camelids subsequently found not to have ulcers and yielding negative results in camelids with overt gastric hemorrhage.

Radiography

Radiographs are commonly used to evaluate the skull and esophagus, and less frequently, other parts of the gastrointestinal system.[40–44,46] In the skull, jaw masses and dental abnormalities are the most common indications. To localize lesions, lateral, dorsoventral, and oblique views are useful. Use of a radiolucent speculum aids in the separation of the dental arcades. Patients must be heavily sedated to allow proper alignment, especially for the dorsoventral and oblique views. Intraoral radiography may be useful for demonstrating cranial jaw lesions.

Cervical and thoracic radiography is often used to assess esophageal function.[43] Normally, the esophagus is scarcely recognizable on plain films. Liquid contrast material fed by dose syringe or mixed with grain or feed pellets highlights the mucosa and allows assessment of lumen size and emptying function. We commonly use up to 120 mL of barium sulfate (60% weight/volume) in adults and 20 to 50 mL in neonates. Healthy camelids usually clear the barium within a few seconds, so retention of all but a superficial layer after 30 seconds is considered abnormal.

Conventional radiography of the abdomen is most useful in the neonate because in juvenile and adult camelids, the feed-filled C1 obscures most other GI organs. In younger camelids, this compartment is not so large, and the intestinal loops can be seen more distinctly in the caudodorsal abdomen. Also, crias are more prone to gaseous distension of bowel, which contrasts with the feed-filled organs. Occasionally, obstructions and distended bowel may be visible in older camelids. In older camelids, gastroliths may be visible as rows of mineralized concretions over the cranial and caudal saccular regions of the first compartment. Mineralized phytobezoars or sand may be likewise visible.

Contrast radiography has been especially helpful with esophageal disorders. Approximately 100 mL of contrast material given to an adult llama is usually sufficient. Contrast may also be used to highlight the first gastric compartment to help distinguish it from other structures in cases of colic in crias. Intestinal passage of contrast material in crias has been described, but in sick crias, contrast tends to remain in the stomach.[45] Contrast normally enters the proximal loop of the ascending colon after 2 to 4 hours but may not enter the spiral colon for over 16 hours.

Advanced radiologic imaging techniques such as computed tomography (CT) have seen limited application in camelids but will doubtless see more application in the future.[47,48] Current reports on CT use are mainly descriptive studies of normal camelids, or examinations of the head. CT may also be used to identify vascular ring anomalies in the chest or thickened bowel or mass lesions in the abdomen.

Ultrasonography

We have performed abdominal ultrasonography on a number of camelids with GI disease, but more work is needed to establish parameters of normal and abnormal findings.[49] We have used both linear array and sector scanners with 3.5 to 7.5

megahertz (MHz) probes. The 5-MHz equipment commonly used for reproductive evaluation of camelids is usually adequate.

Two approaches can be used: *transrectal* and *transcutaneous*. Transrectal ultrasonography is of limited value because it is difficult to visualize structures cranial to the bladder and reproductive tract in small camelids and most structures that can be scanned can also be palpated per rectum (i.e., the spiral colon) in large camelids or viewed transabdominally. This technique is most useful for investigating solid masses such as intrapelvic hematomas, abscesses, or tumors, or urogenital abnormalities.

Transcutaneous abdominal ultrasonography has been used to diagnose GI disorders in horses, cattle, and small animals but has seen limited usage in camelids. We have found it very useful, especially in camelids too small for rectal palpation. Examination can often be performed easily through the flanks or the caudoventral abdomen because of the small amount of hair, whereas examination through the cranial abdomen and the ventrum or between ribs requires clipping of the fleece. It is especially useful in differentiating free fluid accumulation from visceral distension in camelids with abdominal distension.

Examination may be performed with the animal in the standing position or in lateral recumbency.[31] In the craniocaudoventral abdomen, the first and third gastric compartments (Figure 40-11) appear as large, thick-walled, fluid-filled viscera on the left and right sides of midline, respectively. The space between them, usually 3 to 7 cm right of midline, contains hypoechoic peritoneal fluid. The first compartment extends the length of the abdomen, with the saccules clearly visible as semicircular, thin-walled hyperechoic protrusions from the compartment wall caudal to the transverse pillar and cranial to the umbilicus. The compartment and saccular lumens often contain numerous hyperechoic densities with shadows caused by bezoars, feed material, and gas. These prevent clear imaging of the lumen. The third compartment can be followed from the xiphoid past the umbilicus, where it curves dorsad and craniad. It can be recognized by its curved walls in cross-section. Third compartment ingesta is usually less echogenic and more uniform in appearance than the first compartment fluid and enables good imaging of the wall. It is important to remember that the wall normally thickens from 4 mm in the cranial regions to 7 to 10 mm in the true gastric region.

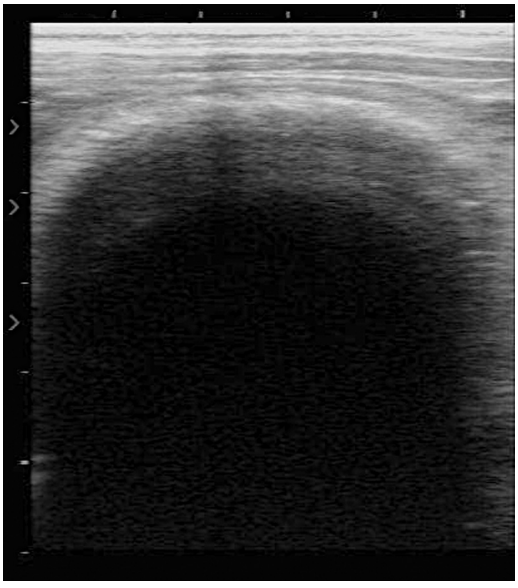

Figure 40-11 Transcutaneous ultrasonographic appearance of the normal third compartment.

Peritoneal fluid is usually anechoic and, except for the normal accumulation between the compartments and the peritoneum, rarely can be imaged. Ultrasonography may be highly useful in identifying pockets of fluid to be retrieved by abdominocentesis and also for determining the severity of ascites.

The right caudoventral and caudolateral abdomen (through the paralumbar fossa) contains most of the scannable intestine, including most of the jejunum, ileum, cecum, spiral colon, and descending colon. It is very difficult to identify individual intestinal structures because most of the loops are of similar size and very little fluid is present within the intestinal lumen, giving the various structures and their contents a heterogeneous, hyperechoic appearance. Occasionally, a peristaltic wave may be seen as either the hyperechoic bowel moves in the surrounding peritoneal fluid or as a fluid bolus (rarely wider than 1 cm or longer than 3 cm) is moved through the small intestine.

Endoscopy

Endoscopy has been used to image the pharynx, esophagus, and cardiac region of the stomach. Oral endoscopy and passing any endoscope through the mouth is not recommended because of the potential of the patient's sharp teeth to damage the scope. Nasal endoscopy limits the diameter to about 6 mm in neonates and 1 cm in adults. Imaging the esophagus is most useful in cases of suspected esophageal obstruction, megaesophagus, and esophagitis. Imaging the cardia of C1 has limited application but may be used to look for ulcers in the region or foreign bodies in the stomach. Sedation should be considered because camelids tend to regurgitate during prolonged endoscopy.

Exploratory or Laparoscopic Surgery

Exploratory surgery is a useful test in that it allows direct or camera-mediated inspection of various organs, correction of a variety of maladies, or collection of biopsy samples. For most purposes, ventral midline or right paralumbar fossa approaches are satisfactory. Obviously, if the surgeon desires to enter the first compartment, the left flank is preferable to the right, and if terminal C3 is of special interest, a right paracostal approach allows the best inspection of a variety of poorly mobile regions. These are discussed in detail in Chapter 57.

Oral Disorders

Ulcerative and Proliferative Lip Lesions

A variety of factors affect the lips, particularly in the region of the mucocutaneous junction. Those that affect multiple mucocutaneous junctions are covered more thoroughly under dermatologic conditions (Chapter 35).

Fibropapilloma

Proliferative, unpigmented, sessile lesions on the lips, external nares, and occasionally cheek skin appear to occur as a result of infection with a camelid papilloma virus.[50] This virus appears to be poorly transmissible, as spread within herds is slow and rare, and may depend on some inherent immunodeficiency of the host to cause visible lesions. Although some camelids develop a solitary nodule, most develop several, often on different regions of the lips and nose. They do not affect attitude or appetite, although they may become ulcerated.

Biopsy may be used to confirm the fibropapillomatous characteristics of the lesion, and polymerase chain reaction (PCR) may be used to confirm the presence of papillomavirus. The camelid virus appears to be unique but has the greatest homology with bovine papillomavirus-1.

Resolution of lesions may be problematic. Most do not regress spontaneously, and some may grow quite large. Removal may be curative or may lead to aggressive spread and regrowth, so the first attempt should be made with a suitable margin. Regrowth may also occur in the form of granulation tissue, so subsequent biopsies may not reveal the original lesion. Various treatments including cryosurgery, laser excision, and intralesional injection with chemotherapeutic agents may help prevent regrowth.

Oral Mucosal Lesions

Camelids are susceptible to many of the oral vesicular or erosive diseases of regulatory importance, including foot-and-mouth disease and vesicular stomatitis. However, camelids are generally not exposed to these agents outside of their native environments and appear to be minor hosts. Also, most described lesions are on the feet, and the mouth is generally spared. The first reports of suspected bluetongue are now coming out of Europe and California. Oral lesions may be present along with more severe systemic disease. In the case of oral mucosal lesions, the appropriate samples should be taken to ensure that diseases of regulatory importance are not missed.

Oral ulcerative lesions are most commonly traumatic or bacterial (*Actinobacillus*, *Fusobacterium*) in origin (Figure 40-12) and usually respond to short courses of antibiotics, or the appropriate dental correction. Proliferative and ulcerative lesions are commonly attributed to parapox virus infection. In the United States and Europe, these lesions are occasionally linked to camel pox or the camelid papillomavirus. Lip lesions of any sort are more likely to be clinically significant in camelids than ruminants because of the tonguelike role of camelid lips in prehension.

Ptyalism, Pseudoptaylism, and Cud Spilling

Salivary loss is relatively common and is much more frequently associated with deficits in swallowing than in overproduction. A variety of causes have been described, but half or more of the cases remain unexplained or poorly explained. The cause may be morphologic, as with cleft palate, mismatch in the length of the maxilla and mandible, wry nose, cranial displacement of the parotid duct, or laryngeal malformation.

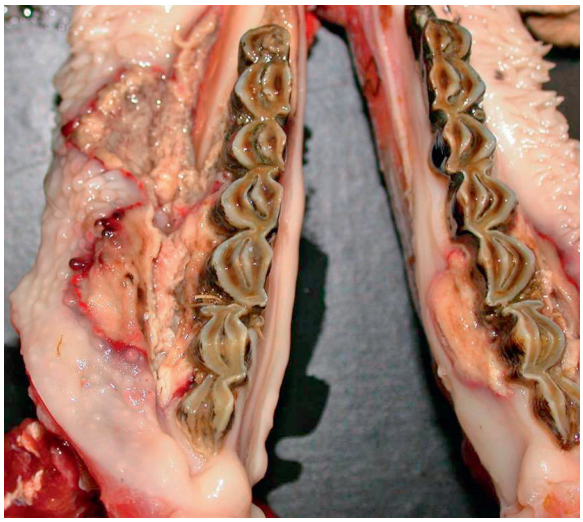

Figure 40-12 Severe oral ulceration with *Fusobacterium* infection.

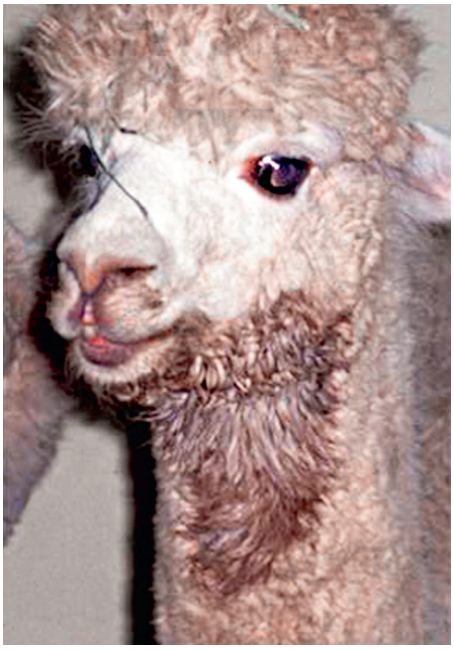

Figure 40-13 Salivary or water staining compatible with pseudoptyalism or a vice.

It may also be functional, as with tetanus, rabies, botulism, lingual paresis, tick paralysis, epiglottic paralysis, pharyngeal edema (a common cause of salivary loss in debilitated, hypoproteinemic camelids) or any source of weakness that causes the camelid to drop its nose or have a sluggish swallowing reflex. Salivary loss may also accompany irritation of oral structures, as with ulcerative stomatitis, glossitis, malocclusion, or intraoral foreign bodies, or obstructive disorders such as choke or megaesophagus. Cud spilling and salivation may also be a conscious or unconscious vice. Some camelids drool absentmindedly or rest their chins in water buckets (Figure 40-13), which leads to a similar appearance. Others appear to lose cuds overnight, possibly during some phase of sleep. If the camelid demonstrates no evidence of dysphagia at other times, cud spilling is usually behavior related.

If generalized weakness is present in addition to salivary loss, the cause should be investigated. If not, the mouth should be examined for irritation, trauma, foreign bodies, malocclusion, the location of the pyloric duct papillae, and conformation of the jaw and oral cavity.

True hypersalivation is far less common than pseudoptyalism. The leading cause is ingestion of slaframine or the "slobber factor." This is a parasympathomimetic indolizidine alkaloid produced by the fungus *Rhizoctonia leguminicola*, which colonizes legumes, particularly red clover in damp areas and subterranean clover in dry areas. The fungus causes discoloration of the clover known as "black patch." All species of herbivore are affected.

After ingestion, the toxin is metabolized by the liver to an acetyl choline analog. Signs start 3 to 5 hours after ingestion and last about 24 hours. Clinical signs include profuse salivation ("slobbers"), colic, lacrimation, tremors, urination, diarrhea, bloat, dyspnea, and stiff gait. Diagnosis is made by identifying the signs and finding the infected plants. Finding the signs of colic and slobbers in multiple animals is also highly supportive. Clinical pathologic abnormalities have not been reported. Signs usually resolve spontaneously over 24 hours but may last in a diminishing fashion up to 3 days. Treatment is not usually necessary, and the specific combination of signs, especially if multiple animals are affected or the discolored clover can be found, helps the attending veterinarian be confident about the diagnosis. In severe cases, atropine may provide some relief.

Camelids that lose saliva rapidly become acidemic and hypokalemic. Except in the instance of choke, these electrolytes may be replaced orally. Cud spilling is less life threatening and rarely requires treatment beyond that which addresses the primary cause.

Malocclusion

Incisor and molar malocclusion are relatively common, especially in alpacas. Incisor problems usually relate to teeth that project too horizontally and fail to contact the dental pad (see Figure 40-2). In some cases, malocclusion occurs secondary to jaw length abnormalities or to deformities, including brachygnathism, prognathism, wry nose, tumors, or bony abscesses. Although incisor malocclusion theoretically could affect the camelid's ability to graze, the cosmetic abnormalities usually far outweigh any functional disturbance.

Molar malocclusion is also common, especially in older camelids.[42]Although it may lead to very irregular points and ridges on the occlusal surfaces of the molars, these changes may also be more cosmetic than pathogenic (see Figure 40-3). Facial and jaw deformities may be contributing factors. On rare occasions, halitosis, cud retention, quidding, and weight loss have been blamed on malocclusion. Generally, we are skeptical that irregular molars affect function unless they traumatize the oral mucosa. Because the inside of the back part of the mouth can be difficult to examine, both malocclusion and mucosal trauma may be difficult to establish on physical examination.

Molar malocclusion can be assessed by radiographic examination of the occlusal surfaces. This may be difficult in live animals because of the overlapping structures and is therefore best evaluated on oblique views. Most camelids show relatively flat occlusal surfaces for the first 4 years of life, after which curves and irregularities develop progressively in many. Most changes are bilateral and are most likely caused by the camelid's dominant transverse chewing pattern. These changes have not been linked to any particular health event.

Obstetric wires, canine nail trimmers, files, hand saws, and rotary cutting tools all have been used to cut the long incisors. Some alpacas require this intervention yearly. Various files have been used on molars. As some degree of irregularity of the molars is normal, it is important to establish the necessity for this procedure (weight loss, dysphagia, mucosal trauma) before performing it.

Jaw Masses

Bony facial masses are a common finding in camelids. These may be caused by tooth root abscesses, osteomyelitis (lumpy jaw), or in rare cases fractures, cysts, or tumors.[40,41,51–55] Tooth root abscesses are much more common than lumpy jaw in camelids and most commonly involve the second molar (30%, mainly the caudal root), the first molar (23%), the third molar (15%, mainly the cranial root), or the last premolar (13%) of the mandible.[40,41] Incisors (7%), canines (3%), and maxillary teeth (6%) are affected much less commonly. Approximately half the affected camelids have multiple infected teeth, and about 20% of affected camelids have infected teeth on more than one hemiarcade.[40]

Lumpy jaw is much less common, perhaps representing 2% of all bony swellings. It tends to cause a plaquelike enlargement of one hemimandible and should be suspected when multiple camelids in a herd develop characteristic signs over a short period. Lumpy jaw is most easily confused with tooth root abscessation of multiple adjacent cheek teeth, when the bony swelling is more generalized (Figure 40-14).

Tooth root abscesses and lumpy jaw have very similar clinical appearances, and radiography is necessary to differentiate them. Both cause hard, asymmetrical facial swelling that may drain thick pus intermittently, both tend to grow if left

Figure 40-14 A large, plaquelike tooth root abscess. Such broad lesions may be difficult to differentiate from lumpy jaw.

Figure 40-15 The typical focal bony swelling seen with abscessation of a single mandibular molar.

Figure 40-16 A maxillary tooth root abscess with drainage on the face and out of the nose.

untreated, and neither commonly leads to dysphagia or weight loss. Abscesses usually lead to more focal bony enlargement, but this may become generalized with chronicity or if multiple teeth are affected (Figure 40-15).

Occasionally, other signs are noted in conjunction with facial swelling or, in very rare cases, before the swelling occurs. These signs may include salivary loss, quidding, weight loss, and anorexia. Firm palpation of the mass may elicit a pain response. Also, maxillary tumors may lead to deformation of the face under the eye and unilateral purulent nasal discharge (Figure 40-16). In rare cases, systemic effects are noted. Although bacteremia secondary to tooth root abscessation appears to occur in the minority of cases, a small percentage will develop infection or abscessation in the internal organs, including the liver, kidneys, spleen, or lungs.

No consensus exists with regard to the causes of tooth root abscesses and mandibular osteomyelitis. Grass awns, foxtails, or other foreign bodies may inoculate the deeper tissues with bacteria. Fractured teeth or a fractured jaw may allow similar

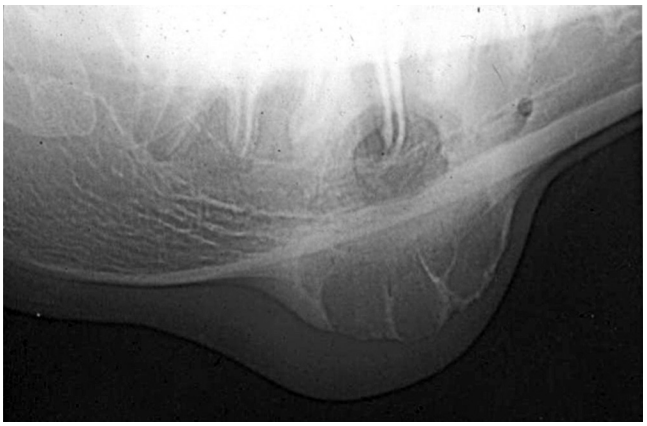

Figure 40-17 Radiographic appearance of a tooth root abscess. Note the lytic center around the root and the proliferative bone.

penetration. Periodontal disease, which is generally underappreciated in camelids, may similarly facilitate deep infection.[42] Tooth trimming has been incriminated in the occasional incisor or canine abscess, but some camelids have abscesses in front teeth that have never been trimmed. Another possibility that has been raised is infection through the tooth root. Patent infundibulum has been identified in approximately 20% of abscessed teeth.[43] Immunodeficiency also may contribute in some cases.[40]

Diagnosis of tooth root abscess can usually be made on the basis of clinical signs, but radiography is necessary to definitively differentiate this from the various other uncommon possibilities. Tooth root abscesses are characterized by lysis and proliferation of bone with a radiolucent pocket around the affected root or roots (Figure 40-17). In contrast, lumpy jaw has lysis and proliferation without associated changes to the tooth root. Bacterial culture may be helpful in selecting antibiotics and definitively diagnosing lumpy jaw. Biopsy is recommended if a significant soft tissue component seems to exist.

A variety of treatment options are available. In our area, most owners prefer to attempt to sterilize the infection by using antibiotics. Because the infection usually involves single or multiple species of facultative anaerobic organisms, penicillin (22,000 to 44,000 units per kilogram [units/kg], subcutaneously [SQ], q24h for 6 to 12 weeks) and ceftiofur (2 to 4 mg/kg, SQ, q24h for 6 to 12 weeks) have been used most extensively. Oral sulfa antibiotics have also been used in an attempt to avoid the need for injections, but their absorption and efficacy are usually disappointing. Recently, florfenicol (20 mg/kg, SQ, q48h for 10 treatments), isoniazid (20 mg/kg, orally [PO], q24h for 4 to 12 weeks), and long-acting ceftiofur crystalline free acid (6.6 mg/kg, SQ, q7d for 6 weeks) have shown promise as more convenient yet effective treatments. Although it has not been reported in New World camelids, isoniazid has been associated with fatal marrow suppression in camels.[56] As an adjunct to antibiotic therapy, draining tracts should be flushed with antiseptic solutions and possibly curetted out. This same treatment approach may be used for lumpy jaw as well.

Antibiotic treatment is especially useful in camelids with multiple infected teeth and those in advanced pregnancy or when the owner has financial limitations and surgery is a less

feasible option, but it is also a reasonable way to start therapy for any camelid with an affected tooth. It has been effective in over half of the cases in which it has been used.[40] Efficacy is judged by partial regression or lack of advancement of the bony lesion when evaluated physically or radiographically, cessation of pus drainage, or resolution of other signs.

Surgical treatment is another option and may be better when antibiotic treatment has failed, long-term injections are problematic, or when the owners desire a rapid, more cosmetic result.[40,41,51,57] General anesthesia is strongly advised, although not mandatory. Tooth repulsion is the most common procedure, followed by hemiextraction, apicoectomy, and the rare root canal. Curettage and removal of bony sequestra may also be performed during the procedure or may be the primary treatment for lumpy jaw. Care must be taken during extraction, especially in alpacas, not to fracture the jaw. More details on the procedures may be found in Chapter 63. After surgery, patients are usually administered analgesics and antibiotics for up to 14 days. Whether medical or surgical treatment has been used, recurrence of the infection, or emergence of infection of another tooth is always a possibility.

Other bony masses are rare, and most of these have considerable soft tissue expansion.[52–55] Radiography shows thin layers of displaced bone surrounding this soft tissue density, and biopsy of the soft tissue is usually necessary to make a diagnosis. Sinus cysts have been seen to deform the maxilla in juveniles and young adults. Ameloblastomas or odontomas may affect the mandible or maxilla, mainly in the young but occasionally in older camelids as well. An expansile, multiloculated, firm soft tissue mass may be visible or palpable within the oral cavity. Because of the propensity of these tumors to deform the dental arcade, mastication is more likely to be affected than with abscessed teeth. Local or regional excision may eliminate the mass, but failure to excise adequate margins may result in regrowth. Radiation therapy was attempted in one llama with unsatisfactory results. Squamous cell carcinoma and anaplastic sarcoma have also been described and are less amenable to treatment.

Nonbony masses are less common, which leads us to think that all jaw masses are tooth root abscesses, until proven otherwise. Soft tissue abscesses are rare except with caseous lymphadenitis, and therefore animals should be examined carefully before any such structure is lanced. The two most common solid, soft tissue masses of the face are retained cuds (Figure 40-18) and lymphoma (Figure 40-19). Diffuse swelling is seen with various insect and reptile bites. Unlike in ruminants, camelids rarely develop submandibular edema with hypoproteinemia, although they may develop pharyngeal edema.

Figure 40-18 Facial enlargement caused by a retained cud.

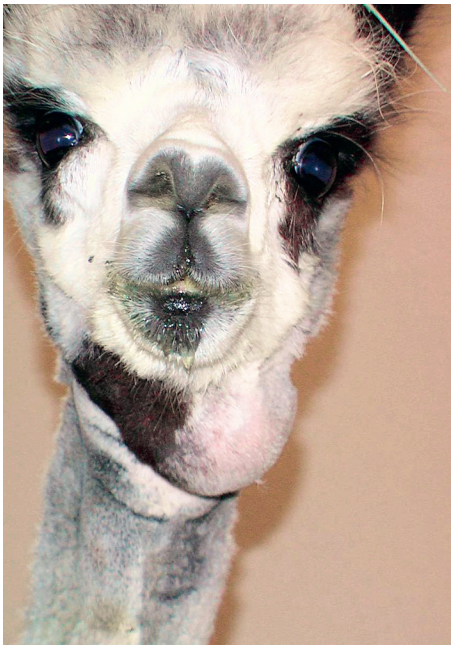

Figure 40-19 Enlarge submandibular lymph node compatible with lymphoma.

Esophageal Disorders

Choke

Juvenile and adult camelids must regurgitate gastric ingesta up the long and partially vertical esophagus for repeated chewing. To accommodate this, the esophagus is lined with mucus glands and ringed with well-developed bands of striated muscle.

Because camelids are usually selective eaters, and often more browsers than grazers, primary choke is uncommon. It may be seen with weak or excessively hungry camelids, especially when fed pellets or cereal grains, or rarely in neonates who overindulge on long-stem grass or hay. More commonly, choke occurs secondary to some other form of partial esophageal obstruction or neuromuscular dysfunction of the esophagus.

Partial esophageal obstruction is relatively rare. In younger camelids, it may occur secondary to a vascular ring anomaly.[58] This has been recognized in an adult camelid as well.[59] In older camelids, diverticula, strictures, foreign bodies, or tumors of the esophagus or cardia may obstruct passage of

Figure 40-20 Salivary loss compatible with Slaframine intoxication or choke.

ingesta. Blackberry canes, fibroma, and squamous cell carcinoma have all been observed.[60]

Clinical signs of choke are similar, regardless of the cause. Affected camelids lose saliva out of the mouth and occasionally the nose. The salivary discharge often contains bits of chewed feed (Figure 40-20). The head is often held in a low position to facilitate regurgitation. Intermittent appetite is usually retained, exacerbating the obstruction and regurgitation. A fluid wave may be visible on the left side of the neck. Aware camelids usually protect their airway fairly well, although they may cough and have moist sounds on auscultation of the larynx and trachea. Compromised or sedated camelids, neonates, or camelids restrained with their head elevated are at greater risk for developing aspiration pneumonia, leading to depression, loss of appetite, and intermittent fever.

Clinical pathology abnormalities are usually mild. Metabolic acidosis, azotemia, and hyperalbuminemia develop with severe salivary loss. Stress leukogram may be replaced by inflammatory changes with feed aspiration.

Diagnosis may be based on clinical signs, but a further workup may be helpful in confirming the diagnosis and establishing the presence or absence of underlying lesions. Plain radiography of the neck may reveal a soft tissue density, possibly with a mottled opacity, at the site of obstruction. Contrast radiography is not recommended before the obstruction is resolved because of the risk of aspiration of contrast material. Thoracic radiography or ultrasonography may also reveal information concerning the health of the lung. Transcutaneous cervical ultrasonography may reveal the feed mass and fluid above it. Esophagoscopy is the most direct method and allows direct inspection of the obstruction and the mucosa around it.

Choke may be relieved by a variety of techniques, with the basic tenet of avoiding esophageal damage or feed aspiration. Allowing an aware adult a few hours without feed, water, or edible bedding may allow for self-resolution. Light tranquilization may be beneficial, but sedation may inhibit the camelid's ability to protect its airway. We prefer butorphanol to xylazine. If the obstruction does not resolve, with the camelid tranquilized and restrained with its head depressed below the level of the thoracic inlet, a regular or cuffed stomach tube may be introduced to the level of the obstruction. Water or air may be used to distend the esophagus or break up the mass, or the mass may be pushed gently into the stomach. Alternatively, the mass may be picked apart using the endoscope and biopsy tool. If success is not achieved in 15 to 30 minutes, the amount of effort seems excessive, or the camelid is becoming unduly stressed, the procedure should be halted and the camelid allowed to recover without food or water for an hour or two. If the camelid becomes dehydrated, intravenous (IV) fluids should be administered, possibly with supplemental bicarbonate to correct acidosis. If aspiration is suspected, an aggressive course of broad-spectrum antibiotics and antiinflammatory agents should be administered. After resolution, endoscopic evaluation of the esophageal mucosa is helpful in assessing the risk for strictures, and further workup may be helpful in investigating underlying disorders.

Megaesophagus

The most common underlying cause of choke is megaesophagus, which may also create a variety of other disease signs (see Figure 40-20). In most cases, megaesophagus is idiopathic.[43] Endocrine disorders and myasthenia gravis have not been linked to this disorder in camelids. Because of the importance of concerted muscular activity during swallowing and regurgitation, lesions of the vagus, its offshoots, and the muscles it innervates are suspected in most cases. Neck trauma caused by fight wounds, dog bite wounds, running through fences, electroshock collars, or other penetrating injuries appears to be the principal identifiable cause of cervical esophageal neuromuscular dysfunction. Trauma from overly invasive venipuncture or jugular catheterization attempts could potentially be causative, but this has not been confirmed. Muscular function of the esophagus may also be compromised by *Sarcocystis*, *Toxoplasma*, trypanosomes, warble flies, botulism, hypocalcemia, or any other process that compromises systemic function. Organophosphate toxicity was blamed in one case.[43] Megaesophagus may also occur secondary to the chronic obstructive lesions such as tumors, diverticula, and vascular rings.[58,59] Some of these lead to temporary dysfunction, but most cause permanent, progressive functional failure. In spite of all these possibilities, no specific etiology has been identified in over half the cases.

It is likely that megaesophagus starts as a focal lesion, but persistent choke or chronic distension may lead to mechanical widening of the affected zone. Regurgitation is affected before swallowing. The earliest sign is mild salivation, probably induced by a cud which failed to make it past the lesion.[43] The next sign is often cud loss, probably induced when the camelid loses track of a cud while it passes through the lesion (Figure 40-21). Other signs which appear in the first few weeks or months are recurrent choke and the development of a palpable or visible recurrent or persistent fluid wave in the left ventral neck. The distended esophagus may be palpable. Weight loss is progressive, and feed aspiration is always a risk. Respiratory sounds may be abnormal with aspiration. Also, gurgling of gas and fluid in the esophagus may sound like GI sounds in the thorax.

Megaesophagus is seen most commonly in adults, but camelids around weaning time represent a distinct subset of cases. It is likely that these younger crias have had the lesion for a

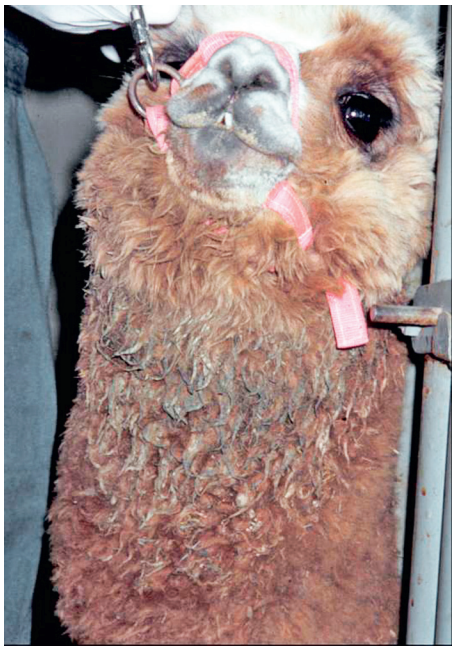

Figure 40-21 Feed staining compatible with regurgitation caused by megaesophagus or choke.

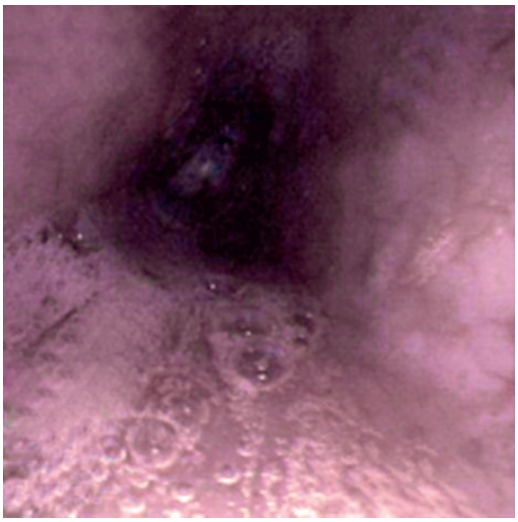

Figure 40-22 Esophageal endoscopy demonstrating dilation and flaccidity.

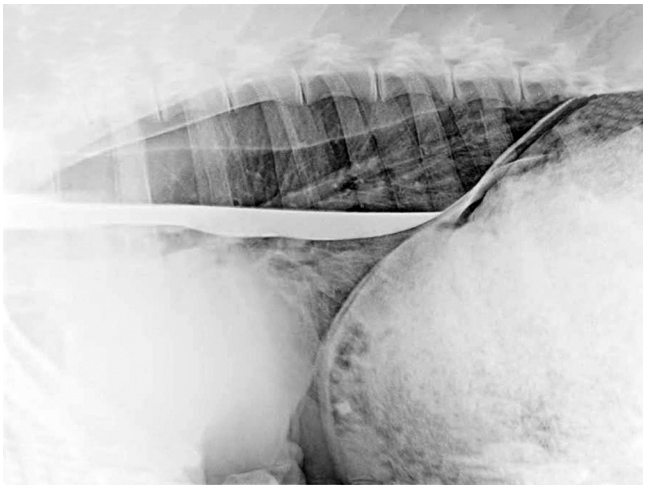

Figure 40-23 Retained contrast material within the dilated esophagus of a camelid with megaesophagus.

while, possibly since birth, and that clinical signs developed as the cria transitioned from a milk diet to solid feeds. Hereditary megaesophagus has been described in other species, but we have attributed a high proportion of those cases to vascular ring anomalies, which may, in and of themselves, be heritable.[58,59]

A tentative diagnosis may be reached by identification of the characteristic signs. Except in cases of aspiration or the rare cases where an underlying inflammatory lesion is present, clinical pathology changes are usually mild. Esophageal endoscopy may reveal feed distension, or a dilated, flaccid esophagus (Figure 40-22). Plain or contrast radiography, fluoroscopy, and CT are the best confirmatory tests. Plain-film radiography shows the feed-filled esophagus, especially within the thorax. Contrast material helps highlight the feed impaction or constriction (see Figure 36-1), but should be avoided with complete obstruction because of the risk of aspiration. In the unobstructed esophagus, delayed passage of the contrast material is generally diagnostic—normally it clears almost instantly—but it must be remembered that severely compromised or heavily sedated camelids will have poor esophageal function (Figure 40-23). Fluoroscopy is useful to demonstrate the functional defect, especially with vascular ring anomaly.

No specific treatment for megaesophagus exists, particularly because lesions are usually chronic at diagnosis. Parenteral sulfa antibiotics may be helpful for *Sarcocystis* or *Toxoplasma* infection, and ivermectin may be helpful against larger parasites. Ponazuril and related compounds may also have value in treating protozoal infections. Anecdotally, anti-inflammatory medications, including corticosteroids, have led to resolution in some cases. Even if a primary obstructive lesion is identified, correction of that may not occur within the time necessary to prevent the secondary muscle damage to the esophagus. Because camelids with megaesophagus have more difficulty with regurgitation than with swallowing, feeding from a height may aid in keeping the esophagus clear of food but is unlikely to help during rumination. The most beneficial treatment appears to be feeding highly digestible, small particle feedstuffs such as complete pelleted feeds or alfalfa pellets from a bucket raised above the level of the back in small quantities frequently. Others report success with long-stem hay. Frequently, it is a matter of finding a feedstuff that the camelid tolerates without developing choke and then monitoring weight. Camelids that lose weight have an understandably worse prognosis than those that do not. With successful dietary management, some camelids may survive for years, whereas others who do not tolerate any diet will usually succumb in a number of months.

Esophagitis

Esophageal ulcers resulting in vomiting and pseudoptyalism may occur after ingestion of irritating plants, particularly rhododendron, laurel, azaleas, and Labrador tea.[61,62] Some of

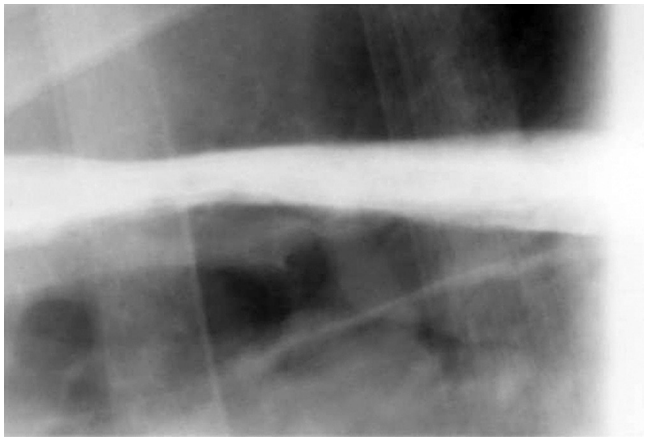

Figure 40-24 Close-up view of a thoracic contrast esophagogram. Note the irregularity of the ventral margin of the contrast column, indicative of esophagitis.

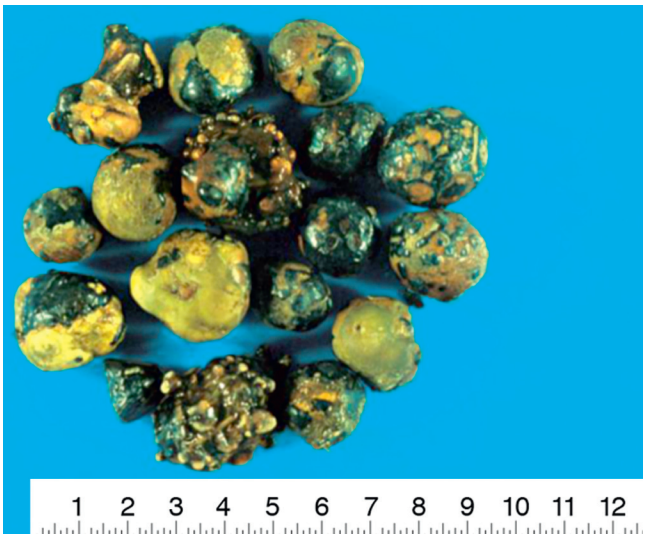

Figure 40-25 Mineralized gastroliths from the saccules of a llama. Such concretions are usually incidental findings but may be associated with weight loss and, in rare instances, may lead to saccular rupture.

these are typically grown as ornamental shrubs, and others grow wild. Camelids are exposed to the ornamentals either by experimenting with unfamiliar plants or when dried leaves blow onto other feedstuffs. Camelids are exposed to the wild forms during off-season packing trips, when these evergreens may be the only green plants available. Actual esophagitis is not a commonly reported lesion with these plants but has been confirmed in cases seen at our clinic. Other irritants such as undiluted phenylbutazone or amprolium may also cause ulcers but are rarely given in sufficient quantities to cause clinical signs. Blister beetle ingestion is another possibility, but this has not yet been described in New World camelids. Lesions with all these irritants are worst in the esophagus and may extend into the forestomach. The mouth is usually unaffected. Although the toxic principals of the irritant plants are cardiac glycosides, heart irregularities appear to be uncommon.

Affected animals eat readily but quickly retch out the cuds. Weight loss and feed aspiration may ensue. Diagnosis is by clinical signs, history of exposure, and finding of poisonous plant leaves in the vomitus. Contrast esophagography may demonstrate poor emptying or irregularity to the mucosa (Figure 40-24). Esophagoscopy allows direct visualization of the linear ulcerated lesions, which often involve much of the length of the esophagus.

Coating drugs may be of some therapeutic benefit, but the most important treatment is supportive care during the healing period. Sucralfate (20 to 40 mg/kg, PO, q6-8h) or bismuth subsalicylate may coat the lesions and allow them to heal. IV fluids and nutrition may be necessary during the acute phase, but dietary management similar to what is used for megaesophagus may help minimize choke and weight loss. Prognosis is generally good, but camelids may vomit for up to 6 weeks before complete resolution occurs.

Gastric Disorders

Mineralized Gastroliths and Foreign Bodies

The nonkeratinized epithelium of the gastric saccules is protected from the abrasive action of ingested fiber by muscular sphincters coated with keratinized squamous epithelium. If large particles pass this sphincter, they are frequently trapped in the saccular lumen. Wedge-shaped grains, particularly sunflower seeds, appear more prone to do this. Once in the lumen, mineral is laid around the objects, forming stones (Figure 40-25). These stones, with the indigenous name "luan cura," were once purported to be of medicinal value to humans.[63,64] They invariably contain a nidus of plant material, usually a seed surrounded by a mineralized matrix, and may be seen as checkerboard rows of mineral densities on radiography of the abdomen. Wires and other foreign bodies that get trapped in the saccules undergo similar mineralization, perhaps preventing penetrating injury. The outer surface of mineralized objects may be smooth or rough and yellow-green or black in color. The size usually varies from about 5 mm to 30 mm per side, although kilogram-weight stones have also been described. Mineral analyses of such bezoars at our laboratory and elsewhere have suggested that they may contain different layers of mineral and consist mainly of calcium or magnesium phosphate salts.[65] It has been suggested that the microenvironment of the saccule contributes to magnesium phosphate salt crystallization because of the high bicarbonate and phosphate concentrations in saccular fluid and that the bezoar helps maintain this microenvironment by blocking evacuation of the saccule during forestomach contraction. Small bezoars may initially act as valves that occlude outflow and prevent saccular eversion and large bezoars often completely fill the saccular lumen and may eventually rupture through.

Currently, our knowledge about the importance of these bezoars is rudimentary. We find them most commonly on radiographic or postmortem examination of aged camelids and cannot always associate them with clinical signs. Colic and gastric perforation may occur with large, sharp saccular bezoars, but this is uncommon.[66,67] It is a common belief, although unproven, that camelids with a large number of saccular bezoars become unthrifty. As most volatile fatty acids

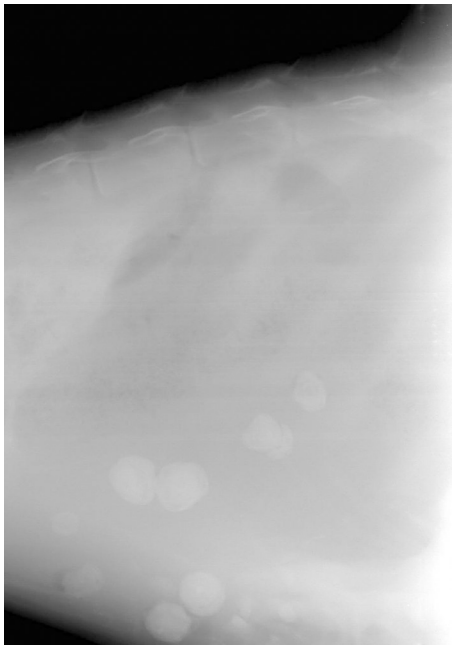

Figure 40-26 Radiographic appearance of saccular gastroliths.

from the forestomach liquor seem to be absorbed across the saccular membranes, it follows logically that blocking these membranes could result in decreased digestive efficiency, acidification of the forestomach liquor, and increased delivery of osmotic particles into the lower GI tract. These effects could lead to chronic weight loss and diarrhea.

Diagnosis is usually incidental. Radiography reveals them as lines and rows of mineralized densities (Figure 40-26). Necropsy also reveals the liths. We have not attempted therapy for saccular bezoars. Acidifying the forestomach contents with a high energy diet could be attempted to inhibit precipitation of magnesium phosphate crystals, but this treatment is not recommended because of the risk of forestomach acidosis. Surgical removal would also be difficult because of the number and the embedded nature of the bezoars. Because the mineral matrix is often brittle, the bezoars could be broken down in situ to facilitate removal. Grain (seed) feeding may increase the likelihood of stone formation, but this is unknown. Stones have been found in wild camelids and may even be more common in those species, so other sources are also possible.[64]

Intraluminal bezoars may mineralize with time. This is a rare finding, but it was linked with abdominal distension and ill-thrift in one juvenile alpaca. Because of the mineralization, the foreign bodies were detectable on radiography of the abdomen. Surgical removal would theoretically have been possible, but this particular alpaca succumbed before that was attempted.

Grain Overload or Forestomach Acidosis

Camelids are adapted to survive in harsh, nutrient-poor environments. Their gastric fermentation of fiber is among the most efficient of hoofstock species. It is likely that the nonkeratinized epithelium of their gastric saccules contributes to this efficiency by providing surface area for rapid absorption of ionized volatile fatty acids from the gut lumen. However, as addressed in the previous discussion of physiology, camelids appear to lack capacity to increase salivary buffering or gastric surface area substantially, and claims for enhanced forestomach buffering were probably overrated. Additionally, as camelids appear to absorb most of the VFA in its ionized state, the higher acidity and higher percentage of unionized VFA may not enhance absorption, as it does in ruminants. For all these reasons, it is likely that camelids are as susceptible or more susceptible than ruminants to the deleterious accumulation of acidic fermentative products after rapid ingestion of highly fermentable feeds.

Forestomach acidosis occurs after the ingestion of fermentable carbohydrates. Specific causes vary geographically and with feed availabilities. Cereal grains are most commonly implicated, with other carbohydrate-rich foods such as fruits and incompletely processed beet pulp also being potentially causative factors.[37] Rabbit chow was implicated in one small outbreak.[68] Feed exposure may be intentional or accidental. Intentional exposure may involve uninformed personnel or managers trying to increase body condition, make animals more approachable, or compensate for higher energy demands by feeding palatable, energy-rich feeds. Unintentional exposure may involve camelids escaping into feed stores or orchards, maturation of fruit in a paddock, or overingestion by individuals of feed meant for a larger group. Group housing may increase competition between animals, encourage aggressive feeding behavior, and increase the risk of forestomach acidosis. Even when the overall amount of grain appears reasonable if equally divided, certain aggressive eaters may get more than their share.

When the rate of acid production in the forestomach outpaces buffering and absorption, the pH of the forestomach fluid decreases, the microbial population changes, and microbial production shifts from VFA to lactic acid. Some of the lactic acid is absorbed into the systemic circulation, and the rest acts as an osmotic agent, drawing body water into the forestomach. Systemic dehydration and acidosis and chemical injury to the gastric mucosa ensue (Figure 40-27).

Clinical signs in the peracute stage of forestomach acidosis include obtundation, tachycardia, dry mucous membranes, weakness, ataxia, GI atony, and forestomach distension. Forestomach gas distension and splashiness, common signs in ruminants, are uncommon in camelids. Because of the staggering gait, eventual recumbency, and head retroflexing over the back, signs are occasionally misinterpreted as evidence of neurologic disease or colic. Rapidity of development and progression of signs depend on the amount of fermentable carbohydrate ingested. Severely affected animals may collapse into lateral recumbency or die within a few hours of ingestion. Less severely affected camelids may show some obtundation and reluctance to stand or eat for a few days, but they do not develop more severe disease. In survivors, within 2 to 3 days, some lactate passes into the lower GI tract, causing convalescent osmotic diarrhea. Polioencephalomalacia-like signs have also been reported.[68]

In the subacute or chronic form, the amount of carbohydrate fed is enough to elicit signs but not enough to induce shock. The common history for this is the poor-doer fed a moderate amount of supplemental grain, which ironically often continues the cycle of ill-thrift. Signs include cycles of

Figure 40-27 Massive first compartment ulceration with acute forestomach acidosis.

weight gain and loss, intermittent diarrhea, and occasional fever.

Diagnosis of all forms is best made by determination of forestomach fluid pH.[37] Normal forestomach pH in camelids is between 6.4 and 6.8. These values tend to climb with anorexia or salivary contamination. Camelids with acute acidosis often have values of 4.0 to 4.5; these may climb to 5.0 or more with time. Camelids with the subacute or chronic form have pH values around 5.5. In general, anything under 6.0 should be considered suspicious.

Metabolic acidosis is the most consistent blood abnormality. Other common biochemical abnormalities include hyperchloremia, hyperlactemia, hyperglycemia, azotemia, and hyperalbuminemia. Stress leukogram is also common, whereas neutropenia and toxic changes provide evidence of mucosal injury.

Treatment follows the guidelines established for ruminants. Correction of dehydration and systemic acidosis is critical, as is prevention of continued forestomach acid production and systemic absorption. Treatment with IV fluids containing bicarbonate based on the calculated deficit and cold oral fluids containing magnesium hydroxide (up to 1.25 mg/kg per dose) usually is adequate, although gastric lavage or gastrotomy may be necessary with severe disease, or if IV fluids are impractical. Nonsteroidal antiinflammatory drugs (NSAIDs; flunixin meglumine, 0.5 to 1.1 mg/kg, IV, q8-24h) may be helpful to treat toxemia and shock. Treatment with antibiotics (penicillin or ceftiofur at standard doses) and thiamine HCl (10 mg/kg, IM or IV, up to q4h) also are recommended to prevent secondary complications. When gastrotomy is not performed, it is possible, but rare, for acidosis and associated clinical signs to recur over the next 72 hours, as further gastric fermentation occurs. Gastric transfaunation 2 to 3 days after the initial episode may help restore a more normal microbial population. Most affected animals survive with treatment. A small percentage become chronic poor-doers with hypoproteinemia and weight loss, most likely due to deep, nonhealing ulceration of the gastric mucosa (Figure 40-28).

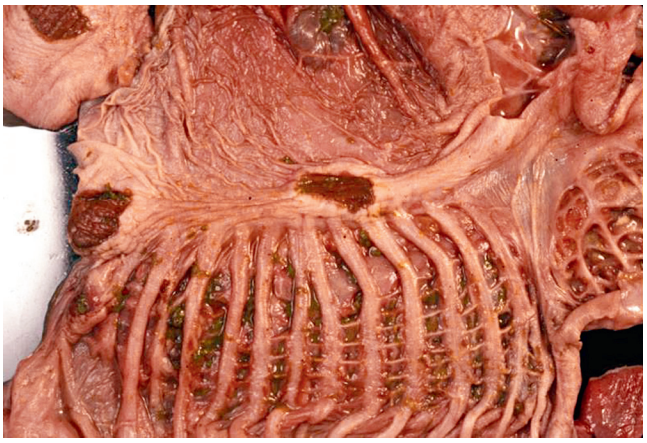

Figure 40-28 Focal, chronic first compartment ulceration.

As with ruminants, dividing grain feedings over the day, making feed changes slowly, providing adequate roughage, and preventing individual animals from overfeeding on grains are useful methods to prevent forestomach acidosis. Additionally, deleting or decreasing the more fermentable grains such as barley and processed corn from the mix has been helpful in prevention in affected herds.

Ulcerative Gastritis

First Compartment Ulcers

First compartment ulcers are a poorly recognized entity, in part because they are near-impossible to recognize before death. Ulcers may be focal, multifocal, or diffuse. They usually occur along the transverse pillar or other raised areas of the squamous mucosa (see Figure 40-28). The etiology is unclear. Previous bouts of forestomach acidosis, ingestion of caustic or abrasive feeds and substances, presence of sand, hair balls, or fiber balls, tumors, or infection by membrane-damaging agents may initiate the lesions, which are then perpetuated by superinfection with fungi such as *Aspergillus* or bacteria such as *Fusobacterium*.

Signs are vague and include poor growth or body condition, anemia, weight loss, or intermittent diarrhea. Blood abnormalities include hypoproteinemia, mild to moderate anemia, toxic changes in the neutrophils, and alterations in the WBC profile, reflective of blood loss, toxemia, or microbial invasion. Ultrasonography of the forestomach may reveal areas of decreased mucosal thickness, but they are often focal and obscured by feed or gas. Radiography may reveal radiodense contents such as sand. Further diagnostics may reveal metastasis of infection to other organs, particularly the liver and lungs. On rare occasions, catastrophic change occurs with gastric rupture. More commonly, a slow decline is followed by a rapid terminal bout of bacteremia and shock. Treatment is usually based on a surmise, as the lesions are not identified before death. If sand is recognized or suspected, psyllium-based feed supplements may be of some value, and serial radiography may be used to assess sand removal.

Antibiotic or antifungal agents may have some value in treating all forestomach ulcerative lesions.

Third Compartment and Duodenal Peptic Ulcers

GI peptic ulceration has long been considered one of the main health concerns in llamas and alpacas, but we actually know little about the incidence or importance of this disorder.[69] In fact, referring to these as "peptic" ulcers is entirely an extrapolation but is useful in differentiating these lesions from GI ulcers thought to be caused by microbes, parasites, or external agents. Various sources estimate the prevalence of GI ulcers in necropsied North American camelids to be approximately 6%, but this includes primary, secondary, and incidental lesions. Interestingly, the occurrence of peptic ulcers appears to be diminishing, possibly because of the "maturation" of the industry and an increased knowledge of feeding and management practices among veterinarians and owners.

In spite of the frequency of diagnosis of ulcers, relatively few scientific reports exist. Most case reports describe camelids with chronic diseases, and it is impossible to determine when in the course of the illness the ulcer developed, and what role it played in disease signs.[70–72] The pathogenesis likewise remains a speculation. The third gastric compartment is long and tubular, running craniocaudal in the right ventral abdomen. Within the first two thirds to 80% of its length, fermentative digestion takes place, and the pH is approximately 6.4. At its caudal extreme, where the organ abruptly turns dorsal and cranial, a small region of hydrochloric acid secretion exists. Peptic ulcers typically occur at this caudal flexure or near the pylorus and into the cranial duodenum, areas where the luminal contents are most acidic or subsequently neutralized (Figure 40-29). Most ulcers occur along the greater curvature. The usual suspects—stress, high grain diets, NSAIDs, and concurrent diseases—have been suggested as possible causes, but most camelids known to have ulcers do not have good evidence of any of these. Through retrospective study of case records, we have identified seven historical factors associated with perforating GI ulcers; it is likely that others exist and that some factors vary in importance with geographic location. These specific factors are (1) frequent harassment by a dominant camelid, (2) recent transport, (3) recent delivery of a cria (this may relate to transport, as this was most frequently seen in camelids that delivered on one property and were subsequently moved to another), (4) grazing lush spring pasture, (5) enteritis or septicemia, (6) GI foreign body, and (7) bowel ischemia. The last three factors were associated with focal ulceration and perforation at any point along the gastrointestinal tract while the first three were associated with third compartment or proximal duodenal ulceration. In addition to these factors, forestomach acidosis may cause nonperforating, but severe, ulceration in the forestomach and mild ulceration in the third gastric compartment. GI tumors are associated with focal nonperforating ulceration at any point in the GI tract, and copper poisoning may cause massive, diffuse gastric ulceration. Factors associated with nonfatal, nonperforating ulcers are unknown, but it should be noted that chronic disease conditions, hospital stays, and long-term and short-term treatment with NSAIDs were not identified as risk factors for perforating ulcers.

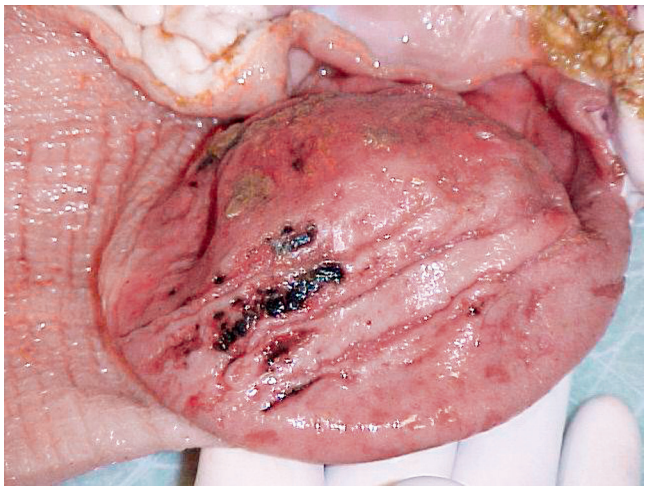

Figure 40-29 Linear ulcerations in the gastric portion of the third compartment.

One study suggested that the specific mechanism leading to ulcers is lack of progressive gastric or duodenal motility.[73] Motility in that area is peristaltic. In normal camelids, fermented ingesta frequently move into the fundic region, diluting the hydrochloric acid, and the mixture is regularly expelled into the pyloric region and duodenum, where the acids should be neutralized. With poor progressive motility, the acid contents remain unneutralized and possibly damage the mucosa. Most of the identified risk factors such as anorexia or intermittent eating (as may occur with sick, transported, or bullied camelids), dehydration, electrolyte or acid-base abnormalities, and gastric hyperacidity (feeding too much grain, or even overindulgence on lush spring grass) could plausibly be linked to poor gastric emptying. Reflux of bile from the duodenum could exacerbate acid injury or may simply provide a marker for lack of progressive outflow.

Low mucosal blood flow may contribute by adversely affecting mucosal health and healing. Camelids are very good at masking and tolerating dehydration, so these abnormalities may not be detected during the period of ulcer formation. Many of the same risk factors that are suggested to be inhibitors of gastric emptying are also likely to decrease blood flow.

Ulcers may be single or multiple, pinprick or extensive, superficial or deep, and possibly perforating (Figure 40-30). Approximately half the total cases of dead camelids had a perforation, but the prevalence of nonperforating ulcers is probably larger in the live population. Clinical presentation relates to the nature of the lesion. Nonperforating ulcers are usually blamed for causing anemia, depression, inappetence, colic, sensitivity to right cranioventral abdominal palpation, recumbency, and death in camelids of all ages, as in the clinical disorders associated with these lesions in horses, cattle, and humans. However, the reality is that most nonperforating ulcers are clinically silent, even when extensive. Camelids show those particular signs with a multitude of disorders (or in complete health in the case of resenting palpation) and rarely show any signs, except perhaps mild anemia, with ulceration. Chronic ulceration, sometimes with replacement of the entire acid-secreting mucosa by granulation tissue, is an exception, often leading to weeks or months of progressive ill-thrift. Fecal occult blood tests are not a reliable method of diagnosing nonperforating gastric ulcers, and melena is more common

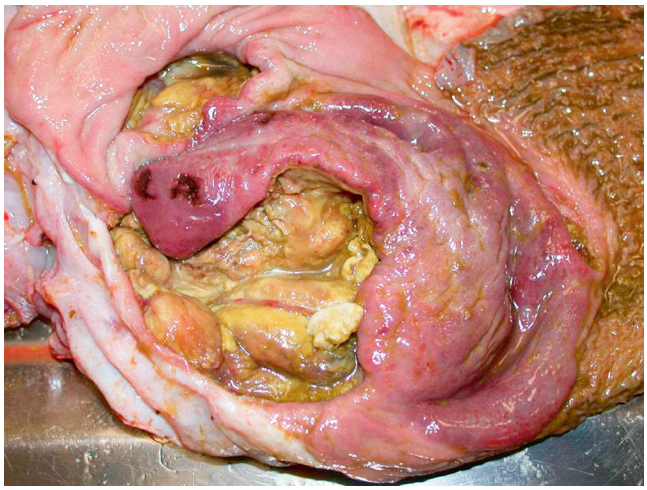

Figure 40-30 A ruptured third compartment ulcer and surrounding peritonitis.

with gastric masses or massive ulceration caused by copper toxicosis. Thus, it is difficult to identify nonperforating ulcers, and their frequency is unknown.

Although we are uncertain of the significance of superficial ulcers, the importance of perforations cannot be doubted. Peritonitis and ulcers as separate diagnoses accounted for a total of between 9% and one third of camelid deaths according to two surveys conducted by the International Lama Association. Rupture of gastric, duodenal, or colonic ulcers in camelids confers a grave prognosis, with most camelids showing anorexia, severe depression, increased recumbency, and signs of septic shock. Fecal production and gastric motility are greatly reduced. Owners rarely report having seen active colic in the days preceding this acute decline, but colic signs are occasionally seen after the perforation has occurred. In a small minority of cases, affected camelids are chronic poor-doers for months before perforation, but most are outwardly completely healthy until perforation. Blood and peritoneal fluid may provide evidence of septic inflammation, but these changes often normalize with time, particularly with localized peritonitis. Clinical changes may also largely normalize and cause more of a chronic ill-thrift syndrome, although appetite rarely returns to normal.

Diagnosis of nonperforating ulcers remains speculative. To date, the affected regions are not accessible by endoscopy, ultrasonography is rarely sensitive enough, and blood work changes are nonspecific. Anemia and hypoproteinemia may be seen but are common findings in many sick camelids (a substantial number of which have unfortunately died over the years and been confirmed not to have had ulcers). Various fecal occult blood methods have been evaluated anecdotally, with none providing consistent, helpful results. The same is true for serum pepsinogen determination. Abdominal fluid is usually unremarkable. Feces are usually normal, albeit possibly scant. Melena is extremely uncommon and more often linked to a tumor or copper poisoning than simple ulceration, with or without a perforation.

All of these findings may also be true for perforating ulcers, especially if abdominal contamination is confined to the omental bursa. Clinical disease is more apparent, but the diagnostic tests may be frustratingly unhelpful. Complete blood cell count (CBC) may reveal toxic changes and immature forms of neutrophils. Abdominal fluid may reveal bacteria or fungi, high or low cell counts, high protein content, and toxic changes. Ultrasonography may reveal thickening of the omentum, flocculation of fluid and pockets of fluid; the caveat is that fluid accumulations are often fairly normal fluid around the lesion, rather than abnormal fluid from the actual lesion. Exploratory surgery or postmortem examination may reveal overt contamination and the hole in the GI tract. Except for surgery, however, all of these tests may also yield normal or equivocal results.

Treatment of nonperforating ulcers is controversial, particularly because the diagnosis is usually speculative. Camelids showing the signs popularly ascribed to ulcers may, in fact, have other diseases, many of the putative risk factors for ulcers have not been substantiated by broad clinical experiences, and many of the camelids with ulcers show no outward signs of illness, calling into question which population to treat. Several common medications have been shown to be ineffective at reducing gastric acidity at conventional dosages. These include cimetidine (up to 25 mg/kg, intramuscularly [IM]), ranitidine (1.5 mg/kg, IV), oral omeprazole (up to 12 mg/kg), and intravenous omeprazole at 0.2 mg/kg.[69,74–77] Effective treatments have included high doses of intravenous omeprazole (4 to 8 mg/kg) and pantoprazole (1 mg/kg, IV or up to 2 mg/kg, SC).[75,77] It is speculated that H_2-blockers are effective at extremely high intravenous doses, but this has not been substantiated scientifically, and oral forms of these agents are likely to face severe impediments to systemic absorption. Misoprostol is also effective but has a high rate of unacceptable adverse reactions and therefore cannot be recommended.[75] Oral sucralfate, whose efficacy is difficult to establish because it is not absorbed systemically and does not affect gastric acidity, has also been used (20 to 40 mg/kg, PO, q6-8h). In spite of these data and the problems with diagnosis, anecdotal reports of improvement after use of the "ineffective" agents are common.

Although specific pharmaceutical treatment of nonperforating ulcers has been fairly unrewarding—it is difficult to identify which camelids to treat and most antiulcer medications are poorly efficacious in camelids—overall good medical treatment may substantially contribute to ulcer prophylaxis. On the basis of the identified risk factors and putative pathogenesis, correction of underlying diseases, maintenance of good mucosal blood flow and gastric emptying, and avoiding causes of stress or anorexia are probably the best way to avoid and treat nonperforating gastric ulcers. Palatable, forage-based rations may be prokinetic, epitheliotropic, and nonacidogenic. Fluids should support mucosal health and good GI function. Analgesic medications may increase appetite and decrease pain-mediated ileus. Effective management, with readily available food and water and a noncompetitive environment, is the cornerstone. Philosophically, the practitioner needs to decide whether all sick or at-risk (bullied, new introductions, transported, competing breeding males) camelids need preemptive intervention or if the lack of effective treatments or infrequency of clinical progression (most perforations occur in outwardly healthy camelids, not poor-doers) precludes the use of specific antiulcer treatments. Following the principles of promoting healthy gastric emptying suitably

addresses both circumstances. Prophylactic and therapeutic goals of restoring or maintaining perfusion; acid–base, mineral, and electrolyte homeostasis; and physical stimulation of progressive gastric motility through the regular provision of good-quality roughage that the camelid can eat comfortably and throughout the day appear to minimize the chance of perforation and perhaps the prevalence of nonperforating ulceration.

Treatment after perforation is usually ineffective. Camelids do not always die acutely; many are able to surround the lesion with omentum, fibrin, and adhered loops of bowel, but these camelids do not do well in the long term. Partial gastrectomy and local resection of contaminated tissue may be of benefit, if the damage is narrowly contained. Medical treatments include fluids, antibiotics, antiinflammatory or analgesic medications, and appetite stimulants. The use of specific antiulcer medications is probably no longer pertinent, although inhibition of acid production may allow small lesions to heal. Overall, the prognosis for camelids with perforated ulcers is grave. As ulcers have been seen occasionally in small outbreaks, if one camelid in a herd develops a perforation, the others should be scrutinized for risk and treated accordingly.

Neoplasia

Squamous cell carcinoma is the most commonly reported primary neoplasm affecting the gastrointestinal tract of camelids, while multicentric lymphoma is the most commonly reported neoplasm in camelids overall.[78–80] Internal squamous cell carcinoma usually affects the first compartment (Figure 40-31), and most affected camelids are adults, many older than 10 years. Lymphoma more commonly affects the caudal end of the third compartment (Figure 40-32) but may also affect the first or second compartment and appears to be common in all ages of camelid. Other neoplasms, particularly carcinomas of glandular tissue and sarcomas of smooth muscle origin, occur sporadically and also tend to occur in older adults. In general, neoplastic disorders cause gradual weight loss with progressive anemia, hypoproteinemia, lethargy, and anorexia. Melena is occasionally seen when gastric ulceration is severe. Diarrhea might be noted early in the course of the disease or terminally. Lymphoma may cause palpable masses on other body parts; it is covered more extensively in Chapter 36. Often, the early clinical signs are missed, and the llama is either found dead or extremely debilitated. Antemortem diagnosis and successful treatment of malignant internal neoplasms are rare but could be improved by earlier identification of the sick animal, greater awareness of the tumors, and a diagnostic workup directed toward neoplastic disorders.

Clostridial Gastroenteritis or Enterotoxemia

Clostridium perfringens infection has been associated with fatal third compartment gastritis and enteritis in neonatal and adult camelids. As with some other clostridial diseases, the question remains whether *Clostridia* are primary pathogens or secondary invaders that cause easily recognizable and usually lethal disease. Clostridial organisms are gram-positive, motile, spore-forming, facultatively anaerobic rods. They may be present in low numbers in the gut contents of healthy animals and proliferate under certain conditions. High fermentable carbohydrate in gastric contents and slow gut transit are generally thought to promote clostridial overgrowth. Clostridial organisms are also toxin producers, and slowed transit or organism overgrowth may allow these toxins to reach pathogenic concentrations. Other diseases may facilitate clostridial disease by slowing gut transit, changing the gut microbial population, or damaging the mucosa to allow for clostridial invasion and greater systemic absorption of the toxins. Specifically, peptic ulcers or gastric nematodes are thought to facilitate gastric disease, and nematodes, coronavirus, *Giardia*, and coccidia are thought to facilitate intestinal disease.[81,82]

In both North America and South America, clostridial gastroenteritis has been associated with herd outbreaks of death. These tend to be associated with damp weather, which also tends to be most conducive to the proliferation of nematode and protozoal pathogens. Crias are generally considered to be more susceptible compared with adults, and outbreaks in South America are associated with a death rate of up to 70% of newborn crias in bad years, but adults are also occasionally affected.[83,84]

Several types of *C. perfringens* exist. These were traditionally defined by the toxins secreted but now are typically defined by the toxin genes present. Type A is best known for its α-toxin, whereas type C additionally secretes β-toxin. ε-toxin is the main effector of type D. Type A is usually blamed for

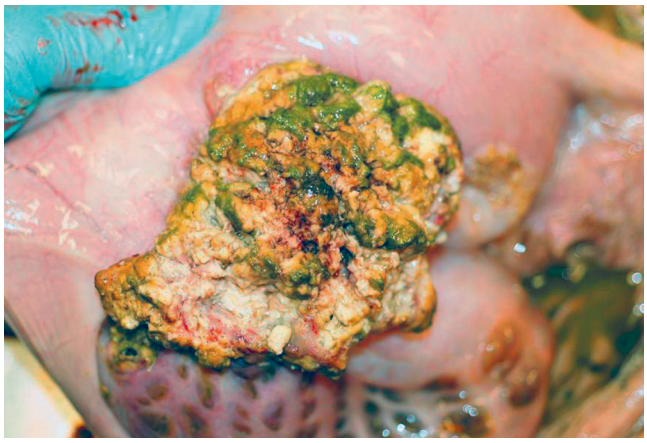

Figure 40-31 Squamous cell carcinoma of the first compartment in a llama.

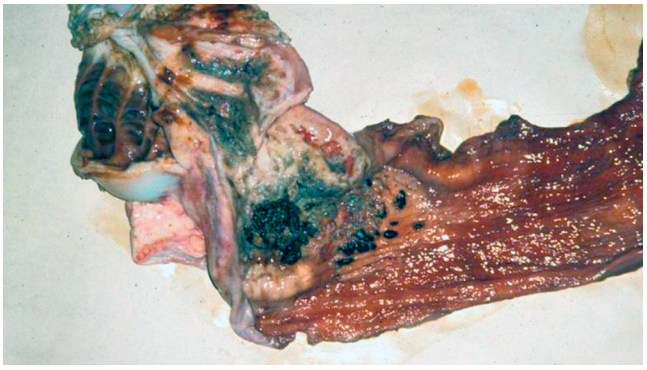

Figure 40-32 Lymphoma of the caudal flexure of the third compartment. Note the deep ulceration leading to melena.

neonatal enteritis. This is supported by one Peruvian study, in which all *C. perfringens* isolates from crias suspected of dying from enterotoxemia had the α-toxin gene and extremely few had the genes for toxins typical of types B, C, D, or E.[85] In Oregon, type C also appears to play a role.

Types C and D cause diseases resembling enterotoxemia in young ruminants. β-toxin is inactivated by trypsin and thus usually has a narrow window in which to affect the neonate. Type D typically affects animals older than 3 days. Newer research has also shown that some toxins important for disease may not be included among those used to classify the types, such as the β-2 toxin.[85,86] In contrast, enterotoxin appears not to play much of a role.

Clostridial toxins cause local necrosis, which may become generalized with systemic absorption. The infection itself may also cause hemorrhage, necrosis, and gas gangrene of the gastric or intestinal wall. Clinical signs include severe depression, shock signs, colic, abdominal distension, possibly fever, and fetid or hemorrhagic diarrhea. Diarrhea is usually seen only if the camelid survives at least 12 hours after the onset of clinical signs. Signs are often peracute and severe, but with clostridial disease superimposed on some other preceding digestive disorder such as forestomach acidosis, coronavirus infection, and GI parasitism, signs of that other disease may have been present for days to weeks before the rapid worsening. Some camelids die suddenly, others within hours of the onset of severe signs, and still others within days. Survivors are the minority in spite of aggressive treatment.

Premortem diagnosis is usually presumptive. Fecal toxin assays developed for use in other species are not always accurate, and rapid assays do not exist for many of the important toxins. Fecal culture followed by toxin or genetic analysis is possible but usually too time consuming to have any impact on treatment, except in herd outbreaks. Radiography of the abdomen may reveal gas-distended loops of bowel. Ultrasonography may reveal gas-distended viscera and possibly thickening and gas shadowing of the gastric or intestinal wall.

Hematologic abnormalities include neutrophilia in most cases and neutropenia in some. Lymphopenia, anemia, toxic changes, and immature neutrophils are often present. Hyperfibrinonemia may develop if the camelid survives more than a day. Biochemical abnormalities include hyperglycemia, metabolic acidosis with hyperlactemia, often hypoproteinemia, and potentially other indicators of shock. Anemia, hypoproteinemia, and electrolyte changes may be present because of the preceding disease.

Postmortem examination tends to yield impressive lesions. The mucosa of the third compartment is thickened, necrotic, hemorrhagic, edematous, and emphysematous. Small intestine might also be affected, and ingesta may be hemorrhagic. Body cavity fluids tend to be blood tinged. Differentiating premortem changes from terminal or postmortem changes may be difficult. *Clostridia* tend to overgrow with lack of GI motility, particularly after death. Significant hemorrhage is usually good evidence that changes occurred after death. Culturing *C. perfringens* from lesions and demonstrating the presence of toxin support the diagnosis.

Identifying the disease in the individual camelid is less important than recognizing the need for aggressive treatment. IV fluids, antiinflammatory drugs (flunixin meglumine; 0.5 to 1 mg/kg, IV, q12h), and antibiotics are essential. Although *Clostridia* themselves are best treated with agents such as penicillin, ampicillin, or ceftiofur, the uncertainty of the diagnosis and the likelihood of gut damage dictates broad-spectrum antimicrobial coverage. Penicillin (44,000 units/kg with q6h IV, or q12h SQ or IM) and ceftiofur (4 to 8 mg/kg IV, SQ, or IM, q8-12h) are typically used at high doses, twice the usual amount. Oral penicillin (22,000 units/kg, PO, once or q12h) may have some value as an adjunct to decrease gastric overgrowth. Affected camelids may also require plasma transfusion. Antisera against clostridial toxins may have some value, if type C is involved but may not counteract other important toxins. Use of commercial toxoid vaccines is likewise only partially effective unless the vaccines contain specific alpha toxoid.[87] Autogenous vaccines made during outbreaks may be more effective, although they also might not protect against all causative toxins.

Gastric Impaction

Gastric impaction is relatively uncommon but can be frustrating to diagnose when it occurs. Either the first or third compartment may be affected. The impaction may be principally sand, plant fiber, or hair fiber, with hair impactions being more common in the young. Clinical signs develop because of the slow distension of the stomach and progress over several weeks to signs of obstruction. First compartment impactions tend to cause progressive left-sided abdominal distension, loss of appetite, weight loss, and general malaise. Mild colic may be an occasional feature. When the stomach fills to the level of the lower esophageal sphincter, abnormal regurgitation and eructation begin, and the camelid may develop bloat. Gastric motility is diminished, although some degree of normal fecal passage is usually retained. Sand tends to be more abrasive, leading to more CBC abnormalities, intermittent fever, and deeper bouts of depression. Without resolution, the stomach may eventually rupture, or the camelid may develop sepsis or aspiration pneumonia.

Third compartment impaction tends to have a more acute onset. Abdominal distension is milder and more liquid, and the depression is partially caused by the dehydration, alkalosis, and hypochloremia associated with gastric reflux. Appetite and fecal production diminish over 2 to 3 days. Colic may be more evident with this than with first compartment impaction.

In younger camelids, radiography may reveal evidence of the inappropriate feed-filled mass, but this is harder to determine in adults. Sand impaction is more visible radiographically than in the other type, and contrast material may be used to enhance visualization of less radiopaque masses. Ultrasonography may reveal an unusual intraluminal echogenicity, but this is especially difficult to judge in the first compartment, where the hyperechoic mucosa–gas interface tends to obscure all deeper detail. Exploratory surgery has been most revealing, especially for third compartment impaction. Unless the first compartment is palpably enlarged, appreciation of greater fibrosity may be subjective. Emptiness of the aborad intestinal tract with concurrent gastric fibrous distension may provide some indirect evidence of impaction but also may reflect poor forestomach function in general.

If the impaction can be appreciated on exploratory surgery, gastrotomy may be performed to remove the mass. With first

compartment impaction a ventral midline approach is preferred, although a left paralumbar approach may also provide acceptable exposure. With third compartment impaction, a right paracostal approach is recommended for best exposure. The mucosa should be examined at the same time for ulcerations. Postoperative care includes antibiotic therapy and feeding small amounts of easily digestible feeds. Alfalfa leaves or pellets, blackberry leaves, and fresh grass are all nutritious, palatable, and low-residue feeds. Oral agents such as bismuth subsalicylate, sucralfate, or kaolin-pectate may be helpful if the mucosa is irritated or ulcerated.

Parasitic Gastroenteritis

A variety of parasites affect the GI tract of New World camelids. Some of these are unique to camelids, but many also infest or infect ruminants, other domestic animals, cervids, or other wildlife species as well. In areas where camelids are kept in relative isolation, the camelid-specific parasites may predominate, but where camelids have regular contact with cervids or ruminants, the shared parasites often become more important. Thus, parasite control often must go beyond detection and treatment of parasites in the camelid herd. As a rule, parasitic infections are more associated with ill-thrift than more specific and overt signs of GI disease such as diarrhea or colic, but as such, they are among the most common causes of poor-doing in domestic camelids. In spite of this, there are strikingly few scientific reports of disease caused by most of these parasites.

Strongyles

A number of important nematode parasites primarily affect the peptic region of the third compartment (Figure 40-33). The major ones are *Haemonchus, Ostertagia, Teladorsagia, Trichostrongylus, Camelostrongylus,* and *Marshallagia*. These cause disease in camelids much as they do in domestic ruminants, that is, by erosion and ulceration of the gastric mucosa, and by destruction of the gastric pits. Infection starts when third-stage (L3) larvae are ingested and burrow into the gastric mucosa. They shed their cuticle to molt into L4 larvae within a few days and begin to feed on blood (mainly *Haemonchus*) or proteinaceous exudates. Maturation into adults may occur within 3 to 4 weeks or be delayed for several months (hypobiosis, arrested development); that is, the prepatent period, and potentially prepatent disease, may extend over several months. With most of these parasites, two main types of disease exist: (1) type 1 infestation, which is caused by larvae and adult worms soon after ingestion of large numbers of infective larvae, and (2) type 2 disease, which occurs upon massive resumption of growth of arrested L4 larvae. Although type 2 disease is the focus of the scant reports, both types are seen in clinical practice.[88,89]

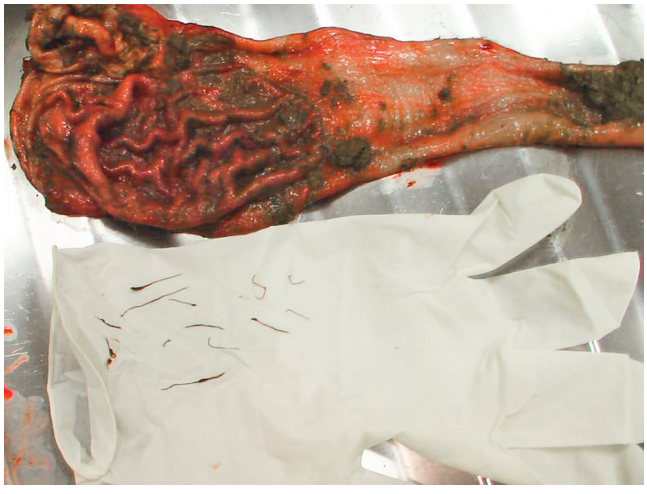

Figure 40-33 Parasitic gastritis and the causative worms.

The life cycle for most GI nematodes is completed when adult females release eggs, which pass with feces to hatch out on pasture. Larvae on pasture undergo two molts until they become infective L3 larvae. These are susceptible to extreme temperatures, starvation, and desiccation, so they gather in water droplets on blades of grass or leafy forage, where they are eaten by the next host.

Haemonchus, or the barber pole worm, is generally thought to be the most pathogenic of the gastric nematodes because it feeds on blood. The L4 larvae and adults consume approximately 0.05 mL blood per worm per day. With a typical alpaca blood volume of around 3.5 L, each worm consumes 0.0014% of blood per day, and 1000 worms consume 1.4% of blood per day. Blood feeding commences approximately 11 days after ingestion. The name "barber pole" comes from the double helix of the red, blood-filled intestine and the white, egg-filled ovaries of the adult female worm.

A number of other nematodes affect the intestinal tract. The major small intestinal strongyles include *Cooperia, Nematodirus, Trichostrongylus, Strongyloides,* and *Lamanema chavezi*. The major large intestinal strongyle is *Oesophagostomum*, which is found infrequently. Of the intestinal strongyles, *Lamanema* is reported to be the most pathogenic in South America.[90] It is considered rare outside of South America but recently has been associated with disease in New Zealand and may be important in North America as well.[91] For the most part, these worms undergo a similar life cycle as those of gastric worms except that they damage the intestinal mucosa instead of the stomach. In addition, *Lamanema* L3 and L4 larvae undergo extensive enterohepatic migration over 2 to 4 weeks and may thus also cause damage to the liver and occasionally the lungs.[90,92] Viscachas (South American rodents in the family Chinchillidae) are thought to be the normal host.[93] In the United States, *Nematodirus battus* has been associated with severe disease.

The life cycle of *Strongyloides* may be complicated. Rhabditiform larvae or larvated eggs are passed in feces. These larvae mature on pasture to become free-living, reproductive adults. Alternatively, rhabditiform larvae mature within the gut to filariform larvae to reinitiate infection through the gut wall (internal autoinfection) or penetrate the perirectal skin (external autoinfection). Last, rhabditiform larvae, either from feces or from the eggs of free-living adults, may mature to filariform larvae in the environment and penetrate the skin of another host. Filariform larvae, regardless of the route of infection, migrate into the lungs, where they are coughed up, swallowed, and settle in the intestine. There, they mature to adult female worms.

Non-strongyle worms affecting the GI tract include *Capillaria (Aonchotheca), Trichuris* (whipworms), and *Moniezia* (tapeworms, non-nematodes). *Trichuris* particularly appears to

be clinically important. Whipworms affect the colon, whereas most of the other worms affect the third compartment or small intestine. By affecting the colon, *Trichuris* is more likely to cause diarrhea and straining than the other worms. It is also a blood-feeder, so it is more likely to cause moderate anemia, although still unlikely to cause the single-digit packed cell volume (PCV) results seen with *Haemonchus*.

The life cycle of most camelid non-strongyle-type worms has not been specifically determined. Considering other *Trichuris* spp., it is likely that whipworm infection occurs after ingestion of larvated eggs and that eggs require several weeks to months in the environment to larvate. Larvated eggs may survive for several years in the soil. Hosts also typically shed embryonated *Capillaria* eggs in feces and ingest larvated eggs. Both *Trichuris* and *Capillaria* have lengthy prepatent periods in ruminants and may cause prepatent disease in camelids. *Capillaria* is known to be an intermittent shedder. Low egg counts may be found in heavily parasitized camelids.

With *Moniezia*, eggs or segments containing eggs are passed in feces. Eggs are then eaten by soil mites, in which they develop into infective cysticercoids. Camelid (or livestock) hosts eat the infected mites, and the cysticercoids mature into adults in the small intestine.

Except for *Haemonchus* and *Trichuris*, the main signs of infection by GI nematodes are poor weight gain or ill-thrift. These signs may progress to weakness, lethargy, anorexia, increased time spent in the recumbent position, and eventually signs of sepsis and a comatose state. Diarrhea is uncommon, except with *Trichuris*. Panhypoproteinemia is common, and other evidence of shock or sepsis will develop as disease severity increases. Mild anemia is common, with *Trichuris* and *Haemonchus* specifically causing moderate to severe anemia as well. In addition to standard measurement of PCV or RBC count, and physical evidence of mucous membrane pallor, weakness, and tachycardia, subjective indices, such as FAMACHA scoring have been tested in camelids.[94] This involves comparing conjunctival color to standards on a card, and using the result to estimate anemia. In spite of overt blood or protein loss, overt peripheral edema is rare; abdominal distention and increased respiratory effort provide more reliable evidence of hypoproteinemia. Analysis of peritoneal or pleural fluid reveals that these are transudates.

Protozoa

Awareness of the importance of protozoal enteritis has been growing steadily. This is reflected both in the number of scientific publications, and the overall recognition that parasite control strategies must extend beyond anthelmintics. Also, once considered diseases of crias, certain protozoal enteritides are now widely recognized as important disorders of all ages of camelids.

Giardia—A flagellate parasite. *Giardia* infection primarily occurs from contaminated water sources, where oocysts can survive about 3 months in water at 4°C. The types (assemblages) of *Giardia* affecting camelids appear to have host ranges involving mainly hoofstock in some cases, or more generally infective in others.[95–97] Thus, the zoonotic and cross-species potential varies from one situation to another. *Giardia* seems to thrive on certain farms, probably related to local conditions.[98] Such farms typically have outbreaks during their wet season, or after a heavy rain, particularly during times of year that young crias are present. This highlights that contaminated puddles and standing water may serve as temporary reservoirs, not just permanent water courses. Filtering drinking water may reduce exposure in some camelids, but has limited impact if they have access to contaminated pasture.

Ingested *Giardia* cysts release 2 trophozoites each after exposure to stomach acid. These attach to small intestinal (usually duodenal) mucosal cells and reproduce asexually by binary fission. Rounds of replication recur until inhibited by the host response, each time potentially doubling the number of trophozoites. Some trophozoites eventually encyst, and both cysts and trophozoites are passed in the feces. Only cysts have the potential for becoming infective to other animals. They are infective at passage or soon thereafter, so direct transmission from infected feces is possible.

Unlike Eimeria, *Giardia* remain extracellular. There is reactive intestinal inflammation and villus atrophy, and cell function is impaired, but the mucosa remains essentially intact. Therefore *Giardia* feed off intestinal contents, particularly glucose, not exudates. Clinical signs include diarrhea, weight loss, occasionally colic, passage of gas, or steatorrhea, and in the worst cases, general progression toward death. The incubation period for disease is approximately 5 days, but has lasted as long as 21 days in humans. With longer incubation periods, the disease can be seen 7 to 10 days before patency. To date, *Giardia* is primarily a clinical problem in crias up to about 7 months of age,[98–100] potentially because of the dearth of intestinal glucose once fermentative digestion has matured. It was identified in 18% of 45 cases of diarrhea in unweaned llama and alpaca crias in Pacific Northwest[97] and in 33% of 58 hospitalized crias in Ohio.[101] It has also been identified in the feces of non-clinical older camelids, but usually only in low concentrations.[96] Risk factors for oocyst shedding included having more than 10 yearlings on the property, smaller pen sizes, and large unit sizes of more than 20 animals. Prevalence of shedding may be underestimated, because diarrhea is not always found in camelids with severe giardiasis, and it is feasible that adults with ill-thrift may have covert *Giardia* infections.

Cryptosporidium—Cryptosporidium spp. are small zoonotic protozoal pathogens that can cause severe and sometimes fatal outbreaks of diarrhea in neonates as well as immunocompromised older animals. *Cryptosporidium parvum* and *ubiquitum* affect camelids and several other species, including man, and transmission from alpacas to humans has been documented.[102] *Cryptosporidium parvum* has been investigated most extensively, and is believed to be responsible for most mammalian disease.[103] It was found by fecal immunofluorescent assay in 9% of cria diarrhea cases in the Northwest U.S.[98] Another US-based study using an immunocard assay found infection in 25.9% of crias with diarrhea.[101] In the UK, *Cryptosporidium* was found in 8.8% of cria diarrhea samples,[104] and in Peru, 13% of healthy crias up to 15 days old were found to be infected by Ziehl-Neelsen modified acid-fast staining, with the highest prevalence in crias 12 to 15 days old.[105] *Cryptosporidium* may be a secondary pathogen, but one recent study found no other gastrointestinal tract pathogens in 13/20 alpaca crias diagnosed with cryptosporidiosis, suggesting a role as a primary pathogen.[106]

After ingestion of a sporulated cyst, four sporozoites are released in the small intestine. These attach to the microvillar

membrane, which engulfs them. They remain at this margin of the cell, and are referred to as intracellular but extracytoplasmic. Within this vacuole, the organism is shielded from both the gut lumen environment and intracellular defenses, and presumably from many chemotherapeutic agents. *Cryptosporidium* also develops a unique feeder organelle membrane on the inner side of the vacuole to facilitate nutrient uptake from the host cell. They mature to trophozoites, and multiply asexually (schizogony). These new forms (merozoites) are released into the gut lumen, where some of them reattach to epithelial cells and repeat the cycle of multiplication. Others, upon entry into the brush border of a new epithelial cell, mature into Type II meronts, which release Type II merozoites. These invade new cells and mature into the male (microgamont) and female (macrogamont) forms. Microgametes from the microgamonts are released to penetrate macrogamonts, effecting non-multiplicative, sexual reproduction. Fertilized zygotes mature to oocysts. *Cryptosporidium* is relatively unique among coccidia, in that 20% of oocyst are thin-walled and likely to rupture within the GI tract, and that thick-walled oocysts are usually sporulated, and hence infective, immediately on passage. Rupture of thin-walled oocysts releases a new round of sporozoites and starts the process of infection over. This can repeat until inhibited by the host immune response.

Over the course of infection, *Cryptosporidium* tends to move from the upper small intestine to the jejunum, ileum, cecum, and proximal ascending colon. Although it is not as overtly damaging to epithelial cells as larger coccidia, cell function is certainly disturbed, potentially to the point of cell death. Additionally, the inflammatory immune response is likely to further cell damage, increase cell turnover, and promote secretion over absorption. *Cryptosporidium* has also been found in the biliary, renal, and respiratory epithelium of some immunocompromised patients of other species.

Cryptosporidiosis is primarily considered a disorder of crias <14 days old, but has been seen in older crias, and occasionally adult camelids as well.[102,104,106–108] Recently, it has been implicated in several herd outbreaks of cria diarrhea around the U.S,[102,104] with clinical disease ranging from mild to lethal. Adult herdmates occasionally develop diarrhea at the same time as the cria outbreaks, and may at that time be found to be positive on fecal examination; isolated adult cases have been seen as well. Hence, adults must be considered a possible, if minor, source of infection. Larger herds with a large number of births close together are most likely to have outbreaks of cryptosporidiosis, presumably due to passage between camelids. In smaller herds with sporadic cases, other infected species may by introducing the parasite. The fecal material at presentation varies in consistency from watery to pasty, and in color from white, yellow, or green, to tan or green if crias were old enough to be grazing. Other clinical signs of affected crias include reduced fecal production, lethargy, weakness, reduced gastrointestinal sounds, failure to thrive, weight loss to emaciation, and abdominal pain. Relapses often occur after 4 to 7 days of improvement. These are due to autoinfection, a reinitiation of infection by thin-walled cysts in the gut. In crias with diarrhea, common clinicopathologic abnormalities include acidemia, hyperlactemia, azotemia, and hyperglycemia and high aspartate transaminase and gamma-glutamyltransferase activities.[104] It is interesting to note that whereas evidence of sodium and chloride loss are common in calves with clinical cryptosporidiosis, they are inconsistent findings in crias, emphasizing the need for a good diagnostic evaluation before initiating treatment, when possible.

Effective specific treatment for cryptosporidiosis remains elusive. The mainstay of therapy involves supportive care with intravenous fluids and possibly parenteral nutrition due to the malabsorption and maldigestion which occurs with this disease. Plasma may be necessary as well. In one recent case series, 20 alpaca crias with cryptosporidiosis generally exhibited clinical signs for at least 7 days, but the infections were mostly self-limiting; good supportive care resulted in a successful outcome in 16 crias.[104] Supportive care included partial parenteral nutrition in 19 of the 20 crias. Weight loss and refractory azotemia were associated with unsuccessful treatment.

Eimeria—Larger coccidian parasites. New World camelids appear to be susceptible to at least 5 species. Coccidia appear to be camelid-specific, unlike many worms, *Giardia*, and *Cryptosporidium*. Thus, transmission from ruminants or wildlife is not thought to be important. These 5 species of coccidia do, however, appear to affect all species of New World camelid, and whether they also affect Old World camels is an open question. Whereas coccidiosis is primarily thought to be a disease of the young in many species, and is well publicized as a cause of illness and death in crias in South America, it is becoming more commonly recognized worldwide as a cause of adult morbidity and mortality as well.

With *Eimeria* and the other gastrointestinal protozoa, severity of infection relates to the ingested dose, the pathogenicity of the parasite, and the ability of the host immune response to limit the parasite's reproductive success. High concentrations of naïve hosts can lead to rapid environmental build-up of infective oocysts; overcrowding, environmental fecal contamination, and eating off the ground contribute to risk. Pathogenicity may relate to size: *E. lamae* is reported to be the most damaging small coccidia,[109] and there is increasing evidence that *E. macusaniensis* is the most damaging overall (Figure 40-34). The propensity for neonatal or juvenile disease may relate to the parasite's ability to survive the functional forestomach and the host's immune response. In environments where these parasites are common, early exposure is essentially guaranteed, and continued reinfection likely promotes a vigorous immune response in survivors.[110,111] In those environments, disease is most common in the naïve crias, which may shed pass-through oocysts within 5 days of birth. In cleaner environments, level of exposure is less certain, and some camelids may remain naïve and susceptible into adulthood. In all cases, overwhelming exposure, often facilitated in individual animals (newcomers to a herd) eating off the ground, or compromise of the immune response may allow disease, even in older animals.

Camelid coccidia can be lumped into categories of small and large. The small coccidia are relatively conventional in appearance and life cycle, and will be familiar to anyone acquainted with coccidial disease in domestic poultry or ruminants. The large coccidia of camelids are relatively unique. The small coccidia include, by increasing size of oocysts, *Eimeria punoensis* (17 to 22μ in length), *E. alpacae* (22 to 26μ), and *E. lamae* (30 to 40μ).[112] Oocysts become more ovoid as the species gets larger, so that *E. punoensis* is about 16% longer

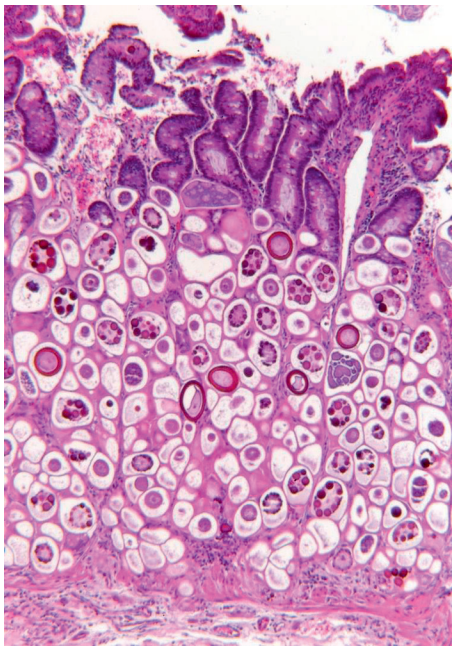

Figure 40-34 Ileal mucosa of an adult alpaca obliterated by *Eimeria macusaniensis* at various stages of development.

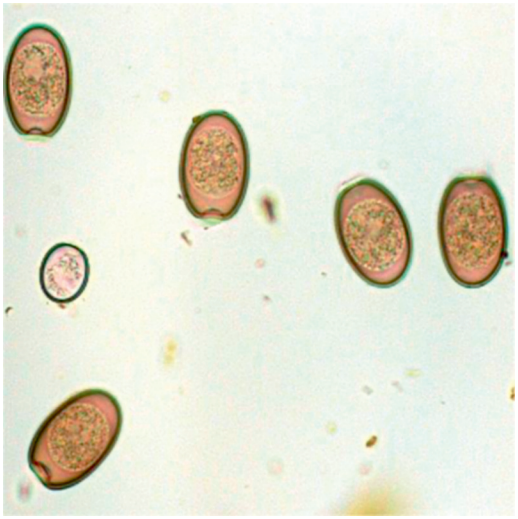

Figure 40-35 Microscopic appearance of *Eimeria punoensis* (single round oocyst) and several *E. lamae*.

than it is wide without a clearly visible polar cap (by standard light microscopy), whereas *E. lamae* is about 60% longer than it is wide and has an obvious cap (Figure 40-35). These follow the same general lifecycle as *Cryptosporidium* with a few notable exceptions. *Eimeria* oocysts are not thin-walled and are not capable of autoinfection. They do not sporulate and become infective until they have spent 4 to 12 days (for *E. lamae*) or more outside of the host.[111,113] After ingestion, sporulated oocysts usually release 8 sporozoites, which penetrate epithelial cells. The host cell nucleus and organelles are marginalized and the cell ruptures with maturation of each parasitic stage. Thus, mucosal loss can be widespread, particularly during the early, multiplicative stages of infection. The prepatent periods are approximately 10 days for *E. punoensis*,[114] 15–16 days for *E. lamae*,[109] and 16–18 days for *E. alpacae*.[114]

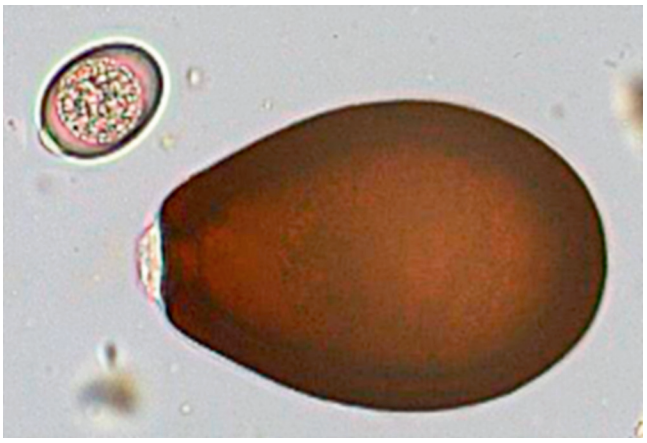

Figure 40-36 Size comparison of the smaller *Eimeria lamae* and the larger *E. macusaniensis*.

The small coccidia are best associated with hemorrhagic, watery diarrhea progressing to weakness, lethargy, weight loss or poor weight gain, feed refusal, dehydration, and eventually shock, coma, and death.[109] Colic, respiratory distress, and cerebral signs are uncommon or late findings. The gut, particularly the terminal jejunum and ileum, is occasionally hemorrhagic or markedly edematous, and may have areas of mucosal hemorrhage, fibrinonecrotic pseudomembranes, or punctate white lesions, but is more commonly grossly non-remarkable.[109,115] Histologically, lesions are most pronounced in the villi. There is mucosal loss and villus shortening. Immature and mature forms of the coccidia may be present. The submucosa is often filled with hemorrhagic or eosinophilic infiltrates. In severe cases, the mucosa is lost to the basement membrane. Protein loss is considerable, and hypoproteinemia is the most consistent blood abnormality. Anemia, hyponatremia, and hypochloremia are other common abnormalities.

The small coccidia primarily cause clinical disease in crias up to around 8 months of age. South American crias are usually shedding by around 23 days of age, with earliest shedding by 15 days, meaning they become infected shortly after birth.[110] Shedding increases until 40 to 50 days of age, then gradually tapers off. Illness usually occurs in those first 2 months. Under rare circumstances, clinical disease is seen in older crias or adults. This usually reflects overwhelming exposure or a poor immune response.

The large *Eimeria* of New World camelids are *E. macusanensis*[116] and *E. ivitaensis*.[117] These are 3 to 4 times larger than small coccidia (Figure 40-36), and are approximately 80 to 100µ in length (Figure 40-37). As such, they resemble *E. leuckarti* of horses, E. camelli of camels, and other large *Eimeria*. *E. ivitanensis* oocysts are elongated ellipses, whereas *E. macusaniensis* is ovoid and pyriform, resembling a cut avocado or watermelon seed in shape. Both have an obvious polar cap. There is some heterogeneity in size and shape of *E. macusanensis*, and it is possible that future research will reveal that distinct species exist. *E. macusanensis* also has a thick wall (approximately 8.5 to 11µ), which makes the cyst extremely durable;[116,118] identifiable cysts have survived approximately 10,000 years in mummies.[119]

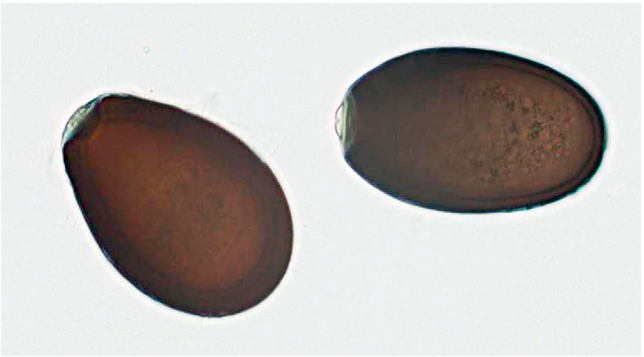

Figure 40-37 Oocysts of *Eimeria macusaniensis* (*left*) and *E. ivitaensis* (*right*).

The life cycles of the large coccidia resemble those of small coccidia, except that everything generally takes longer. The prepatent period for *E. macusaniensis* is from 32–43 days;[109,120,121] that from *E. ivitaensis* has not been reported. Sporulation times for *E. macusaniensis* range from 2 to 3 weeks, with faster times under warmer conditions.[111,120] Sporulation appears to arrest at 7°C or below. Sporulation time for *E. ivitaensis* has not been reported, but appears to be in the 7 to 10 day range in our laboratory. The longer lifecycle means that patent infections appear later than with small coccidia,[110,120] but not necessarily that disease occurs later. Severe disease and death appear to be able to occur within 3 weeks of initial exposure and 2 weeks before establishment of patency. There is growing evidence that crias shedding small *Eimeria* oocysts or showing signs of enterotoxemia, may actually be dying of prepatent *E. macusaniensis* infection.[81,82,122] Additionally, there are increasing reports of prepatent or patent disease in adult camelids.[81,118,123,124] Some of these are long-time herd residents, but most have a history of transportation and mixing with new groups of animals. Whereas shows, sales, and movement for breeding may cause stress and inhibit the immune response, the simplest explanation may lie in eating habits: new entrants in a herd are more likely to eat off the ground than out of feeders, or more likely to eat in the less desirable areas of pasture. Thus, they may ingest larger doses of the parasite and be more likely to show disease signs.

The characteristics of clinical disease associated with *E. macusaniensis* have been studied and reported more extensively than any other parasitic gastroenteritis in camelids.[81,123–125] It is likely that much of this information pertains to other coccidial infections and to parasitic gastroenteritis in general. In younger camelids, gastroenteritis is more likely to result in clinical diarrhea, whereas in older camelids, diarrhea is often absent or easy to miss. In fact, in adults, it is frequently counterintuitive to directly link the presenting signs to GI illness. In addition to or instead of diarrhea, general signs include weight loss or poor weight gain, ill-thrift, and increasing lethargy, weakness, and loss of appetite. It is one of the most common causes for weakness, weight loss, hypoproteinemia, or ill-thrift in our area. Some animals show colic signs, probably more with *E. macusaniensis* than with any other parasitic gastroenteritis, and coccidioisis may contribute to some intestinal accidents.[126,127] As the disease progresses, hypoproteinemia worsens without commensurate anemia; mild anemia is common, but hypoproteinemia and hypoalbuminemia often become severe (such as 3.8 to 2.2 mg/dl for serum total protein and 1.8 to 0.6 mg/dl for serum albumin concentrations). There is also often some reduction in serum sodium and chloride concentrations, and hypokalemia becomes more marked with anorexia. With worsening disease, the animal becomes susceptible to translocation of bacteria or toxins through the damaged mucosa, the effects of hypoproteinemia, and circulatory shock. Signs of other organ systems are seen: camelids may develop ascites, hydrothorax, hydropericardium, pharyngeal edema, and cerebral edema, with ensuing abdominal distention, increased respiratory effort, lethargy, tachycardia, hypothermia, dysphagia, salivary loss, and cerebral signs. Eventually, there is also clinicopathologic evidence of the systemic disease including azotemia, metabolic (usually lactic) acidosis, high liver enzyme activity, hyperbilirubinemia, and increases in blood fat fractions. Abdominocentesis usually yields a transudate. Abdominal imaging is also inconclusive: colicky camelids may have ileus and fluid-distended intestine, though usually to a lesser degree than camelids with GI obstruction. Overtly thickened bowel wall is rare (<10% of cases). In some cases, signs are insidious, and the animal is simply found dead.

Good management practices and maintenance of hygienic facilities for young animals should be considered the most important factors in prevention of coccidiosis. This includes limiting exposure of young crias to older crias or adults and timely removal of feces. Strategic use of anticoccidial drugs may also be considered as a supplemental means of controlling coccidiosis. Since outbreaks of coccidiosis are common in the wetter months, prophylactic drug regimes should be used during these times of year, especially if they coincide with the first 6 months of a newborn's life. Consideration should be given to application of preventative measures before and during stressful events such as weaning, shearing, or herd movements. Treatment medications may be used intermittently for control. Alternatively, decoquinate may be added into feed at 0.5mg/kg/day for 28 days. Use of ionophore antibiotics such as monensin or salinomycin is discouraged without further research regarding safety and efficacy, because they have been associated with toxicosis in camelids.[128]

Balantidium coli, which is an amoeba, has been described in camels and anecdotally in camelids.[129] In the Middle East, camels are postulated to be the reservoir population for human infections.[130] In alpacas, *Balantidium* is thought to cause diarrhea without weight loss in adults, and occasionally diarrhea in crias.

Diagnosis of Parasitic Gastroenteritis

For most parasitic disorders of the GI tract, fecal diagnostic assay of some type is the only definitive premortem test. For *Cryptosporidium*, detection is most commonly achieved by light or fluorescent microscopy of prepared fecal specimens. Formalin-ethyl acetate sedimentation appears to be the best method for concentrating oocysts but is not in common use in general practice.[131,132] Flotation and flotation–centrifugation procedures using flotation solutions with specific gravity (SG) of 1.18 or less (zinc sulfate [$ZnSO_4$], saturated saline, some sucrose solutions) work reasonably well, especially if the animal is shedding heavily. Denser solutions float more debris and may obscure oocysts. Acid-fast, auramine, acridine orange or immunofluorescent staining enhance the visibility of the

oocysts. Fecal enzyme-linked immunosorbent assay (ELISA) has been tested but does not appear to improve detection, whereas fecal PCR appears to offer an alternative to microscopic methods, especially for detecting low-level shedding.[133,134] In camelids, usually crias, with clinical cryptosporidiosis, shedding is usually at a high enough level to make detection relatively straightforward.[98] In crias beyond the acute phase of disease or in adults, more sensitive methods may be warranted.

For *Giardia*, light microscopy of a direct fecal smear (a small amount of feces swabbed or rubbed on a drop of water or saline on a microscope side, or simply a swab of watery feces) may reveal the motile trophozoites. Both trophozoites and cysts may be disrupted by flotation solutions, so the direct smear is indicated if *Giardia* is suspected. Zinc sulfate solution (33%) appears to float cysts (SG = 1.05) well with minimal disruption, whereas saturated saline rapidly collapses them, and sucrose solution may be too viscous. Immunofluorescent or Wheatley trichrome staining may be used to aid in cyst detection (Figure 40-38). Antigen detection assays have been replacing microscopic methods of detecting giardiasis.[135] Although these have not been tested extensively in camelids, the large host range of *Giardia* suggests that they may be of some value. In all cases, and especially in chronically infected animals, shedding is inconsistent, so serial tests may be indicated.

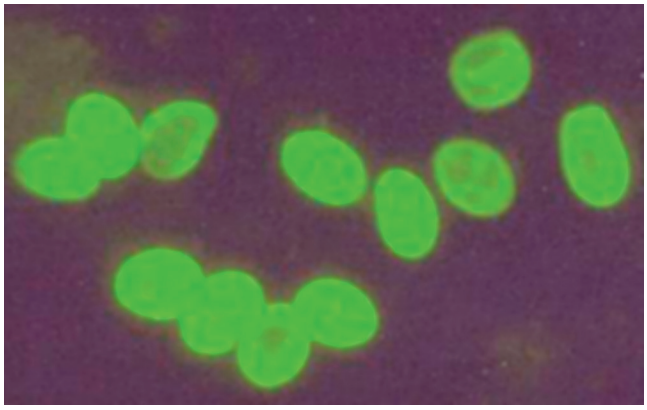

Figure 40-38 Fluorescent microgram of *Giardia* in a cria with diarrhea.

For coccidia and most helminths, light microscopy of fecal samples is also the standard method of parasite detection. A number of pitfalls, namely, prepatent or peripatent disease, intermittent shedding, and ova or oocysts with a range of densities, exist. Prepatent disease is more common in the parasites with longer life cycles, that is, the helminths and *E. macusaniensis*. Prepatent and shedding periods for the most important camelid GI parasites are listed in Table 40-1. With *E. macusaniensis*, clinical disease and death may occur up to 2 weeks before the appearance of oocysts in feces.[81] In the case of helminths, the gap may be several weeks as well, or even longer for *Capillaria* and *Trichuris*. For small coccidia, *Cryptosporidium*, and *Giardia*, clinical disease usually appears around the same time as passage of fecal cysts but may precede it by a day or two. If prepatent disease is suspected, serial fecal analyses at 2- to 4-day intervals may be used to confirm the organism; presumptive treatments may be instituted in the interim. When looking for prepatent or peripatent shedding of protozoan parasites, it is important to keep the shedding curve in mind; *Eimeria* oocyst passage resembles a bell-shaped curve, with few at the earliest stages, peaking in 1 to 2 weeks before tapering off. The same is true for *Giardia* and *Cryptosporidium* except that shedding may occur in multiple waves. Shedding curves are less of an issue with helminths, whose adults are relatively long-lived.

Intermittent shedding is a pronounced feature of *Giardia*, *Capillaria*, *Moniezia*, and possibly other worms. Two to four serial samples at 2- to 4-day intervals may help reveal these infections. Along with intermittent shedding, certain helminths appear to shed low numbers of ova overall; *Nematodirus*, *Capillaria*, and *Lamanema* are especially known for this.

TABLE 40-1 Prepatent Periods for Important Camelid Parasites

Parasite	Prepatent	Patent	
Eimeria punoensis	10 days		Illness rare
E. lamae	15–16 days		Illness with patency
E. alpacae	16–18 days		Illness rare
E. macusaniensis	31–43 days	20–40 days	Illness possible 2–3 weeks before patency
E. ivitanensis	Unknown		Illness rare
Cryptosporidium spp.	3–7 days	4–5 days	Illness with patency
Giardia duodenalis	5–21 days	months	Illness possible 7–10 days before patency
Balantidium coli	Unknown		Illness rare
Strongyle worms	17–36 days or longer		Prepatent disease possible
Nematodirus battus	14–21 days		
Trichuris tenuis	30–60 days?		Prepatent disease possible
Capillaria spp.	42 days?		
Moniezia	5–6 weeks		
Dictyocaulus spp.	22–25 days?		Prepatent disease possible
Fasciola hepatica	70–84 days?		
Dicrocoelium dendriticum	49–79 days?		

With regard to oocyst and ovum density, *Giardia*, *Cryptosporidium*, small *Eimeria*, *E. ivitanensis*, and most helminth ova appear to float readily in flotation solutions with a SG of 1.18 (saturated saline, 33% $ZnSO_4$).[136] The notable exceptions are *E. macusaniensis* and *Trichuris,* which require solutions with SG of 1.027 or greater. *Balantidium coli* cysts are also dense but appear to be extremely rare in llamas and alpacas.

The focus of this section is illness in the individual, and individuals with clinical disease caused by parasites are likely to have high parasite loads. Camelids in a herd are not uniform in their parasite loads. The old tenet is that 10% of the animals have 90% of the parasites. These animals usually reflect those that have lower immunity (the old, young, and infirm) or have higher exposure (outcasts, dirt-eaters, crias). Depending on the severity of the problem in the herd and the prevailing conditions, these heavily infected animals may easily exceed the 10% and may even represent the majority. Whatever the case, if a severe parasitic problem is detected in an individual or individuals from a herd, some investigation of the burden in herdmates may be warranted.

With most parasites, severity is estimated by fecal egg (ova or oocyst) count. On the basis of the caveats above, this is an imperfect estimate but remains unchallenged by a better fecal examination method. When in doubt, other historical, physical, or clinicopathologic evidence should be used to support or refute a diagnosis of endoparasitism.

A variety of methods of fecal analysis have been described, with endless variations. The moniker "modified" has been taken to the extreme in some instances, where the "modified" versions of some tests bear little resemblance to their previous versions. The preferred test depends to some degree on the parasite we wish to detect, and unfortunately, no one test optimally detects all parasites.[136] Many tests completely miss certain parasites.

1. *Direct smear.* This is a simple test, in which a small amount of feces is liquefied with a small amount of saline solution and smeared onto a microscope slide. Only categoric quantification is possible. This is best for detecting motile protozoa such as *Giardia* and *Balantidium* trophozoites and adequate for most types of worm ova at high concentrations.[129] It is not particularly effective for detecting parasites when only low numbers of ova or oocysts are present.
2. *Saturated saline flotation.* This test relies on the difference in SG between saturated (1.18) or more dilute saline and the oocysts or ova. About 2 to 5 g of feces are dissolved in 10 mL of flotation solution in a 15 mL tube. The tube is filled with more solution to form a positive meniscus and a cover slip placed across the top. After 15 to 60 minutes, the coverslip is removed, placed on a slide, and examined under light microscopy. Saline will not float *Trichuris, E. macusaniensis*, or liver fluke eggs. Many other salts such as 33% $ZnSO_4$ solution have a similar SG to saline. Saline distorts *Giardia* rapidly but may be the preferred flotation solution for *Nematodirus* and small *Eimeria*.
3. *Sucrose flotation*. This test is similar to the one above. Sheather solution (454 g of sucrose in 355 mL of hot water plus 6 mL of formalin [optional] to prevent microbial growth) has a higher SG than saline (1.27) and thus will float all but liver fluke eggs. The high viscosity of this solution may inhibit flotation of small coccidia or *Nematodirus* but may be better than saline for *Cryptosporidium* and *Giardia*.
4. *McMaster counting chamber.* Rather than floating the eggs in a tube onto a coverslip, this test involves putting a slurry into a counting chamber. A 4-g fecal sample is liquefied in 26 mL of saturated saline or sucrose solution by using a mortar and pestle. The suspension is funneled through a screen into a 50-mL tube. The filtrate is then used to fill two chambers of a counting slide, which is examined under light microscopy. Multiplying the number of eggs of each type by 25 yields an approximation of eggs per gram of feces. Most parasites can be seen this way, but this test is not particularly sensitive for detecting low levels of infection. The denser parasite eggs sink very quickly in saline and will be missed if any delay occurs before counting.
5. *Double centrifuge technique.* A 2-g fecal sample is liquefied in 98 mL of water by using a mortar and pestle or a similar system. The sample is allowed to dissolve completely, preferably overnight, and then the sample is stirred to create a homogeneous solution. Ten milliliters of the sample is poured into a graduated centrifuge tube and spun at 200× gravity for 5 minutes. The supernatant is removed and a few milliliters of sugar solution (as above) added to resuspend the pellet. The tube is refilled with 10 to 12 mL of the sugar solution and then centrifuged again at 200× gravity for 5 minutes. The tube is filled until a positive meniscus forms, and a coverslip is placed on top. After a suitable time, the coverslip is removed and placed on a glass slide for counting. For most parasites, peak ova or oocysts concentrations are found 4 to 6 hours later, but most positive samples will have been revealed within 1 hour. Multiplying the counts of all of each type of ovum or oocyst seen by 5 yields an approximation of ova or oocysts per gram of feces. The advantages of this technique are that the longer dissolving period enables more eggs to be released from fecal matter and the pelleting concentrates the eggs. This technique appears to be the best way to find small numbers of eggs, as with *Nematodirus, Capillaria* or many parasites at the onset of their shedding period.
6. *Sedimentation.* Feces are dissolved in water and either left to settle or pelleted in a centrifuge. A drop or two of the sediment is placed on a slide for microscopic examination. This is the most used technique for detecting fluke eggs, which do not float in most solutions. The Flukefinder is a specialized system for sedimenting fluke eggs. Sedimentation is also useful for detecting *Balantidium coli*.[129]
7. *Baermann test.* This test is most commonly done on fresh feces to identify lungworms but may also be used for detecting *Strongyloides*. If the sample is allowed to sit for a few days, strongyle larvae may hatch out of their eggs and confound the diagnosis. This test involves suspending a fecal sample in cheesecloth within a funnel or a cone-shaped vessel. The vessel is filled with warm water and left to sit for at least 8 hours. Free-living larvae will concentrate at the bottom of the cone and can be pipetted out for examination under a microscope.

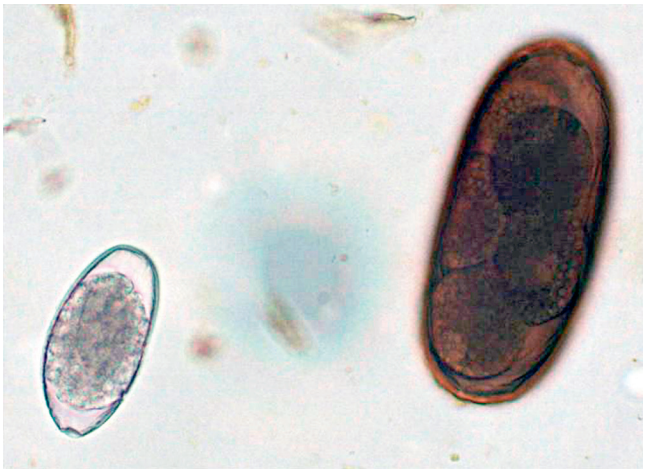

Figure 40-39 *Nematodirus battus* (*right*) and a smaller strongyle-type egg (*left*).

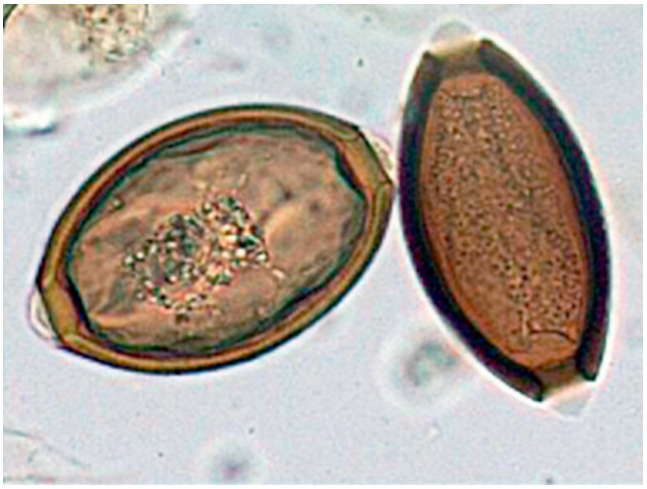

Figure 40-40 Ova of *Capillaria* (*left*) and *Trichuris* (*right*).

8. *Immunologic methods.* Fluorescent antibody and enzyme immunosorbent tests are available to aid in the detection of *Cryptosporidium* and *Giardia*. A systemic (blood) antibody response to the flukes occurs within about 30 days of exposure and may be useful in detecting prepatent disease.
9. *PCR.* Fecal PCR has gained acceptance as an accurate method of detecting cryptosporidiosis. It is less reliable for other coccidial diseases because of the durability of their thick-walled cysts. Nevertheless, preliminary reports suggest fecal PCR may have some value in detecting coccidiosis both in the prepatent and patent stages of infection.

Identification of parasites is based on recognition of the characteristic appearances of their ova or oocysts. Most of these resemble their analogs affecting other species of animal. Most strongyle-type worms release normal strongyle (trichostrongylid)–type eggs, and differentiating them requires experience, fine measurement, genetic tests, or hatching assays. Because of its individual importance, differentiating *Haemonchus* from other strongyles may be critical. A straightforward fluorescent-staining technique has recently been made available and appears to be useful for this purpose.[137]

Nematodirus and *Lamanema* have double-sized, strongyle-type eggs. For the less pathogenic *Nematodirus*, these are relatively symmetric and clear around their central cells. For *N. battus* and *Lamanema* (Figure 40-39), one side appears flatter than the other, and they are a darker brown around their central cells.

Capillaria ova are about 80% the length of a trichostrongylid ovum or the *E. macusaniensis* oocyst (Figure 40-40). They are ovoid, with slight tapering to the polar cap at each end (American [Rugby] football-shaped; length <2× width). *Trichuris* is more slender than *Capillaria*, slightly longer (length >2× width) and of a darker brown; it also has two polar caps. *Moniezia* eggs have a typical pyramid shape (Figure 40-41).

Sizes and shapes of *Eimeria* oocysts have been discussed above. Large *Eimeria* oocysts are approximately the same size as standard strongyle ova. Small *Eimeria* are one third to one fifth the size. Judging the importance of a positive fecal test is almost as challenging as debating the reliability of a negative

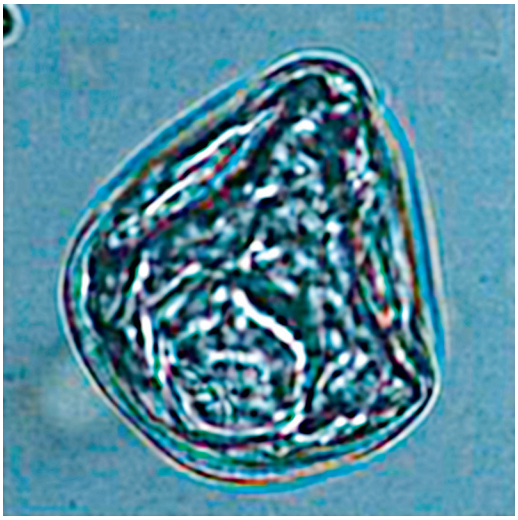

Figure 40-41 *Moniezia* egg.

one. If history, signs, and the diagnostic evaluation support a diagnosis of endoparasitism, it should be considered regardless of egg count. By the same token, finding a few ova or oocysts in clinically healthy animals rarely reflects an emergency situation. Ambient conditions are also important in judging "normal" shedding. Most worms and small coccidia follow the general pattern described for other herbivores, that is, older camelids are generally resistant to disease. Therefore, higher oocysts counts should be expected in camelids less than 1 year of age, and up to a few hundred coccidial oocysts per gram can be considered normal. Counts much higher than 200 oocysts per gram are unusual in adults unless the camelid is otherwise debilitated or in an overcrowded environment. Ova counts are similar, with the same caveats, as well as the considerations that certain parasites (*Nematodirus battus, Capillaria, Lamanema*) may shed few eggs and that many are also associated with prepatent or peripatent disease. With *Giardia*, the more cysts found in feces, the more likely it is that *Giardia* is the cause of diarrhea. Shedding may by episodic, corresponding to waves of replication.

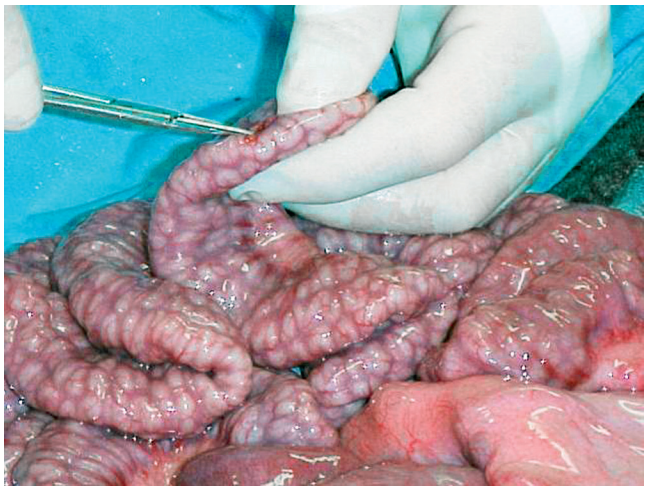

Figure 40-42 Thickened "cobblestone" intestine associated with *Eimeria macusaniensis* infection, hypoproteinemia, and edema of the bowel wall.

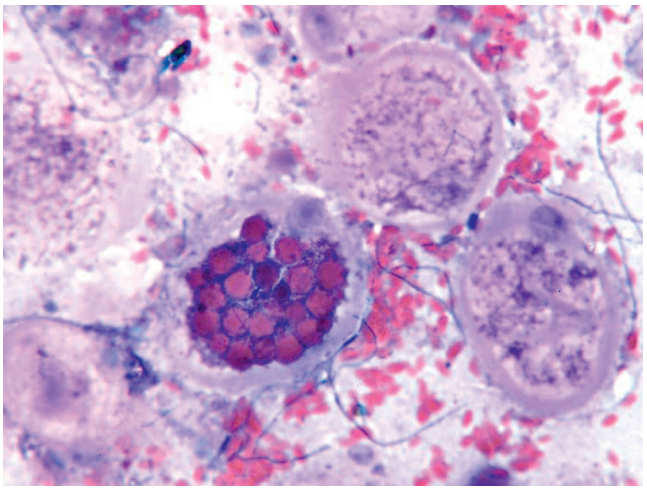

Figure 40-43 Postmortem impression smear of the ileal mucosa, revealing numerous *Eimeria macusaniensis* dwarfing the surrounding erythrocytes.

If an animal succumbs or is euthanized, postmortem examination may be informative. Fecal-style tests may be done on stomach or intestinal contents, or mucosal scrapes. Most worms are macroscopic and may be found on close inspection of the mucosa and its adjacent contents (see Figure 40-33). The terminal C3 (*Haemonchus, Ostertagia, Telodorsagia*) and the cecocolic area (*Trichuris*) are the most important areas to check. Female *Haemonchus* (barber pole) and *Trichuris* (long, coiled or whiplike head) may be macroscopically distinct from other nematodes. If adult and larger larvae are not visible, histopathologic examination may be required.

Giardia readily detaches itself from the mucosa and thus may be missed on histopathologic examination. *Cryptosporidium* and *Eimeria* are more easily visible, but infection is often segmental. Six to seven samples, from the duodenum, duodenal-jejunal junction, mid-jejunum, jejunoileal junction, ileum, cecum, and proximal loop of the ascending colon, should be obtained to minimize the risk of missing the infection. It is important to remember that coccidiosis and some other parasitic infections may cause bowel wall thickening (Figure 40-42), punctate areas of inflammation, or hemorrhagic or fibrinous exudates but frequently cause no macroscopic lesions; samples should be taken for histopathology if these disease are considered at all likely.

For faster postmortem diagnosis of coccidial infections, impression smears of the intestinal mucosa may be taken and stained with most convenient cytostains. *E. macusaniensis* enormously outsizes any host gut or blood cell (Figure 40-43). Small *Eimeria* are comparable with larger epithelial cells but are morphologically distinct. The ileum is the best spot for obtaining impression smears, but multiple smears taken along the small intestine will minimize the chance of missing a segmental infection.

Treatment

If a camelid has confirmed or suspected *clinical* endoparasitism, individual treatment may be warranted. Herd control will be discussed elsewhere in this text. The treatment decision may be triggered by a clinical sign, diagnostic test result, or clinical suspicion; regardless, the decision should incorporate goals to improve the health of the patient and potentially to decrease risk to other animals in the herd. Every egg on fecal testing does not warrant a treatment response, but equally, some camelids with negative fecal tests will require aggressive treatment.

A number of specific antiparasitic agents have been used in camelids with varying results. The standard disclaimer that these medications have *not* been evaluated scientifically for safety or efficacy, including the safety in pregnant animals, is worth repeating here. *Use at your own risk! Cryptosporidium* continues to resist attempts to develop a consistently effective treatment. Paromomycin (25 to 100 mg/kg, PO, q12h) and azithromycin (7.5 to 10 mg/kg, PO, q24h) have both been used clinically, but all positive results are anecdotal.[106] Fenbendazole (10 to 50 mg/kg, PO, q24h for 1 to 3 days) and metronidazole (15 mg/kg, PO or per rectum [PR], q8h; or 25 mg/kg, PO or PR, q12h, for 5 days) have both been used for treatment of *Giardia* and appear to be safe and effective. Oral dosing of metronidazole should be avoided in adults and once crias develop a functional forestomach (about 3 to 4 months of age).

The most commonly used anticoccidial medications for camelids in North America are amprolium (10 mg/kg at the label dilution, PO, q24h for 5 days) and sulfa antibiotics.[81] Both are more effective against the immature forms of the parasite in ruminants and should not be expected to immediately reduce fecal shedding. Efficacy in camelids remains empirical, and pharmacokinetic trials on sulfa antibiotics suggest that uptake (and therefore efficacy) is negligible once the forestomach develops.[138–140] Likewise, amprolium included in the water supply is often not ingested in sufficient quantities to be effective; individual animal dosing is preferred. Treatment with amprolium during the prepatent period does appear to reduce subsequent shedding and also decrease the viability of oocysts. Some concern exists that protracted treatment with amprolium may cause polioencephalomalacia, but we have not seen that with the 5-day course. Milling decoquinate (0.5 mg/kg for 28 days) into pelleted feeds may also be helpful if the crias are old enough to eat the pellets.

Benzeneacetonitrile compounds have been gaining popularity as a coccidia treatment in the United States and are heavily used in other countries. Ponazuril and toltrazuril (5 to 20 mg/kg, PO, q24h for up to 3 days) are effective against multiple stages of the parasite and rapidly stop shedding. The higher doses and longer courses are for treatment of individuals; the lower doses or single treatment may be used for control. Diclazuril has seen limited use; in cattle, gastric absorption is lower than for toltrazuril.

With sick animals with presumptive or confirmed clinical coccidiosis, therapeutic courses are indicated. Contact animals may also be treated if considered at risk. This varies tremendously on the basis of individual circumstances, especially in adults. Eating off the ground is a major risk factor. With crias, it is relatively ubiquitous, as they approach their surrounds with curiosity and ingest a variety of substances. Grazing adults or those fed on the ground are likewise at a universal level of risk. Adults or juveniles fed out of feeders may have dominant animals eat off the ground, whereas more submissive animals eat leavings off the ground or are fed separately, which increases the risk of those individuals. In cases where the risk is universal, treatment of the entire group may be warranted on identification of a single sick animal. Where the risk is heterogeneous, selection of high risk animals for treatment is more efficient. If risk is unknown, simple tests such as body condition scoring, weight measurement, or blood protein determination may be used to identify animals suffering disease consistent with coccidiosis.

Prophylactic treatment strategies also depend on the circumstances. Younger animals have the lowest immunity and the greatest tendency to eat off the ground, including the ingestion of feces. They are also often born during or shortly before the onset of wet, mild weather (spring or fall), when conditions are most conducive to coccidiosis. Mass treatment 2 to 8 weeks after birth may be indicated, with the 2- or 3-week treatment reserved for operations at extremely high risk. Similar guidelines apply to groups of animals put on pasture or feed on the ground, especially during periods of mild, damp weather. Otherwise, mass medication should be based on tangible evidence of a widespread problem, for example, property history or fecal samples with high oocyst counts.

The common rule of thumb when treating camelids for worms is to use bovine or sheep dewormers at approximately 1.5 times the label dose. The rationale for this is that camelids have greater lean body mass per unit weight and thus a greater volume of distribution for aqueous agents. Oral dewormers typically get absorbed more slowly than in ruminants but persist longer in blood. Common treatments include fenbendazole (7.5 to 10 mg/kg, PO, for 1–3 days), albendazole (10 to 20 mg/kg, PO, once; to be avoided in early pregnancy), thiabendazole (100 mg/kg, PO, for 1–3 days), ivermectin (0.3 mg/kg, PO or SQ, once), and pyrantel pamoate (18 to 25 mg/kg, PO, for 1–3 days). Tapeworms may be treated with fenbendazole or albendazole. Praziquantel has also been used.

Concern about drug-resistant nematode populations in certain parts of the world is increasing. In particular, resistance to ivermectin and fenbendazole appears to be a growing problem, particularly in the eastern United States, where frequent deworming to combat *Parelaphostrongylus tenuis* may have contributed. Approaches include nonchemical management strategies, which use agents at higher doses or for longer courses or use novel agents. These are less of a concern in the individual control strategies than in herd control strategies.

Many camelids with clinical endoparasitism require treatment beyond specific antiparasitic agents. These camelids are weak and lethargic and thus require shelter and noncompetitive access to food and water or alternative sources of nutrition. They have lost blood or blood components and may thus require blood or plasma transfusions or some delivery of fluids. They may have compromised mucosal integrity and may benefit from antibiotic coverage or antiinflammatory medications. Other medical conditions may arise and require specific treatment. Often, intensive care is required. With this, even severely affected camelids may survive (see Table 40-1).

Diarrhea

Diarrhea is one of the more puzzling conditions in New World camelids. It simply means an increase in the frequency of defecation and fluid content of feces. The two often go together, and basically, it is usually the increase in water content that fuels the increase in defecation frequency.

A variety of conditions may lead to an increase in fecal water. These include microbial and parasitic infections, dietary factors, runaway inflammation, and systemic diseases. These conditions may be transient or life-threatening, sporadic or endemic, seasonal or epidemic, unique to individual camelids or important to the whole herd, and acute or chronic. Trying to differentiate among causes may be difficult, time consuming, and expensive, but it is often warranted.

Camelids are naturally good at preserving their body water. This has enabled both large and small camelid species to survive in very dry environments. Body water preservation occurs through a variety of mechanisms, including greater gastric digestion, slow gastric emptying, and extensive colonic water absorption. The end result is the dry fecal pellet. Camelid feces comprise 40% to 60% water. Cattle feces comprise about 85% water. Thus, the amount of water in camelid feces must roughly double before the pellets become as soft as cattle feces and must contain even more water to become liquid.

The colon is the most important location for dehydration of feces. Over 25% of the GI water entering the camelid colon is absorbed before defecation, compared with 10% in cattle. Roughly half this absorption occurs up to and through the spiral colon, and the other half in the transverse and descending colon. In contrast, water content of ingesta is about the same at the beginning of the small intestine as at the end.

Factors that increase small intestinal secretion or inhibit absorption may result in excess water being delivered to the large intestine, but the relatively high amounts of water absorbed through the colon may compensate for this abnormality. Thus, small intestinal and gastric diseases do not reliably cause diarrhea in camelids, unless (1) the damage is severe, (2) a systemic complication such as hypoproteinemia exists, or (3) the colon is also affected. In contrast, colonic diseases often cause diarrhea. Thus, the severity of the diarrhea is a poor reflection of the severity of gut damage and often more an indicator of location.

In addition to the above, diarrhea may not reflect gut or systemic disease at all. The amount of water in feces is affected by the number of undigested and unabsorbed particles of feed making their way into the intestine and by the water content

of the diet. Camelids drinking more water or eating a diet high in water such as fresh pasture grass often become maximally hydrated and leave some of this water in the gut. This may be noted as the absence of formed pellets on green pasture. Additionally, some diets contain materials that are poorly digested, for example, lignin or other fibers. Other diets are poorly digested by the current population of gastric microbes, as frequently is the case in the adaptation period after a feed change. Still others, particularly those high in soluble carbohydrate as is found in cereal grains, sugar, or molasses, may promote lactate production. About half the microbial lactate is poorly absorbed. In all these circumstances, a greater than normal number of undigested or unabsorbed particles remain in the gut, drawing in water and loosening the feces, without any real change in the wall of the gut.

The more troublesome diseases involve morphologic changes in the gut wall. The most common abnormalities are the erosion or ulcer. Erosions are areas of superficial loss of the cells lining the gut. Ulcers are deeper. Erosions result in a loss of the cells most important for absorption and, to some extent, digestion. These cells may be lost as a result of bacterial, viral, or parasitic infection, chemical injury, lack of blood flow, or toxic effects. The most important causes of erosive diarrhea are listed in Box 40-1.

Other pathologic processes may lead to diarrhea as well. Some of these promote ulceration, whereas others affect water or substance transit across the intact gut lining. Infectious thickening of the gut wall may be caused by mycobacteria, particularly *Mycobacterium avium* ssp. *paratuberculosis* (Johne disease), or *Yersinia* spp. Mycobacteria lead to granulomatous changes, which decrease absorption and promote protein loss. *Yersinia* thickens the wall with microabscesses. Cellular infiltrates are also seen with lymphocytic–plasmocytic or eosinophilic cellular infiltration. These infiltrations are thought to be triggered by some immune stimulus such as a food allergy or reaction to parasites, but their causes are rarely identified. Neoplastic infiltrates cause more focal but similar lesions.

Inflammation around the bowel (peritonitis) also decreases absorptive function and may be caused by gut leakage (usually from a ruptured ulcer or obstructed bowel) or bacterial infection such as with *Streptococcus equi* ssp. *zooepidemicus* infection. Pancreatic inflammation may decrease digestive function and affect the adjacent bowel through inflammation. Additionally, diseases which decrease blood protein or increase hydrostatic pressure may increase fecal water. Diseases which decrease blood protein include most of the chronic or erosive diseases in camelids. Disorders associated with an increase in hydrostatic pressure include portosystemic shunt, renal disease, hepatic cirrhosis, and right heart failure.

BOX 40-1 The Most Important Causes of Erosive Diarrhea in Camelids

BACTERIA
- *Salmonella enteritidis*, *Escherichia coli*, *Clostridium perfringens*, *Yersinia* spp., *Mycobacterium avium* spp., and possibly *Campylobacter* spp., *Bacteroides fragilis*, and *Lawsonia intracellularis*.

VIRUSES
- Coronavirus, rotavirus, and possibly parvovirus and bovine viral diarrhea virus

PARASITES
- Protozoa: *Eimeria* spp., *Cryptosporidium* spp., *Giardia lamblia*, *Balantidium coli*
- Helminths: *Trichuris* spp., and to a lesser extent *Nematodirus battus*, and the other gastric and small intestinal worms

CHEMICAL INJURY
- Lactate associated with forestomach acidosis, grayanotoxin (andromedotoxin) from azalea, rhododendron, or laurel; oleandrin from oleander

TOXIC EFFECTS
- Endotoxin; other bacterial toxins associated with sepsis; arsenic

Diarrhea in Crias

Neonatal and juvenile diarrhea is a common complaint from owners of cattle, sheep, pigs, horses, and goats. Microbial causes are usually blamed, although in some cases nutritional or other considerations come into play. The most commonly identified pathogens are bacteria, viruses, and protozoa. These infections are relatively self-limiting, and clinical signs are more related to fluid and electrolyte loss than anything else. For ruminants and pigs especially, various products have been developed which specifically address water, base, and salt loss. Various antibody and vaccine preparations are available to directly combat the causative organisms, but with the exception of *Eimeria* and coliform diarrhea, antimicrobial treatment is usually not considered necessary.

In contrast, earlier reports say little about diarrhea in crias or suggest that the disorder is rare but more complicated than in domestic ruminants. The perceived rarity may have been more a reflection of management (fewer large farms and infrequent births) than existence of pathogens. Large farms are now more common, as are neonatal problems. Earlier reports focused mainly on protozoal and helminth causes of diarrhea. This occurred partially because of (1) the ease of diagnosis of these problems with a light microscope; and (2) the lack of suitable techniques for diagnosing viral diseases. Bacterial disorders with multisystemic signs, for example, coliform sepsis and clostridial enterotoxemia, also received a fair amount of attention. These are certainly important disorders that occur under certain conditions but result in clinical pictures that are very different from typical uncomplicated neonatal or juvenile diarrhea in other domestic farm animals. Because of the emphasis on bacteria and systemic diseases, treatment recommendations usually emphasize antimicrobial drugs. Without contrary recommendations or data, these practices have continued.

Clinical Syndromes in Camelids of All Ages

Diarrhea in domestic farm animals may be roughly divided into two categories: (1) sporadic, noncontagious, individual

animal problems and (2) contagious herd outbreaks. The distinction may be blurred by management conditions that cause multiple animals to develop noncontagious conditions at the same time, lack of enough same-aged stock for an outbreak of an infectious disorder to occur, or slow-acting infectious agents that strike individual animals at infrequent intervals. Other sporadic conditions include sepsis, peritonitis, or noninfectious ulcerative disorders. These may become commonplace in herds living in conditions of poor hygiene or with a high prevalence of failure of passive transfer. Contagious conditions may be caused by bacteria, viruses, and protozoa. Camelids are different from other domestic livestock in that many of the contagious causes of diarrhea appear to strike a variety of age groups, from neonates to adults.

Historically, the most common clinical presentation has been the affected individual. Often, the camelid appeared systemically ill, and diarrhea was neither profuse nor watery. The diarrhea typically appeared to have blood in it or was fetid. Other signs included fever, tenesmus, abdominal distension, severe obtundation, recumbency, anorexia, tachycardia, injected mucous membranes, and possibly colic. Clinical pathology evaluation revealed electrolyte or acid–base disturbances that were usually mild and could not account for the degree of obtundation. Abnormalities in the leukogram were often more remarkable: leukopenia or leukocytosis, left-shift, and toxic changes. Thus, the original data about bacterial condition appeared to be justified. Indeed, many of these animals died in spite of treatment, and postmortem examination revealed hemorrhage, inflammation, and necrosis in the gut wall, occasionally full-thickness perforations of GI viscera, and systemic signs of sepsis. Coliform organisms, occasionally *Clostridia* and a host of other gut bacteria, could be recovered from these lesions.

Occasionally, a camelid would present with a more typical picture of diarrhea, including profuse, watery diarrhea, dehydration, inappetence, and general lack of evidence of systemic disease. Clinical pathology was often similar to what is seen in calves, usually at the milder end of the hyponatremia and acidosis spectrum. Efforts to identify pathogens in the feces of these crias were often unsuccessful. *Eimeria*, *Cryptosporidia*, *Giardia*, and helminth ova were found occasionally, but with the exception of *Eimeria* in certain herds, repeat isolates from individual herds were rare.

Herd outbreaks may have been avoided by management practices as much as anything else. Most camelid farms have a relatively small number of animals, with few crias born each year, and these may be spaced over several months. Thus, the large numbers of susceptible hosts to multiply the pathogen in the environment may have been lacking in most instances.

Since about 1998, herd outbreaks of profuse, watery diarrhea have become much more common. These were first noted in the Pacific Northwest but have since been reported in various states in the United States. Camelids of all ages are affected, with most outbreaks starting with alpacas coming home from shows, sales, or trips to other premises for reproductive purposes. Llamas may also be affected, but most outbreaks start with affected alpacas. Crias, which are not usually taken to shows, appear to be more affected by dehydration and electrolyte loss compared with older animals. The most common isolates from these outbreaks are coronavirus in all age groups and *Cryptosporidium* in crias. Rotavirus may play a role as well, especially in crias.

As a last syndrome, soft, nonprofuse, unformed feces are common in crias and adults showing no depression. Pathogens are rarely isolated. Poor fecal formation in these crias appears to be caused by abnormal digestive function, that is, dietary indiscretion or lack of suitable gut flora. In adults or juveniles, it often relates to lush forage, through either high water intake or subclinical acidosis. Some people have had good success treating these with probiotic agents; almost all resolve in 7 to 10 days without treatment.

As camelids age, resistance to some pathogens develops. Additionally, the function of the spiral colon matures. This does not decrease the likelihood of infection or gut damage but makes the clinical sign of diarrhea less likely. As a rule, protein loss and weight loss with enteric infections and parasitism are far more likely to develop in older camelids than diarrhea. Exceptions are chiefly disorders which affect the ileum or colon.

Dietary influences are also different in younger and older camelids. In older animals, water content of feed and digestion by forestomach microbes play major roles. Softening and clumping of fecal pellets occurs in camelids under stress or after diet changes, but true, nonpathologic diarrhea becomes less common as camelids grow older.

Acute Diarrhea

Bacteria

Most bacteria are associated with the more severe syndromes of enteritis with systemic signs. *Escherichia coli* and other coliform organisms have been isolated from blood and postmortem tissues in a number of cases, but it is always difficult to interpret whether they are primary invaders or colonizers of preexisting lesions. Coliform enteritis is most common as a manifestation of neonatal sepsis, which is covered in Chapter 42. Convincing evidence of a predisposing ulcerative process such as coccidiosis, GI worms, forestomach acidosis, impaction, or a bezoar usually exists in older animals. Failure of passive transfer and a dirty environment may increase the risk in crias. Lesions are usually ulcerative and highly inflammatory. Therefore, most coliform infections appear to be opportunistic and affect individual animals, not groups, and preexisting gut damage may facilitate infection. No particular reservoir population has been identified. Since *E. coli* is a common fecal organism, any number of animals may serve as hosts.

Salmonella is a relatively rare problem, but prevalence appears to be increasing with larger herd sizes. It is also an organism of sepsis, and diarrhea is usually mild compared with the degree of depression. All ages are affected.[141–143] Contact with cattle may increase herd risk, but many other animals may also serve as reservoir or transport hosts. *Salmonella* is generally regarded to be a virulent pathogen and requires no co-infection or previous damage.

C. perfringens is probably the best known cause of enteritis in crias but appears to be substantially less common or less important in North America than in South America. It is described in more detail earlier in this chapter. Growing evidence in camelids suggests that *C. perfringens* is a secondary invader of tissues damaged by coccidiosis, giardiasis, or

coronavirus infection. Crias often have a 4- to 14-day history of diarrhea without severe accompanying signs, most likely related to the original gut pathogen, followed by rapidly progressive depression, anorexia, recumbency, abdominal tympany, colic, coma, and usually rapid death associated with secondary clostridial disease. The volume of diarrhea usually decreases as clostridiosis becomes the more dominant condition, while the character of it becomes fouler and bloodier. Affected crias are often those that received the most aggressive treatment for their primary disorder, especially large numbers of oral antibiotics and adjuncts, and these oral agents possibly affect the gut microbial population to favor clostridial overgrowth.

Viruses

Rotavirus. Rotavirus is a double-stranded RNA virus in the family Reoviridae. By electron microscopy, it appears as a thin-rimmed wheel with short spikes. Rotavirus is blamed for diarrheal disease in neonates of most farm animal species, and also occasionally causes disease in adults. In New World camelids, it has been isolated from diarrhea outbreaks involving neonatal crias[144] or adults, and the occasional older crias.[100] Rotavirus was detected by ELISA and confirmed by electron microscopy in 2 of 53 fecal samples collected during a diarrhea outbreak among 1-day to 4-month-old Patagonian guanacos.[87] ELISA failed to detect coronavirus in any of those samples, but the sensitivity of ELISA for either virus in camelids is unknown. This outbreak affected 100% of the crias in 2 separate herds, being fatal in 83% of cases, but other pathogens, including *Salmonella*, were isolated from some cases. Animals were kept in small yards and raised on powdered cow's milk. In all cases disease was acute in onset and the stool was dark-green. Dehydration occurred and death followed within 2–6 days. Affected animals were 7 to 40 days old, and a high proportion of them were also shown to be seropositive to rotavirus.

Rotaviruses in general show a fair amount of antigenic variation, and strains have been reported to cross between species. Non-clinical carrier animals or clinically ill ones may introduce the virus to new susceptible hosts. Incubation periods are between approximately 18 and 96 hours, with the primary site of replication being the absorptive epithelial cells of the small intestine. In some cases, large intestinal cells are also affected. Rotavirus exposure appears to be fairly ubiquitous in South America, even among wild camelids, which may have a higher prevalence of anti-rotaviral antibodies than antibodies against most other livestock viruses.[144–147] This suggests rotavirus may be endemic in many populations of both wild and domestic South American camelids. The strain analyzed in one outbreak resembled bovine strains of the virus,[148] but transspecies cross-infectivity has not been confirmed.

Coronavirus. Coronavirus is a large single-stranded RNA virus in the family Coronaviridae. Coronavirus is characterized by a spherical virion surrounded by a fringe of radiating peplomers. Coronavirus is blamed for diarrheal disease in neonates of most farm animal species, and also occasionally causes disease in adults. In New World camelids, it has been associated with periodic outbreaks involving all ages,[100] followed by relatively quiescent periods when cria diarrhea is still common. Coronavirus was the most common pathogen identified in unweaned crias with diarrhea in one Oregon study.[84] It was identified by electron microscopy in 42% of cases and affected 64% of herds studied, with an age distribution from 10–150 days at the time of diagnosis. Another U.S. study found coronavirus in only 6.9% of 9- to 94-day-old crias with diarrhea and no rotavirus,[86] but electron microscopy was not performed in all cases. Since both those studies were conducted, there have been repeated outbreaks of diarrhea in North American camelids, including crias, with coronavirus identified more commonly than any other potential pathogen. Clinical infection has been confirmed in crias as young as 1 day of age. Rotavirus has also been identified in some cases. Shows appear to be a common site for transmission between herds. It is genetically relatively conservative, but strains appear to be species-specific. The camelid strain appears to be related to bovine and porcine coronavirus, but represents a unique strain.[149] Whether it originally arose from a bovine or porcine strange is unknown. Non-clinical carrier animals or clinically-ill ones may introduce the virus to new susceptible hosts. Incubation periods are between about 18 and 96 hours, with the primary site of replication being the absorptive epithelial cells of the small and large intestine. Compared to rotavirus, it appears to damage a greater length of intestine, and more severely affect the regions it infects.

Vaccines and monoclonal antibody preparations against enteric viruses are available for calves and lambs in the U.S. Some contain antibodies from hyperimmune bovine colostrum, and should be given orally within the first 12 hours of life. Others are modified live vaccines that can be given either orally to calves as soon as possible after birth, or to late-term pregnant cows by injection via a 2-dose course that is also required at subsequent pregnancies. These may be used in camelids on farms experiencing outbreaks of viral diarrhea, but they are of unknown safety and efficacy in these species.

Other Viruses. A variety of other potential pathogens have been found on different occasions and linked to GI disease. These include bovine viral diarrhea virus (BVDV), parvovirus, and others. Evidence for their importance in causing diarrhea is still lacking.

Parasites

Parasitic gastroenteritis is covered separately in this chapter. Most parasites cause ill-thrift or weight loss more than diarrhea. The most common parasites associated with actual watery or bloody malformed feces include *Cryptosporidium*, *Giardia*, *Eimeria*, and *Trichuris*. Of these, *Cryptosporidium* is mainly a disease of crias less than 3 weeks old, *Giardia* mainly affects crias 10 to 60 days old, small *Eimeria* primarily affect crias 2 weeks to 8 months of age, and *Trichuris* and *E. macusaniensis* affects all New World camelids over about 2 weeks of age.

Forestomach Acidosis

Forestomach acidosis characteristically has an acute onset and affects more than one animal. Camelids have adapted to survive on a very nutrient poor diet and thus are not particularly adapted to high energy feeds. Previous conclusions to the contrary, that camelids are actually protected against hyperfermentation, are probably erroneous and misleading. Some outbreaks are related to classic overfeeding or accidental exposure (leaving the door to the grain room open), but others are

caused by a few animals eating more than their share of a group's ration. Diarrhea is one of the later signs with acute forestomach acidosis, showing up 2 to 3 days after the signs of the metabolic crisis, which include neurologic signs (stumbling gait, head arched over the back), abdominal distension, and weakness. Other aspects of this disorder are discussed elsewhere in this chapter. With subacute or subclinical acidosis, intermittent bouts of weight loss, depression, and diarrhea may occur, divided by periods of apparent normalcy.

Peritonitis

Separate from sepsis, acute abdominal inflammation may lead to diarrhea. Peritonitis leads to irritation and inflammation of the gut wall, affecting gut function, and often resulting in diarrhea. It also affects the transit of fluid and protein through the peritoneal space, affecting the gut's ability to transport fluid out of ingesta and into circulation. The four major causes of bacterial peritonitis that we see are (1) translocation of bacteria through a damaged gut or urogenital viscus wall, (2) direct introduction of bacteria through a wound or penetrating instrument, (3) release of bacteria through a ruptured gut, uterus, or urinary structure, and (4) hematogenous infection of the peritoneal cavity. Wounds are less common than the other three causes, which often are difficult to distinguish; by the time the animal's abdomen is examined before or after death and is found to have peritoneal exudate, reddened and thickened gut walls, and a spot of leakage or rupture, it is impossible to say which lesion led to the other two. The most common organisms isolated from abdominal fluid of camelids are *Streptococcus* and *Escherichia*. *Streptococcus* infection is usually hematogenous, and hence the primary pathogen, whereas *Escherichia* may come from a variety of sources. Specifically, streptococcal peritonitis is part of the "alpaca fever" complex, a cause of fever, diarrhea, colic, and respiratory disease covered in Chapter 37. Being hematogenous, streptococcal infections may not have caused much physical damage by the time of diagnosis and may respond well to treatment. In contrast, coliform infections usually involve major morphologic changes to a viscus wall, changes that may be irreversible or require surgical resection.

Diagnosis

Diagnosing the cause of acute diarrhea involves careful collection of historical, physical, and laboratory evidence. Foremost, it should be clarified whether diarrhea is the primary problem or part of a larger syndrome. Camelids with primary diarrhea tend to have more profuse, watery feces and less evidence of systemic disease. Signs of systemic disease that suggest more than simple enteritis include fever; injected mucous membranes; other GI signs such as colic (peritonitis, slaframine or arsenic intoxication), vomiting (poisonous plant ingestion), abdominal distension (may reflect enteritis with protein loss or problems with the hepatic or urogenital systems); or dyspnea (streptococcal pleuritis in addition to peritonitis, or sepsis in general). Laboratory evidence of more complex disease includes neutropenia or a degenerative left shift, hyperfibronogenemia, high hepatocellular liver enzyme activities, and abnormalities (high cell count, protein concentration, or both; presence of microbes) of peritoneal fluid.

Outbreaks are usually attributable to highly contagious agents, including coronavirus, *Cryptosporidium*, and possibly rotavirus, *Salmonella* or *Streptococcus*, overingestion of fermentable carbohydrate, or ingestion of toxins. Even so, with smaller camelid herds and their dietary selectivity, these agents may also cause sporadic disease in individual animals. Other parasites causing diarrhea, chiefly *Eimeria*, *Giardia*, and *Trichuris*, may affect multiple animals simultaneously, but more commonly affect them in a more enzootic than epizootic fashion; sporadic or clumps of cases appear over a few weeks to months.

Flecks of blood may point to *Eimeria*, *Trichuris*, or coronavirus, whereas clumps of blood are more suggestive of intussusception. Otherwise, the nature of profuse diarrhea depends more on the diet than on the agent. The more profuse and watery the diarrhea, the more commonly it leads to electrolyte and acid–base disturbances. Hypochloremia, hyponatremia, and metabolic acidosis are reasonably common findings with viral enteritis and may be severe. With protozoal enteritis, these abnormalities are less common or severe but still may be present, especially in neonates. Because electrolyte loss is relatively rare with camelid illness, in general, these findings, even if mild, point strongly to an enteric disorder. With more severe dehydration or shock, lactic acidosis and azotemia develop. With any sick camelid, hyperglycemia is common; urinary loss of excess glucose may exacerbate dehydration, especially if water intake is inadequate. Water loss may, therefore, outstrip electrolyte loss, leading to more severe evidence of dehydration (tachycardia, obtundation, weakness, feed refusal) than would be expected in a ruminant with diarrhea and a similar degree of hyponatremia and hypochloremia. Thus, physical evidence may trump laboratory evidence regarding the severity of the disorder and need for aggressive treatment.

To diagnose specific agents, it is important to obtain good stool samples early in the course of the disease. These samples must also be kept cool and transported to a laboratory quickly, especially for the viruses. If the camelid does not defecate readily, a gentle rectal lavage with up to 10 mL of warmed saline may aid in procuring a sample. If the lavage method is used, cyst and egg counts will be artificially low. It is also important to recognize that some disorders develop in the prepatent period for parasites. Repeating fecal examinations every 2 to 3 days may be helpful if a risk of coccidiosis or whipworms exists.

Detection of parasites is discussed elsewhere in this chapter. Identification of virus by electron microscopy (EM) is the gold-standard diagnostic tool, but EM is not available in all areas. Coronavirus is also fairly labile, and requires rapid processing for correct identification. Diagnostic assays available for use in other species have not been validated for camelids, and most are actually designed for human group A rotaviruses, the same group that causes most enteric infections in calves and foals.[150] Nonetheless, several studies have found these tests to be useful for veterinary applications.[151–154] A rapid enzyme immunoassay test (ImmunoCard STAT! Rotavirus, Meridian Bioscience Inc., Cincinatti, USA) is available and has been recommended for use in cattle.[155] Rapid latex agglutination test kits are also available (Rotavirus Latex Agglutination Test, Oxoid, UK, and Virogen Rotatest Test, Inverness Medical Professional Diagnostics, Princeton, NJ, USA). The Virogen Rotatest kits have been shown to have good sensitivity compared with virus isolation (87.8%) while specificity was 73.3%.[151] The rapid tests are quick and easy to use, but it must

be stressed that they have not been validated on camelid fecal samples. Polyacrylamide gel electrophoresis (PAGE) testing for rotavirus can also be performed on feces and is not bovine specific, so it may have an application in camelid neonatal diarrhea. Testing for coronavirus in cattle is achieved by enzyme-linked immunosorbent assay (ELISA) for fecal antigen, available in Europe: again, it is not known whether this test is sensitive for camelid coronavirus but strains isolated from alpacas suggest that they are closely related to some bovine strains.[149] PCR techniques may also be used.[156] Further research into the most appropriate diagnostic testing methodologies for viral pathogens in camelids is required. Until this is available, electron microscopy remains the most reliable technique.

Treatment of viral diarrhea is mainly supportive. It is important to rule out, and if necessary, treat for other pathogens which may be present. Intravenous fluid therapy is likely to be required in order to counter fluid and electrolyte losses. Systemic antibiotics are indicated to prevent secondary bacterial complications resulting from compromised intestinal epithelial integrity and immunosuppression.

Salmonella and *E. coli* may be grown by bacteriologic culture. *Salmonella* may also be detected with PCR, and PCR may additionally be used to detect virulence factors on *E. coli*. Positive growth of *Salmonella* on any biologic sample is usual considered significant. Growth of *E. coli* from feces is not enough to confirm its role in disease. Growth from blood or peritoneal fluid is compatible with sepsis and more confirmatory. Diagnosis of peritonitis is by analysis and culture of peritoneal fluid or surgical exploration. Imaging studies may offer evidence but usually require a confirmatory test.

Clostridial infections are difficult to definitively diagnose before death and sometimes after death. The rapid clinical course (12 to 48 hours) limits the value of fecal culture. Newer molecular toxin assays have eased typing and potentially made it easier to interpret positive culture results. Empirical diagnosis, aided by results from previous losses from death, is still the most common method. Postmortem culture of ingesta or lesions may also be misleading. Clostridia rapidly overgrow and invade the dead gut. Demonstrating host reaction is necessary to confirm the premortem importance of *Clostridia*.

Frequency

Giardia, *Eimeria*, *Cryptosporidium*, and helminths together account for about half of our cases of diarrhea in crias. They are more common on overcrowded farms and have some potential for cross-species (all but *Eimeria*) or zoonotic (*Giardia*, *Cryptosporidia*) infections. Of these, *Cryptosporidium* is the most likely to cause life-threatening fluid loss, and the most common pathogen in crias less than 21 days old. Uncontrolled *Eimeria* or *Giardia* infections may also lead to severe disease. Helminth parasites are less commonly a cause of diarrhea because they are controlled by medications, because crias take some time to develop pathogenic loads, and, most importantly, because ill-thrift is a far more common manifestation of a high nematode burden compared with diarrhea.

Self-limiting bacterial enteritis is rare. Most bacterial infections associated with diarrhea are either enteropathogenic as with *Clostridium* spp. and *Salmonella* or invasive as with *Listeria*, *Escherichia*, or other opportunistic organisms causing neonatal sepsis.

Coronavirus and possibly rotavirus account for the other half. Coronavirus specifically has been linked to several outbreaks of diarrhea in the northwestern states and elsewhere across North America, affecting camelids of all ages. Crias with their smaller fluid reserves are most susceptible to severe diseases. These are not thought to be novel pathogens; rather our ability to isolate them is improving. BVDV and a parvovirus have also been found, and their significance in both neonatal and adult camelids deserves further investigation.

The juvenile period in camelids bridges the time between the true neonatal period (up to 3 weeks) and the period of peak endogenous immunity (between 8 and 18 months, approximately). As such, juveniles are often still susceptible to late onset variants of the neonatal disorders but also are susceptible to many of the adult conditions. Coronavirus remains a problem in all age groups, especially with outbreaks. *Eimeria* spp. are the most common parasitic cause of diarrhea in camelids more than 3 weeks old. *Cryptosporidium* and *Giardia* may affect older crias but appears most commonly before age 3 weeks.

As camelids age, resistance to some pathogens develop. Additionally, the function of the spiral colon matures. This does not decrease the likelihood of infection or gut damage but makes the clinical sign of diarrhea less likely. As a rule, in older camelids, protein loss and weight loss with enteric infections and parasitism are far more likely to develop than diarrhea. Exceptions are chiefly disorders which affect the ileum or colon (*Eimeria, Trichuris,* and coronavirus).

Dietary influences are different in younger and older camelids. In older animals, water and carbohydrate content of feed and digestion by forestomach microbes play major roles. Softening and clumping of fecal pellets occurs in camelids under stress or after diet changes, but true, nonpathologic diarrhea becomes less common as camelids grow older. Viral diarrhea continues to be a common cause of diarrhea in adults, especially in relation to outbreaks. Parasitic enteritis in adults is more commonly caused by large coccidia or whipworms than the *Giardia* and *Cryptosporidia* of the very young. *Cryptosporidium* has been reported in adults in the midst of outbreaks, but that finding appears to be rare.

Treatment

General treatment of diarrhea follows the principles used in other species. However, electrolyte loss, acidosis, dehydration, and hypoglycemia are less severe or less common under most circumstances, and empirical correction of unconfirmed abnormalities may lead to complications that are worse than the original disease. Unless the animal is convincingly dehydrated or azotemic, the initial fluid bolus does not usually exceed 5% of body weight in crias and 2% in adults. Larger boluses are permissible if the camelid can tolerate fluid loading but may result in pulmonary edema if given to animals with hypoproteinemia. Subsequent fluid rates of up to 5% (juveniles and adults) to 10% (neonates) of body weight over 24 hours are usually sufficient, except in the presence of profuse watery diarrhea.

Acidosis may be the result of bicarbonate loss and hence responds to bicarbonate administration. If blood pH or bicarbonate cannot be measured directly, estimating the base deficit at half of what a calf with comparable signs would have is usually safe. Hypernatremia is more common than

hyponatremia, and a real danger of spontaneous or iatrogenic hyperosmolar syndrome exists. In the presence of dehydration, shock, or hyperadrenocorticism, even unsupplemented, standard electrolyte replacement fluids, combined with a high rate of sodium retention, may lead to rapid increases in blood sodium. Ideally, blood sodium is monitored during treatment to avoid serious complications.

Except in rare cases, blood glucose is normal to high and does not need to be supplemented. Spontaneous or iatrogenic hyperglycemia without adequate fluid replacement is a major contributor to hyperosmolar disorder, often preceding it. Camelids with either hypernatremia or hyperglycemia should be monitored carefully. If hypernatremia appears to be worsening, diluted oral or IV fluids, milk, or diluted milk replacer offer options to provide water without much glucose or sodium. Calf oral electrolyte solutions mixed using label directions should generally be avoided because they provide too much sodium and glucose. Diluting those solutions two- or threefold, or using human pediatric electrolytes may be helpful. If giardiasis is suspected, even dilute oral glucose could potentially promote protozoal replication.

Because systemic or invasive bacterial infections are more common compared with less pathogenic ones, depressed camelids with diarrhea often require treatment beyond that for dehydration and acidosis. Antibiotics, NSAIDs, and plasma may be necessary. When clostridiosis is suspected, treatment should consist of fluids and antiinflammatory medications for shock, antibiotics, potentially plasma, and specific antiserum against β-toxin and ε-toxin. If the primary enteric pathogen can be identified, treatment against it should be initiated as well. Although nursing small, frequent meals is probably acceptable, boluses of milk or milk replacer should be avoided. Penicillin-class antibiotics at 1.5 to 2 times the normal dose are recommended. A single oral dose of penicillin on first identification may be helpful.

Gram-negative infections are much tougher to treat because they often reflect a severely compromised gut. Mixed infections are even worse. Vigorous antibiotic treatment, NSAIDs, fluids, plasma, lavage of the abdomen, and resection or oversewing of damaged gut may be necessary.

Specific treatments for some of these disorders such as parasitic and *Streptococcus* infection may be found in the pertinent sections in this chapter.

Prevention

Most of the pathogens of diarrhea are spread through fecal–oral transmission. Therefore, general hygiene, including periodic cleansing of facilities and shared equipment and good pasture management, are keys to prevention. Parasitic pathogens usually require a fairly large infective dose except in the very young.

Infectious pathogens also prey on the young, although naïve or immunocompromised older animals are also at risk. Boosting immunity and preventing exposure of highly susceptible animals are key in the fight against these organisms. Quarantine periods for visitors, new animals, or stock that has left the property and general separation between these animals and neonates are desirable.

Colostral antibody provides some local protection against the enteric pathogens, but the concentration of antibody in colostrum drops too quickly to protect the neonate for more than a couple of days. After that period, absorbed antibody and the neonate's own secretory immunity take over. Continued colostrum feeding or oral antibody preparations may prolong the protection period, but they would have to be continued for several weeks to guide the neonate through the highest-risk period.

Vaccination of cattle has been shown to increase colostral antibody, but the duration of the protection remains short. Vaccination of the neonate is unlikely to provide protection for several weeks and also does not adequately stimulate secretory immunity. Data from sheep suggest that they do not develop significant protection with vaccination before age 7 weeks. Cattle vaccines against enteric viruses may provide some protection, but antigenic variability may limit that protection and vaccine complications have been reported anecdotally.

Chronic Diarrhea and Weight Loss

Several GI causes of chronic diarrhea and weight loss in New World camelids have been described. Of these, Johne disease (paratuberculosis) has received the most attention, and fear of a positive diagnosis of Johne disease has made many producers reluctant to pursue a definitive diagnosis for camelids displaying these clinical signs. However, some of these disorders are treatable, and most are not known to be contagious. It is the veterinarian's role to educate the client on the importance of establishing a definitive diagnosis for camelids showing these signs. Identification of animals with contagious disease will help reduce risk of transmission, and timely identification of animals with treatable disorders will allow for specific therapeutic measures to be instituted.

Because of their thick fleece, aloof attitude toward people, and stoicism, camelids often conceal the signs of chronic enteric diseases until they are beyond treatment, particularly when diarrhea is less of a feature than weight loss. Daily observation of their eating, defecating, and activity level, periodic weighing or assessment of body condition score, and inspection of the dung pile for evidence of diarrhea are useful management practices to detect these diseases in the early stages. If the diarrhea or weight loss is missed, the owner or veterinarian may see sudden death, chronic disease that does not respond to standard field treatments, or fatal secondary infections instead. Although the individual causes of chronic diarrhea are uncommon, it is likely that the veterinarian with a fair-sized camelid practice will encounter at least one of these diseases on an annual basis.

Most of the GI causes of chronic diarrhea or weight loss involve chronic inflammatory or infiltrative disorders thickening one or more of the layers of the gut wall. This inhibits absorption of nutrients from the gut lumen and leads to loss of protein through mucosal ulceration or from obstructed lymph channels. Granulomatous inflammation may occur with mycobacterial infections. *M. avium* ssp. *paratuberculosis* (paratuberculosis or Johne disease) is the best known cause in North America and Oceania.[157,158] Johne disease is also found in Europe but is somewhat overshadowed by *M. bovis*, especially in Great Britain and Ireland.[159–161]

Nongranulomatous inflammatory cell infiltrates are more common in most areas. Eosinophilic infiltrates are the most frequently diagnosed. Care must be taken because camelids,

like ruminants, normally have large numbers of eosinophils in their villus crypts. Lymphocytic–plasmocytic enteritis is similar but less common. Clinical signs may wax and wane for years, with animals repeatedly dropping and gaining weight. Noninflammatory, nonneoplastic thickening of the gut is rare. We have seen one alpaca with idiopathic muscular hypertrophy. All these conditions require surgical biopsy to attain a premortem diagnosis and thus are frequently missed.

Neoplastic growths within the intestine cause weight loss and hypoalbuminemia but are usually too focal and cranial to result in diarrhea. Because these masses often ulcerate and are often gastric, melena may occur. We have seen severe melena more often in camelids with gastric neoplasms than all other conditions combined. Lymphoma, squamous cell carcinoma, and adenocarcinoma are the most common GI neoplasms, in that order.

This is a far from comprehensive list. However, it points out that most of the diseases we recognize as causing chronic diarrhea in New World camelids are severe, multisystemic disorders. Because of this, veterinarians should try to diagnose and treat these disorders quickly and aggressively.

Neoplastic Disorders

Relatively few reports of neoplastic disorders in camelids have been published. Squamous cell carcinoma is the most commonly reported primary neoplasm affecting the GI tract, whereas multicentric lymphoma, which commonly invades the gut wall, is the most commonly reported neoplasm overall. Glandular tumors of the intestine and leiomyoma have also been reported. In general, neoplastic disorders cause gradual weight loss with progressive lethargy and anorexia. Diarrhea might be noted early in the course of the disease or terminally. Often, the early clinical signs are missed, and the llama is either found dead or extremely debilitated. Antemortem diagnosis and successful treatment of malignant internal neoplasms are rare but could be increased by earlier identification of the sick animal, greater awareness of the tumors, and a diagnostic workup directed toward neoplastic disorders.

Lymphoma. Lymphoma is covered more extensively in Chapter 36. This section will focus on GI involvement. Lymphoma, or more broadly, malignant round cell neoplasia, is often multicentric, and thus multiple organs and multiple sites within single organs may be affected.[81,82] In one study, 5 of 23 camelids had gastric and local lymph node involvement, and they did not have lesions in any other organs.[82] However in another study, 2 of 10 affected camelids had GI tumors as part of a multicentric disorder.[81] Of the 5 gastric tumors, 4 stained as B-cells versus 7 of 19 nongastric tumors.[82]

The most common GI site is terminal C3 (see Figure 40-32). Intestinal and C1 tumors have also been seen. Usually, a diffuse, multilobulated mass exists in the GI wall with ulceration of the overlying mucosa. Smaller satellite masses are also common.

Lymphoma may be found in camelids of any age. External lymphadenopathy is supportive but not present in all cases. A variety of clinical signs are possible, depending on the extent of masses and organs involved. Weakness, weight loss, recumbency, inappetence, and tachypnea are common signs. Neurologic dysfunction, particularly posterior paresis, is seen in about 20% of cases. Specific GI signs are rare. Ulcerated neoplasms of the wall of C3 or the small intestine may cause severe melena and allow for bacterial invasion of tissues. Diarrhea may be caused by neoplastic infiltration of the liver or gut wall.

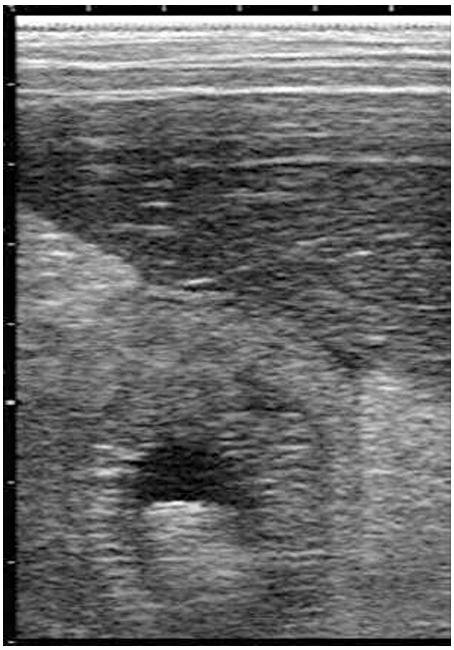

Figure 40-44 Transcutaneous ultrasonographic appearance of a gastric carcinoma in an alpaca.

Diagnosis is achieved by identifying neoplastic lymphocytes on cytologic examination of body cavity fluid, blood, bone marrow, a tissue aspirate, or an impression smear done on a biopsy sample or by histologic examination of a tissue biopsy sample. If no external masses are palpable, ultrasonography of the internal organs may reveal masses or diffusely infiltrated tissues for sampling. Third compartment masses have been the easiest GI tumors to find (Figure 40-44) because of their proximity to the right ventral body wall.

Experiences with chemotherapy are limited. Camelids appear to respond favorably to protocols used in other domestic mammals; however, the caveat is that the advanced stage of the diseases on presentation and the likelihood of mucosal ulceration make secondary bacterial infections likely. Lengthy survival has not been described.

Squamous Cell Carcinoma. Squamous cell carcinoma is the second most commonly reported malignant tumor of camelids, with about half the affected camelids having skin masses and the other half having tumors of the first gastric compartment.[80,81,162,163] Unlike lymphoma, gastric squamous cell carcinoma appears to be a tumor of mature animals (ages 5 to 10 years or older), but one 2-year-old with an unusual distribution of lesions has been reported. Common clinical signs include weight loss, lethargy, depression, anorexia, or mild colic and often are noted for up to a month before death. Abdominal distension with ascites or respiratory difficulty with hydrothorax also are common; although diarrhea is less common with neoplastic disorders than with inflammatory or hypertrophic bowel disease, melena has been seen with squamous cell carcinoma. Diarrhea often is present when ascites becomes severe. In one camelid, antemortem

diagnosis of neoplasia was made by microscopic examination of the peritoneal fluid, although most fluid samples from camelids with neoplasms are normal. In all camelids, the definitive diagnosis was made on the basis of postmortem examination. The primary tumor mass in most camelids was found in the cranial compartment of C1 (see Figure 40-31), with metastases throughout the abdominal and pleural cavities. Other sites, including the squamous C1-C2 junction and the gastric region of C3, have been reported. The tumor in this last llama was unusual because it occurred in a young animal and arose from a site not normally lined with squamous epithelium. Because of the tumor's location, an endoscope could be used to examine and biopsy the mass, but we are unaware of any attempt to do this. Reports on ruminal squamous cell carcinoma in cattle suggest a toxic etiology, particularly bracken fern toxicosis. We have not seen a sufficient number of camelids with this disease to make a similar inference, but bracken fern toxicosis has been associated with squamous cell carcinoma of the urinary tract in camelids. Attempts to identify a causative virus have been unrewarding. Because of the difficulty in making an antemortem diagnosis of this disease, no reports of treatment of these tumors have been published.

Nonneoplastic Inflammatory Bowel Diseases

Paratuberculosis (Johne disease) and Other Chronic Bacterial Disorders. *Paratuberculosis* (Johne disease [JD]) is caused by infection with *M. avium* ssp. *paratuberculosis*. It is the most important GI mycobacterial disease in countries where tuberculosis is reasonably controlled. Other natural hosts include cattle, sheep, goats, deer, other wild ruminants, humans, and possibly horses and rodents. Additional hosts may act as mechanical vectors, allowing the organism to multiply in the gut and be shed in feces without colonizing the gut wall. This disease appears to be rare in domestic camelids, although small outbreaks have been reported.[157,158,164] Culture-positive feces have been obtained from wild camelids.[165] Anecdotally, Australia and Pennsylvania appear to have reported the largest numbers of camelids with clinical disease. In Australia, rigorous regional control programs are in place, and these programs have greatly reduced the prevalence of this disease in camelids and ruminants.

Three strains of the Johne organism are known, one "bovine" and two "ovine." The "bovine" strain appears to be the more common isolate in both wild and domestic camelids, but the ovine strain has been found as well.[164–167] The organism is shed in the feces of infected animals and may survive on pasture or in lagoons up to a year, especially under cool, moist, acidic conditions. As environmental survival is limited, soil contamination is unlikely to be the source of infection for camelids on pasture unless ruminants have recently grazed on it. The prevalence of chronic shedder or carrier camelids also appears to be low, although more sensitive diagnostic techniques may reveal more subclinical shedders.[164] Therefore, contact with an actively shedding ruminant or contaminated ruminant product appears to be the most likely source of infections in camelids. Of ruminant products, goat or cow colostrum is the most widely used in camelids and also is likely to be ingested by the most susceptible host, the neonate. Conventional pasteurization procedures do not completely kill *M. avium* ssp. *paratuberculosis* in colostrum, so efforts should be made to procure ruminant colostrum from animals not infected with the organism.

The organism infects macrophages and causes granulomatous reactions primarily within mesenteric lymph nodes and the mucosal and submucosal layers of the gut wall. Grossly, mesenteric lymph nodes may be several times their normal size, and infected gut may be ropelike with rugose mucosal folds. A number of other mycobacterial organisms cause lymphadenopathy and granulomas in various parenchymal organs, but gut wall granulomas are most common with the *M. avium* complex and *M. bovis*.

The most common clinical signs are weight loss, diarrhea, progressive weakness, and death.[157,158,164,168] Diarrhea is often a near-terminal finding and only seen in about half the cases. All ages of camelid may be affected; younger animals are likely to develop disease in areas of high exposure. Total clinical course is rarely longer than 8 weeks; up to 3 months has been reported.[162] The duration of subclinical infection is unknown. Clinical signs likely are the result of nutrient malabsorption and protein loss by the thickened gut; hypoalbuminemia and hypoproteinemia are the most common blood abnormalities. Evidence of secondary bacterial infection is common with advanced disease. Thickened gut might be seen on transabdominal ultrasonography or a cross-sectional imaging study, but thickened gut is present in only about half the cases.

Most patients succumb within a few days to weeks of diagnosis. Others are euthanized for poor condition, poor treatment response, or suspicion of a contagious disorder. On postmortem examination, mesenteric or abdominal lymphadenopathy is usually found, and about half the affected camelids have grossly thickened small intestine and ascending colon (Figure 40-45). Histopathologic examination of the gut and lymph nodes reveals granulomatous inflammation, with epithelioid macrophages, giant cells, and lymphocytic and plasmocytic infiltrates. Numerous acid-fast positive bacilli are found within the macrophages. Occasionally, ulceration of the overlying mucosa and necrosis of the lamina propria are seen. Granulomas are also occasionally found in the liver.

Diagnosis of Johne disease may be difficult. In ruminants, animals with clinical disease only make up the minority of

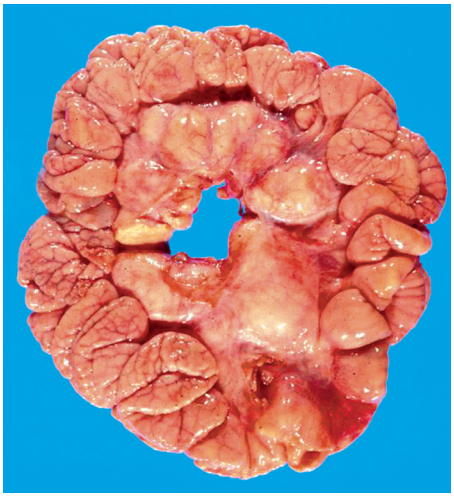

Figure 40-45 Thickened bowel and enlarged mesenteric lymph nodes in a llama with Johne disease.

animals infected with *M. avium* ssp. *paratuberculosis*. Most premortem diagnostic tests are more accurate for animals with clinical disease, making subclinical carrier animals difficult to identify. Fecal culture remains the standard, but false-negatives do occur, and positive results may take 4 to 12 weeks.[164] Culling all culture-positive animals appears to have greatly reduced the prevalence of this disease in Australia.

Conventional agar gel immunodiffusion and complement fixation tests designed for the diagnosis of Johne disease in ruminant species have given mixed results in infected camelids.[157,158] Newer PCR probes and ELISA test may prove to be more reliable and more convenient for herd screening.[164,169] Histopathologic examination of surgical biopsy tissue samples for characteristic lesions has not been used extensively in camelids but could be a useful technique for identifying animals with subclinical disease, for establishing a definitive diagnosis for animals with clinical disease, or for diagnosing those without overt disease but with a positive test result.

Although various treatments have been attempted in some genetically superior cattle with Johne disease, the benefit of treating the individual camelid should be weighed carefully against the risk of maintaining a potential source of infection for other animals on the farm. Isoniazid or rifampin is the most common antimicrobial used, and clarithromycin may have some value as well.

A single report of *M. avium* ssp. *avium* causing disease indistinguishable from Paratuberculosis in a juvenile alpaca has been published.[170] This is a more sporadic infection in ruminants compared with *M. avium* ssp. *paratuberculosis* infection and may come from infected birds, ruminants, cervids, or other mammals. Practically, it is important because it may mimic Johne disease in many ways, potentially triggering an unnecessary regulatory response, and also because it may yield negative results on some specific *M. avium* ssp. *paratuberculosis* tests, confounding diagnosis. *M. bovis* has also been found to occasionally cause intestinal granulomas in infected animals.[159] These are a minor feature of this infection and not typically associated with diarrhea. Tuberculosis is covered more completely in Chapter 37.

Yersinia enterocolitica and *pseudotuberculosis* both have many mammalian and avian hosts, including humans. Disease is relatively rare in camelids. Transmission is thought to occur through the fecal–oral route. Mucosal breaks may facilitate entry of *Yersinia* into deeper tissues. Rarely, the animal develops acute diarrhea and toxemia and succumbs within hours. More commonly, the animal develops chronic weight loss with terminal, potentially melenic, diarrhea. Postmortem examination reveals thickening of the gut wall and regional lymphadenopathy; abscesses may be present in the gut wall, lymph nodes, or other organs. Microscopic examination reveals numerous microabscesses in the gut wall and mesenteric lymph nodes. Diagnosis can be made by performing culture of lesions or demonstrating a rising antibody titer. Tetracycline is the most commonly used drug to treat this infection, but diagnosis is usually too late for effective treatment.

Eosinophilic or Lymphocytic–Plasmocytic Enteritis (Inflammatory Bowel Disease). Eosinophilic enteritis or enterocolitis is an inflammatory bowel disease (IBD), which closely resembles Johne disease clinically.[171] In areas where mycobacterial diseases are rare, eosinophilic enteritis may be the most common cause of chronic diarrhea and weight loss. Lymphocytic–plasmocytic enteritis is similar but far less common. Historically, camelids with either form of IBD often have a longer clinical course, with recurrent bouts of diarrhea and weight loss lasting several years in some instances.

The stimuli for IBD are unknown in most cases. Feed allergies, as well as aberrant reaction to helminths, have been postulated. Whether these allergies are against common camelid parasites or aberrant ones is also unknown.

In addition to chronic diarrhea and weight loss, most affected camelids also have hypoalbuminemia and hypoproteinemia. Peripheral eosinophilia is rare, as is eosinophilia in peritoneal fluid; when those are found, they may represent that inflammation has developed deeper than the GI lamina propria. Indeed, if inflammation is confined to deeper gut tissues, diarrhea is a less consistent finding, and evidence of ascites (abdominal distention, fluid pockets noticed on ultrasonography) may predominate. Ultrasonography findings or other imaging results have not been described, but even if thickening of bowel were detected, a number of differential diagnoses would have to be considered.

Definitive diagnosis requires histologic examination of affected bowel tissue. Rectal tissue is often unaffected, so surgery would be required to obtain antemortem samples. The jejunum and the ileum are usually the best sites unless another distinct area of thickening exists. Samples should be processed routinely, including the appropriate tests to rule out mycobacterial infection. Care must be taken to interpret biopsy results correctly. Camelids, like ruminants, normally have large numbers of eosinophils in their crypts of Lieberkühn.[172] Abnormal camelids will have large numbers of eosinophils or lymphocytes and plasmocytes in the villus tips, dilated villus lacteals, and submucosal edema, or these cellular aggregates and local edema will be found in the serosal region (Figure 40-46).

Even though eosinophilia is not part of the described response to coccidia infection, some camelids with IBD of either the eosinophilic nature or lymphocytic-plasmocytic nature also have evidence of several coccidiosis, including prepatent disease first detected by tissue biopsy.[173] In those animals, or any camelid with evidence of another GI disease, those diseases should be addressed and response observed before initiating any specific treatment for IBD. Likewise, mycobacterial diseases should be thoroughly investigated and ruled out before initiating any immunosuppressive treatment.

Once a diagnosis is achieved, initial treatment includes thorough deworming, including potentially treatment of contact small animals or other possible sources of aberrant parasites. Feed changes may be instituted on an empirical basis, but this has not been particularly rewarding in the past. Supportive care, including plasma transfusions, may be necessary for severely compromised patients. Once all of this has been addressed and infectious agents have been ruled out, immunosuppressive treatment may be attempted. Sick camelids are prone to developing secondary bacterial infections when treated with steroids, so antibiotic coverage should be considered, at least in the initial stages of treatment. Immunosuppressive treatments have not seen extensive use in camelids. One possible regimen involves 3 weeks of prednisone

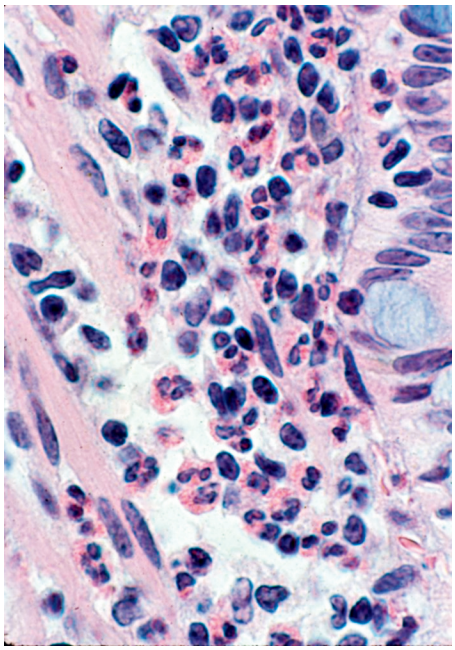

Figure 40-46 Numerous eosinophils in the small intestinal villus of a llama with eosinophilic enteritis.

(1.1 mg/kg, PO, q12h), followed by tapering doses for a fourth week.

Idiopathic Muscular Hypertrophy. Muscular hypertrophy is a rare condition, which may mimic either IBD or a phytobezoar obstruction in adult camelids.[36] No specific cause has been identified. Early clinical signs include diarrhea and weight loss progressing over several weeks to months. Eventually, the hypertrophy may become severe enough to obstruct passage of ingesta, leading to acute colic and intestinal or gastric distention. Blood abnormalities include severe hypoalbuminemia; with obstruction, hypochloremia, metabolic alkalosis, and azotemia develop. The most severe lesions are in the adoral sections of the small intestine, which is different from the predilection for ileal hypertrophy in horses with this disorder.[174] The cranial distribution makes palpation per rectum challenging, but the thickened loops and possibly gastric wall may be detected with ultrasonography. Exploratory laparotomy may be performed to obtain a biopsy sample, diagnose the cause of the chronic weight loss and diarrhea, and potentially relieve the obstruction. Grossly, the thickening differs from IBD or Johne disease in that the affected intestine does not have thickened rugose mucosal folds. Histopathologic analysis reveals marked hypertrophy of the inner longitudinal muscle layer of the small intestine with lesser hypertrophy of the outer circular layer. Affected intestine is usually too extensive to resect, so a bypass procedure may be necessary.

Internal Parasitism

The major clinical signs seen with most severe internal parasite infestations are poor growth and fiber coat, weight loss, and death. Submandibular edema and diarrhea, which are common in ruminants, are rare or late signs in camelids. Anemia is a common abnormality but well hidden by camelids until the PCV drops to less than 8%. Helminths, flukes, and *Eimeria macusaniensis* are the most common internal parasites associated with chronic disease. These infestations are discussed elsewhere in this text.

Colic

The subject of abdominal pain in camelids has been addressed a number of times over the years, with the discussion becoming progressively more detailed as clinicians gained more experience with these species.[36,44,175–182] A variety of diagnostic approaches, medical treatments, and surgical interventions have been proposed. Over the years, opinions have been in both extremes, ranging from, "We had better do surgery because the patient is worth so much" to "Surgery is useless because the patient will die anyway." The bases for these extreme views were the high proportion of underlying lesions that were a major morphologic defect, for example, a ruptured ulcer, and the general uncertainty on how to properly assess the severity and nature of the disease in a sick camelid. As clinicians have become more knowledgeable about camelid diseases and more proficient in and comfortable with dealing with these animals in general, a more systematic, evidence-based approach has developed.

One of the greatest dilemmas in the study of colic in camelids has been how to define it. By definition, *colic* means acute, paroxysmal abdominal pain and is not organ specific. Practically, we have extended the definition to include all animals with spontaneous, demonstrable abdominal pain. Most causes of abdominal discomfort in camelids involve the GI tract, although a number of disorders of other organ systems may contribute. Confounding the issue are disorders that have signs often associated with colic, for example, awkward gaits, inappetence, or increased recumbency, caused by mechanisms such as tick paralysis, neuropathy, and musculoskeletal disease, which are independent of abdominal pain, and these disorders must be differentiated from colic.

The fundamental mechanisms of abdominal pain are the same in camelids as in other species. These include activation of stretch-pain receptors in the mesentery, various ligaments, or organ capsules, muscle spasms, inflammation, and ischemia. Behavioral, anatomic, and dietary features make these mechanisms more or less operative in camelids compared with other animals, as do the characteristics of the infectious and parasitic agents affecting the camelid gut.

With most obstructive lesions, activation of the mesenteric stretch receptors is the principal cause of pain. The degree of traction on the mesenteric root relates to the degree of intestinal fluid distension. Bowel diameter may increase somewhat, but, more important, long sections of bowel are distended to their maximal diameter. Pain from mesenteric stretch is frequently more severe with colonic or distal jejunal obstruction than with duodenal obstruction because in the latter case, much of the intestinal fluid may be refluxed into the gastric compartments. In this way, camelids are like ruminants and unlike horses: The large capacity gastric compartments may sequester a considerable amount of fluid without becoming notably distended. Since these compartments already essentially sit on the floor of the abdomen, their omental and mesenteric attachments also do not become particularly taut.

Similar findings are seen with nonstrangulating entrapments. With strangulating obstructions, or advanced

obstructions, ischemia and inflammation augment the pain. With bezoars, damage to the intestinal mucosa through pressure and abrasion may lead to inflammation, weakness, and eventual rupture. This damage is most obvious when the bezoar has been pushed aborad: hemorrhagic linear erosions often are found on the oral side of the obstruction, whereas the aboral side appears healthy and empty.

Inflammation and ischemia are common mechanisms of pain with inflammatory lesions. Unfortunately, these make up a greater proportion of the total than in horses and even cattle; that is, colic in camelids is more likely to have a basis in damaged tissue than in those species.

Specific Causes

Camelids are selective eaters. They use their sensitive upper lip, rather than a broadly sweeping tongue, to guide them toward preferred feedstuffs and naturally eschew fibrous stems in preference for leafy plants with high protein content. They regurgitate forestomach contents frequently and appear to chew thoroughly, even though their molar surfaces are often irregular. Their selectivity greatly lowers their risk of ingesting a dangerous foreign body. "Hardware disease," as described in cattle, is extremely rare in camelids; one notable exception involved a llama who swallowed a Steinman pin from its jaw fracture repair.[183] The few wires we have found in the stomach are coated with mineral and show no evidence of penetration. Other obstructing, non–feed related, foreign bodies are likewise rare.

In spite of their selective eating, colic related to obstruction by *phytobezoars* is common in camelids of all ages (Figure 40-47).[36,44,182,185] Thorough chewing reduces most plant particles to manageable size, but some larger particles apparently survive. They form aggregate phytobezoars, presumably in the gastric compartments, which collect there or intermittently migrate into the lower GI tract. The C2 canal between the esophagus or C1 and C3 is never as narrow as the reticuloomasal orifice in ruminants and may not be as effective in preventing passage of large particles. Obstructing bezoars are generally slightly larger than the pyloric diameter or the maximally distended intestinal diameter, and they lodge at a site of narrowing. Additional bezoars may be found in C1 or C3, and in occasional cases, the entire gastric compartment or proximal loop of the ascending colon is filled with a *feed impaction*. Even more rarely, impaction may be caused by ingestion of sand.[46]

Bezoars most commonly lodge where the mesentery is short and the GI tract narrows: the pylorus, the duodenum aborad from the ampulla, and the loops of the spiral colon.[36] The pylorus represents the first major reduction from the gastric diameter (see Figure 40-7). The cranial duodenum has a significant reduction in diameter after the ampullae. Bezoars are also occasionally found in the jejunum, but irritation to the gut mucosa orad from the obstruction usually suggests that the obstruction originated in the duodenum and migrated. The diameter of the ascending colon decreases by over 80% from its origin by the cecum to its termination in the transverse colon (Figure 40-48). Ingesta is dehydrated over this same course from an oatmeal consistency to formed fecal pellets. The aboral centrifugal loops of the spiral colon are buried within the fat and mesentery of the wide centripetal loops, limiting the visibility, mobility, and distensibility of these later loops. Larger bezoars (>2 cm diameter) usually plug the pylorus, whereas the more common, 1- to 2-cm-diameter bezoars usually lodge in the duodenum, jejunum, or colon. Additional sites of anatomic narrowing may occur with pyloric stenosis, tumors, IBD, fungal granulomas, tapeworms, strictures, or muscular hypertrophy.

Small intestinal bezoar obstruction is the colic lesion for which we have identified the strongest age and species predilection (alpacas <1 year old). Overall, small intestinal obstruction appears to be more common in alpacas than in llamas, whereas spiral colon obstruction is more common in llamas. This may be related to the difference in absolute intestinal diameters between those species of camelids; however, duodenal phytobezoars obstruction has also been reported in an adult camel, which presumably had a larger small intestinal diameter than even a llama.[184]

Overall intestinal mobility may also play a role. The first several loops of duodenum are tightly adhered to the greater

Figure 40-47 A typical obstructing phytobezoars from the third compartment in an alpaca.

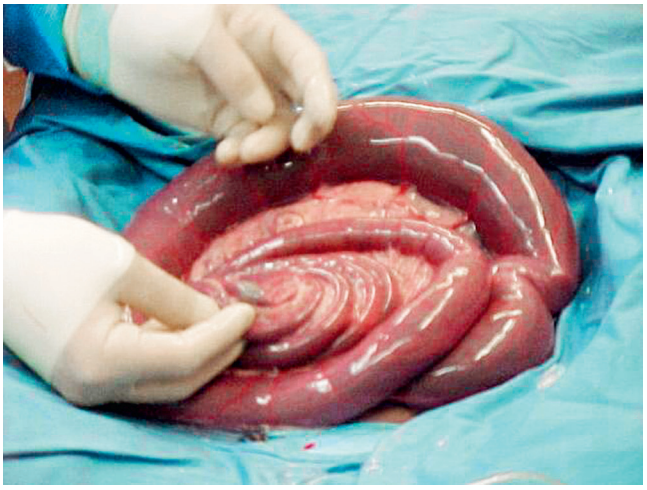

Figure 40-48 Fluid distention of the spiral colon associated with a bezoar. The obstruction can be seen at the tip of the surgeon's right index finger.

curvature of the pyloric antrum, and the remainder of the duodenum and the first two thirds of the jejunum also hang from a relatively short mesentery. The mesenteric root lengthens into the terminal jejunum, ileum, cecum, and ascending colon. Motility may be impaired by other lesions; abdominal adhesions are not uncommon, particularly around the spiral colon. Cecocolic impaction in a small number of camelids from the Netherlands and Missouri has been blamed on *dysautonomia*.[186–188] This may be a regional phenomenon or simply unrecognized in other places.

The makeup of obstructing bezoars has led to speculation as to their origin. The two most notable components are large pieces of plant stem and strands of hair. The plant fiber particles suggest that the camelid is not chewing correctly, is not ruminating adequately, has poorly regulated transport of particles into the intestinal tract, or is on a ration with poorly digestible fiber. Anecdotally, long-stem spring pasture grass was linked to an outbreak of obstructions in one alpaca herd in the Pacific Northwest. In addition to fiber length, incisor malocclusion or poor molar grinding, hurried eating behavior, anxiety, weakness, diet, and other stressors may all play a role in phytobezoar development.

The contribution of hair fiber to *trichophytobezoars* is the most significant exception to the dietary selectivity of camelids. Hair has been found in numerous GI bezoars, particularly in younger weanlings.[36,44] It is surmised that these crias ingest hair after separation from their mothers, but some preweaning hair eating and sucking has been seen as well, especially in Suri alpacas. Boredom, ectoparasites, and other skin irritants may also promote hair ingestion. Occasionally, large *trichobezoars*, or loose aggregates of hair, are found, usually in the first compartment, but these are more likely to cause mucosal irritation, inappetence, and possibly gastric distention, rather than obstruction and colic (Figure 40-49).

Forestomach fermentation in adult camelids decreases the likelihood of aberrant intestinal fermentation, and hence gas colic is uncommon. The cecum is so small that it is doubtful that much fermentative digestion occurs within it. However, aberrant fermentation may occur throughout the intestines in younger camelids or in adults with depletion of their normal gastric microbial population. In crias, this "milk colic" may be severe, but usually lasts only a few hours. As in other species, poorly digestible milk replacers or viral damage to the intestinal brush border may contribute to aberrant fermentation. In adults, gas colic is extremely rare. In all ages, infection by gas-producing bacteria may contribute to colic signs with some forms of enteritis.

Strangulations and *entrapments* are relatively infrequent, although entrapments are more common in camelids that have had previous abdominal surgery or infection, and anecdotal evidence suggests that volvulus is more common in camelids with coccidiosis or chronic abdominal inflammatory conditions.[36,126,189–192] The gastric compartments and their contents are generally immobile; the first compartment is weighed down with ingesta, and the long, tubular third compartment is anchored by the omentum along its entire greater curvature to the caudoventral first compartment. Displacement or volvulus of the gastric compartments has not been described. The short mesentery of the cranial small intestine and descending colon limits the mobility of these organs as well. The cecum is fixed along its entire mesenteric and antimesenteric length to adjacent structures and thus is unlikely to be involved in entrapments independently.

Most strangulations and entrapments involve the caudal flange of the jejunum, ileum, cecum, or ascending colon, including the spiral colon, all of which are suspended from long, narrow mesenteric attachments. These structures may migrate to essentially every unoccupied point in the abdominal cavity. Entrapments occur when one or more of these structures move through the epiploic foramen, a mesenteric rent, a tight band formed by fibrous adhesions, or a defect in the body wall or diaphragm.[36,189–192] Adhesions without entrapment and torsion of the mesenteric root of less than 360 degrees often result in signs resembling those of entrapped bowel.[36,190]

Any entrapment, as well as torsion of the mesenteric root of 360 degrees or greater (Figure 40-50), may lead to strangulation of bowel.[36,126] Torsion near the base of the mesentery is likely to involve the jejunum alone, the ileum, cecum, and colon together, or all the structures, whereas lesions further out on the mesenteric stalk may exclude the jejunum, ileum, and cecum. Multiple rotations of the

Figure 40-49 A hair-based impaction in the stomach of a young alpaca.

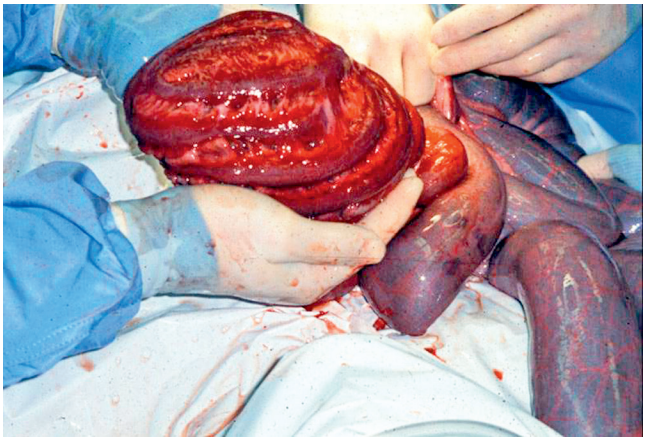

Figure 40-50 Strangulation of the spiral colon caused by mesenteric volvulus. This section of colon was successfully resected.

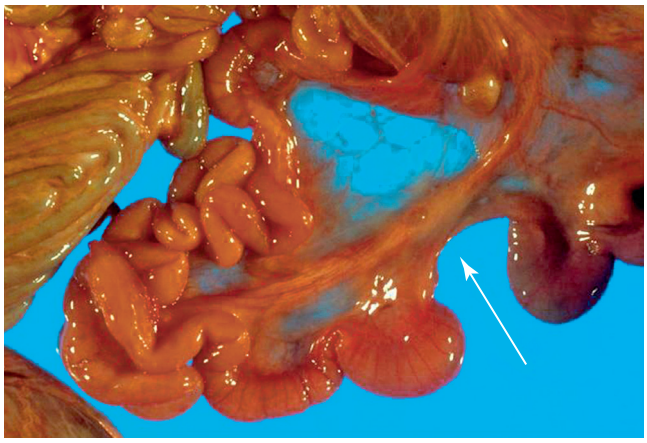

Figure 40-51 Jejunal atresia (*arrow*) in a newborn cria.

mesenteric root are common. *Intussusception* causes lesions that resemble both intraluminal obstruction and strangulation. Jejunojejunal, ileocecal, and ileocolic intussusception have been described.[36,127,193]

Risk factors for entrapments and strangulations are very different from those for intraluminal obstructions. Congenital, acquired, or iatrogenic (surgical) defects of the mesentery or walls of the peritoneal cavity are responsible for most. Adhesions related to previous surgery, abscesses, or bacterial peritonitis facilitate most of the rest. Intussusception and mesenteric torsion may be accidents or may be caused by altered intestinal motility. We have noted coccidial diarrhea in several camelids that subsequently developed torsion of the root of the ascending colon. Intussusception is rarer but may occasionally be preceded by a bout of diarrhea.

In neonatal camelids, *atresia* may lead to signs of obstruction. Atresia ani or recti are the most common, followed by atresia coli and the truly rare atresia jejuni (Figure 40-51).[194,195] Poor development of the caudal GI tract is often linked to pelvic or urogenital malformations; these possibilities should be investigated before repair is attempted. Heritability in camelids is unknown.

Slaframine poisoning is an uncommon but important cause of spasmodic colic. It presumably has the same pathogenesis as in other species and is associated with ingestion of the parasympathomimetic indolizidine alkaloid (slaframine) produced by the fungus *Rhizoctonia leguminicola*. This fungus colonizes legumes, particularly red (*Trifolium pratense*) or white (*T. repens*) clover in damp areas and subterranean clover (*T. subterraneum*) in dry areas, causing discoloration of the clover known as "blackpatch." Other plants may be susceptible as well. Camelid exposure appears to be most common in the damp seasons, particularly spring and fall, as moisture favors growth of both clover and the fungus. Exenatide, a glucagon-like peptide 1 analog, has also been associated with colic possibly related to gastric cramping in camelids.[196]

Further toxic ingestion leading to colic has occurred in camelids eating *rhododendron* or *oleander*.[63,64] Colic is a small part of the clinical presentation of this poisoning, which is discussed elsewhere in this text.

Inflammatory diseases are among the most common causes of colic in New World camelids and are often the most difficult to diagnose and treat appropriately. The causative lesions

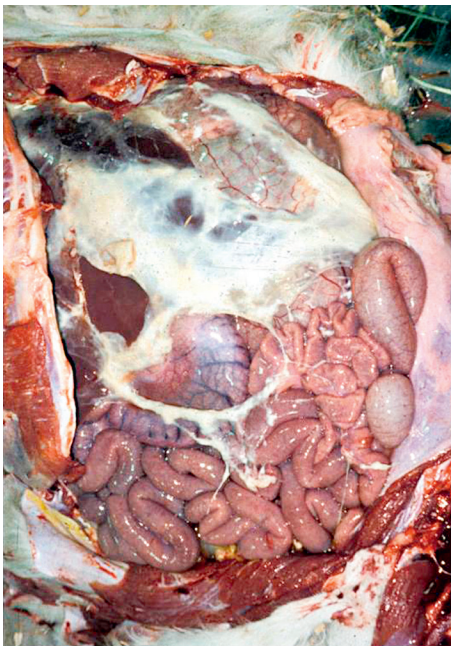

Figure 40-52 Purulent, fibrinous peritoneal fluid in a llama with *Streptococcus equi* ssp. *zooepidemicus* infection.

range from almost certainly fatal to easily survivable, but it is often not easy to judge which end of the spectrum is present in an individual camelid. The frequency and severity of the more life-threatening inflammatory lesions have been largely responsible for the perception that "colic," as a broad definition, has a poor prognosis in camelids.

Inflammatory diseases frequently cross categories between GI diseases and other abdominal or systemic diseases. One may lead to the other: Chemical, infectious, and mechanical erosive conditions of the bowel may lead to translocation of bacteria and inflammation of other abdominal structures. Peritonitis caused by various processes may lead to serosal irritation and inflammation of the bowel. *Streptococcus equi* ssp. *zooepidemicus* peritonitis is the most common cause of this (Figure 40-52).[36,197–199] With advanced lesions, it may be difficult to distinguish between primary and secondary lesions.

Inflammatory lesions that originate in the bowel are common, but many of them do not cause overt pain signs unless inflammation extends into the peritoneal cavity or leads to fluid distension of the bowel. Examples of these include forestomach acidosis, fungal gastritis, gastric saccular bezoars, ulcerative gastritis, parasitic gastroenteritis, and bacterial or viral enterocolitis. These diseases may be subclinical or cause clinical signs such as depression, weight loss, and diarrhea. However, with the exception of *Eimeria macusaniensis* infection, they rarely cause noticeable abdominal pain.[83,125] If colic is associated with one of these diseases, usually the bowel wall is necrotic or perforated (i.e., the ruptured gastric ulcer) and the abdomen has been contaminated. Hence, again with the exception of *E. macusaniensis* infection, a grave prognosis is associated with this combination of lesions and clinical signs.

Necrotizing enteritis or *enterocolitis* are among the most serious causes of acute abdomen because most affected camelids die.[36] Lesions may be diffuse, segmental, or multifocal.

The affected bowel is often firm and thickened, and its serosal surface is usually intensely red or purple, often with gray or gray-green discolored patches. Blood vessels supplying affected bowel frequently contain thrombi. Pain is likely from ischemia and the severe inflammatory reaction occurring within the bowel wall, as well as irritation of adjacent mesentery and peritoneum. Recent evidence suggests that many cases of necrotizing enterocolitis are progressions of simpler disorders such as coronavirus infection or coccidiosis.

Inflammation associated with a lesion that commonly causes colic (i.e., obstruction, entrapment, or strangulation of the bowel) likewise is associated with a worse prognosis. Inflammation with these lesions typically signifies breakdown of the mucosal barrier to bacterial translocation and weakening of the bowel wall. Gut rupture and gross contamination of the abdomen become more likely. In our estimation, such changes occur 3 to 5 days after intraluminal obstruction or entrapment and sooner (< 24 hours) with strangulations. In other case, the perforation is acute, as with rectal trauma or a uterine tear, but the end result is the same: peritonitis causing pain.[34,200]

Inflammation of the bowel secondary to peritonitis is also a common finding. The leading cause of peritonitis is sepsis; other less frequent causes include toxemia (snakebite), hepatitis, pancreatitis, urogenital leakage or infections, invasive veterinary procedures, and penetrating injuries. Signs specific to other affected organ systems may be more obvious than colic.

A number of causes of colic signs exist in camelids without primary or secondary GI lesions. They are worthy of mention because of the need to differentiate them from GI disease and to initiate correct treatment and because some of them are common. These lesions usually cause activation of capsular or ligamentous stretch-pain receptors or inflammation without peritonitis. The most common of these involve the urogenital tract; uterine torsion and urolithiasis may be the most common causes of abdominal pain encountered in some practice areas. Other urinary disorders, particularly developmental abnormalities and nephritis or ureteritis may cause pain as well. These disorders are covered in detail in Chapter 39. Hepatic distension caused by lipidosis, bacterial infection, or fasciolia-sis is an uncommon cause of colic, as is pancreatitis. These are covered in Chapter 41.

Transient colic lasting a few minutes to less than an hour is a common finding but is an infrequent cause for referral. These camelids recover, often without treatment. Most do not show any other abnormalities, except for a few that develop diarrhea. In rare instances, these colic episodes last several hours and thus make the camelid indistinguishable from one with a more severe lesion.

The reason for most transient colic is unknown. The best known cause is slaframine ingestion, which may cause episodes lasting up to 24 hours. Forestomach fermentation in adults should prevent aberrant intestinal fermentation and gas production, but neonates appear prone to milk fermentation and infection with gas-producing bacteria. Other camelids with transient colic may develop "enteritis-like" or "obstruction-like" clinical pathology abnormalities, which suggests that those pathologic processes contribute to some cases.

Transient colic tends to differentiate itself from the more insidious causes of colic by bouts of normalcy, a healthy appetite in the patients, and often some degree of normal defecation or diarrhea.

Frequency

The types of lesions described here have been blamed for up to 19% of deaths in camelids. It is possible that the percentages are even higher because colic signs may not have been recognized in all camelids, even if they were present. In our practice, colic is responsible for approximately 5% of camelid admissions. The percentage is likely to be lower in general practice, although poor recognition of signs may lead to underdiagnosis. The prevalence of individual diseases is likely to vary, depending on geographic, dietary, breed prevalence, and other factors. With many of these diseases, colic signs are transient and subtle and may not be noticed without careful, constant observation.

Diagnosis

A thorough, systematic examination of camelids with colic is helpful in differentiating the cause, appropriate treatment, and prognosis. Because of the relative infrequency of transient lesions versus more serious ones, both the diagnostic investigation and the treatment often start at a higher level of aggressiveness than in horses with routine colic. Not all of the following tests are essential in every case, but enough testing should be done to gain sufficient understanding of the health of patient and the likelihood of a particular type of lesion.

Clinical Characteristics

Except for intoxications, colic is almost always an individual animal condition in camelids. If multiple animals are affected simultaneously, especially if they also show evidence of salivation or diarrhea, slaframine, oleander, or rhododendron ingestion should be considered. If multiple animals are affected over several weeks, the list is longer, and includes postweaning trichophytobezoars, spring grass phytobezoars, *Streptococcus equi* ssp. *zooepidemicus* infection, parasites, and gastric ulcers.

To the practitioner most acquainted with colic in horses, tachycardia, abdominal distension, absence of appetite, borborygmal sounds, and fecal passage, as well as activities, including pawing, restlessness, and stretching the abdomen, are frequently taken as signs of colic. Although all of these signs may be present in some colicky camelids, many have neither tachycardia nor active signs of pain. Camelids have been described as being stoic animals that try to hide signs of illness and pain. Although it is true that signs in camelids are less visible and violent than in horses, their behavior is restive and abnormal enough to be apparent to the careful observer. Specific signs of colic include lying in an abnormal position (Figure 40-53): sternal recumbency with one or more legs extended to the side or lateral recumbency (Figure 40-54); repeatedly getting up and lying down; kicking at the abdomen; and frequently changing position while recumbent.[36] Lying quietly in sternal or lateral recumbency is not a specific sign of colic.

Level of pain and presence or absence of other signs may be used to estimate the likelihood of the various categories of lesions. Small intestinal obstruction often leads to more subtle

Figure 40-53 The subtle appearance of colic in a llama. Note the abnormal hindlimb position. This animal also appeared restless and shifted position every few minutes.

Figure 40-54 An alpaca in lateral recumbency because of colic. Note the disturbance of the straw from rolling.

signs than other types of lesions because fluid is refluxed into the gastric compartments and inflammation is minimal. This is most true for obstructions near the pylorus; the further into the jejunum the bezoar is pushed, the more tension exists on the mesentery and the more painful the camelid appears. Thus, in addition to the importance of recognizing pain signs, it is important to remember that camelids with mild to subtle signs still may have life-threatening obstructive lesions.

Very cranial obstructions may cause several other abnormalities that help differentiate them from other lesions. Fluid accumulation in the forestomach leads to bilateral, ventral abdominal distension, and a pot-bellied appearance (Figure 40-55). Gas may be eructated until the gastric fluid level reaches the cardiac sphincter, causing muscular spasms. After that time, bloat may be noticed. Other signs include dehydration, depression, and in rare cases, vomiting. Abdominal

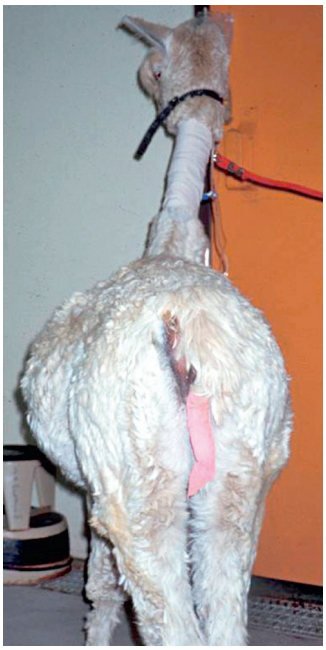

Figure 40-55 Abdominal distention in an alpaca with a midjejunal intussusception.

distension is difficult to assess in full-fleeced camelids; direct external palpation ("bear-hug") may be necessary. Other conditions, including advanced pregnancy, effusive peritonitis, venous congestion (as with strangulation), and enteritis may lead to abdominal distension without gastric engorgement. These conditions may be distinguished by other characteristics. It is also appropriate to mention here that subjective assessment of cranial abdominal pain by palpation of the flanks is extremely difficult to interpret; most camelids do not like being touched in that area, and it is natural for them to flinch, jump, or lie down when touched.

Dehydration and depression with small intestinal obstruction are caused by sequestration of ingested and secreted fluids, with minimal functional intestinal absorptive surface area. Signs worsen with proximity to the pylorus, completeness of the obstruction, and time. Thus, a camelid with duodenal phytobezoar obstruction is likely to have subtle colic signs that abate after a few days because of progressive dehydration and depression. In contrast, a camelid with a colonic bezoar obstruction that leaves most of its intestinal absorptive epithelium functional is likely to have more obvious pain signs early in the course of the disease and to develop minimal dehydration.

Camelids with strangulating obstructions or sepsis also develop signs of shock, dehydration, and depression (and also distension, as previously mentioned) over 6 to 24 hours because of venous pooling, peritoneal effusion, and the inflammatory response. Such camelids usually appear to be more in pain and differ in other aspects of the diagnostic evaluation but may be extremely difficult to differentiate from camelids with jejunal obstruction or any small intestinal obstruction with inflammatory complications.

In addition to the more obvious colic signs and possible dehydration and depression, camelids with inflammatory or

cecocolic diseases may also show tenesmus. Camelids usually defecate and urinate at a communal dung pile several times a day but rarely spend more than a minute or two at the pile or assume the posture when away from the pile. Tenesmus with obstruction is usually nonproductive, whereas camelids with enteritis or peritonitis may have diarrhea. Thus, tenesmus with diarrhea and colic signs suggests a severe inflammatory condition. Tenesmus is also a common sign with urogenital disorders.

Anorexia, lack of feces, and gastric hypomotility are common findings in camelids with GI disease but lack specificity because they are also seen with diseases of other organ systems. However, finding appetence, normal fecal passage, and normal ruminations in a camelid with colic strongly supports a mild, transient problem, a noninflammatory, non-GI condition (i.e., uterine torsion), or the early stages of more severe disease. These signs should be monitored for resolution or progression.

Fecal passage may provide other insights into the nature of the disease. Camelids with obstructions may pass normal feces for 1 to 3 days (longer with small intestinal obstruction than with cecocolic obstruction), but eventually, they pass dry, mucus-covered feces and later stop defecating altogether. Camelids with entrapments may continue to pass small quantities of normal or blood-tinged feces, whereas camelids with strangulations and necrotic enteritis and peritonitis may pass diarrhea in decreasing quantities. Consistent passage of small amounts of diarrhea in spite of ongoing pain signs for up to 4 days is common with *E. macusaniensis* or peritoneal *Streptococcus* infection. Continued, profuse diarrhea or normal feces are rare in camelids with colic caused by GI lesions requiring surgical repair.

Vital signs have been of little value in determining severity of colic and the presence of surgical lesions. Camelids with all types of lesions have been recorded as having low, normal, and high pulse and respiratory rates. We tend to think that the more normal the vital signs, the better is the prognosis, but this is not always true. High rectal temperatures are most common with peritonitis but are not specific to the cause of inflammation. Low rectal temperatures may occur with most disorders and are usually a sign of circulatory compromise and more severe disease. When interpreting heart rate, it is imperative to remember that most healthy adult camelids have heart rates of 72 beats per minute (beats/min) or less and that values between 72 beats/min and the published high end of 90 beats/min may reflect pain or shock.

Rectal Palpation

Although juvenile llamas and most alpacas may be too small, rectal examination of most adult llamas is possible with effective restraint (preferably in a camelid chute), adequate lubrication, and, if necessary, chemical sedation. Adding 20 mL of a local anesthetic agent (2% lidocaine HCl) to 100 mL of lubricant (for adults) and injecting the mixture into the rectum by plastic dose syringe or insemination pipette relaxes the anal sphincters and caudal rectum sufficiently to allow a thorough rectal examination. Individuals with large hands may not be able to do this and should stop if they encounter too much resistance. Rectal injuries and bladder rupture have been reported when care was not exercised.[38]

Rectal examination of the llama is similar to that of the cow; the bladder, the female reproductive tract, the left kidney, and the caudal first gastric compartment usually can be palpated. The right abdomen usually has no palpable viscera. Rectal palpation is most useful for abnormalities in the caudal abdomen, especially for urogenital lesions. Obstruction, inflammation, entrapment, or strangulation of the terminal small intestine and ascending colon are likely to be detected by the presence of distended bowel or tight mesenteric roots, whereas generalized inflammatory conditions and small intestinal obstructions generally cause no palpable abnormalities.

Except for diagnosing uterine torsion, rectal examination of colicky camelids has been largely supplanted by flank ultrasonography in our practice.

Clinical Pathology

A thorough clinicopathologic evaluation helps identify lesions and select the appropriate treatment. In general, the more normal the evaluation, the better is the health of the bowel and the better is the prognosis for survival. Camelids with hematologic abnormalities are likely to have inflammatory lesions, and camelids with small intestinal obstruction are likely to have metabolic abnormalities.

Complete Blood Cell Count

Because of the frequency of inflammatory lesions in camelids with colic, differential blood counts are frequently abnormal. These changes may be obscured by stress neutrophilia, a common finding in sick camelids. More specific evidence of inflammation includes immature granulocytes, toxic changes in granulocytes, hyperfibrinogenemia, and neutropenia (if this last change is not masked by stress changes in the leukogram). Inflammatory changes are to be expected in camelids with peritonitis or enteritis; in camelids with obstructive bowel diseases they are an undesirable sign of advanced disease.

Acid–Base Balance

Camelids with small intestinal obstruction frequently develop metabolic alkalosis because of the sequestration of gastric HCl within the gut lumen.[36,44,177] Small intestinal entrapments and strangulations and longstanding cecocolic obstruction occasionally cause mild metabolic alkalosis, but such lesions usually cause so much pain that veterinary attention precedes development of this abnormality. Camelids with inflammatory or ischemic conditions have a tendency toward metabolic acidosis. This is more likely to be caused by lactate production than intestinal bicarbonate loss and is often accompanied by hyperlactemia and a high anion gap. Metabolic acidosis may also be seen with longstanding intestinal obstructive lesions as they become more inflammatory; hence, it is a sign of more severe disease.

Electrolytes

Serum chloride is the most important electrolyte for diagnosing GI causes of colic.[38,46,177] Chloride ion is sequestered in the gastric compartments with cranial obstructions but to a much lesser degree with more aboral obstructions. Thus, hypochloremia (<106 mEq/L) is usually present under similar conditions as metabolic alkalosis. Hypochloremia may also be

found with metabolic acidosis in camelids with enteritis; hyponatremia and hypoproteinemia usually accompany it in these cases. *Eimeria macusaniensis* is the most common cause of enteritis associated with colic.[83]

Sequestered chloride may also be detected in gastric fluid. This fluid may be retrieved by stomach tube or paracentesis.[36,38] Issues relating to method of analysis have been outlined earlier in this chapter. When analyzed correctly, forestomach fluid supernatant appears to have a similar chloride concentration as cattle (normal: <20 mEq/L; camelids with anorexia: <30 mEq/L). Higher values may be expected in camelids with obstructions that are more cranial, complete, and longstanding.[36] Values greater than 40 mEq/L almost certainly reflect an obstruction. Smaller increases are seen with strangulating lesions because of their rapid decline in clinical parameters.

Hypokalemia is almost ubiquitous in camelids with GI disease and worsens with duration and severity of anorexia and tendency toward metabolic alkalosis.[180] Thus camelids with small intestinal obstructions have some of the lowest serum potassium concentrations, but this finding should be expected in camelids with other GI disorders as well.

Hyponatremia with hypochloremia but not alkalosis is a relatively rare finding in sick camelids and, with colic, is highly suggestive of enteritis such as *E. macusaniensis* infection.[36,81] Hypoalbuminemia and hypoproteinemia are also common with enteritis.

Other Blood Constituents

Measures of dehydration and renal function are also likely to be most severe in camelids with small intestinal obstruction and also in camelids in the shock phase, which occurs with many of the inflammatory conditions.[36] Creatinine is of more value than urea nitrogen in some cases because urea nitrogen is affected by the rate of ruminal utilization. Albumin and serum total protein also tend to increase with dehydration, but camelids appear to lose or catabolize protein with many GI disorders.[177] Therefore, high and low albumin concentrations are associated with more severe disease. Additionally, protein is lost because of enteritis. Severe hypoproteinemia combined with continued passage of soft feces is suggestive of *E. macusaniensis* infection.

Abdominal Paracentesis

Abdominal fluid may be obtained from most sick camelids by using the right paracostal approach.[35] Ultrasound guidance may improve the yield but is usually not necessary. Protein concentrations and nucleated cell counts of peritoneal fluid are lowest in camelids with the least damage to the bowel and without peritonitis. Thus, they are most normal with early intraluminal obstructions and nonstrangulating entrapments. Abnormal peritoneal fluid with these lesions (often accompanied by abnormal CBC results) indicates progression of the lesion and inflammatory complications. Strangulating obstructions with venous occlusion and intussusception may increase the volume and RBC count of the peritoneal fluid before they increase protein and WBC counts. Very high (<4 g/dL) protein concentrations and nucleated cell counts (<10,000 cells/μL) are indicative of inflammatory diseases, as are low cell counts with toxic changes to neutrophils. Bacteria and plant material may be seen with sepsis and visceral rupture (see Figure 40-51). Peritoneal fluid activities of amylase and lipase higher than serum activities may be associated with pancreatitis.[201]

Peritoneal lactate higher than systemic lactate tends to reflect compromise of the bowel in general, strangulation, or effusive cellular peritonitis. The greater the divergence, the more severe is the lesion. In general, the more normal the peritoneal fluid, the less severe is the lesion. Uroperitoneum in camelids is similar to that of other species. Exceptions to this rule include camelids with mature adhesions, which may have uncorrectable lesions but normal fluid and camelids with certain inflammatory conditions (pancreatitis, streptococcal peritonitis), which may have correctable lesions in spite of very abnormal fluid.

Abdominal Imaging

Radiography

Plain abdominal imaging studies are of limited usefulness in diagnosing the cause of colic. In adult camelids, most abdominal detail is obscured by feed material in the gastric compartments, and few lesions lead to gas accumulation within the bowel. Neonatal llamas have a higher incidence of abnormal intestinal fermentative disorders and smaller gastric compartments; thus imaging studies of colicky crias may reveal elaborate intestinal gas patterns or potentially impactions of the forestomach or colon.

Contrast studies have been performed on few occasions. They may be diagnostic with some obstructive lesions and are useful in distinguishing whether a distended viscus is first compartment or not (Figures 40-56 and 40-57). To highlight gastric structures in crias, 25 to 50 mL of barium paste or liquid or a 50:50 mixture is enough. Unfortunately, intestinal motility is often impaired by the time many camelids are brought to facilities with adequate radiography equipment, making the interpretation of contrast studies difficult. Mineralized bezoars or sand impactions may be visible without contrast.[46]

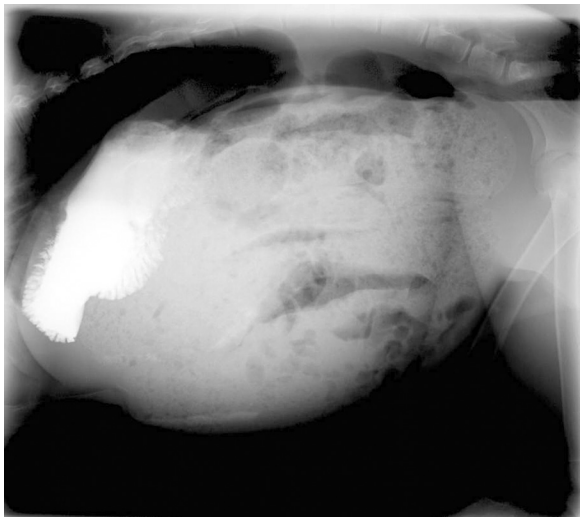

Figure 40-56 Contrast material within the first compartment of a cria with abdominal distention from colonic impaction.

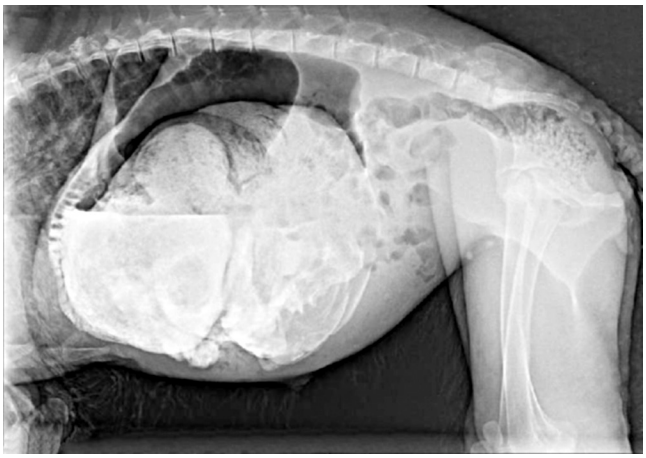

Figure 40-57 Contrast material within the first compartment of a cria with abdominal distention caused by first compartment impaction.

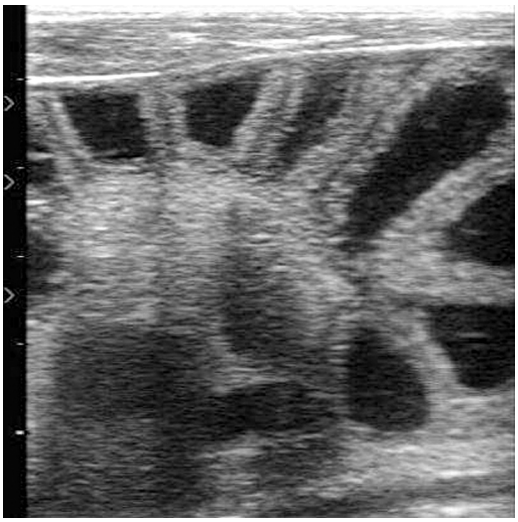

Figure 40-58 Transcutaneous ultrasonographic appearance of fluid-distended bowel associated with bezoar obstruction.

Ultrasonography

Ultrasonography of the abdomen and its viscera may be performed transrectally or transcutaneously. It is ideal for detecting fluid accumulation within the bowel or the abdomen. Transrectal imaging is best for the same lesions as those palpated per rectum, that is, those in the caudal abdomen. This technique has the advantage that it can be performed in many camelids that are too small for palpation. Transcutaneous imaging through the right paralumbar fossa shows the same structures and reveals the size and contents of the gastric compartments, as well as the amount, location, and nature of free fluid.[31]

Ultrasonography allows assessment of bowel contents and motility. Changes in wall thickness are usually subtle. Normal intestines should have intermittent pockets of hypoechoic fluid and clear evidence of peristalsis. Obstructed or damaged bowel has larger, stable pockets of fluid with minimal evidence of progressive motility (Figure 40-58).[49] This is easier to find with lesions of the colon or jejunum because of their accessibility. Cranial obstruction may be inferred on finding fluid distension or atony of the third gastric compartment. Distinguishing obstructed bowel from severely inflamed bowel is difficult; on occasion, the lumen of the inflamed bowel may have gas shadowing and that of obstructed bowel may contain a hyperechoic mass of fiber or intussusception. Inflamed, distended bowel may also show evidence of progressive motility.

A large amount of abdominal fluid is present with effusive peritonitis, severely inflamed bowel, venous occlusion with strangulation, uroperitoneum, and other ascites-causing conditions. The more inflammatory the cause, the more hyperechoic flecks are likely to be present. As mentioned previously, finding large pockets of fluid ultrasonographically aids in successful paracentesis. Ultrasonography may also be used to look for uterine torsion, urolithiasis, renal disease, liver disease, mass lesions, and pancreatitis.

Summary

Table 40-2 summarizes the most characteristic clinical findings for the various types of lesions. The typical camelid with small intestinal obstruction is mildly painful, has abdominal distension, is dehydrated, and has hypochloremic metabolic alkalosis with a high gastric fluid chloride concentration. Because of the mild signs, camelids with this type of lesion often are not noted to be sick for several days and often develop mucosal injury and inflammatory complications before detection. Thus, camelids with small intestinal obstruction have historically done poorly. Camelids with cecocolic obstruction have overt signs of colic, minimal dehydration and metabolic disturbances, and lesions that are easily detectable by rectal palpation or ultrasonography. Because of more timely detection and treatment and less tissue and metabolic compromise, camelids with cecocolic obstruction have a better prognosis. Camelids with entrapments and strangulations generally act like camelids with intraluminal obstruction of the same site except that camelids with strangulating lesions deteriorate more quickly without appropriate treatment. Hyperlactemia and high lactate content in serosanguinous abdominal fluid are characteristics of strangulation. Camelids with inflammatory lesions tend to have overt colic signs, but these may be overshadowed by depression. They tend to have abnormal CBC, blood protein and electrolyte concentrations, and peritoneal fluid but have other clinical features which overlap with those seen with obstructive lesions. Prognosis with inflammatory lesions is variable; most camelids have a poor prognosis, but those with some specific diseases (pancreatitis, *E. macusaniensis* enteritis, streptococcal peritonitis, mild bowel inflammation) may do well.

When evaluating individual camelids, it is important to remember that the severity or presence of these findings varies on a case-to-case basis. It is also important to remember that obstructive type lesions eventually take on more inflammatory characteristics; for example, high peritoneal fluid protein concentration and circulating band cells are not unusual in camelids with longstanding duodenal obstruction.

When assessing camelids with colic, our goals are (1) to select the appropriate treatment and (2) to make a diagnosis. The three treatment options are (1) medical management, (2) surgical exploration and correction, and (3) euthanasia in the face of inability to provide relief using one of the other

two modalities. In general, camelids with transient colic, a portion of those with inflammatory lesions, and a small portion of those with obstructive lesions respond to medical treatment. A portion of those with obstructive lesions and a small portion of those with inflammatory lesions benefit from surgery. Failure of either of these modalities is usually caused by the presence of advanced inflammatory lesions. In some cases, as with ruptured gastric ulcers, these lesions may occur at the same time as the first clinical signs and are therefore unavoidable. In other cases, as with abrasion of the bowel wall obstructed by a bezoar, advanced inflammatory lesions may develop because of failure to recognize the significance of earlier clinical findings.

Treatment

Surgical exploration of a camelid with an acute abdomen can be a difficult decision. Historically, some reluctance to perform surgery exists, mainly because of the perception that most camelids with colic (including many with intraluminal obstructions) eventually have uncorrectable lesions. This perception has been reinforced by poor survival rates following surgery. However, improvements in diagnostic modalities, anesthetic management, and clinical decision making have led to many successful surgeries.

Our experiences with camelids are far less extensive than those with horses and cattle, but using criteria similar to those used in these species appears to give us reasonable guidelines as to when to do surgery in camelids with colic. This includes clinical signs (particularly persistent pain with lack of fresh fecal production) that worsen or do not improve with medical treatment. It is also difficult to determine how long to continue medical treatment before resorting to surgery. Although some evidence suggests that some intraluminal obstructions resolve with medical treatment, other evidence suggests that delaying surgery in camelids with intraluminal obstruction may be detrimental; many intestinal ruptures found at surgery or necropsy occur at the site of a chronic intraluminal obstruction. Our best estimates indicate that gut friability becomes life threatening within 4 days of obstruction. Because cranial obstructions usually cause less overt signs compared with caudal obstructions, damage to the bowel wall may be more advanced on first examination of camelids with cranial obstructions. Abnormalities in the leukogram or peritoneal fluid and signs of sepsis often accompany bowel compromise.

Rising awareness of *E. macusaniensis* as a cause of colic has complicated the use of pain as the major determinant for surgical exploration. Camelids with parasitic enteritis tend to pass some normal feces, have blood work suggestive of enteritis, and have ultrasonographic evidence of progressive motility.

Medical Treatment

Medical treatment of colic focuses on the principles of patient stabilization and maintenance, pain control, treatment of infectious and inflammatory conditions, and occasionally attempts to resolve the obstruction.

Patient stabilization involves shock treatment and restoration of hydration, electrolyte, acid–base, and protein norms. Shock treatment is most vital with strangulation and inflammatory lesions. IV fluids are key. An initial bolus of up to 5% of body weight (10% in neonates) may be administered, if the patient will tolerate it and is not too hypoproteinemic. With alkalosis, 0.9% sodium chloride may be used, but with most disorders, a polyionic, buffered fluid is adequate. Sodium bicarbonate may be added to correct severe acidosis (pH <7.25), but this treatment is not particularly effective in the face of lactic acidosis. Supplemental potassium chloride, administered at less than 0.5 mEq/kg/hr may also be helpful. Because of the small patient size and rarity of severe hyponatremia or hypochloremia, hypertonic saline is rarely necessary. Plasma transfusion or administration of a synthetic colloid may be necessary to treat hypoproteinemia. We typically administer plasma if the blood albumin concentration is less than 2 g/dL or total protein is less than 4 g/dL.

After the initial fluid bolus, we typically continue IV fluids at 2 mL/kg/hr in adults and up to 4 mg/kg/hr in neonates until the camelid is able to obtain its own water.

Stabilization also involves common sense procedures such as protection from the elements; removal from stressful circumstances; provision of heat, water, and possibly feed; and protection from other animals. A bedded, sheltered stall with a controlled temperature is an effective measure.

Pain control may be difficult to achieve and has the temporary danger of masking a surgical lesion. Flunixin meglumine (1.1 mg/kg, IV, q12h) or butorphanol tartrate (up to 0.05 mg/kg, IV or IM, q4-6h) offer relief from mild pain but are usually inadequate to control colic. In one case under our care, butorphanol appeared to allow sufficient patient relaxation for an alpaca to pass an enormous (>5 cm in diameter) bezoar, but that is rare. More aggressive pain management includes lidocaine (1.3 mg/kg IV over 15 minutes as a loading dose, followed by 0.05 mg/kg/min) or ketamine (40 mcg/kg/min) as constant rate infusions. Other options include epidural morphine (0.1–0.3 mg/kg of preservative-free morphine diluted to 12 mL), buprenorphine (3 mcg/kg, IM, q4-6h), or transdermal fentanyl (300 mcg/hr in an adult llama). Fentanyl typically takes too long to control pain to be practical in the treatment of colic. α_2-adrenergic agonists generally should be avoided because of their contribution to cardiovascular compromise and displacement of the soft palate.

Flunixin meglumine is also useful as an antiinflammatory. We typically use a dose of 0.5 mg/kg, IV, q12h, unless circumstances justify a higher dose. Antimicrobials round out the medical treatment plan. Broad-spectrum antibiotics such as penicillin–aminoglycoside combinations may be augmented with metronidazole to increase the anaerobic coverage. Ceftiofur sodium may not have sufficient distribution to the peritoneal cavity to be effective there. If coccidia are suspected, the appropriate medication should be used.

Oral and parenteral agents to help move an obstruction have shown little promise in our practice. Oral agents tend to sit in the atonic, distended stomach, and parenteral agents are not able to move the mass.

Determining how long to attempt medical treatment requires careful assessment of the patient. If we are not able to control the pain, we are reluctant to delay surgery beyond more than 24 hours in any camelid showing evidence of obstruction and diminishing fecal passage. To continue

TABLE 40-2 Known Causes of Colic in New World Camelids and Their Clinical Characteristics

Lesion	Other Signs	Level of Pain	Dehydration/ Shock	Complete Blood Cell Count
Bezoar or impaction, pylorus or small intestine	Obtundation, feed refusal, decreased fecal passage, abdominal distention; ± regurgitation	Low, unless bezoar migrates	Progressive, often advanced by time of detection	Normal or stress, progressing to inflammatory
Bezoar or impaction, large intestine	Feed refusal, decreased fecal passage, ± abdominal distention, tenesmus	High	Unlikely, progressing over 3–4 days	Normal or stress, progressing to inflammatory
Intussusception (small intestinal, ileocecal, ileocecocolic, colocolic)	"Raspberry jam" feces in decreasing amounts, abdominal distention with gurgling	High	Progressive over 2–4 days	Inflammatory
Entrapment hernia, mesenteric rent, epiploic foramen	Progressive abdominal distention, decreased fecal passage ± feed refusal	Mild to moderate	Unlikely, progressing over 3–4 days	Normal or stress, progressing to inflammatory
Strangulation, mesenteric volvulus	Rapidly progressive feed refusal and obtundation	High	Likely, progresses over 6–24 hours	Stress
Rhododendron, oleander ingestion	Vomiting	Low	Rare, except with oleander	Normal or stress
Slaframine ingestion	Salivation	Episodic	Rare, unless salivary loss is severe	Stress
Coccidiosis, especially with *E. macusaniensis*	Weight loss, passage of mildly loose feces to diarrhea	Low to high	Rare unless advanced	Normal to inflammatory, mild anemia
Clostridial enteritis	Gas distention, absent to hemorrhagic feces, severe obtundation ± fever	High to extreme	Moderate to severe	Inflammatory
Uterine torsion	Near-term, intermittent feed refusal, continued fecal passage	High with spasms	Rare, unless rupture, thrombosis	Normal or stress
Gastric or intestinal leakage (ruptured ulcer or other causes)	Obtundation, feed refusal, declining fecal passage	Low to high	Progressive	Normal, stress, or inflammatory
Urolithiasis, urinary tract malformations	Dysuria, straining, urine dribbling	Low to moderate	Progressive over 1–5 days	Normal or stress
Nephritis or ureteritis	Feed refusal, possible continued fecal passage	High to extreme	Progressive	Normal, stress, or inflammatory
Peritonitis	Feed refusal, ± fever, dyspnea, injected mucous membranes	Mild to high	Progressive	Inflammatory, unless localized
Pancreatitis	Feed refusal, ± fever	Mild to moderate	Mild to progressive	Normal, stress or inflammatory
Fascioliasis, hepatic lipidosis	Feed refusal, obtundation	Mild to moderate	Mild to severe	Normal, stress, to mild inflammatory, often with mild anemia

Acid–Base	Blood Biochemistry	Abdominal Fluid	Rectal Examination	Ultrasonography
Progressive metabolic alkalosis, eventual hyperlactemia	Low chloride in blood, high in C1 fluid, progressive hyperproteinemia and azotemia	Normal, progressing to inflammatory	Normal	Enlarged C3 ± distended SI and ileus
Normal, progressing to alkalosis	Normal, progressing to hypochloremia	Normal, progressing to inflammatory	Distended intestine	Distended intestine and ileus
Alkalosis with small intestinal lesions, develops over 2–4 days with large intestinal lesions	Hypochloremia with SI; slower developing with LI	Hemorrhagic, increasingly inflammatory	Distended intestine	Distended intestine and ileus
Alkalosis with SI lesions, develops over 2–4 days with LI	Hypochloremia with SI; slower developing with LI	Normal, progressing to inflammatory	Distended intestine	Distended intestine and ileus
Normal acutely, rapidly progresses, usually to metabolic acidosis	Normal, progressing to azotemia and hyperlactemia	Hemorrhagic, increasingly inflammatory	± distended intestine	Ileus, ± distended intestine, increased free fluid
Acidosis with shock	Normal	Normal, unless rupture	Normal	Ileus
Acidosis from salivary loss	Normal	Normal	Normal	Normal
Normal, unless in shock	Mild hypochloremia and hyponatremia, hypoproteinemia	Transudate, progressing to inflammatory	Normal to mild fluid distension	Normal to segmental fluid distension, progressive motility
Progressive metabolic acidosis	Normal electrolytes, hypoproteinemia	Normal, progressing to hemorrhagic, inflammatory	Normal to mild fluid and gas distension	Segmental fluid dissentions, gas-shadowing, ileus
Normal	Normal	Normal	Normal, except uterus	Normal
Normal to metabolic acidosis	Normal to mild hypochloremia	Normal to inflammatory, occasionally microorganisms	Normal	Normal to areas of mixed echogenicity compatible with leakage
Normal, some alkalosis with rupture or acidosis with shock	Normal, some hypochloremia and hyponatremia with rupture	Normal to inflammatory, copious with uroperitoneum	Gastrointestinal (GI) system normal, pulsing urethra, ± big or absent bladder	GI normal, big to absent bladder, ± urethral distension, mineral shadows, uroperitoneum
Normal, some alkalosis with rupture or acidosis with shock	Normal, some hypochloremia and hyponatremia with rupture	Normal, rarely inflammatory changes	Normal	Mineral shadowing or hyperechogenicity in urinary tract, ± evidence of retroperitoneal fluid leakage
Normal to acidosis with shock	Normal	Inflammatory, occasionally microorganisms	Normal	Increased hyperechoic fluid
Normal to acidosis with shock	Normal	Normal to inflammatory, ± increases in amylase and lipase	Normal	Hyperechoic foci near pancreas
Normal to acidosis with shock, ketonemia	Liver enzyme abnormalities, ± hypoproteinemia, hyperbilirubinemia, and increases in fat fractions	Transudate to normal	Normal	Possible hepatic changes

medical treatment even that long, the camelid must show no evidence of bowel degradation on physical, blood, or peritoneal fluid examinations. If the camelid continues to pass diarrhea or soft feces, possibly has a fever, and ultrasonography reveals evidence of progressive motility, the high prevalence of *E. macusaniensis* infection in our area often warrants extending medical treatment for 2 to 3 days.

Criteria for surgery include persistent pain and absence of feces; finding evidence strongly suggestive of an obstructive lesion, entrapment, or strangulation; and sufficient confidence in ruling out inflammatory conditions such as peritonitis, pancreatitis, enteritis, and hepatitis. Evidence for a small intestinal obstruction includes hypochloremic metabolic alkalosis, a high forestomach chloride concentration, abdominal distension, and possibly regurgitation or vomiting. It must be kept in mind that camelids with obstructions fairly far forward in the intestinal tract may not have marked pain signs but rather may appear progressively more lethargic and dehydrated.

Evidence for a distal small intestinal or colonic obstruction includes rectal or ultrasonographic identification of fluid-distended loops of bowel without good progressive motility in camelids showing overt pain signs. They may also have hypochloremic metabolic acidosis, but that usually signifies that they have been obstructed for several days, making them more urgent surgery candidates. Characteristics of volvulus or intussusception include initial high levels of pain, serosanguinous peritoneal fluid, and high concentrations of lactate in blood and especially peritoneal fluid. Camelids with intussusception may continue to pass feces that are usually visibly abnormal, with evidence of blood.

The important thing is to explore camelids with surgical lesions in a timely fashion and also to address medical lesions with confidence. In choosing the surgical option, ruling out peritonitis, pancreatitis, enteritis, and other medical conditions is as important as diagnosing lesions that are candidates for surgery.

Surgical Approaches

Ventral midline, paracostal, and paralumbar celiotomy all have been performed successfully to diagnose and treat GI disorders in camelids. Left paralumbar laparotomy offers the best exposure to the first gastric compartment but poor exposure to the remaining abdominal viscera. Therefore, it is not recommended unless the lesion is thought to affect those compartments. The choice between ventral midline and right paralumbar exploratory laparotomy often is based on the preference of the surgeon, as both approaches allow for palpation, visualization, and exteriorization of most abdominal viscera, including the spiral colon, cecum, most of the small intestine, and the pyloric antrum of the third gastric compartment. The pyloric antrum may be difficult to exteriorize through a ventral midline incision when the third gastric compartment is distended with fluid. Therefore, a right paracostal or paralumbar incision may be preferable when a gastric or duodenal lesion is suspected.[36,44] Herniation of GI viscera through the body wall incision may occur after ventral midline laparotomy, although we have noted this to occur more frequently after cesarean section in large female llamas than after general surgical exploration of the abdomen of camelids.

Abdominal surgery procedures are covered in depth in Chapter 57.

Postoperative Peritonitis

Diffuse abdominal infections are a common complication after colic surgery. In our experience, they are frequently fatal and may be the cause of death in camelids that underwent an otherwise successful procedure. Affected camelids are depressed, anorexic, spend a large amount of time in the recumbent position, pass scant amounts of feces, and develop respiratory distress. They typically have degenerative neutropenia on leukogram and high peritoneal fluid protein concentrations and nucleated cell counts. Fever is an inconsistent finding. In many cases, the infection arises from bowel compromise before surgery. Thus, it is a consequence of the delay, not the procedure.

If postoperative peritonitis is detected or suspected, it should be treated by following the general guidelines for sepsis or peritonitis treatment. Fluid support, antiinflammatory medications, and broad-spectrum antibiotics diffusing well to the abdomen are the cornerstones. Abdominal drains and local flushing of infected wounds may be beneficial as well.

Atresia

Segmental atresia of the intestinal tract is an uncommon occurrence in neonatal alpacas and llamas. Atresia ani is the most common anomaly, but atresia of the jejunum or colon also occurs. As many camelid owners give enemas to neonates to stimulate meconium passage, the lack of an external orifice, atresia ani, often is noted within a few hours of birth. If not noted, the cria may become clinically ill or develop abdominal distension. Techniques to correct atresia ani in camelids are similar to those used in other species (Figure 40-59). As with neonatal ruminants, although surgical correction usually is fairly simple, camelids with atresia ani often have other

Figure 40-59 Surgically corrected atresia ani in a cria.

developmental abnormalities. Specifically, we have seen atresia jejuni, horseshoe kidneys, renal agenesis, pelvic abnormalities, and skull malformation in conjunction with atresia ani. Thus, in spite of the ease of repair of the one problem, a good prognosis cannot be given until other malformations are ruled out.

Atresia jejuni (see Figure 40-51) and atresia coli are less common but more rapidly lethal than atresia ani. Although crias with these disorders do not pass meconium, they do have an anus, so the lesion is not readily apparent on inspection of the newborn. Atresia jejuni is the most serious segmental disorder because less healthy gut surface is available for nutrient and colostral absorption. This makes the neonate highly susceptible to hypoglycemia and infectious disease. Crias with intestinal atresia are usually bright at birth but develop progressive depression and abdominal distension over the first few days of life. Colic and tenesmus may be seen with atresia coli. No reports of surgical correction of intestinal atresia in camelids have been published, but it is likely that procedures developed for ruminants would be adequate. We have seen a cria with atresia jejuni at two separate sites, so the entire bowel should be examined before surgical correction is performed. Broad-spectrum antimicrobial drugs, IV fluids, and plasma are often required before and after surgery. Although a genetic basis for atresia coli and ani exist in other species, we do not have enough data to assess the genetic basis of these abnormalities in camelids. Greater experience may provide more information about the hereditary risk.

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