Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company’s public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.
Gross Lesions of Alimentary Disease in Adult Cattle

Bradley L. Njaa, DVM, MVSc a,*, Roger J. Panciera, DVM, PhD a, Edward G. Clark, DVM, MVSc b, Catherine G. Lamm, DVM, MRCVS c

NECROPSY EXAMINATION

Gross examination of the gastrointestinal tract (GIT) of cattle is one of the most challenging parts of a necropsy. The GIT is one of the largest and heaviest organ systems of the animal. It is initially divided into the upper GIT (oral cavity, esophagus, forestomachs, abomasum) and the lower GIT (intestines, rectum, and anus). In healthy cattle, the forestomachs occupy more than half the abdominal cavity. The rumen (Fig. 1) alone has a capacity range of 102 to 148 L (which approximates to a similar weight in kilograms), whereas the abomasal volume ranges from 10 to 20 L.1 The intestines occupy a much smaller space in the peritoneal cavity but have a length that is considerable, ranging from 33 to 63 m.1 Thus, a thorough examination is physically demanding because of its size and requires a significant proportion of time to adequately examine its entirety.

In addition, the GIT is one of the few organ systems that normally contain a mixture of commensal and potentially pathogenic organisms. In health, there is continuous interaction between the host and microorganisms resulting in a perpetual steady state

KEYPOINTS

- Gross lesions in the gastrointestinal tract must be incorporated with clinical and historical data to provide meaningful interpretations.
- Red bowel typifies congestion more commonly than hemorrhage or hemorrhagic enteritis.
- Enteritis usually includes, in addition to congestion or hemorrhage, edema, fibrin or feed material adherent to affected mucosa, mucosal erosion, ulcers or necrosis, serosal hemorrhage with adherent fibrin, and luminal contents with abnormal consistency and possibly a miasmic odor.
of controlled inflammation. Once the animal dies, these organisms continue to rapidly proliferate, are exothermic, and release destructive enzymes and gases that lead to rapid autolytic and putrefactive changes that can quickly obfuscate the superficial intestinal mucosa either obscuring lesions or creating pseudolesions. Timely examination of the GIT is essential for the best opportunity to contribute to the diagnostic process.

For ease of examination during necropsy, the gastrointestinal system is divided into three to four sections: the oral cavity and esophagus; the forestomachs and true stomach (abomasum); and the intestines, which can be divided into small and large intestines separately or examined as a continuum. Examination of the GIT begins with a thorough examination of the oral cavity, including the lips, gums, hard and soft palate, teeth, and tongue for erosions, ulceration, hemorrhage, and masses. Remember that many esophageal lesions tend to be in the distal one-third of the esophagus. It is important to be thorough to detect subtle mucosal erosions or ulcers, find stray wire or lead particles in the reticulum, identify parts of toxic plants in all compartments, characterize the feed, or observe substances that are not miscible in water-based rumen contents. These changes can be easily overlooked or missed if this part of the necropsy is given a cursory glance or omitted all together. All four compartments must be thoroughly opened and their contents evacuated to better examine the mucosa.

Ideally, the small and large intestine should be stripped from its mesenteric attachments before or during removal from the peritoneal cavity allowing palpation for masses, recognition of any color variation, and localization of any areas of serosal inflammation or perforations. A good place to begin is where the ileum attaches to the cecum and begin stripping orally toward the duodenum. Identifying the ileum is also important because it is a common location for many intestinal diseases and ensures that Peyer’s patches are located and collected. Resist the temptation to only "spot check" various segments of the intestine because subtle or regionally variable lesions could be easily missed.

While opening segments of intestine to assess contents, the mucosa can be more thoroughly evaluated. The often semifluid to pasty consistency of intestinal contents may obscure the mucosa beneath impairing its thorough examination. It is tempting to scrape this material off of the mucosa with the edge of the necropsy knife, but that damages the underlying epithelium if histopathology is desired. If that is the only means of assessment,
then ensure that other segments with similar gross lesions are sampled for histopa-
thology. Small amounts of water under low pressure or small amounts poured over the
mucosa more gently clear the contents from the surface and allow more detailed exam-
ination. If a pathogen is suspected, representative sections of bowel should be collected
for microbiologic testing before opening or flushing the segments. Ensure that segments
of intestine collected for microbial testing are kept closed, using string to seal the ends of
the segments. When submitting to the laboratory, it is imperative that segments of bowel
are packaged separately from other fresh tissues.

The purpose of the gross necropsy examination of the GIT is to recognize the pres-
ence of lesions, thus requiring a basic understanding of its normal appearance and
anatomy. This article highlights gross changes to the GIT of adult cattle that help place
the disease process into broad categories. Although few gross lesions reach the
zenith of pathognomonic, there are numerous lesions that when considered in aggre-
gate with history (number of animals affected, environment, duration of signs, time of
onset relative to management changes, previous management, and so forth) and clinical
signs can help narrow the spectrum of causes, provide a basis for a strong
presumptive diagnosis, and focus diagnostic test selection.

LESION CATEGORIZATION

There are numerous ways to codify gastrointestinal gross pathology. One of the
most practical methods is to classify lesions using the following broad categories:

- Color variation
- Mucosal surface integrity
- Size variation
- Luminal content features

Each category is briefly introduced in general broad terms. For each functional
and anatomic division, normal is defined followed by application of the method to
each segment. The GIT is divided into the oropharynx, esophagus, forestomachs,
abomasum, and intestines.

GENERAL CONSIDERATIONS

Color Changes

Red mucosa in the abomasum and intestines is often normal or uninterpretable as it
relates to disease or inflammation but is commonly misdiagnosed as hemorrhage
and inflammation (Fig. 2). Many dead cattle can have segments of quite red intestine,
even with bloody contents, that histologically do not translate into hemorrhage or
inflammation. Smudgy red to pink discoloration of gastrointestinal serosa is frequently
encountered in adult cattle with prolonged postmortem intervals caused by lysis of red
cells with leakage or imbibition of hemoglobin into the tissue interstitium. With time
and proliferation of bacteria, imbibed hemoglobin can react with bacteria-derived
hydrogen sulfide to produce iron-containing compounds that impart a dark green to
black tissue discoloration, referred to as “pseudomelanosis.”

Serosal surfaces of the forestomachs, abomasum, and intestines tend to be highly
variable, ranging from pale tan to pink to medium purple, dependent largely on the
postmortem interval. Never judge an animal as pale based solely on serosal surface
pallor. Serosa may be helpful along with subcutaneous tissues in detecting and con-
fiming icterus.

Mucosal Surface Integrity

In general, mucosal surfaces have a glistening sheen that is best mimicked by visual
appearance of “semigloss” to “glossy” finish house paints. When mucous membranes
have a texture that is analogous to “matte” or “eggshell” paint finishes, that is strongly
suggestive of erosion or shallow ulcers. When the center of defects is brighter or
darker red compared with the surrounding glistening mucosa, that is strongly suggestive of ulceration. Adherent feed or fibrin to the mucosal surface is a strong indication of local necrosis and inflammation.

**Size Variation**

Variation in the size of segments of the GIT tends to be the result of changes in wall thickness and luminal diameter. In general, increases in mural thickness can be caused by physiologic hypertrophy to the tunica muscularis or mucosal epithelium or by expansion of selected lamina or all layers of the wall because of accumulation of excess fluid or gas, infiltration by inflammatory cells, neoplasia, or a combination. Mural thickness may be decreased in cases where the luminal diameter is greatly expanded causing mural stretching or in cases where there is mural necrosis.

Excess fluid collected in tissues can be an antemortem physiologic response to hypoproteinemia leading to edema; it can result from localized or systemic inflammation, or possibly a response to excessive intravenous administration of fluid. Postmortem fluid accumulation can occur if the postmortem interval to necropsy is prolonged or if the carcass remains in an environment with high ambient temperatures and relative humidity. Similarly, excessive expansion by gas may be caused by antemortem proliferation of gas-producing bacteria, complications related to previous surgery, or disease. More commonly, excess gas accumulation in a carcass occurs postmortem related to proliferation of gas-producing anaerobic bacteria, such as *Clostridium* spp.

Luminal diameter is dependent on the muscular and neural integrity of the viscus wall, the transit time of the luminal contents, and the balance between absorption and secretion in the various segments of the GIT. Any alteration to one or more of these components can dramatically affect the size of the GIT, especially the intestines. For example, a focal intestinal obstruction can lead to proximal accumulation of fluid and resultant distention along with collapse or narrowing of the distal bowel (**Fig. 3**).

**Luminal Content Features**

Luminal contents are normally variable depending on the segment being examined in terms of odor, consistency, and color. Factors that contribute to this include type of diet (pasture vs dry lot vs feedlot rations); amount and type of water consumption;
and concurrent stressors, such as animal density, environmental stressors, and disease. Ruminal contents tend to have a sweet aromatic scent when cattle are on pasture or fed silage but become more sour and acidic in the face of high-grain diets or in cases of acidosis. Intestinal contents tend to move from coarse to liquid to pasty to semisolid when moving from oral to anal. Deviation from this often correlates with various disease states. Finally, color is extremely variable throughout the bowel and only in few circumstances is it diagnostically useful.

**PATHOLOGY OF SPECIFIC ANATOMIC LOCATIONS WITHIN THE GIT**

### Oropharynx

Oropharyngeal mucosa tends to be highly variable in terms of melanin pigmentation depending on the breed and color variations within particular breeds. Dorsally, the dental pad is a thickened, often fissured area that apposes the lower incisor teeth. Teeth tend to have white enamel with darker green to brown to black staining of the opposing surface of upper and lower arcades.

Oral mucosa typically has a bright, slightly moist sheen. The hard palate has transverse ridges from which caudally project papillae that help guide food in an aboral direction. The soft palate is continuous with the hard palate and tends to have a smooth surface. The cheek mucosa has long, broad, aborally directed papillae that are sharply pointed in health (Fig. 4).

**Fig. 3.** Focal small intestinal obstruction. An intraluminal obstruction (arrow) is present, resulting in a markedly dilated and congested proximal bowel and mostly empty distal bowel. (Courtesy Dr B.L. Njaa, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

**Fig. 4.** Bovine papular stomatitis involving the oropharynx. Numerous circular (arrow) lesions are scattered throughout the surface of the hard and soft palate. Several of the lesions are less circular and have a more proliferative superficial exudation (arrowhead). (Courtesy College of Veterinary Medicine, Cornell University, Ithaca, NY.)
Tongues are their principle organ of prehension in cattle. Overlying the dorsal surface of the tongue are small, fine, sharp papillae, many of which play a sensory role. Approximately three-fifths caudal to the rostral tip is the lingual fossa demarcating the rostral edge of a large dorsal prominence called the torus linguae.

Brown to blackened teeth with hypoplastic enamel is likely caused by chronic fluorosis (Fig. 5). Alternatively, hypoplastic or dysplastic enamel can be the result of in utero bovine viral diarrhea virus (BVDV) infections (Fig. 6). Oropharyngeal mucous membranes can become pale because of anemia or severe blood loss. Alternatively, they can become cyanotic because of cardiovascular compromise and collapse. When a cow is exposed to high nitrate–containing plants (frost-damaged cereal crops, Brassicaceae spp, and Sorghum spp) or fertilizers, the nitrate is converted to nitrite and when it is absorbed converts hemoglobin to methemoglobin, imparting a light brown or “muddy” tincture to blood and tissues (Fig. 7).

Localized and systemic diseases with inherent epitheliotropism may first be visualized when examining the oropharynx but are rarely limited to this location. Shallow erosions tend to be slightly different from the surrounding tissue because of pigmentation changes and loss of the typical sheen (Fig. 8). Similar lesions may be seen in the lingual mucosa (Fig. 9). Causes frequently associated with this lesion include BVDV, bluetongue virus (BTV), herpes viruses that cause malignant catarrhal fever (MCF), rinderpest, and rupture of epithelial vesicles.

Vesicular disease is a category of disease that typically involves a certain degree of alarm and investigation and confirmatory testing by federal veterinarians. In North America, these most often are categorized as foreign animal diseases or transboundary animal diseases. Commonly, intact vesicles or erosions, the result of ruptured vesicles, are the first sign of concern, initially observed on the muzzle and in the oral cavity. Ruptured vesicles and erosions when secondarily infected, can become deep, inflamed, painful, mucosal ulcers. Possible causes include foot-and-mouth disease, vesicular stomatitis, chronic BVD, BTV, and MCF (Fig. 10). Other causes that tend to lead to severe erosion or ulceration include blister beetle (cantharidin) toxicosis; traumatic injury secondary to coarse feeds or caustic chemicals; and uremia. In extreme cases, plant awns or hair may embed themselves in the ulcerated lingual mucosa, especially in the lingual fossa, giving the appearance of lingual “hair” (Fig. 11).

Fig. 5. Periodontal disease caused by fluorosis. Enamel erosion and hypoplasia and black discoloration are caused by excess fluoride ingestion. The white material on the surface of the teeth is what remains of the enamel. (Courtesy College of Veterinary Medicine, Cornell University, Ithaca, NY.)
Fig. 6. Periodontal enamel defect caused by BVDV. This bilaterally symmetric enamel defect in deciduous incisor teeth is caused by a previous *in utero* infection with BVDV. (*Courtesy Dr E.G. Clark, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK.*)

Fig. 7. Methemoglobinemia. The blood on the left is muddy, red brown, collected from an animal exposed to excess nitrates. The blood on the right is from a normal animal. (*Courtesy Dr R.J. Panciera, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.*)

Fig. 8. Hard and soft palate erosions. The oral mucosa of the hard and soft palate is multifocally eroded with areas (*arrowheads*) that lack the normal glistening sheen and are typically darker than the surrounding unaffected mucosa. BVDV was confirmed as the cause in this case based on enzyme-linked immunosorbent immunoassay and immunohistochemistry techniques. (*Courtesy Dr B.L. Njaa, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.*)
Fig. 9. Lingual erosions. There are shallow (arrowhead) and deeper erosions (arrows) affecting the mucosal surface of the tongue. The lingual papillae covering the surface of the torus linguæ (T) are blunted. This is the tongue from the same animal in Fig. 8 that was confirmed positive for BVDV. (Courtesy Dr B.L. Njaa, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

Fig. 10. Ruptured vesicles in the lingual mucosa. The lingual mucosa is focally ulcerated (arrows) in areas where vesicles have ruptured. This animal was a confirmed case of foot-and-mouth disease during an outbreak in Bolivia in 2007. (Courtesy Dr C. Orozco, USDA, APHIS, Bolivia.)

Fig. 11. Necroulcerative lingual gingivitis. At the rostral base of the torus linguæ (T), in the lingual fossa is an area of dark red ulcerative inflammation within which are embedded grass awns mixed with other feed material. (Courtesy Dr E.G. Clark, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK.)
Two of the more commonly referred to ailments of the oropharynx that lead to swelling and variation in size are “lumpy jaw” and “wooden tongue.” The former is also referred to as “actinomycosis,” denoting a severe osteomyelitis that results from *Actinomyces bovis* penetration through the oropharyngeal mucosa with subsequent penetration of the periosteum of the mandible and eventual mandibular osteomyelitis. This results in a severely disfiguring disease (Fig. 12). Wooden tongue, also called “actinobacillosis,” is a lingual infection by *Actinobacillus lignieresii*. The tongue becomes markedly enlarged and painful and may develop ulceration because of its obstructive size and susceptibility to trauma (Fig. 13).

Animals repeatedly exposed to bracken fern forage run the risk of developing squamous cell carcinoma of the upper GIT (Fig. 14). Rarely, other neoplasms may affect the oropharynx leading to cachexia either through obstruction or because of impaired ability to masticate food.

### Esophagus

Once opened and laid flat, the esophageal mucosa tends to be normally quite pale, with a smooth texture, lacking surface folds. The underlying submucosa is normally loosely attached to the overlying mucosa allowing for a moderate amount of laxity when the mucosa is torqued relative to the muscle layer.

At the level of the thoracic inlet is where a clearly demarcated line of congestion abruptly ends in pallor forming what is most commonly referred to as a “bloat line” (Fig. 15). This lesion occurs in cattle that develop markedly increased intra-abdominal pressure as ruminal pressure increases because of excess uneructated gas accumulation. This increased abdominal pressure compresses the thorax and blanches the intrathoracic portion of the esophagus, whereas the esophagus cranial...
to the thoracic inlet tends to be markedly congested. Any reason to prevent eructation of gas can lead to the formation of a bloat line.

Serpentine, pale white to orange red spirurid parasites are commonly found burrowed in the epithelium of the esophagus, and occasionally the lingual mucosa. This is the typical presentation of Gongylonema pulchrum infestations. When present in the ruminal submucosa, they are Gongylonema verrucosum. They are clinically inconsequential (Fig. 16).

Papules that form in the esophageal mucosa and oral mucosa (see Fig. 4) are very common in younger cattle, most often the result of bovine papular stomatitis virus, a member of the genus Parapoxvirus. Transmission probably occurs through direct contact and stress or immunosuppression may precipitate disease. These lesions
are variable in size, typically very round to oval, and can be subtle raised foci or very prominent papules with raised rims and eroded centers and have adherent fibrin and feed material (Fig. 17). Erosions can affect the esophagus with causes similar to those described for the oropharynx (BVD, MCF, BTV) (Fig. 18). Deeper ulcerated lesions may be the result of secondarily infected BVD erosions, mucosal disease, MCF, infectious bovine rhinotracheitis, ruptured vesicles, or mucosal trauma (Fig. 19).  

Fig. 13. Actinobacillosis or “wooden” tongue. (A) The tongue is markedly enlarge with several areas of chronic ulceration along its lateral edge. (B) Cross section through a case of wooden tongue with numerous pyogranulomas (arrow) scattered throughout the tongue. This is typically caused by Actinobacillus lignieresii. ([A] Courtesy Dr R.J. Panciera, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK; [B] Courtesy Dr E.G. Clark, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK.)

Fig. 14. Lingual and pharyngeal squamous cell carcinoma. A large area of ulceration is present at the base of the tongue. This was associated with chronic ingestion of bracken fern. (From Masuda EK, Kommers GD, Martins TB, et al. Morphologic factors as indicators of malignancy of squamous cell carcinomas in cattle exposed naturally to bracken fern (Pteridium aquilium). J Comp Pathol 2011;144:48–54; with permission.)
**Fig. 15.** Bloat line. Cranial to the level of the thoracic inlet (arrow), the esophagus is markedly congested, whereas caudal to this the esophagus is pale. This is an indication that elevated intraruminal pressure could not be alleviated resulting in severe vascular compromise and formation of a “bloat line.” (Courtesy Dr R.J. Panciera, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

**Fig. 16.** Esophageal *Gongylonema pulchrum* infestation. Serpentine mucosal or submucosal parasites in the esophagus (arrow) are typical of *G pulchrum* infestation. When found in the rumen, they are *Gongylonema verrucosum*. (Courtesy Dr M. Czajkowski, College of Veterinary Medicine, Cornell University, Ithaca, NY.)

**Fig. 17.** Bovine papular stomatitis, esophagus. Numerous round to oval papules are present. The largest one is centrally ulcerated. Bovine papular stomatitis virus, often affecting cattle that may have impaired immunity, causes these lesions. (Courtesy Dr B.L. Njaa, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)
Mucosal injury to the esophagus tends to heal by fibrosis and scarring resulting in luminal narrowing and stricture formation near the site of injury (Fig. 20). Injury may be caused by luminal foreign bodies that become lodged or any of the other infectious causes. Proximal to the stricture, the esophagus may become dilated

Fig. 18. Multifocal linear esophageal erosions. Numerous mucosal erosions are scattered throughout this esophagus. With lesions this numerous and shallow, BVD must be strongly considered. This animal was confirmed positive for BVDV. (Courtesy Dr B.L. Njaa, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

Mucosal injury to the esophagus tends to heal by fibrosis and scarring resulting in luminal narrowing and stricture formation near the site of injury (Fig. 20). Injury may be caused by luminal foreign bodies that become lodged or any of the other infectious causes. Proximal to the stricture, the esophagus may become dilated

Fig. 19. Esophageal ulceration. (A) An acute, hemorrhagic, ulcerative lesion affecting the esophageal mucosa. (B) A subacute ulcerative esophagitis with superficial crusting. ([A] Courtesy Indiana Animal Disease Diagnostic Laboratory, Purdue University, West Lafayette, IN; [B] Courtesy Dr E.G. Clark, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK.)
because of slowed or impaired passage of ingesta into the forestomachs. Periesophageal fasciitis that can result from “balling gun” injuries can cause esophageal luminal narrowing (Fig. 21). Traumatic injury from boluses forcibly ejected into the surrounding periesophageal tissue results in localized tissue damage, edema, and inflammation. In addition, pharyngeal trauma associated with this type of injury can result in acquired megaesophagus presumably caused by vagal nerve injury. Rarely, intraluminal, mural, or periesophageal neoplasia can result in obstruction, luminal narrowing or dilation, dependent on either direct or indirect effects on esophageal function (Fig. 22).

**Forestomachs**

Forestomachs represent large, mucosal-lined, fluid-filled, and microbe-laden fermentation vats that are necessary for ruminants to convert the complex carbohydrates of plants into absorbable fatty acids. During the course of the digestion and fermentation

Fig. 20. Esophageal stricture. A focal esophageal foreign body has resulted in areas of ulceration (white arrowhead) and a circumferential area of pallor and constriction (arrows). (Courtesy Dr R.J. Panciera, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

Fig. 21. Pharyngeal and periesophageal cellulitis or fasciitis. Both the trachea (T) and esophagus (E) have been displaced by a regional area of necrosis and inflammation (**). Any type of forceful injury to the pharynx can result in perforation, edema, necrosis, and inflammation. In this case, it was caused by improperly administered medicinal boluses by a mechanical dispensing device (ie, balling gun). (Courtesy Dr E.G. Clark, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK.)
process, gases are produced along with thermal energy. Continued health of ruminants requires coordinated release of the fermentation gases produced and maintenance of rumen flora health. Alterations of this host-environment-microorganism homeostasis are what lead to lesions and disease.

The Reticulum

The reticulum or “honeycomb” is the most cranial, small, relative to the other forestomachs, sac comprising a mucosal web of interconnected, irregularly square, pentagonal or hexagonal, cells that are further divided into even smaller divisions.12 Coarse, large, and heavy materials or objects tend to collect in the reticulum. Sharp objects indiscriminately ingested can accumulate in the reticulum resulting in an increased risk of mural penetration with consequences ranging from focal abscessation to peritonitis to diaphragmatic and pericardial perforation leading to a condition known as “traumatic reticulopericarditis” (Fig. 23). Ingestion of heavy material, such as lead, tends to collect in the mucosal-lined, ridged cells making this an important forestomach compartment to thoroughly examine (Fig. 24). Mucosal changes previously discussed can result in similar changes in the reticulum but are often missed because they are typically too subtle or the diagnosis is made based on lesions in other portions of the GIT (Fig. 25).

Rumen and Omasum

Forestomach mucosae tend to be greenish black because of staining by chlorophyll-containing feed. This staining tends to be relatively uniform throughout these compartments with the exception of the broad rumen pillars, which often are pale, lack papillae, and tend to have a thickened squamous mucosa. When the stained superficial layers of stratified squamous epithelium become blotchy, it is often an indication of disease processes that cause superficial erosions, such as BVD, MCF, or chemical rumenitis (Fig. 26). Similar blotchiness of the omasal mucosa is also typically seen. Any type of hemorrhagic diathesis can result in mural hemorrhages to any tissue or organ system. One cause to keep in mind is the acute hemorrhagic and thrombocytopenic form of acute BVD (Fig. 27).13
Fig. 23. Reticuloperitonitis. The reticulum is firmly adherent to the diaphragm with localized area of peritonitis (*asterisk*). A nail is embedded in the wall of the reticulum (*arrow*). It is most likely that this nail or another sharp, slender piece of metal penetrated through the wall of the reticulum to cause a localized peritonitis. If this inflammation tracks to the pericardial sac, it is called traumatic reticulopericarditis. (Courtesy Dr B.L. Njaa, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

Fig. 24. Lead intoxication. Variably sized, sliver-gray, metallic particles are present within the mucosal compartments of the reticulum. These represent lead shavings obtained from a source of lead in a field, such as used batteries. (Courtesy Dr B.L. Njaa, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

Fig. 25. Bovine papular stomatitis, reticulum. (Courtesy Dr R.J. Panciera, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)
Ulceration and mural necrosis can be seen in cases of necrobacillosis and mycotic infections. Both conditions are typically secondary invasion of the forestomach mucosa by *Fusobacterium necrophorum* or fungal organisms after primary traumatic or chemically (acidosis) induced mucosal injury (Fig. 28). Necrobacillosis invades through the mucosa into the wall but rarely penetrates to the serosal surfaces (Fig. 29). However, mycotic infections can either be superficial mucosal proliferation caused by infections with *Candida albicans* (Fig. 30) or deep and transmural involving the ruminal serosa because of the propensity of many fungi (ie, *Aspergillus* spp, *Mucor* spp, *Absidia* spp, *Rhizopus* spp) to not only proliferate in squamous epithelium but also in the walls of blood vessels causing deep inflammation and infarction (Fig. 31).

Ruminal mucosa varies from lacking papilla along the pillars to being densely covered by rumen papilla that range in length and size depending on the predominant feed-associated volatile fatty acids produced in the rumen fluid. Physiologic thickening and lengthening of the ruminal villi tends to occur with feeds that result in higher...
Fig. 28. Rumenitis. Arborizing islands of adherent mucosa cover small portions of the underlying submucosa (asterisk). The edges of these attached portions of epithelium are thickened and lack rumen papillae. These areas represent areas of rumenitis and epithelial injury. The remaining portions that lack epithelium represent normal postmortem sloughing of mucosa (**). P, rumen pillar. (Courtesy Dr E.G. Clark, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK.)

Fig. 29. Necrobacillosis. This focal area of necrosis is surrounded by a red rim of congestion. This represents a local response to proliferation of *Fusobacterium necrophorum*. (Courtesy Dr R.J. Panciera, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

Fig. 30. Mucosal hyperplasia caused by superficial mycotic rumenitis, from *Candida albicans* proliferation. (Courtesy Indiana Animal Disease Diagnostic Laboratory, Purdue University, West Lafayette, IN.)
levels of propionate and butyrate. Conversely, as the percent of dietary roughage increases, production of propionic and butyric acid decreases while ruminal acetic acid levels tend to increase, and ruminal mucosal papillar hypertrophy and hyperplasia does not occur.11

During necropsy examination of a normal adult bovid, the oral and esophageal mucosa remain firmly and tenaciously attached to the underlying submucosa, whereas the forestomach mucosa tends to normally lift off and away from the underlying, typically reddened submucosa within a few hours of death (Fig. 32).3 Conversely, easily sloughed oral and esophageal mucosa and firmly adherent forestomach mucosa are indications of disease.

Parasitism of the ruminal mucosa can be challenging to observe and confirm without close inspection. Adult flukes that affect forestomachs of cattle from numerous genera are commonly grouped as paramphistome infections (Fig. 33). Adult
parasites are red, pear-shaped, and tend to easily blend in with the rumen papillae but are typically incidental gross findings. Long, keratinized, papillary mucosal projections as the omasum opens into the reticulum and gastric groove are normal and referred to as unguiculliform papillae (Fig. 34). However, proliferative lesions affecting the forestomachs can include papular stomatitis lesions (Fig. 35) and neoplasms, such as mural lymphoma, or mucosal epithelial neoplasms, such as fibropapillomas (Fig. 36).

**Abomasum**

Representing the true stomach of ruminants, this viscus is located caudal, ventral, and to the right of the rumen. It is typically slightly more purple than the forestomachs on its serosal surface and typically has a mucosa that is more red to reddish purple. The mucosal surface has much more of a wet, glistening sheen, lining a series of rugal folds.

![Fig. 33. Rumen flukes. Attached to the rumen surface and partially camouflaged by the rumen papillae are three rumen flukes (arrow). In this case, these are presumptively *Paramphistomum* spp. (Courtesy Mr. Richard Irvine and Dr C.G. Lamm, School of Veterinary Medicine, University of Glasgow, Glasgow, Scotland.)](image1)

![Fig. 34. Unguiculliform papillae. These papillae (arrows) may become quite prominent and gnarly near the reticular groove (G) along the junction of omasum (O) and reticulum (R) but are normal. (Courtesy Dr B.L. Njaa, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)](image2)
Fig. 35. Bovine papular stomatitis, rumen. (Courtesy Dr R.J. Panciera, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

Fig. 36. Rumen papilloma. This focally proliferative, papillary growth is typical of a rumen papilloma. (Courtesy Dr R.J. Panciera, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

Fig. 37. Abomasal hemorrhage. Multifocally throughout the abomasal mucosa are variably sized dark red areas of hemorrhage. Any type of bleeding diathesis can result in this lesion. This particular example was a case of thrombocytopenic BVD. (Courtesy Dr E.G. Clark, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK.)
Red, multifocal to confluent areas of hemorrhage in the abomasal mucosa are most often an indication of an ulcerative or infarction process (Fig. 37). Causes for such changes include various hemorrhagic diatheses including toxins (arsenic); corticosteroids (exogenous and endogenous); and infectious diseases (acute, thrombocytopenic, hemorrhagic BVD) (see Fig. 27). Perforating abomasal ulcers tend to affect young cattle. Less commonly, older animals may develop ulcerative lesions that eventually perforate and cause severe peritonitis (Fig. 38).

Fig. 38. Perforated abomasal ulcer with peritonitis. This is a serosal view of a perforated abomasal ulcer (arrow) that has resulted in a severe fibrinous peritonitis (asterisk). This is more commonly seen in young calves but less frequently can affect adult cattle. (Courtesy Dr B.L. Njaa, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

Red, multifocal to confluent areas of hemorrhage in the abomasal mucosa are most often an indication of an ulcerative or infarction process (Fig. 37). Causes for such changes include various hemorrhagic diatheses including toxins (arsenic); corticosteroids (exogenous and endogenous); and infectious diseases (acute, thrombocytopenic, hemorrhagic BVD) (see Fig. 27). Perforating abomasal ulcers tend to affect young cattle. Less commonly, older animals may develop ulcerative lesions that eventually perforate and cause severe peritonitis (Fig. 38).

Fig. 39. Right displacement and volvulus of the abomasum. (A) The dark red to black viscus is the abomasum. Displacement and volvulus has resulted in severe vascular compromise and tissue death. (B) The mucosal surface of a similarly affected abomasum is extremely dark red and edematous caused by severe congestion and hemorrhage and necrosis. (A) Courtesy Dr E.G. Clark, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK; (B) Courtesy Dr B.L. Njaa, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)
Abomasal displacement and volvulus is a common condition affecting dairy cattle usually around the time of parturition. With the highest incidence in intensively managed herds, the abomasum typically displaces ventrally and to the left of the rumen. Less commonly, the abomasum displaces to the right increasing the chance for abomasal volvulus. The omasum is often involved in abomasal torsions. When severe, blood vessels in the neck of the omasum become obstructed resulting in not only organ distention but also severe congestion and hemorrhage (Fig. 39).\(^\text{15}\)

Focal to diffuse mucosal thickening of abomasal rugal folds is characteristic of parasitism (Fig. 40). The lesion is a combination of mucous metaplasia and hyperplasia with associated chronic inflammation. The causes for this lesion include either *Ostertagia* spp or *Trichostrongylus axei*.\(^\text{11}\)

The most common neoplasm that affects cattle is lymphoma. The abomasal wall is a predilection site (Fig. 41). Infiltration may be diffuse imparting a pale tan tincture to the entire affected area or multifocal, nodular infiltration of neoplastic lymphocytes. Other common sites for infiltration by neoplastic lymphocytes include forestomachs, heart, lymphoid organs, liver, reproductive tract, and spinal canal.\(^\text{16}\)

---

**Fig. 40.** Proliferative abomasitis. This is most commonly the result of *Ostertagia* spp infestation and associated inflammation. (A) Marked proliferation of all abomasal rugae. (B) Higher magnification of another example with focal areas of inflammation. ([A] Courtesy Dr R.J. Panciera, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK; [B] Courtesy Dr B.J. Johnson, California Animal Health and Food Safety Laboratory, University of California, Davis, CA.)
Intestines

In health, intestines vary from pale pink to purple when viewed from the serosal surface and pink-purple to red when the mucosal surface is examined. The mucosal surface has a moist to very wet, velvety, glistening sheen, analogous to a glossy paint finish. Throughout its length, there are mucosal folds designed to increase surface area. Moving aborally, villi tend to decrease in length with no villi present in the large intestine, whereas goblet cell numbers increase such that they are maximal in the distal large intestine.

In general, enteritis is a relatively pedestrian diagnosis histologically but can be very confusing grossly. Redness is only one feature to consider when determining if an animal has enteritis, given that in many cases mucosal redness is a variation of normal. Gross evidence of enteritis is reliably identifiable when there is adherent ingesta mixed with fibrin, erosions, ulcers, and mural necrosis (Fig. 42). Other features include luminal casts of necrotic material and mucosal thickening without loss of the mucosal sheen. Serosal changes to look for include mural edema, serosal congestion or multifocal hemorrhage (Fig. 43), loss of the normal serosal glistening surface, and adherent

Fig. 41. Abomasal lymphoma. (A) Multifocal areas of mural thickening is one manifestation of abomasal lymphoma. (B) Most rugal folds in this abomasum are discolored pale tan and diffusely thickened because of infiltration by neoplastic lymphocytes. (A) Courtesy Dr A.W. Stern, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK; (B) Courtesy Dr S.D. Cramer, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

Intestines

In health, intestines vary from pale pink to purple when viewed from the serosal surface and pink-purple to red when the mucosal surface is examined. The mucosal surface has a moist to very wet, velvety, glistening sheen, analogous to a glossy paint finish. Throughout its length, there are mucosal folds designed to increase surface area. Moving aborally, villi tend to decrease in length with no villi present in the large intestine, whereas goblet cell numbers increase such that they are maximal in the distal large intestine.

In general, enteritis is a relatively pedestrian diagnosis histologically but can be very confusing grossly. Redness is only one feature to consider when determining if an animal has enteritis, given that in many cases mucosal redness is a variation of normal. Gross evidence of enteritis is reliably identifiable when there is adherent ingesta mixed with fibrin, erosions, ulcers, and mural necrosis (Fig. 42). Other features include luminal casts of necrotic material and mucosal thickening without loss of the mucosal sheen. Serosal changes to look for include mural edema, serosal congestion or multifocal hemorrhage (Fig. 43), loss of the normal serosal glistening surface, and adherent
fibrin. If serosal fibrin is mixed with feed material a perforation must be suspected. Finally, enteritis typically results in abnormal fluid contents downstream in the large intestine.

Bright red intestinal mucosa caused by hemorrhage with luminal necrotic debris or clotted blood is in indication of enteritis (Fig. 44). In general, causes to consider include *Eimeria* spp; coronavirus (winter dysentery); BVDV; MCF; *Clostridium* spp; heavy metal intoxication; *Salmonella* spp; bowel strangulations; and *Trichuris* spp (whipworms).\(^3,11\) When large amounts of fibrin are present overlying the mucosal surface, causes to consider include *Salmonella* spp or *Clostridium* spp (Fig. 45). If there is hemorrhage and necrosis of the Peyer’s patches or regional lymphoid tissue, BVD, *Salmonella* spp, or both should be strongly considered as possible causes for this lesion (Fig. 46).\(^11\) Chronically, salmonellosis can manifest as focal areas of necrosis rimmed by fibrosis, commonly referred to a “button ulcers”

![Image](image1)

**Fig. 42.** Enteritis. The mucosal surface of this small intestinal section lacks the normal velvety, glistening sheen and instead is dull, partially covered by fibrin mixed with feed material, and has multifocal areas of mucosal hemorrhage. (Courtesy Dr R.J. Panciera, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

![Image](image2)

**Fig. 43.** Hemorrhagic serositis. There are multifocal red areas over the serosa representing areas of mural hemorrhage and necrosis. This same injury could be observed from the mucosal surface indicating evidence for enteritis. (Courtesy Indiana Animal Disease Diagnostic Laboratory, Purdue University, West Lafayette, IN.)
Intestinal contents tend to have a more putrid, pungent odor caused by the amount of miasma present in cases of some bacterial enteritis, such as salmonellosis. *Mycobacterium avium* subspecies *paratuberculosis*, the causative organism for Johne’s disease, is a resilient intracellular organism that induces a prominent granulomatous reaction. Large numbers of macrophages are attracted to the lamina propria and submucosa of the affected intestine resulting in distinctive corrugation of the mucosa (Fig. 48) with a subsequent reduction in luminal diameter and functional absorptive surface area. In addition, a distinctive serosal and mesenteric response to this organism is granulomatous lymphangitis (see Fig. 48B).

(Fig. 44). Hemorrhagic enteritis. The intestinal mucosa is red and congested with large mats of blood clots mixed with fibrin strands and feed material. This lesion has several potential causes but in this case is an example of coccidiosis. (Courtesy Dr R.J. Panciera, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

(Fig. 47). Intestinal contents tend to have a more putrid, pungent odor caused by the amount of miasma present in cases of some bacterial enteritis, such as salmonellosis.

*Mycobacterium avium* subspecies *paratuberculosis*, the causative organism for Johne’s disease, is a resilient intracellular organism that induces a prominent granulomatous reaction. Large numbers of macrophages are attracted to the lamina propria and submucosa of the affected intestine resulting in distinctive corrugation of the mucosa (Fig. 48) with a subsequent reduction in luminal diameter and functional absorptive surface area. In addition, a distinctive serosal and mesenteric response to this organism is granulomatous lymphangitis (see Fig. 48B).

Fig. 45. Fibrinonecrotizing enteritis. The intestinal wall is mottled red and tan. The mucosa has lost its typical velvety sheen and is overlayed by a mat of fibrin. There is a large luminal clot of fibrin (asterisk). This lesion was caused by *Clostridium perfringens*. (Courtesy Indiana Animal Disease Diagnostic Laboratory, Purdue University, West Lafayette, IN.)
Fig. 46. Peyer’s patch necrosis caused by BVD. (A) Along the antimesenteric edge of this section of small intestine is an area of necrosis and hemorrhage that corresponds to Peyer’s patches. (B) Mucosal surface of small intestine depicting necrosis of Peyer’s patches (arrow) with adherent fibrin and necrotic debris. This lesion is typical for BVDV infections. ([A] Courtesy Indiana Animal Disease Diagnostic Laboratory, Purdue University, West Lafayette, IN; [B] Courtesy Dr R.J. Panciera, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)

Fig. 47. Chronic button ulcers, colon. Numerous nodules are present with raised, rounded edges and central regions of necrosis. These lesions are a mucosal response to chronic Salmonella spp infection, which was confirmed in this case. (Courtesy Dr B.L. Njaa, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK.)
Jejunal hemorrhagic syndrome (or hemorrhagic bowel syndrome) is a relatively poorly understood entity of cattle resulting from chronic and ongoing intraluminal small intestinal hemorrhage without any evidence of concurrent enteritis (Fig. 49). Typically, affected animals present with signs of obstruction or possibly sudden death. The lesion can be segmental with variably sized collections of clotted blood that tend to minimally distend affected loops and yet have an underlying mucosa that is typically grossly normal. Isolation of *Clostridium perfringens* type A from cattle with this condition has been reported but likely does not represent a definitive cause. Others have reported in dairy cattle that intensive management procedures and minimal exposure to pasture in high producing herds are predisposing factors to the development of this syndrome.

Mesenteric and omental fat and other fat stores can become massively necrotic with areas of saponification in cattle (Fig. 50). Typically, affected animals are overconditioned, 2 years of age or older, and frequently of Channel Island lineage. The areas of necrosis and saponification are palpably hard and potentially obstructive. These lesions can vary from incidental findings to a cause of intestinal obstruction.
Fig. 49. Jejunal hemorrhagic syndrome. (A) Unopened loops of small intestine, focally hemorrhagic and mildly dilated because of a luminal blood clot. These luminal blood clots tend to lodge and obstruct. (B) Opened segment of small intestine with luminal clots of blood but a mucosal surface that appears grossly normal. ([A] Courtesy Dr F. Uzal, California Animal Health and Food Safety Laboratory, University of California, Davis, CA; [B] Courtesy College of Veterinary Medicine, Cornell University, Ithaca, NY.)

Fig. 50. Massive omental fat necrosis. This is a lesion that occurs most commonly in Channel Island cattle. Lesions vary from clinically incidental to obstructive. The necrotic fat is typically saponified and palpably hard. (Courtesy College of Veterinary Medicine, Cornell University, Ithaca, NY.)
Sample collection for gastrointestinal disease

- Gastrointestinal tissues autolyze rapidly after death. Therefore, a short postmortem interval is paramount and samples should be collected early in the necropsy procedure.
- For remote necropsies, a few clear digital images of the gross lesions is very helpful when interpreting the case.
- Collect samples for microbiologic testing first
  - Isolate the bowel segments for submission and tie off with string to keep contents intraluminal.
  - Keep gastrointestinal samples separate from other tissue samples when submitting or shipping samples to a diagnostic laboratory.
- Samples for histologic examination
  - Multiple tissues from multiple sites in 10% buffered formalin.
  - Lesional and nonlesional tissues.
  - Ensure that luminal and serosal surfaces are adequately bathed with formalin.

REFERENCES

1. Schummer A, Nickel R, Sack WO. The alimentary canal of the ruminants. In: The viscera of the domestic mammals. 2nd revised edition. New York: Springer-Verlag; 1979. p. 148, 168.
2. Dyce KM, Sack WO, Wensing CJ. The head and ventral neck of the ruminants. In: Textbook of veterinary anatomy. 3rd edition. Philadelphia: Saunders; 2002. p. 636–7.
3. Helman RG. Interpretation of basic gross pathologic changes of the digestive tract. Vet Clin North Am Food Anim Pract 2000;16:1–22.
4. Thompson K. Bones and joints. In: Maxie MG, editor. Jubb, Kennedy, and Palmer's Pathology of domestic animals, vol. 1, 5th edition. Elsevier; 2007. p. 98–9.
5. Brown CC, Baker DC, Barker IK. Alimentary System. In: Maxie MG, editor. Jubb, Kennedy, and Palmer's Pathology of domestic animals, vol. 2, 5th edition. Elsevier; 2007. p. 20–1.
6. Masuda EK, Kommers GD, Martins TB, et al. Morphologic factors as indicators of malignancy of squamous cell carcinomas in cattle exposed naturally to bracken fern (Pteridium aquilium). J Comp Pathol 2011;144:48–54.
7. Radostits OM, Gay CC, Hinchcliff KW, et al. Diseases of the alimentary tract - II. In: Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs, and goats. Saunders Elsevier; 2007. p. 325–36.
8. Soulsby EJ. Nematodes. In: Helminths, arthropods, protozoa of domesticated animals. 6th edition. Baltimore (MD): Williams and Wilkins; 1974. p. 281–3.
9. Bowman DD. Helminths. In: Georgis’ parasitology for veterinarians. 9th edition. St Louis (MO): Saunders Elsevier; 2009. p. 124, 211.
10. Munz E, Dumbell K. Bovine papular stomatitis. In: Coetzer JAW, Tustin RC, editors. Infections diseases of livestock, vol. 2, 2nd edition. South Africa: Oxford University Press; 2004. p. 1289–90.
11. Brown CC, Baker DC, Barker IK. Alimentary system. In: Maxie MG, editor. Jubb, Kennedy, and Palmer's pathology of domestic animals, vol. 2, 5th edition. Elsevier; 2007. p. 39, 42–43, 46–50, 54, 116, 142, 200–1, 285.
12. Konig HE, Liebich HG. Digestive system. In: Konig HE, Liebich H-G, editors. Veterinary anatomy of domestic mammals: textbook and colour atlas. 3rd edition. Stuttgart (Germany): Schattauer; 2007. p. 338–9.
13. Campbell JR. Effect of bovine viral diarrhea virus in the feedlot. Vet Clin North Am Food Anim Pract 2004;20:39–50.

14. Teixeira AF, Kuhnel W, Vives P, et al. Functional morphology of unguiculiform papillae of the reticular groove of the ruminant stomach. Ann Anat 2009;191:469–76.

15. Trent AM. Surgery of the abomasum. In: Fubini SL, Ducharme NG, editors. Farm animal surgery. St Louis (MO): Elsevier; 2004. p. 196–240.

16. Valli VE. Hematopoietic system. In: Maxie MG, editor. Jubb, Kennedy, and Palmer’s pathology of domestic animals, vol. 3, 5th edition. Elsevier; 2007. p. 199–200.

17. Abutarbush SM, Radostits OM. Jejunal hemorrhage syndrome in dairy and beef cattle: 11 cases (2001 to 2003). Can Vet J 2005;46:711–5.

18. Berghaus RD, McCluskey BJ, Callan RJ. Risk factors associated with hemorrhagic bowel syndrome in dairy cattle. J Am Vet Med Assoc 2005;226:1700–6.

19. Bielefeldt-Ohmann H. The pathologies of bovine viral diarrhea virus infection: a window on the pathogenesis. Vet Clin North Am Food Anim Pract 1995;11:447–76.

20. Saliki JT, Dubovi EJ. Laboratory diagnosis of bovine viral diarrhea virus infections. Vet Clin North Am Food Anim Pract 2004;20:69–84.