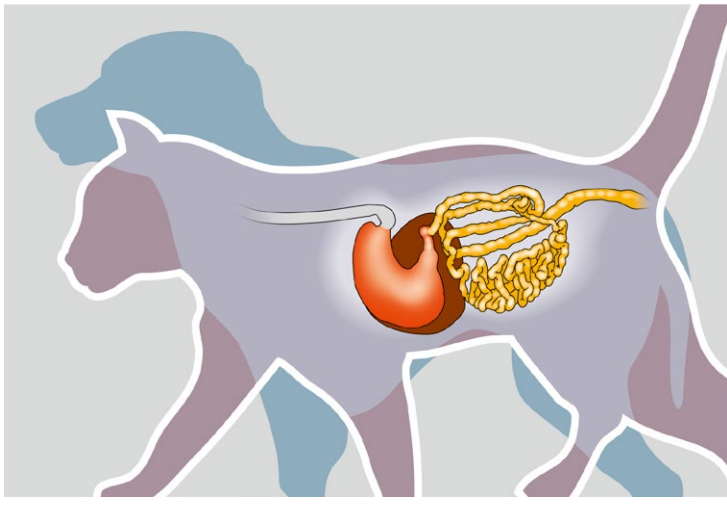
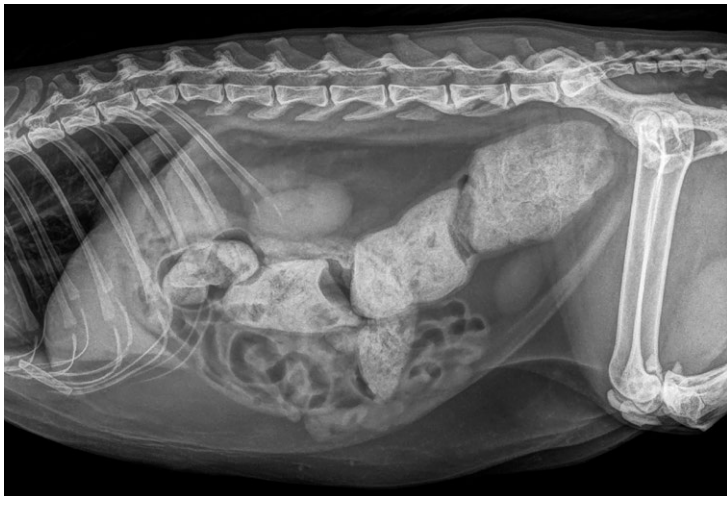
Romy M. Heilmann | Jonathan A. Lidbury |
Jörg M. Steiner (eds.)

Small Animal Gastroenterology

Second, revised and expanded edition





VET EXPERTISE

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Preface

Herakles is quoted as stating that "Change is the only constant in life", and indeed, wherever we cast our glance, the world around us is changing. Some change may be challenging or even threatening, while other changes make our lives easier. A lot has changed over the last 15 years in the field of small animal gastroenterology, and in fact, the field has expanded and many more veterinarians have become interested in this field. The second edition of this book is a testament to this change. The list of authors has grown, and the three editors of this edition feel a sense of gratitude and humility to have had the chance to work with such a formidable group of authors – they have made this project what it is – a comprehensive and up-to-date practical reference text for small animal gastroenterology.

As was the case for the first edition, the first part of this edition is again dedicated to diagnostic modalities that are crucial for working up patients with gastrointestinal signs. We hope the reader will find useful tips for everyday practice in this section. The second section is dedicated to the systemic work-up of the most commonly encountered gastrointestinal signs in dogs and cats. The third section of the book is dedicated to a systematic review of specific diseases categorized by the segment of the gastrointestinal tract that it affects. All sections of the book are carefully referenced so that the reader can quickly find the primary literature on which the information provided is based.

We hope that you find this book useful in your everyday practice. It has been a privilege to edit the second edition of this text!

Texas and Leipzig, Spring 2024
Jörg M. Steiner, Romy M. Heilmann, Jonathan A. Lidbury

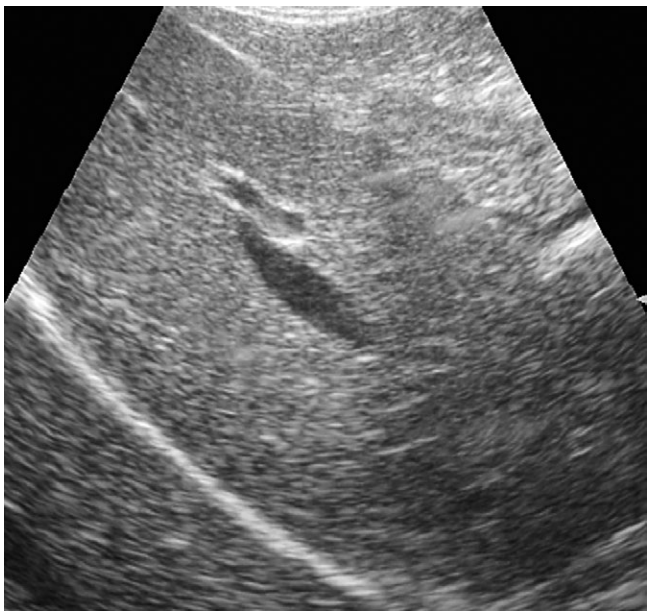


Fig. 1-47 Normal canine liver

The liver has a medium echogenicity. Both portal veins (hyperechoic walls) and hepatic veins (no walls) are visible.

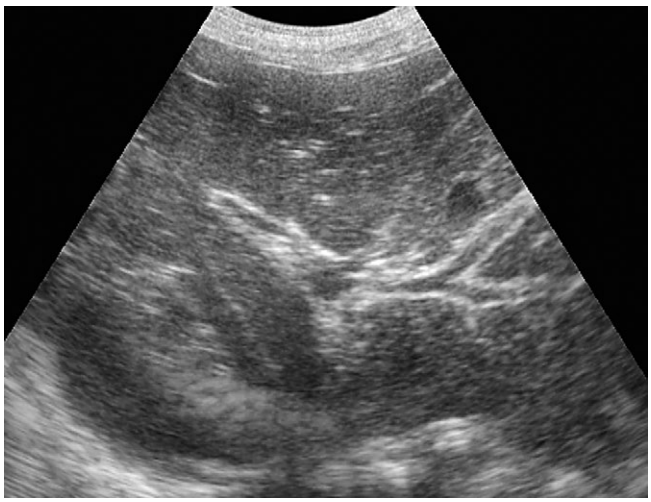


Fig. 1-48 Cholangiohepatitis

Ultrasound image of a hypoechoic liver in a 2.5-year-old male cairn terrier with icterus and vomiting. The liver is enlarged, hypoechoic, and the portal veins are very prominent and hyperechoic throughout the entire liver. Histopathologic diagnosis: cholangiohepatitis.

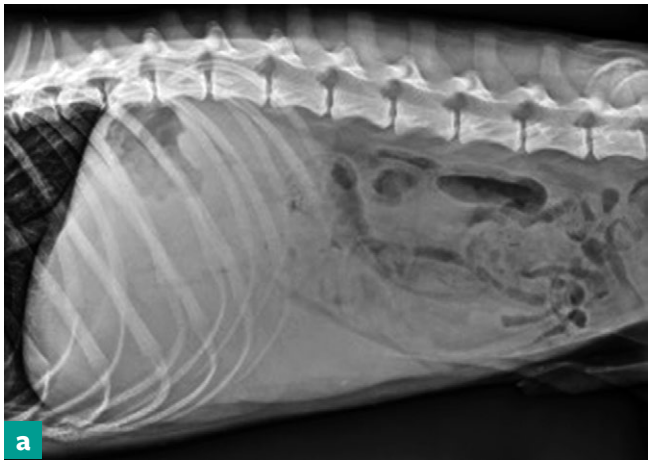
1.4.7.1 Hepatic parenchymal disease

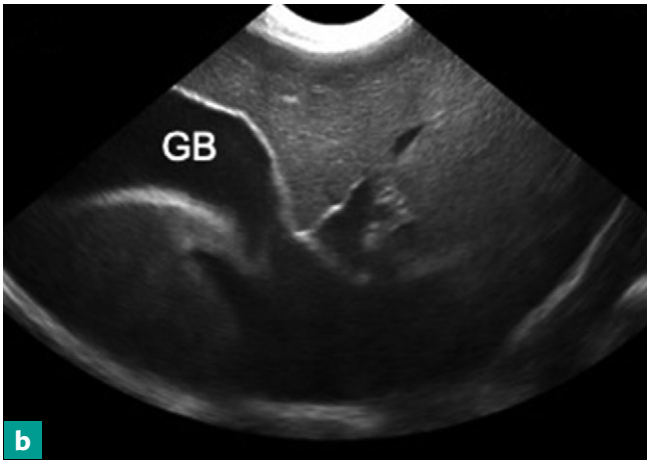
Abdominal radiography may show an enlarged liver in the cranial abdomen, but this finding is nonspecific, and differentiating generalized disease vs. a mass can be difficult. Ultrasonographic changes in patients with hepatic parenchymal disorders are generally focal or diffuse. Diffuse processes are more difficult to recognize than focal or multifocal processes, and a change in the echogenicity of the liver is the main ultrasonographic finding. The most common diffuse diseases in dogs and cats are inflammatory diseases, round-cell neoplasia, non-round-cell pre-nodular metastatic neoplasia, lipidosis, and vacuolar hepatopathy. Ultrasound criteria of echogenicity, diffuse or patchy hyperechoic or hypoechoic echotexture, uniform vs. coarse echotexture, portal venous clarity, and liver lobe geometry have not been found to be discriminatory for the various causes of diffuse liver disease and have a mean accuracy of only 36.5%.[95] This underscores the importance of obtaining samples for cytological or histological analysis in dogs and cats with diffuse liver disease.[96]

In patients with **acute liver injury**, ultrasonography is important for assessing the size and architecture of the liver. The liver may appear normal or enlarged with a generalized decrease in echogenicity. When the echogenicity is decreased, the portal veins appear much more visible, i.e., more hyperechoic than usual (▶ Fig. 1-48). This finding is nonspecific, and differential diagnoses may include toxic injury, infectious hepatitis, metabolic disease, trauma, vascular compromise, cholangiohepatitis, amyloidosis, lymphoma, and passive congestion.

Increased echogenicity of the liver can be detected in a number of conditions, including vacuolar hepatopathy (e.g., hepatic lipidosis in dogs or steroid hepatopathy in cats) and chronic hepatic diseases, such as cholangitis, hepatitis, cirrhosis, and neoplasia, such as histiocytic sarcoma, mast cell tumor, or lymphoma (▶ Fig. 1-49).[94,97–99]

Feline hepatic lipidosis is a common cause of intrahepatic cholestasis that can be recognized ultrasonographically (▶ Fig. 1-50). The liver of affected cats is enlarged, has rounded borders, and shows an increased echogenicity equal to or greater than that of the spleen. In addition, the liver may appear isoechoic or hyperechoic compared with omental fat and hyperechoic to falciform fat.[100] Beam attenuation may also occur, and the dorsal region of the liver may be difficult to visualize. The appearance of vascular structures is also diminished. It is important to remember that in obese cats, the liver will often be diffusely hyperechoic as an incidental finding.[101] CT has also been used to diagnose feline lipidosis.[102] Attenuation (as measured in Hounsfield units) in cats with induced lipidosis has been reported to be decreased compared with healthy controls.





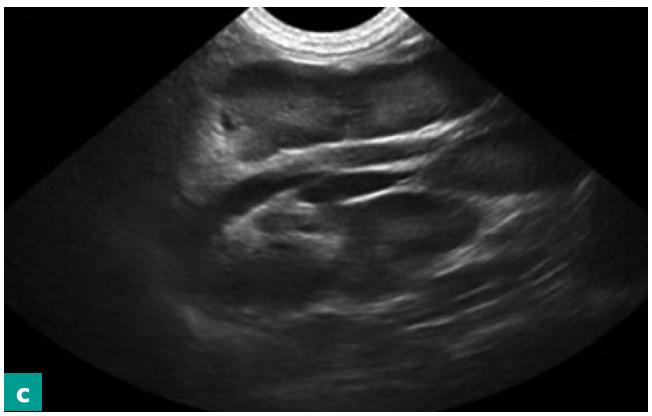


Fig. 1-49 Lymphoma

a Lateral radiograph of the abdomen of a 1-year-old male Standard poodle that presented with lethargy and icterus. The liver and spleen are both enlarged.

b Ultrasound image of the liver. The liver is enlarged and diffusely hyperechoic such that the portal vein markings are not visible. **GB** = gallbladder

c Multiple enlarged jejunal lymph nodes

Diagnosis: intestinal lymphoma.

Cirrhosis and **end-stage chronic inflammatory disease** are less common in cats than in dogs, but hepatic fibrosis may be seen in cats. Ultrasonographic features of cholangitis (formerly termed cholangiohepatitis) range from normal, hyperechoic, hypoechoic, or heterogenous and shows a liver that is either normal in size or enlarged.[103] If portal hypertension is present, peritoneal effusion and splenomegaly can occur. Hepatic cirrhosis and chronic hepatitis are difficult to diagnose by ultrasound due to a large overlap of ultrasonographic features with those of other diseases and can have a similar appearance to those of neoplastic disease.[104] In dogs with idiopathic chronic hepatitis, the liver may be small or normal in size and may contain nodules. Ascites may or may not be present. Regenerative nodules tend to be round and distinct, and the surrounding liver may be of normal or increased echogenicity. Ultimately, a biopsy or fine needle aspiration is needed to arrive at a more definitive diagnosis.

In the **presence of ascites** without hepatic venous congestion, spectral Doppler evaluation of the portal vein can be used to rule out portal hypertension. The velocity of portal venous flow in patients with cirrhosis is generally reduced.[105] In the presence of portal hypertension and ascites, secondary

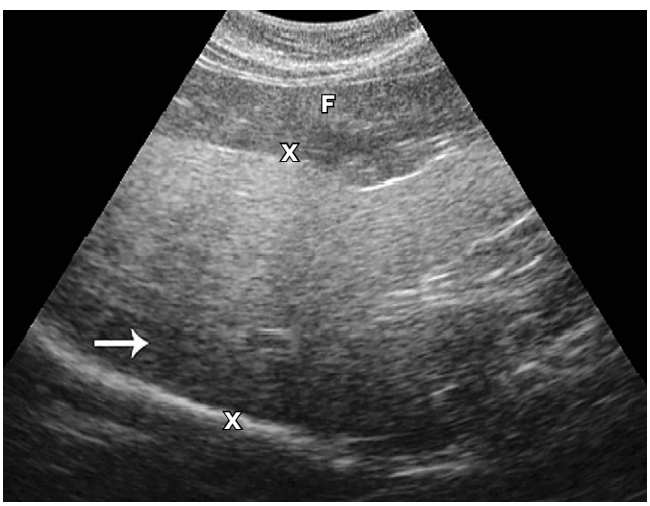


Fig. 1-50 Hepatic lipidosis

Ultrasound image of a hyperechoic liver in a 16-year-old male European short-haired cat that presented with anorexia. The hyperechoic liver (**between x–x**) is homogenous, and few vessels are visible. The echogenicity is equal to or greater than that of the spleen and surrounding mesentery and falciform fat (**F**). Note the hypoechoic appearance of the liver dorsally (**arrow**) due to beam attenuation. Differential diagnoses include hepatic lipidosis, diabetes mellitus, and neoplasia. Cytologic diagnosis: hepatic lipidosis

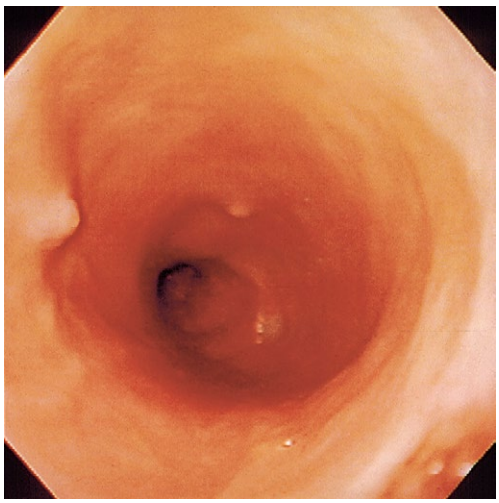


Fig. 1-71 Duodenal papilla

Endoscopic view of a duodenum showing the duodenal papilla at the 9 o'clock position. (Used with permission from Fossum T, 2002[33])

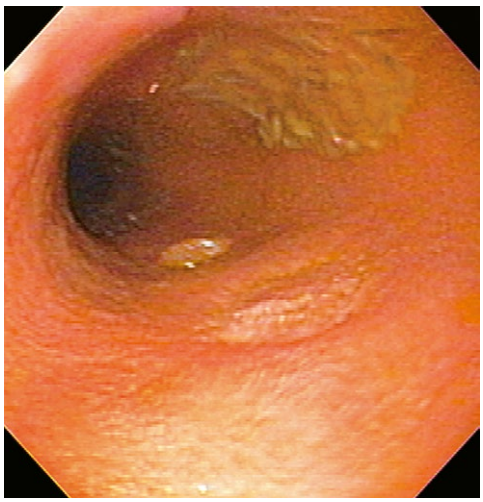


Fig. 1-72 Lymphoid follicle

Endoscopic view of a duodenum showing a crater-like depression at the 5 o'clock position, which represents a normal lymphoid follicle.

1.5.4 Endoscopical findings

1.5.4.1 Physiologic findings

The normal esophagus has a smooth texture, except in the cat, where the distal esophagus has a ribbed texture, where the striated muscle is replaced with smooth muscle.[1,7] Black pigment is usually visible in chow chows or shar-peis. The lower esophageal spincter (LES) may have a reddened area protruding slightly into the esophageal lumen, which is normal. The stomach should have a smooth texture.[2] One may see numerous dots on the mucosa in some patients. The duodenum has a definite, fine texture due to the villi.[3] One can typically see the duodenal papilla (▶Fig. 1-71) and depressions that represent lymphoid follicles (▶Fig. 1-72). When using a video endoscope, the examiner can often appreciate individual villi.

1.5.4.2 Abnormal findings

1.5.4.2.1 Esophagus

Although endoscopy may help to detect a grossly **distended esophagus** (i.e., megaesophagus, ▶Chap. 5.3.8), endoscopy is a poor diagnostic tool for esophageal weakness.[7] Only patients with marked esophageal dilation will be obvious during endoscopy; fluoroscopy is a much more sensitive (and less invasive) way to diagnose esophageal weakness. Also, some preanesthetics (e.g., ketamine or xylazine) can cause the esophagus, stomach, and intestines to become flaccid.[20]

Some **esophageal tumors** present as distinct masses, while others cause strictures (▶Chap. 5.3.12).[7,21] Sarcomas may be caused by *Spirocerca lupi*. *S. lupi* granulomas appear as nodules, with some having a small "crater" or "nipple" from which a red worm may occasionally protrude. Sarcomas, carcinomas, and melanomas are all easily diagnosed by biopsy. Leiomyomas are typically submucosal and are covered with normal mucosa (▶Fig. 1-73). It is often impossible to diagnose these tumors with flexible endoscopic forceps because such forceps cannot cut through the stratified esophageal mucosa. In some dogs, leiomyomas at the LES may only

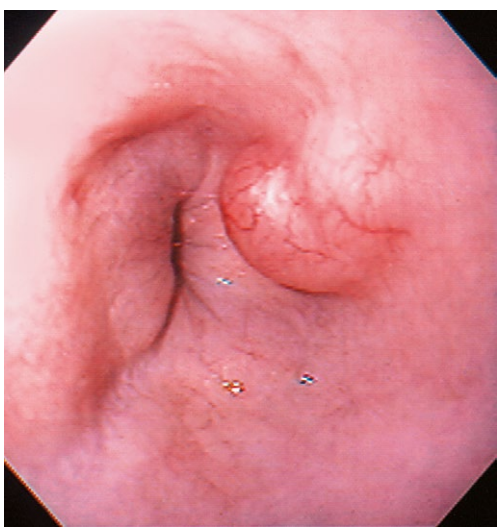


Fig. 1-73 Submucosal leiomyoma in a dog

(Used with permission from Nelson R, 2003[32])

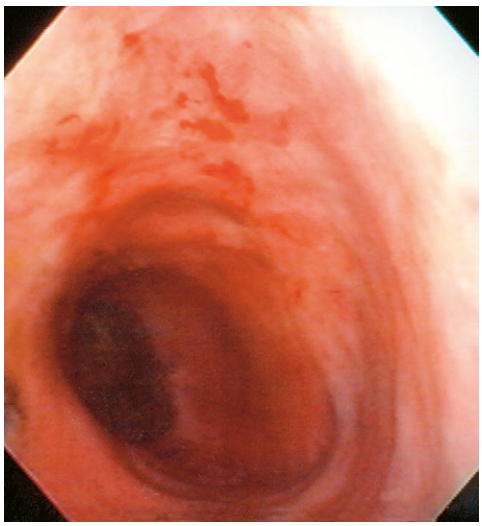


Fig. 1-74 Esophagitis
Note distinct areas of hyperemia.

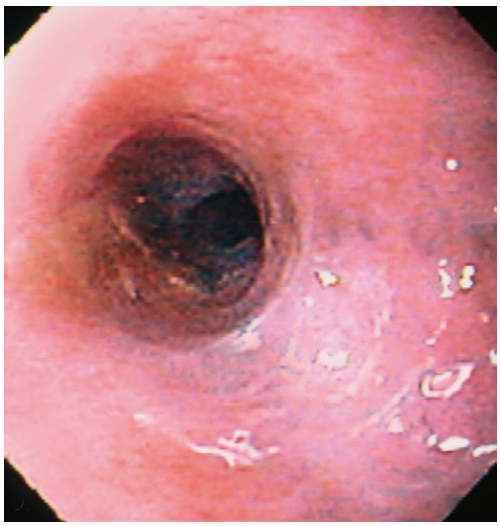


Fig. 1-75 Esophageal stricture
Note the narrowing of the lumen and the white tissue (scar tissue) at the 4 o'clock position.

be visible through the retroflexed position from inside the stomach. Finally, esophageal polyps are rare and, if found, may represent an underlying malignancy that has produced a benign, adenomatous growth over it. A deep biopsy (i.e., surgical or performed with rigid biopsy forceps) that includes the submucosa is necessary for diagnosis.

Esophagitis is usually obvious (▶Chap. 5.3.2). The mucosa is roughened, hyperemic, bleeding, or a combination of those (▶Fig. 1-74).[21,22] Care should be taken to not further damage the esophagus by excessive or rough endoscopic technique. The esophageal mucosa may be biopsied to confirm the diagnosis. Rare patients may have fungal infections (especially pythiosis). The clinician should always seek to identify the cause of esophagitis and carefully examine the stomach and intestines in affected patients for concurrent lesions. Hiatal hernias are sometimes responsible and may be obvious or occult at endoscopy (▶Chap. 5.3.9). Obvious hiatal hernias can have a wide opening at the LES with gastric mucosa protruding into the opening. However, not all patients with a hiatal hernia have esophagitis.

Strictures secondary to esophagitis are usually apparent (▶Fig. 1-75).[23,24] They may occur anywhere but seem more common near the LES. Larger animals may have their esophageal lumen decreased by >75% and still allow the endoscope to easily pass through it. Strictures adjacent to the LES may be mistaken for the LES, especially if the endoscope readily passes through it. Benign and malignant esophageal (i.e., intramural) strictures must be distinguished from extramural strictures (e.g., vascular ring anomaly, ▶Chap. 5.3.11).

1.5.4.2.2 Stomach

Many animals with moderate to severe gastritis have grossly normal-appearing gastric mucosa. Therefore, the gastric mucosa should be biopsied in any vomiting or an anorexic patient undergoing gastroduodenoscopy. Most gastric lesions are not uniformly distributed throughout the entire gastric mucosa. Therefore, one must carefully inspect the entire gastric mucosal surface and biopsy multiple spots throughout the stomach.[7] Any food or water in the stomach should be aspirated, and hairballs or other foreign material removed so that the entire mucosal surface can be examined. If blood is present, one should aspirate as much as possible so that all bleeding lesions may be identified and more closely examined. The operator can also infuse water into the stomach through the tip of the endoscope to help wash out the stomach. The inside of the pylorus is perhaps the hardest place to visualize adequately, but ulcers and *Physaloptera rara* are sometimes found there.

Tumors (▶Chap. 6.3.4) can cause obvious proliferations (▶Fig. 1-76). However, gastric antral mucosal hypertrophy (▶Fig. 1-77) and benign gastric polyps can closely mimic their appearance.[25] Tumors can also cause mucosal ulcerations without any noticeable proliferation (▶Fig. 1-78). Lymphosarcoma is usually easily diagnosed with flexible biopsy forceps, but scirrhous tumors, leiomyomas, or pythiosis may be impossible to diagnose with flexible biopsy instruments. A gastrostomy site can be seen as an elongated

cold LED light sources are now preferred for laparoscopy.[1] A video camera is attached to the telescope and allows the image to be viewed on a video monitor.

A Veress needle is the traditional means of insufflation of the abdominal cavity.[5] The needle consists of an outer cannula with a sharp cutting tip. Within the needle is a spring-loaded obturator that retracts into the needle shaft as it traverses the abdominal wall. Once through the abdominal wall, the blunt-tipped obturator springs forward, preventing needle injury to internal abdominal organs. The Veress needle is then connected to the automatic CO_2 insufflator. Most automatic insufflators are similar and function to dispense gas at a prescribed rate while maintaining a predetermined intra-abdominal pressure. Carbon dioxide is used to prevent air emboli and spark ignition during cauterization.[1]

The traditional cannula units are required to enter the abdominal cavity and are of a corresponding size to receive either the telescope or the biopsy instruments. Several different types of cannula units are available, but the most common ones consist of a sharp trocar housed in an outer cannula. Together they are used to penetrate the abdominal wall. Once in the abdomen, the trocar is removed. Simultaneously, the cannula remains in place, traversing the abdominal wall, and it becomes a portal for the introduction of the telescope or instruments into the abdominal cavity. Cannulas include a one-way valve for maintaining the pneumoperitoneum when instruments are removed.

Common accessory instruments include a palpation probe used to move and palpate abdominal organs and biopsy forceps. The author prefers a 5 mm diameter biopsy forceps with oval biopsy cups to obtain liver, spleen, abdominal mass, and lymph node biopsies. A variety of other biopsy forceps, tissue graspers, and aspiration needles are also available for diagnostic laparoscopy. A "true-cut" type or similar biopsy needle is used both for kidney and deep tissue biopsies. Biopsy needles are passed directly through the abdominal wall and guided to the area to be sampled, much like ultrasound-guided techniques, except the biopsy is visually directed to the area of interest.

1.6.3.1 Procedural considerations

The patient should be fasted for at least 12 hours before the procedure, and the urinary bladder should be evacuated. Laparoscopy is commonly performed using general gas anesthesia, and most patients tolerate the anesthesia and laparoscopy well.[6,7] In some situations, ventilators are used to overcome the potential respiratory effects of abdominal gas distention.

To select the appropriate cannula portal placement sites, one must first determine the objectives of the laparoscopic procedure. The two most common approaches are the right lateral and ventral midline approaches. The author used the right lateral approach for diagnostic evaluation of the liver, gallbladder, right limb of the pancreas, duodenum, right kidney, and right adrenal gland. A ventral approach is useful for many operative procedures and offers good visualization of the liver, gallbladder, pancreas, stomach, intestines, reproductive system, urinary bladder, and spleen. In the ventral approach, visualization is sometimes hindered by the location of the falciform ligament. A complete step-by-step description of the laparoscopy procedure is beyond the scope of this chapter and has been previously described.[1]

1.6.4 Biopsy techniques

1.6.4.1 Intestinal biopsy

Full-thickness small intestinal biopsies can be obtained using a laparoscopy-assisted technique. This involves selecting a portion of the intestine to biopsy, grasping the intestine using atraumatic Babcock forceps, and then exteriorizing the bowel loop through the cannula port site in the abdominal wall. The intestine is then biopsied as it would be done when performing a standard open surgical procedure.[1] The intestine is then returned into the abdomen.

It is often necessary to "run" the bowel first with two grasping forceps to select a section of the bowel for biopsy. The antimesenteric border is then firmly grasped with the Babcock forceps, and the intestine is pulled to the cannula (▶Fig. 1-91). Using a scalpel blade, the grasping forceps cannula incision is extended large enough to exteriorize the loop of bowel. One should visualize the scalpel blade enter the abdominal cavity parallel and adjacent to the cannula shaft. The blade then cuts away from the cannula, increasing the length of the abdominal incision. The cannula, forceps, and intestine all in one are then withdrawn through the incision. Small Gelpi retractors are helpful to keep the incision open. When a 3–4 cm loop of intestine is exteriorized, stay sutures are placed in the intestine to prevent falling back into the abdominal cavity. A small full-thickness biopsy is then obtained in the same manner as one would do when performing an open laparotomy. Following the biopsy and closure of the intestine, the intestinal loop is then returned to the abdominal cavity.

An intestinal biopsy should always be the last laparoscopic procedure to be performed as the pneumoperitoneum is

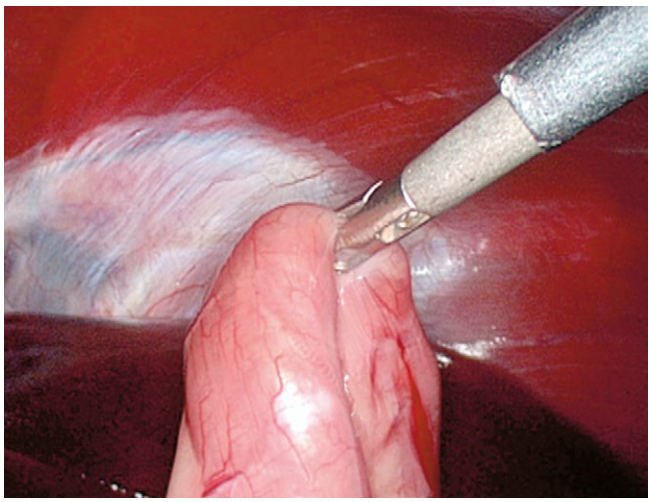


Fig. 1-91 Small intestinal biopsy

View of grasping forceps holding a portion of the small intestine to be exteriorized for a full-thickness biopsy.

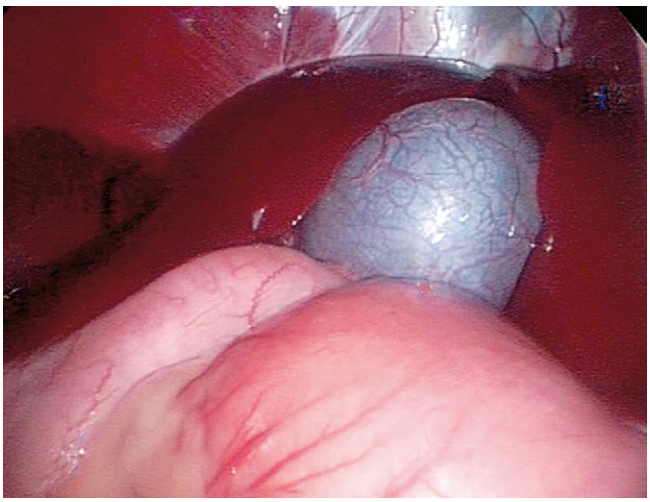


Fig. 1-92 Liver, gallbladder, and intestine

Laparoscopic view through a right lateral abdominal approach showing the liver, gallbladder, and intestines of a normal dog.

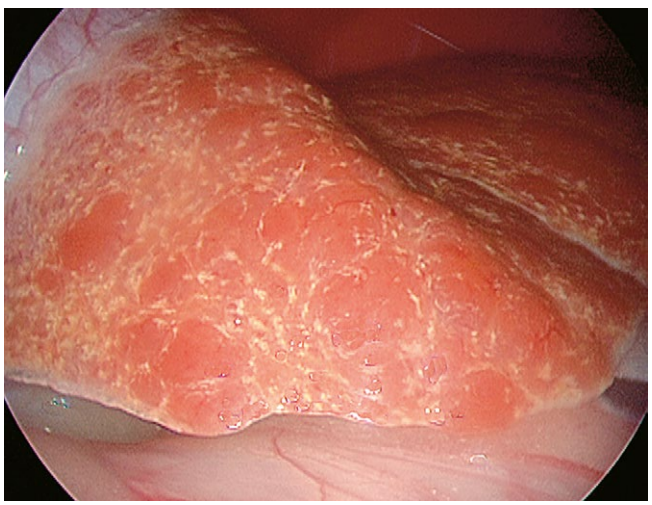


Fig. 1-93 Nodular liver

View of a nodular liver in a dog with hepatocutaneous syndrome.

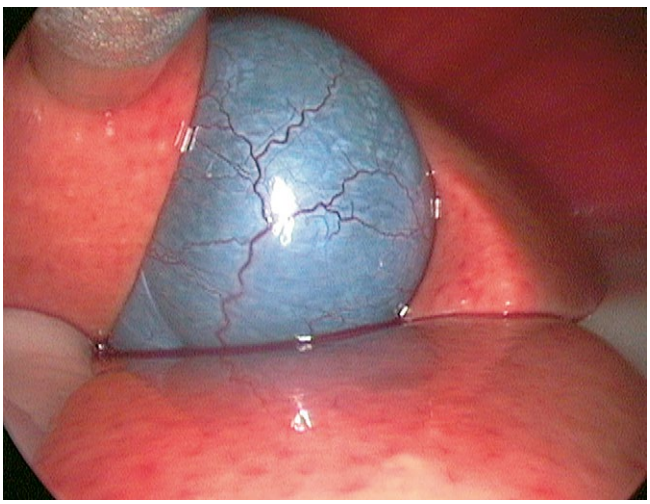


Fig. 1-94 Hepatic lipidosis

View of the liver and gallbladder of a cat with idiopathic hepatic lipidosis.

lost during the procedure. If additional intestinal biopsies or other laparoscopic procedures are to be performed, the trocar cannula must be reintroduced through the abdominal incision, and the incision must be sealed around the cannula to reestablish a pneumoperitoneum. Some use a towel clamp or forceps to close the abdominal wall around the cannula.[8]

1.6.4.2 Liver biopsy

Laparoscopic liver biopsy is considered by many clinicians to be the preferred method of liver biopsy.[9,10] Often, other diagnostic modalities do not provide sufficient tissue or information on the gross appearance of the liver and adjacent organs (▶Fig. 1-92, ▶Fig. 1-93 and ▶Fig. 1-94). Either a right lateral or ventral approach can be used to evaluate most of the liver and extrahepatic biliary system. One can often examine approximately 80% of the hepatic surface but not the entire liver. An ultrasound examination prior to laparoscopy may help plan entry sites and areas to examine. Laparoscopic liver biopsies also provide enough tissue for culture, metal analysis, or other diagnostic uses (▶Chap. 1.8.2 and ▶Chap. 9.3).[1,11]

Prior to liver biopsy, coagulation parameters, including a buccal mucosal bleeding time, are frequently evaluated, but results do not always correlate with the risk of post-biopsy

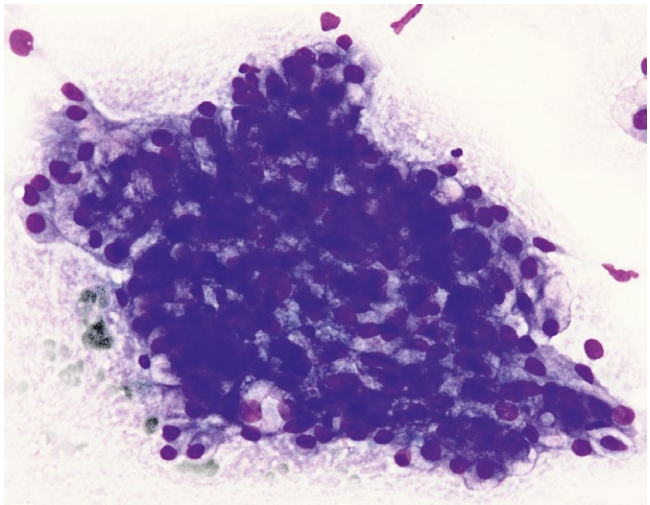


Fig. 1-122 Well-differentiated hepatocellular carcinoma (confirmed by histopathology) in a 13-year-old mixed-breed dog with a surgically removed massive (10 cm diameter) liver mass 1 year prior to fine-needle aspiration (FNA)

Note the large uniform-appearing hepatocyte cluster. Mild diffuse vacuolar changes are present in the cytoplasm (May-Grünwald-Giemsa stain, original magnification 500x).

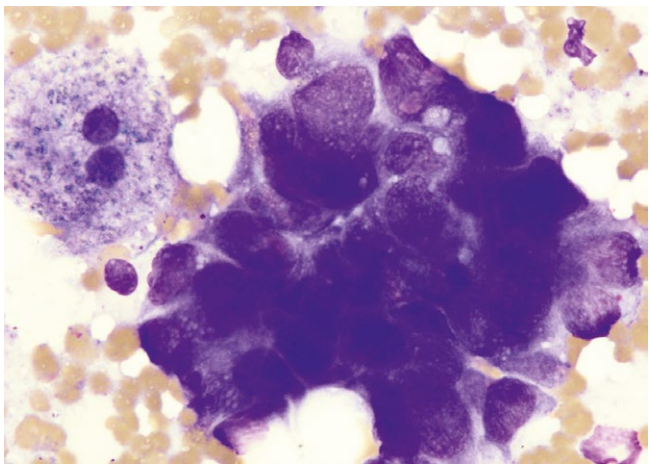


Fig. 1-123 Hepatocellular carcinoma in a 13-year-old female mixed breed dog with multiple hypoechogenic liver masses noted during ultrasonographic examination

Note the moderate anisocytosis, anisokaryosis, prominent nucleoli, variable nuclear: cytoplasmic (N:C) ratio, and nuclear molding. One well-differentiated binucleated hepatocyte is present in the upper left corner of the image (modified Wright's stain, original magnification 1000x).

1.7.3.3 Neoplasia

Hepatic neoplasia (▶Chap. 9.5.7) is classified either as primary (commonly reported neoplastic conditions are hepatocellular adenoma, hepatocellular carcinoma, cholangiocellular [biliary] adenoma, cholangiocellular [biliary] carcinoma, hepatic carcinoid, and hemangiosarcoma) or secondary neoplasia (arising in another organ and metastasizing to the liver).[20] Morphologically, primary hepatic tumors can be subclassified as massive (a single large tumor involving only one liver lobe), nodular (multiple tumors located in different liver lobes), and diffuse (seen as either multifocal nodular changes in several liver lobes or diffuse changes throughout the liver parenchyma).[20,21] The most common primary hepatic tumor in dogs is hepatocellular carcinoma (▶Fig. 1-122 and ▶Fig. 1-123) and, in cats, cholangiocellular carcinoma (▶Fig. 1-124).[20,21] Metastatic tumors in the liver are observed more often than primary liver tumors (▶Fig. 1-125), and metastasis occurs in more than 30% of malignant neoplasms in dogs.[20] The most common metastatic hepatic tumors in dogs are lymphoma (▶Fig. 1-126), pancreatic adenocarcinoma (▶Fig. 1-127), and hemangiosarcoma (▶Fig. 1-128).[20]

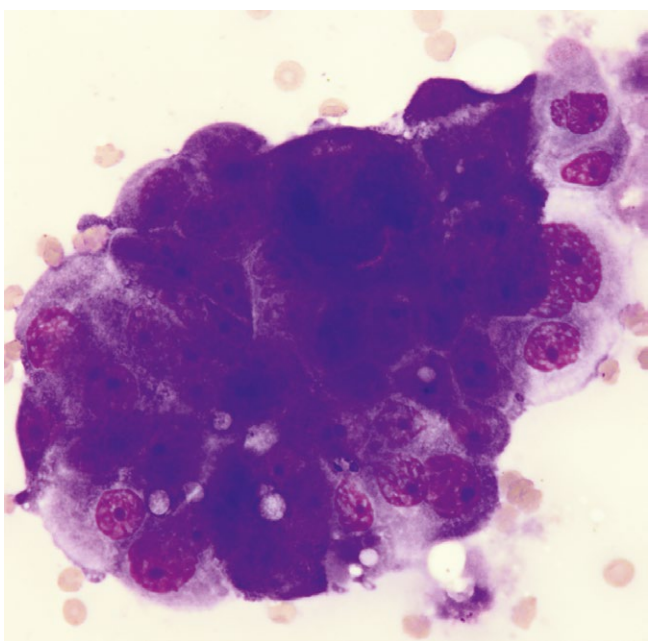


Fig. 1-124 Cholangiocellular carcinoma in a 16-year-old male Persian cat with ascites and marked ultrasonographic changes of the liver parenchyma

Note the moderate anisocytosis, anisokaryosis, and prominent round nucleoli (modified Wright's stain, original magnification 1000x).

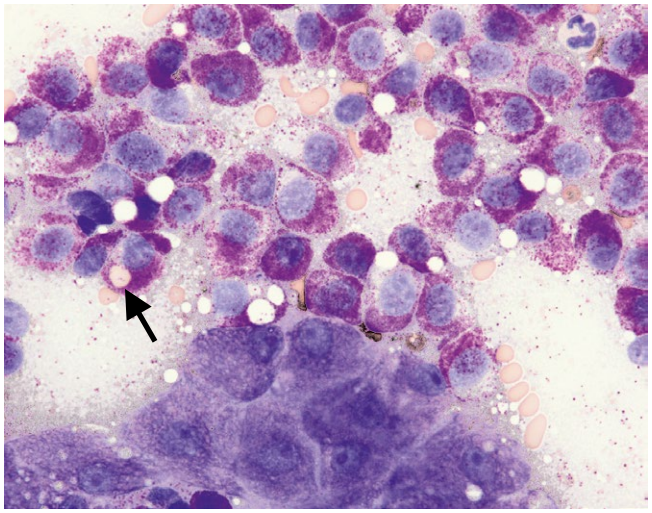


Fig. 1-125 Mast cell tumor in the liver of a 9-year-old male cat with multiple cutaneous mast cell tumors, splenomegaly, and hepatomegaly

Note that some mast cells are erythrophagocytic (**arrow**). A hepatocyte cluster is present at the bottom of the image (modified Wright's stain, original magnification 1000x).

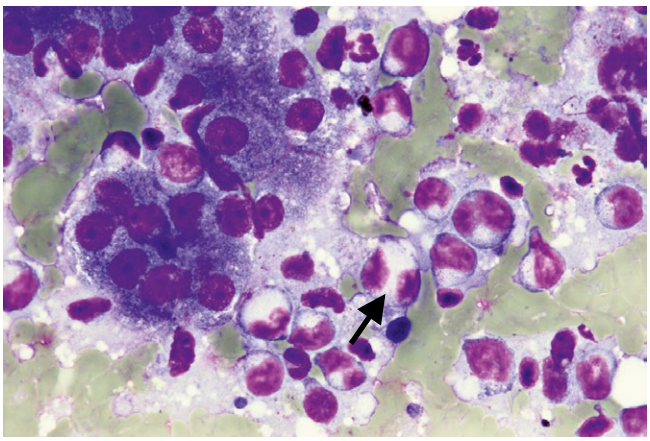


Fig. 1-126 Lymphoma in an 8-year-old male mixed breed dog with hepatomegaly and increased liver enzyme activities

Large immature lymphocytes (likely of T-cell origin) are seen between hepatocytes. Some lymphocytes contain small magenta cytoplasmic granules. Lymphoglandular bodies (cytoplasmic fragments from the neoplastic lymphocytes) are noted in the background. A bizarre mitotic figure [**arrow**] is seen in the center (modified Wright's stain, original magnification 1000x).

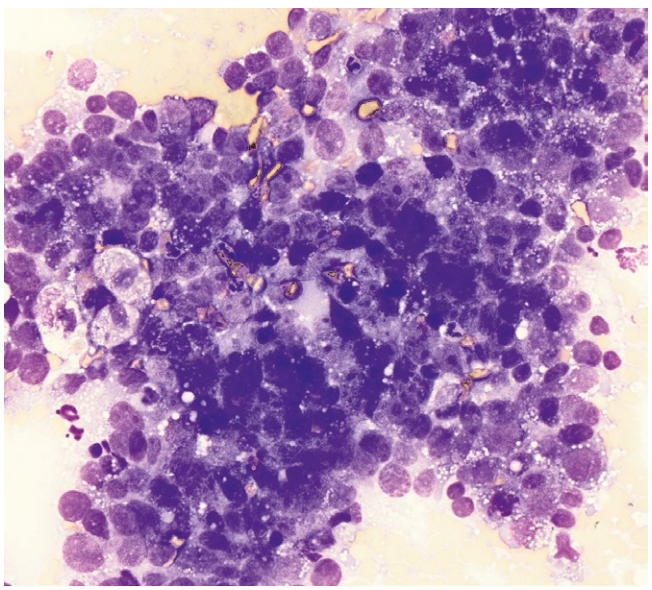


Fig. 1-127 Metastatic pancreatic adenocarcinoma in the liver of an 8-year-old male Labrador retriever with increased liver enzyme activities, hyperbilirubinemia, hypoproteinemia, and hypoalbuminemia

Pancreatic adenocarcinoma with hepatic, intestinal, lymph node, and mesenteric metastases was diagnosed on histopathology. Fine-needle aspiration (FNA) cytology is highly cellular, containing clusters of epithelial cells that fulfill cytologic criteria of malignancy (modified Wright's stain, original magnification 500x).

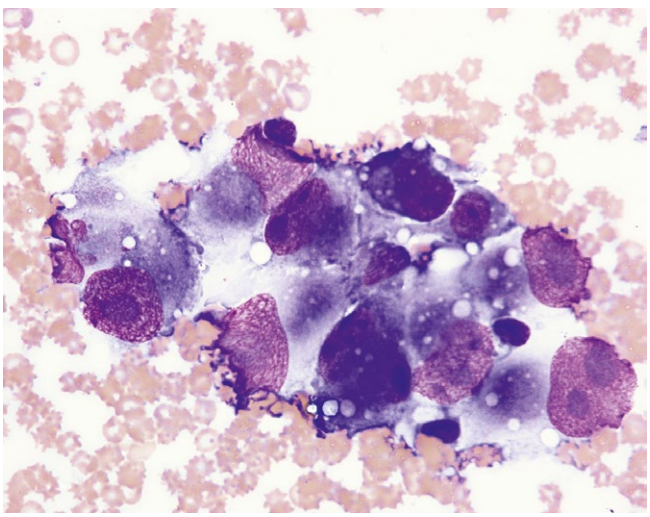


Fig. 1-128 Hemangiosarcoma in the liver of a 10-year-old male German shepherd dog with a splenic mass, enlarged and ultrasonographically heterogeneous liver, and increased liver enzyme activities

Note the large spindle cells with stippled chromatin, multiple prominent nucleoli, macronucleoli, and a plump cytoplasm (modified Wright's stain, original magnification 1000x).

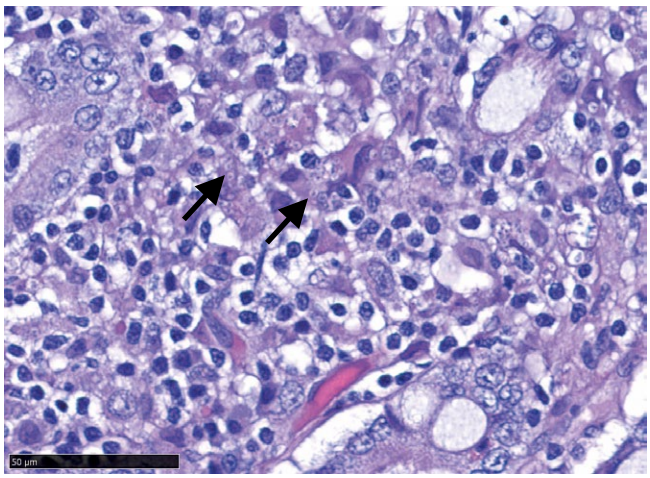


Fig. 1-148 *Histoplasma* spp. infection in the colon of a dog
Macrophages and lymphocytes infiltrate the lamina propria. The cytoplasm of macrophages occasionally contains round, 2 µm-diameter yeasts surrounded by a clear halo (**arrows**) (H&E stain).

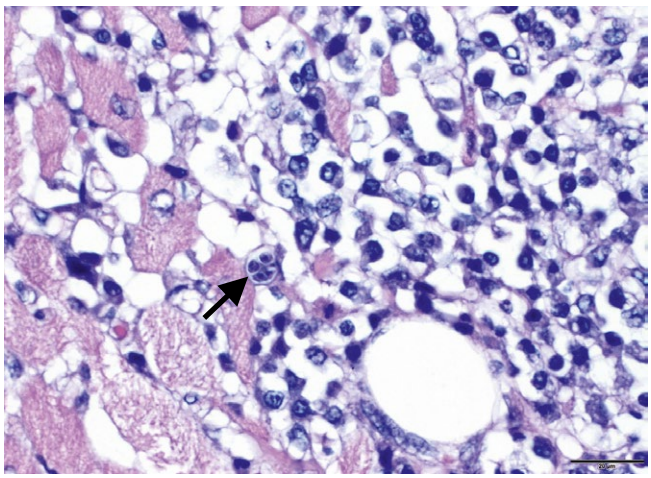


Fig. 1-149 *Prototheca* spp. infection in the colon of a dog
Macrophages, plasma cells, and lymphocytes infiltrate the external muscularis layer. A 7 µm-diameter, oval alga with endospores (**arrow**) is seen within the inflammation (H&E stain).

In endemic areas, canine leishmaniasis (*Leishmania infantum*) may uncommonly manifest as large bowel diarrhea associated with histiocytic colitis.[93] The transmural inflammatory infiltrate is predominantly histiocytic with variable numbers of plasma cells, lymphocytes, and neutrophils.[93] Protozoal amastigotes are round to ovoid, 2.5–5 µm × 1.5–2 µm in size, and found in the cytoplasm of macrophages, often contained within round, clear, parasitophorous vacuoles.[94] *Leishmania* spp. amastigotes need to be differentiated from yeasts of *Histoplasma* spp., with the protozoa having a small, rod-shaped structure (kinetoplast) adjacent to the nucleus that may be difficult to visualize on histologic tissue sections. The protozoa typically stain with Giemsa, but not GMS, and may not stain or stain faintly with PAS.[94] The diagnosis can be confirmed with PCR or IHC.[93,94]

Prototheca spp. is a rare cause of pyogranulomatous colitis in dogs due to disseminated systemic infection (▶Chap. 7.3.1.5.3). Algae are unicellular, spherical to oval, with sporangia ranging from 7–13 µm (*P. wickerhamii*) to 14–25 µm (*P. zopfii*). Algae undergo endosporulation, exhibiting internal septations (▶Fig. 1-149). The organisms might be difficult to identify with routine stains but are revealed by PAS and GMS stains.[95]

Granulomatous colitis and enteritis are rarely associated with gastrointestinal and/or systemic mycobacterial infections in dogs and cats.[96,97]

In endemic areas for bovine tuberculosis, the gastrointestinal manifestation may be seen in cats that ingest raw bovine milk or viscera contaminated with *Mycobacterium*.

Mycobacterial lesions are typically nodular and transmural, corresponding to aggregates of epithelioid macrophages and multinucleated giant cells. In these cases, an involvement of the regional lymph nodes is common. A variable number of acid-fast bacilli are detectable with Ziehl-Neelsen or other acid-fast stains.[97] Sporadic intracellular bacilli are seen in infections with members of the tuberculosis complex (*M. tuberculosis, M. bovis, M. microti*). In contrast, numerous intracellular and/or extracellular bacilli are seen in infections with the non-tuberculous mycobacteria (e.g., *M. avium, M. malmoense, M. fortuitum, M. branderi/shimoidei*).[96,97]

In **canine schistosomiasis** (▶Chap. 7.3.1.4), caused by the trematode *Heterobilharzia americana*, multiple mineralized granulomas may be present throughout the large and small intestinal wall, sometimes concentrated in the submucosa.[98] These granulomas can also be seen in the liver and pancreas, among other organs.[99] The trematode eggs incite an inflammatory response while migrating from the venules to the intestinal lumen. The eggs vary from 50–75 µm in diameter, contain a yellow to brown, refractile eggshell with an internal miracidium (▶Fig. 1-150), and are often mineralized and degenerated.[98] Adult trematodes are rarely detected within the veins.[99]

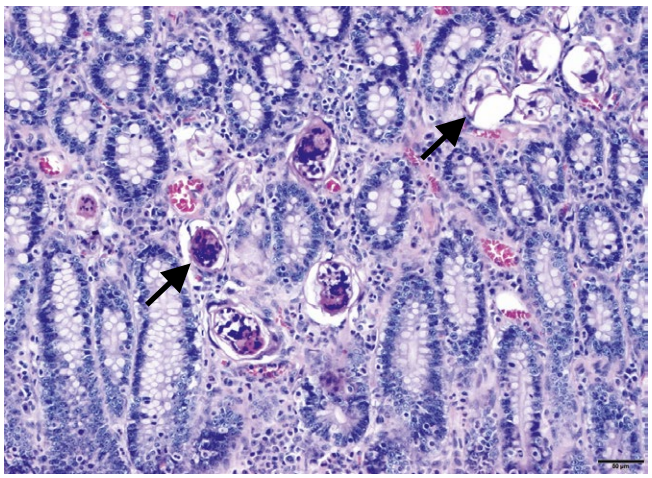


Fig. 1-150 *Heterobilharzia americana* infection in the jejunum of a dog

Multiple 40–50 µm-diameter trematode eggs with refractile shells (**arrows**) are seen in the lamina propria and within the lumen of veins (H&E stain).

Trichuriasis (▶Chap. 8.3.1.1), caused by the whipworm *Trichuris* spp., can result in typhlitis or typhlocolitis in dogs and cats.[100,101] The severity and composition of the inflammatory infiltrate are variable and characterized by eosinophils, neutrophils, lymphocytes, and/or plasma cells with adult nematodes and larvae on the mucosal surface or embedded in the mucosa. Rarely, *Trichuris* spp. penetrate the submucosa and tunica muscularis. Adult nematodes contain coelomyarian musculature, bacillary bands, and stichosomes; eggs contain bipolar plugs.[101]

In cats, the nematode *Strongyloides* spp. can penetrate the colonic mucosa and produce nodular lesions ranging from 1–8 mm in diameter with a central depression. The nodules extend from the mucosa to the submucosa and consist of adenomatous hyperplasia of the glands associated with a lymphoplasmacytic and eosinophilic inflammatory response to the nematode adults, rhabditiform larvae, and eggs. Adult females are approximately 100 µm in diameter with platymyarian musculature, paired genital tracts, and an intestine composed of uninucleate cells.[71]

The protozoa *Tritrichomonas blagburni* may cause lymphoplasmacytic and/or neutrophilic colitis in young cats, resulting in chronic large bowel diarrhea (▶Chap. 8.3.3). On histopathology, the trichomonads are elongate, teardrop- to crescent-shaped, ranging from 4–8 mm long with eosinophilic cytoplasm and a faint, hyperchromatic, round-to-oval nucleus. In a study including naturally infected cats, the organisms were histologically detected in approximately 50% of the examined tissue sections.[102] When present, the trichomonads are more frequently seen on the mucosal surface adjacent to the epithelium and occasionally in the lumen of the crypts or invading the lamina propria. On histopathology, the trophozoites cannot be differentiated from those of *Giardia* spp., requiring fecal culture or PCR analysis.[102]

Amoebiasis is a rare cause of ulcerative, fibrinonecrotizing typhlocolitis in dogs and cats.[103,104] The disease caused by *Entamoeba histolytica* appears to be more severe in animals with concurrent *Ancylostoma* spp. or *Trichuris vulpis* infection. Initial lesions are characterized by superficial mucosal erosions, while established colitis cases exhibit a characteristic flask-shaped, deep ulcer with a narrow neck through the mucosa and a broad base in the submucosa.[8] Amoebae may be seen at the margins of the lesions or admixed with cellular debris. The trophozoites are approximately 20 µm in diameter (ranging from 6–50 µm), spherical to oval-shaped, with a nucleus containing a central karyosome. The trophozoite cytoplasm stains with PAS.[103]

Coccidiosis is caused by *Cystoisospora* spp. in dogs and cats. Multiplication of the organism occurs mainly in the small intestinal epithelium but is occasionally seen in the large intestine.[76,77]

Enteropathogenic *E. coli* is a cause of enterocolitis in cats and dogs, especially in kittens (▶Chap. 7.3.1.2.5). The associated lesions described in kittens include lymphoplasmacytic or neutrophilic inflammation in the ileum and colon, with epithelial injury and occasional crypt dilation. Gram-negative bacterial rods can be seen attached to the superficial and cryptal epithelium.[66] Pathogenic *E. coli* must be differentiated from Gram-positive enterococci (*Enterococcus hirae*), are considered commensals, and are seen adherent to the intestinal epithelium of healthy kittens.[105] The definitive diagnosis of enteropathogenic *E. coli* requires fecal bacterial culture, with a selection of *E. coli* colonies and screening for pathogenicity determinants, because *E. coli* can be cultured both from healthy and diarrheic animals.[66] Other strains of attaching and effacing *E. coli* 0157:H7 can cause hemolytic-uremic syndrome. *E. coli* colonizing the intestinal tract can release Shiga-like toxins that circulate systemically, resulting in anemia, fibrinous glomerulonephritis, and skin lesions (Alabama rot).

Tyzzer's disease is caused by *Clostridium piliforme*. It is a condition of young animals (▶Chap. 9.5.2) and uncommonly affects immunosuppressed kittens and puppies.[106,107]

1.9 Assessment of gastrointestinal (GI) motility

Frédéric P. Gaschen

1.9.1 Introduction and physiology of GI motility

The scope of this chapter is limited to gastric, small, and large intestinal motility in dogs and cats. It is focused on non-obstructive disorders of GI motility which frequently occur in dogs and cats but may remain unrecognized. Often, they are secondary to acute or chronic inflammation of the GI tract, neighboring organs (e.g., pancreas), or the peritoneum. GI motility may also be perturbed by electrolyte disturbances (e.g., hypokalemia), drugs (e.g., opiates), or abdominal surgery and anesthesia. Finally, more rarely, primary disorders of GI motility may occur (e.g., dysautonomia). In addition, recent studies have shown that disturbances of the gut microbiome may negatively impact GI motility through their effects on the tryptophan pathway that generates serotonin, an essential neurotransmitter in the enteric nervous system.[1]

Gastrointestinal motility is a complex series of events that aims at

1. storing and
2. grinding ingested food into particles of a determined size in the stomach before they are propelled into the small intestine;
3. moving the ingesta through the small intestine while allowing the digestive and absorptive processes to take place;
4. optimizing conditions for fermentation and water reabsorption processes in the proximal colon; and
5. storing and coordinating the evacuation of fecal material from the distal colon and rectum.

GI motility is dependent on smooth muscle contractions that are regulated by a combination of myogenic, neural, and hormonal factors.[2–4] A detailed discussion of the physiology of gastric, small intestinal and large intestinal motility is beyond the scope of this chapter and can be found in the listed references.

1.9.2 Indications for assessment of GI motility

Abnormal GI motility may complicate the clinical presentation and compromise the outcome of diseases, such as acute or chronic gastric or intestinal inflammation, acute pancreatitis or critical illness and delay recovery from abdominal surgery. Clinical signs of decreased/absent GI motility are not specific and vary depending on the circumstances. They may include persisting hyporexia/anorexia, signs of nausea, vomiting, or regurgitation (e.g., following gastroesophageal reflux), cranial abdominal discomfort, abdominal distension, and bloated abdomen. Intestinal sounds are usually absent on abdominal auscultation. If dysmotility is suspected, a basic assessment of GI motility is recommended using widely available techniques, such as survey abdominal radiographs and abdominal ultrasonography (▶Chap. 1.9.3). If these initial investigations remain inconclusive, more advanced techniques can be used based on their availability.

1.9.3 Available methods for evaluation of GI motility

Various techniques can be used to assess GI motility in different segments of the GI tract, as described below and summarized in ▶Tab. 1-23. An ideal technique would be easily accessible in the clinical setting, would require minimal manipulations or blood samplings from the dog or cat, and would offer rapid analysis of biological samples as needed to

Tab. 1-23 Characteristics of available methods for assessment of gastrointestinal motility in dogs and cats

Method	Stomach	Small intestine	Colon
Radiography			
Survey radiography	+	+	+
Liquid barium	+	+	+
Barium meal	+	+	–
BIPS	+	+	+
Ultrasonography			
Qualitative	+	+	–
Quantitative	+	–	–
Scintigraphy			
Nuclear scintigraphy	+	+	+
Tracer studies			
Plasma	+	+	–
Breath	+	+	–
Capsules			
Wireless motility capsule	+	+	+
Videoendoscopy capsule	+	+	–

BIPS = barium-impregnated polyethylene spheres

accurately assess transit times and motility in the GI segment of interest (stomach, small or large intestine). Not surprisingly, such a method is not available at this time; therefore, the strengths and weaknesses of existing methods are listed in ▶Tab. 1-24.

Transit times reported in the literature for the same technique vary widely because of a lack of standardization of the size and caloric content of administered meals as well as of the protocol applied. In addition, transit times may be prolonged because of hospital-associated stress and may also vary from day to day in the same animal.[5,6]

1.9.3.1 Radiographs

Radiographic studies are easily accessible and can provide valuable information. Survey radiographs are required to rule out gastrointestinal obstruction. They may also reveal the presence of non-obstructive ileus (adynamic, functional, or paralytic ileus). A description of the radiologic features of ileus can be found in ▶Chap. 1.4.5.1.

Liquid barium has been widely used to assess GI transit times, and details of the procedure can be found in ▶Chap. 1.4.3. With the exception of mechanical obstructions due to foreign bodies or other space-occupying lesions obstructing the gastric or intestinal lumen, assessment of gastric emptying (GE) of liquids does not accurately reflect GE of solids. When barium is mixed with food, it can easily dissociate from the test meal and empty into the duodenum even though the solid food is still in the stomach, making the study unreliable.

Barium-impregnated polyethylene spheres (BIPS) have been used for the evaluation of GI transit times in dogs and cats. They come in two sizes (1.5 and 5 mm diameter) and can easily be used in practice. However, a correlation between the GE of BIPS and the reference method (radionuclide scintigraphy) has been disappointing in dogs and in cats.[7,8]

1.9.3.2 Ultrasonography

Ultrasound machines are widely available, and transabdominal ultrasound examination of the GI tract is a routine exam in dogs and cats presented with GI signs. As described in ▶Chap. 1.4.4, ultrasound can be useful for detecting morphologic gastric wall abnormalities and qualitatively evaluat-

Tab. 1-24 Strengths and weaknesses of available methods for assessment of GI motility in dogs and cats

Method	Strengths	Weaknesses
Radiography	• Equipment widely available • Survey and contrast studies allow the identification of mechanical GI obstructions. • Survey films may allow diagnosis of severe adynamic ileus	• Survey films are insensitive for assessment of GI motility • Barium contrast studies (liquid barium or barium meal) do not accurately reflect solid-phase GE • GE emptying of BIPS correlates poorly with the gold standard
Ultrasonography	• Equipment widely available • Qualitative: rapid assessment of gastric and small intestinal motility • Quantitative: accurate, real-time assessment of actual GE and gastric motility	• Difficult to perform in obese, painful, or otherwise uncooperative patients or when gas is present in the GI tract • Qualitative: operator needs to be familiar with abdominal ultrasonography • Quantitative: skills are required for identification and observation of the antrum over time. Full studies are time-consuming
Nuclear scintigraphy	Reference method for GE evaluation	• Limited availability • Strict radiation protection protocols must be implemented • License may be required to handle radioactive isotopes
Tracer studies	Accurate assessment of gastric or orocecal transit times	• Repeated sampling of blood or breath air is required • Blood or breath air analyses require specialized equipment that is not easily available in veterinary diagnostic labs • Results are only valid if intestinal and liver functions are normal
Capsules	• Can be used in the home environment • WMC: Pressure curves and motility index provide information on motility • VEC: easily available	• Use limited to dogs > 15 kg • WMC: Significant investment required to purchase equipment • VEC: unsure how fasted transit times correlate with postprandial transit times

GI = gastrointestinal; **BIPS** = barium-impregnated spheres; **GE** = gastric emptying; **VEC** = videoendoscopy capsule; **WMC** = wireless motility capsule

ing contractile activity. A normal stomach containing some food should contract 4–5 times per minute.[9] However, the frequency of antral contractions depends on the degree of filling and time since the last meal; an empty stomach can be in a resting state and show fewer and weaker contractions.[10]

Small intestinal luminal contents and peristalsis can also be evaluated. Reported normal rates of peristalsis are approximately five contractions/minute for proximal duodenum and 1–3 to 5.2 contractions/minute for the rest of the small intestine.[9] As noted for the stomach, these rates may vary depending on the timing of the last meal.[10]

Quantitative assessment of GE is performed using sequential evaluation of antral size after a meal and relies on the postprandial measurement of the cross-sectional area or estimated volume of the relaxed pyloric antrum over time. Animals are positioned in dorsal or right lateral recumbency. As the stomach empties, the size of the antrum decreases and ultimately returns to the fasted dimension. Values are plotted over time to obtain GE times (▶ Fig. 1-178). The technique allows determination of solid-phase gastric emptying time as well as the evaluation of motility parameters, including frequency and amplitude of antral contractions and motility index.[3] It was shown to correlate well with a breath tracer method in dogs[11] and with radionuclide scintigraphy in cats.[12] However, it cannot be reliably quantitatively evaluate small intestinal motility.

1.9.3.3 Radionuclide scintigraphy

Radionuclide scintigraphy is the reference technique for evaluating GE in people, dogs, and cats.[3] The animal is fed a solid meal mixed with radiolabeled technetium (^{99m}Tc) bound to albumin, colloid, disofenin, mebrofenin, or sulfur. Postprandial scans are performed at regular intervals with a gamma camera to allow quantification of the radioactivity present in the stomach. A time curve is drawn, and $t_{1/2}$ (time at which one half of the initial radioactivity remains in the stomach) and $t_{50\%}$ (time at 50% of the area under the curve) of GE can be calculated (▶ Fig. 1-178).[6,12] Another radioactive isotope, ^{111}Indium bound to DTPA (diethylenetriamine pentaacetate), can be used for the assessment of colonic transit time. Unfortunately, scintigraphy is only available at academic institutions and select referral centers and is used mostly for research purposes.

GE $t_{1/2}$ (time at which the gastric radioactivity had decreased to 1/2 of the maximum) ranged from 100–182 minutes in six healthy purpose-bred dogs of approximately the same size fed 30% of their daily caloric requirements with dry kibbles and baby food.[6] It was 196 ± 45 minutes in eight healthy purpose-bred cats after a meal supplying approximately 20% of their daily caloric requirement.[12]

1.9.3.4 Tracers

These techniques use plasma or breath tracers. Markers are mixed with the meal and used for postprandial assessment of GE and/or intestinal transit.

In plasma tracer studies of GE, acetaminophen/paracetamol is rapidly absorbed in the proximal duodenum, and postprandial time plots of the serum concentrations are used to extrapolate GE times. The technique has been used successfully for the assessment of GE but has not yet been validated by comparison to other techniques in dogs.[13] In addition, the accuracy of the method for assessing solid-phase

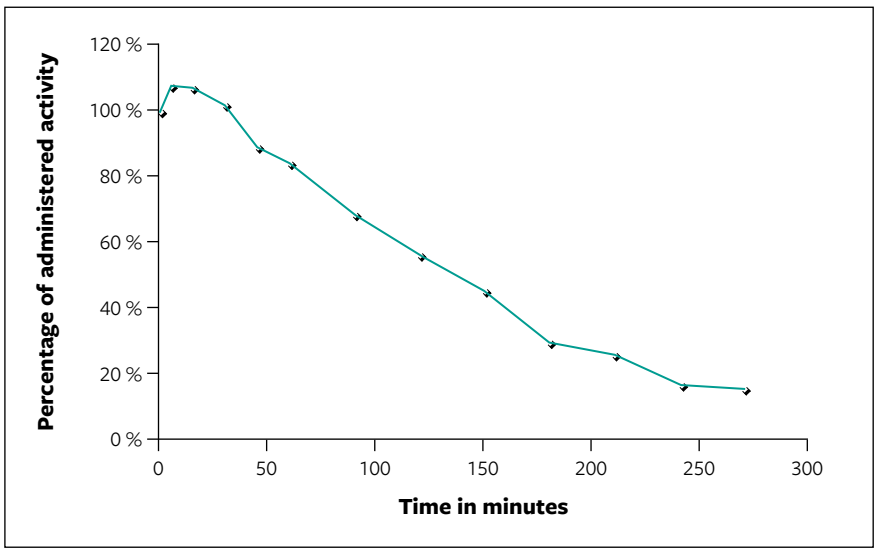


Fig. 1-178 Gastric emptying curve obtained by radionuclide scintigraphy in a healthy dog

Cumulative scintigraphic residual radioactivity in the gastric area plotted against time after ingestion of a dry kibble meal providing 30% of the daily caloric requirements. Gastric half emptying time (GE $t_{1/2}$; time at which the gastric radioactivity had decreased to 1/2 of the maximum) was calculated to be 107.7 min.

2.4.8 Diagnostic investigations

Due to the extensive differential diagnosis list, an array of diagnostic tests may be needed to diagnose the underlying cause. However, this should be staged, starting with a complete blood count, biochemistry, total T4 concentration in cats ≥8 years of age, and urinalysis. If this does not reveal any abnormalities, then fecal flotation, diagnostic imaging with abdominal ultrasonography, serum cobalamin (vitamin B_{12}) and folate (vitamin B_9), baseline cortisol, and trypsin-like immunoreactivity concentrations, and a therapeutic dietary trial should be considered as the next diagnostic steps. For those cases with specific clinical signs, diagnostic investigations should be tailored depending on these signs. An algorithm for the diagnostic investigation of chronic weight loss is presented in ▶ Fig. 2-6.

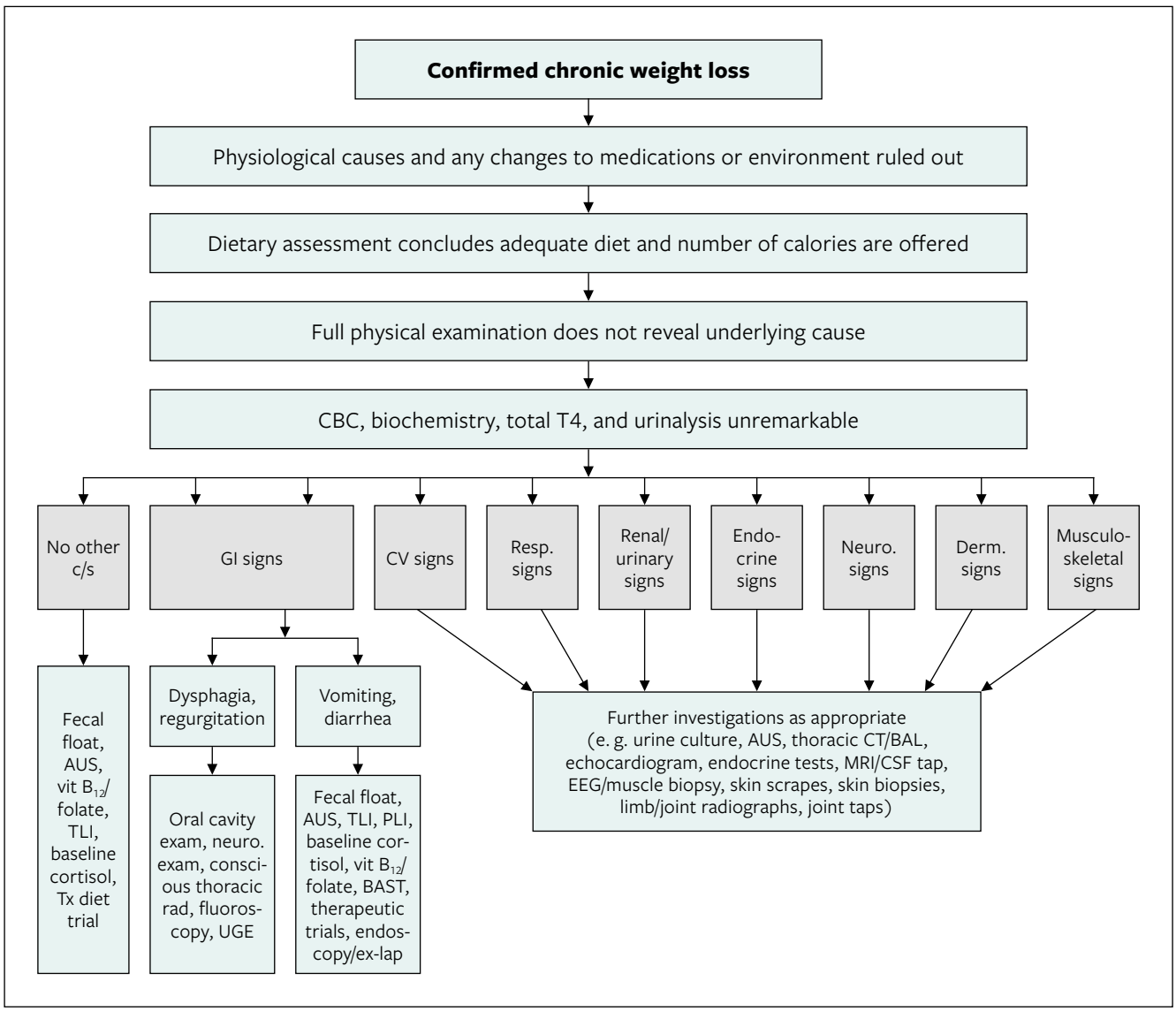


Fig. 2-6 An algorithm for the diagnostic investigation of chronic weight loss in dogs and cats

AUS = abdominal ultrasound, **BAL** = bronchoalveolar lavage, **BAST** = bile acid stimulation test, **CBC** = complete blood count, **c/s** = clinical signs, **CSF** = cerebrospinal fluid, **CT** = computed tomography, **CV** = cardiovascular, **derm** = dermatological, **EEG** = electroencephalogram, **ex-lap** = exploratory laparotomy, **GI** = gastrointestinal, **MRI** = magnetic resonance imaging, **neuro** = neurological, **PLI** = pancreatic lipase immunoreactivity, **rad** = radiograph, **resp** = respiratory, **TLI** = trypsin-like immunoreactivity, **Tx** = therapeutic, **UGE** = upper (± lower) gastrointestinal endoscopy, **vit** = vitamin

2.4.9 Treatment

Treatment should be aimed at addressing the underlying cause of chronic weight loss. However, if treatment is likely to be prolonged or no definitive treatment is available (e.g., idiopathic megaesophagus), assisted feeding via an enteral feeding tube may be needed to ensure the animal meets its daily caloric intake to try to prevent further weight loss. Appetite stimulants such as mirtazapine may be considered prior to the placement of an enteral feeding tube, although this can have varied individual effects and may not increase voluntary intake to meet 100% of the resting energy requirement.

KEY FACTS

- Weight loss of less than 5% body weight is characterized as mild, between 5–10% as moderate, and above 10% as severe. Syndromes related to unintentional weight loss include cachexia and sarcopenia.
- Historical assessment should include the age of the animal, social factors, medical history, a comprehensive diet history, and an assessment of the quality and daily quantity of diet.
- A full physical examination assessing all body systems should be performed to try to ascertain the system primarily responsible for the weight loss.
- Body weight, BCS, and muscle condition should be measured with a standardized scoring system.
- Differential diagnoses for chronic weight loss are extremely broad, encompassing many categories of disease, such as neoplasia, gastrointestinal, pancreatic, hepatic, endocrine, infectious, cardiovascular, pulmonary, neurological, renal, dermatological, dental, musculoskeletal or medications, as well as non-disease related, such as physiological, dietary, and social.
- Treatment should be aimed at addressing the underlying cause of chronic weight loss. However, assisted feeding via an enteral feeding tube may be needed, and appetite stimulants may be considered.

References

The complete list of references for this chapter can be downloaded from vetline.de:
svg.to/9842-02-04

2.5 Clinical evaluation of patients with constipation

Silke Salavati Schmitz

2.5.1 Definition

The normal frequency of defecation is not well defined in dogs and or cats. It is influenced by many internal (activity level, body condition, age, behavior, food intake) and external factors (stress, litter box competition), which cause inter-individual variation. However, most small animals will defecate at least once daily. **Constipation** is defined as infrequent, difficult, or painful evacuation of feces without permanent loss of function, while **obstipation** is defined as a complete inability to pass feces, often indicating permanent functional impairment. The most common clinical signs are therefore straining, tenesmus, and the production of small (often dry and hard) feces or no feces. Generalized dilation and hypomotility of the colon is termed **megacolon**.

NOTE

It is important to differentiate tenesmus associated with constipation from that due to colitis or lower urinary tract disease.

2.5.2 Pathophysiology and differential diagnoses

The main function of the **colon** (apart from immunological functions based on interactions with the microbiota) is the mixing/segmentation and propulsion of feces while retaining water and electrolytes (▶Chap. 8.2). The **rectum** serves as a short-term reservoir. Dilation of the rectal wall induces stronger tonic contraction of the inner rectal sphincter until a threshold is reached that will initiate defecation. This is consciously aided by relaxation of the outer rectal sphincter and tension of abdominal muscles.

The two main mechanisms of constipation are **decreased contractile pressure or increased resistance to the passage of feces**. Causes of increased resistance can be extramural (outside the colonic/rectal wall), mural (within the wall), or luminal, while decreased pressure is always of mural origin and can be of neuromuscular, endocrine, or metabolic origin, and can also be idiopathic (▶Tab. 2-5).

Tab. 2-5 Causes of constipation

Mechanism	Location/origin	Cause	Species difference
Increased resistance	Extramural	Pelvic misalignment (fracture)	Cat >> dog
		Prostatic enlargement or paraprostatic cysts	Dog
		Intrapelvic neoplasia	Dog > cat
		Perineal hernia	Dog
	Mural	Benign solitary mass (e.g., polyp)	Dog >> cat
		Malignant solitary mass (e.g., adenocarcinoma)	Dog = cat
		Diffuse neoplasm (e.g., lymphoma)	Dog = cat
		Stricture (trauma, inflammatory, neoplastic, idiopathic)	Dog > cat
	Luminal	Foreign body or material (e.g., bony meal)	Dog >> cat
Decreased pressure	Neuromuscular	Idiopathic megacolon Hypo- or aganglionosis (Hirschsprung's disease)	Cat >> dog Dog = cat
		Lower motor neuron/spinal cord disease L4-S2: • Cauda equina, intervertebral disc herniation • Sacral spinal cord deformity • Neoplasm	 • Dog >> cat • Cat (Manx) • Dog >> cat
		Dysautonomia (Key-Gaskell syndrome)	Cat >> dog
		Colic ganglioneuritis	Dog = cat
	Endocrine	• Hypothyroidism • (post-treatment for hyperthyroidism)	• Dog • Cat
	Metabolic	Electrolyte abnormalities (e.g., hypercalcemia, hypokalemia)	Dog = cat

There are several **systemic or external causes** of constipation, like obesity, lack of physical activity (e.g., being hospitalized), a change in diet, or administration of drugs that reduce colonic motility (e.g., anticholinergics, opioids) or absorb fecal water (e.g., aluminum hydroxide, sucralfate, barium sulfate). Both general and local **pain** (e.g., bite wounds, anal gland inflammation, perineal hernia) can also cause a reduction in defecation frequency (avoidance behavior).

2.5.3 Diagnostic approach

2.5.3.1 Signalment, history, and physical examination

Signalment can help to pinpoint more likely differential diagnoses: In neonates, congenital abnormalities like hypo-/aganglionosis (Hirschsprung's disease) or *atresia ani* must be considered, while elderly animals are more likely to have neoplasia. In uncastrated male dogs, prostatic disease can lead to constipation. Breed-specific conditions include perianal fistulas in German shepherd dogs (▶Chap. 8.4.3), inflammatory colorectal polyps in miniature dachshunds,[1] intervertebral disc disease in chondrodystrophic dog breeds, and sacral malformations in Manx cats.

Specifics of the **patient history** should be aimed at establishing if a cause is likely local to the rectoanal or perineal area or if more extensive, neurological, or systemic illness is present. The duration of signs and any possible inciting causes (known traumatic events), any drug history, of changes in food or habitats (competition or limited access to litter trays) are important. Some animals can be erroneously presented to the veterinarian for diarrhea (▶Chap. 2.3) or fecal incontinence (▶Chap. 8.3) when only liquid fecal content can be evacuated.

The most important parts of the **physical examination** are abdominal palpation (to assess the severity of constipation/diameter of the colon), an inspection of the perineal region, and neurological and rectal examinations.

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Physical examination is helpful in diagnosing **dysautonomia (Key-Gaskell syndrome)** (▶ Fig. 2-7). This condition is rare, but in endemic regions (Midwestern United States and the United Kingdom) patients (cats are more commonly affected than dogs) present with multiorgan involvement.

Findings include unresponsive mydriasis, ptosis, and third eyelid protrusion, dry rhinarium, reduced tear production (Schirmer tear test <5 mm/min), dry mucous membranes, bladder atony or overflow incontinence, and hypomotility of the gastrointestinal tract resulting in megaesophagus, small intestinal ileus, and megacolon (▶ Fig. 2-7).[2] 0.05% pilocarpine eye drops will induce miosis and retraction of third eyelids in an affected animal but have no effect in healthy animals. Prognosis is poor, and not many animals recover despite supportive treatment.

2.5.3.2 Laboratory testing and diagnostic imaging

The diagnostic approach to patients with constipation will vary depending on the findings from signalment, history and physical examination. Performing a **minimum database** (hematology, serum biochemistry, urinalysis) to detect systemic effects of illness (dehydration), comorbidities, and any metabolic causes (hypokalemia, hypercalcemia) is warranted.

Diagnostic imaging is nearly always indicated. Abdominal radiographs allow assessment of intraabdominal and intrapelvic structures, the severity of constipation, and colorectal content. Abdominal ultrasound allows further characterization of abnormalities (e.g., prostatic enlargement) and detection of intramural intestinal changes.

Fine-needle aspiration (FNA) of abnormal structures can be performed simultaneously. Advanced imaging procedures (CT, MRI) are appropriate to further characterize complex intrapelvic abnormalities or detect intervertebral or musculoskeletal disease.

NOTE

- Abdominal radiographs must be performed with two orthogonal views. Care should be taken to include the entire caudal abdomen and the pelvis.
- If initial radiographic findings are unclear and additional diagnostic imaging modalities are not available, repeat radiographs after instillation of negative or positive contrast per rectum can be informative.

2.5.3.3 Tissue biopsy

Biopsy of intra-abdominal or intrapelvic structures should be considered if the cytologic examination of FNAs is non-diagnostic. For mural changes (strictures, masses), **colonoscopy** is the method of choice as it allows assessment of the extent of disease, less invasive biopsy, and potentially treatment (dilation of stricture, removal or debulking of masses) simultaneously. Full-thickness colonic biopsies are not often performed due to the risk of postoperative complications, but to the author's knowledge, there are no studies investigating complication rates of large intestinal full-thickness biopsies only. Often, pathology in the intestinal muscle layers (e.g., due to feline idiopathic megacolon; FIM) or the enteric nervous system (e.g., hypo- or aganglionic megacolon, colic ganglioneuritis) is only diagnosed after colectomy or on postmortem examination.

Feline idiopathic megacolon (FIM) (▶ Chap. 8.3.6) is a diagnosis of exclusion. Radiographic abnormalities may begin in the descending colon and progress over time to involve also the ascending and transverse parts. The radiographic ratio of maximal diameter of the colon to L5 length can help in differentiating constipation from true megacolon: A ratio <1.28 is consistent with a normal colon, while a value of >1.48 is a good indicator of megacolon (sensitivity 77%, specificity 85%).[4]

2.5.4 Treatment and management

Treatment of constipation is guided by the underlying cause. Alleviating constipation or treating obstipation is warranted in addition in most cases. Specific therapeutic plans depend on the severity of constipation. Long-term management of idiopathic constipation or FIM with a combination of dietary

4.2.2 Eosinophilic granuloma complex

Lara Boland

Eosinophilic granuloma complex (EGC) describes a cutaneous, mucocutaneous junction, or mucosal reaction pattern commonly observed in cats and rarely in dogs.[1,2] Hence, this chapter focuses on EGC in cats.

EGC is divided into three distinct clinical subtypes with similar histopathological lesions: indolent (eosinophilic) ulcers, eosinophilic granulomas, and eosinophilic plaques.[3] Histopathological lesions may occur alone or in combination.

4.2.2.1 Etiopathogenesis

Most cases of EGC are believed to be due to hypersensitivity reactions, including fleas, food, or feline atopic skin syndrome (FASS).[3–7] Primary bacterial or immune-mediated causes of EGC are unlikely.[8] In a retrospective study of feline dermatologic diseases, only 2.9% of cats were diagnosed with idiopathic EGC.[9] The same study identified approximately one-third of cats with skin disease to have hypersensitivities, including flea, food, mosquito, and FASS. In two other retrospective studies, EGC accounted for 10–11% of feline oral cavity lesions.[10,11]

4.2.2.2 Physical examination

Physical examination of cats with indolent ulcers typically reveals unilateral or bilateral upper lip lesions involving the mucocutaneous junction at the level of the upper canines and philtrum. Lesions are well demarcated and vary from small and superficial to extensive deep ulcerations. Lesion margins may be raised, and the ulcerated surface can be erythematous or purulent. Lesions are usually not painful or pruritic.

Eosinophilic granulomas may occur in the oral cavity or elsewhere (chin, caudal thigh). Oral locations include the hard palate, tongue, and lower lip. Lesions are usually well-demarcated and vary from small to extensive, raised to nodular and/or ulcerated erythematous lesions, which may be exudative (▶Fig. 4-2). Hard palate ulcerations can cause acute or chronic hemorrhage requiring blood transfusion and surgical intervention.[12]

Eosinophilic plaques typically involve the ventral abdominal skin and appear as well-demarcated, raised erythematous lesions that may be ulcerated and pruritic.

Fig. 4-2 Eosinophilic granuloma of the tongue and palate of a cat
Note the extensive raised, erythematous and exudative lesions. (Courtesy of Dr. Nicolle Kirkwood, University of Sydney, Australia.)

4.2.2.3 Differential diagnoses

Depending on the appearance, location, and geographic region, differential diagnoses include neoplasia (particularly squamous cell carcinoma), feline herpesviral 1 (FHV-1) dermatitis, cowpox, dermatophytosis, mycobacterial infections, and trauma.[3,11,13,14]

4.2.2.4 Diagnosis

Cytology of oral cavity lesions usually identifies eosinophilic inflammation, but mixed inflammation and/or secondary bacterial infection may be observed. Histopathology is required to diagnose EGC. However, identification of the underlying cause requires the collection of a detailed dermatological history. Flea control and dietary elimination trials support the diagnosis. Histopathology may not be warranted in cats with classic upper lip indolent ulcers. However, other oral cavity presentations cannot be differentiated from neoplasia grossly. EGC lesions are rarely reported in dogs, and biopsy is required for diagnosis.

Differential diagnoses can be excluded (dependent on the clinical subtype observed) by the following:

- Malassezia dermatitis: sticky tape preparations
- Mite infestations: skin scrapes
- Dermatophytosis: Wood's lamp examination, trichography, fungal culture
- FHV-1 infection: histopathology, immunohistopathology, PCR
- Neoplasia: cytology, histopathology

Intradermal allergen testing and allergen-specific IgE serology may support a diagnosis of flea allergy or atopy when combined with other findings and treatment responses.[4,15]

4.2.2.5 Treatment

Treatment focuses on identifying and managing the underlying cause, when possible, for example, through strict parasite control and elimination diet trials. The use of H_1-receptor antihistamines and/or allergen-specific immunotherapy for FASS has moderate to good efficacy based on limited evidence.[6,15] Cats that do not respond may require immunomodulatory or immunosuppressant drugs.

EGC lesions usually respond rapidly to systemic glucocorticosteroids (1–2 mg/kg PO q24h).[6] Titration to the lowest effective dose for chronic use should be aimed to reduce glucocorticosteroid side effects. Alternative treatments should be considered if this is not possible. Synthetic progestins and depot corticosteroid preparations, although effective, should be avoided if possible due to their diabetogenic and other glucocorticosteroid side effects, which can be prolonged.

EGC lesions usually respond well to treatment with cyclosporine (7 mg/kg PO q24h until clinical signs resolve, then dose q48h to twice a week).[6,16,17] Side effects of cyclosporine include gastrointestinal signs, secondary infections, malignant neoplasia, and gingival hyperplasia.[18–20]

Off-label use of oclacitinib, a Janus-kinase inhibitor, is described in cats with FASS to have good efficacy, although not superior to glucocorticosteroids.[21–23] However, there is limited evidence about the response to treatment, medication side effects, and recommended dose.[6]

Eosinophilic plaques can reduce in size or resolve with amoxicillin-clavulanate, where indolent ulcers show less of a response, likely by treating secondary bacterial infection.[24] The author does not recommend antimicrobials as first-line or sole treatment for EGC, given the suspected allergic etiology and concerns of antimicrobial resistance development.

4.2.2.6 Prognosis

EGC lesions can spontaneously resolve or be recurrent or persistent without appropriate treatment.[25] Treatment can be curative and may be required intermittently or persistently.

KEY FACTS

- Eosinophilic granuloma complex describes a cutaneous, mucocutaneous junction, or mucosal reaction pattern, mostly due to a hypersensitivity reaction.
- Treatment focuses on identifying and managing the underlying cause, and immunomodulatory or immunosuppressant drugs may be required.

References

The complete list of references for this chapter can be downloaded from vetline.de:
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4.2.3 Salivary disorders

Ana C. Castejon-Gonzalez,
Alexander M. Reiter

4.2.3.1 Introduction

Salivary gland disease is rare in small animals (0.3%), but it should be included in the differential diagnosis list for patients with pain and swelling of the head and neck.

Salivary gland neoplasia is more frequent in cats than in dogs.

Sialocele, sialadenitis, sialolithiasis, sialadenosis, necrotizing sialometaplasia, and neoplasia usually involve the major salivary glands (mandibular, sublingual, zygomatic, and parotid glands). However, the minor salivary glands (distributed within the oral mucosa) can sometimes be affected.[1]

4.2.3.2 Salivary gland diseases

Sialocele is an accumulation of saliva in the subcutaneous or submucosal tissue caused by leakage from a salivary gland or corresponding ducts. The leakage results in an inflammatory

reaction in the surrounding tissues. Trauma, foreign bodies, and sialoliths have been identified as causes, although the cause remains unknown in most instances. A possible immune-mediated etiology has also been reported. Young dogs and poodles are predisposed,[2,3] and the monostomatic portion of the sublingual salivary gland is most often affected. Sialoceles can be classified as cervical, pharyngeal, sublingual, or retrobulbar, depending on the location of the swelling. The soft, fluid-filled swelling is often not painful (particularly in chronic stages). However, because sialoceles can be present with sialadenitis, sialadenosis, necrotizing sialometaplasia, and tumors, pain can be present in some cases. Patients with pharyngeal sialocele may show signs of upper airway obstruction, and those with zygomatic sialocele show retrobulbar swelling extending periorbitally.[4,5]

Sialadenitis is inflammation of a salivary gland secondary to infection, trauma, or foreign bodies. Pain may be elicited on palpation of the affected region, opening the mouth, or during deglutition. Thick and purulent discharge from the corresponding ducts, sialoliths, and sialoceles might be seen or be developing.[1,2]

Sialoliths (composed of calcium carbonate, calcium phosphate, and/or calcium oxalate) and **foreign bodies** can obstruct the salivary duct causing dilation and/or rupture of the duct and sialadenitis.[1,6]

Sialadenosis is an idiopathic, painless, non-inflammatory, non-neoplastic enlargement of the salivary glands.[1,7] It tends to be bilateral. The mandibular glands are most frequently affected, but it is not unusual to see an enlargement of multiple glands. The cause is unknown, although autonomous nervous system dysfunction and an unusual form of epilepsy are suggested as possible etiologies.[8,9] Dogs may present with clinical signs of lethargy, anorexia or hyporexia, dysphagia, and weight loss. Retching, gulping, snorting, lip-smacking, hypersalivation, and regurgitation are also reported. A firm cervical swelling (affected mandibular and/or parotid glands) can be palpated on physical examination. When the zygomatic salivary gland is involved, exophthalmos, decreased retropulsion of the eye, and a palpable swelling within the soft palate can be identified.[1]

Necrotizing sialometaplasia, also known as salivary gland infarction or necrosis, is characterized by squamous metaplasia of the mandibular gland parenchyma and ducts. Unlike sialadenosis, patients with necrotizing sialometaplasia tend to show pain (on palpation and when opening the mouth) and more severe clinical signs of systemic disease (weight loss, lethargy, and anorexia), respiratory and gastrointestinal disease (retching, gulping, gagging, regurgitation, vomiting, coughing, tachypnea, and increased respiratory effort).[1,2,10] The cause is unknown, but parasympathetic stimulation is believed to contribute to the pathogenesis.

Salivary gland **neoplasia** is uncommon. The disease primarily affects older dogs and cats. The mandibular and parotid glands are most frequently affected, but other glands (including the minor salivary glands of the oral mucosa) can also be affected. Malignant tumors such as simple or mixed adenocarcinomas are most common. Clinical signs and physical examination findings include dysphagia, halitosis, and the presence of a firm mass in the affected region. Adenocarcinoma of salivary glands in the oral mucosa is frequently ulcerative.[11,12]

Young adult dogs are predisposed to sialocele, middle-age dogs to sialadenosis and necrotizing sialometaplasia, and older dogs to sialadenitis and neoplasia.

4.2.3.2.1 Diagnosis

Fine-needle aspiration of sialocele fluid typically reveals a low-cellularity, stringy, clear to blood-tinged to brownish fluid. Because sialoceles can be present with other diseases of the salivary glands, a diagnosis of sialocele does not exclude any of these, and a final diagnosis should be based on the combination of clinical presentation, diagnostic imaging, and other diagnostic test results. Fine-needle aspiration and incisional biopsy of salivary gland tissue can support a presumptive diagnosis. Bacterial culture and sensitivity testing aim to target the antibiotic treatment in cases of infectious sialadenitis.[1,13,14]

Radiography and ultrasonography can identify sialoliths. Sialography can localize the gland and/or duct of origin of a sialocele. Sialoceles are easily diagnosed by ultrasonography. However, CT and MRI can provide more detailed information and show the full extent of salivary disorders. CT and MRI can identify tumors, sialoceles, lesions consistent with sialadenitis (heterogeneous contrast enhancement), and sialadenosis (enlargement with no other abnormality). Differentiating between inflammation and neoplasia is not always possible, and the definitive diagnosis requires a biopsy in these cases.[14,15] Tumor staging (size, lymph node involvement, and distant metastasis) is a prognostic factor in dogs with salivary gland neoplasia.

Determining the salivary gland involved in cervical sialoceles (parotid vs. mandibular or sublingual gland) can be

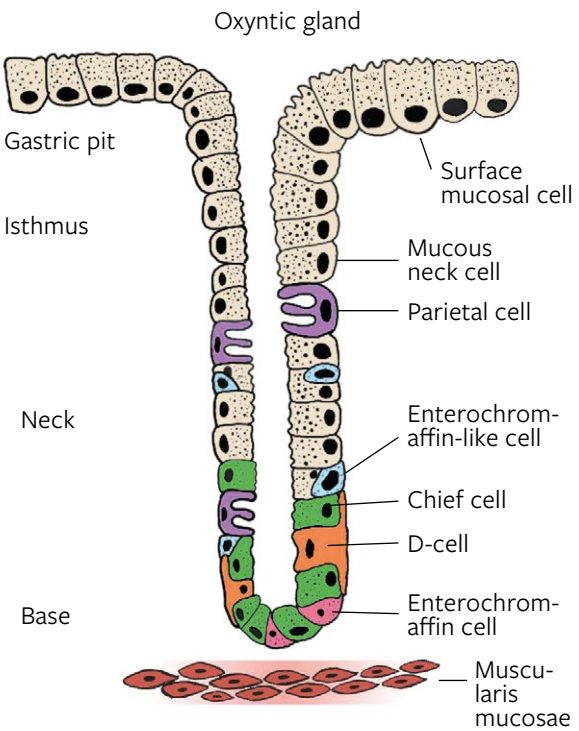

Fig. 6-2 Structure of a gastric fundic gland

Fundic glands are found in the corpus and fundus of the stomach and consist of various cells responsible for regulating acid and mucus secretion.

Pyloric glands contain mucus-secreting cells and gastrin-producing endocrine cells (G-cells). Gastrin stimulates parietal cells to produce gastric acid and is a trophic factor for the gastric mucosa.

KEY FACTS

- There are five anatomical regions of stomach that play varying roles in enzymatic and mechanical digestion.
- Gastric glands produce important chemical agents, including gastric lipase, gastrin, ghrelin, hydrochloric acid, pepsinogen, R-protein, and trefoil factors.

References

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6.2 Physiology

Panpicha Sattasathuchana

6.2.1 Gastric emptying and motility

Ingesta pass to the stomach from the esophagus through the lower esophageal sphincter. The first part of the stomach that contacts the ingesta is the cardia. The fundus and the corpus can actively expand to accommodate the ingesta. In the postprandial phase of gastric motility, the antrum functions to mechanically crush and reduce the size of the ingesta. The pylorus acts as the "gatekeeper," preventing any particles >2 mm from entering the duodenum.[1] Ingesta-particles >2 mm are propelled back into the antrum. This repeating a to-and-fro movement of ingesta in the antrum leads to the mechanical breakdown and stirring of food known as trituration, resulting in the passage of digestible solids into the duodenum. The rate of gastric emptying of solid food depends on the size of the particles and caloric density. Liquids empty from the stomach more rapidly than digestible solids.[1] Non-digestible solids are emptied later in the interdigestive phase of gastric motility.

6.2.2 Gastric acid secretion

Acidic gastric juice with a pH of 1–2 is required for digestion in the stomach. The acidic conditions in the stomach result from hydrochloric acid secreted by parietal cells in the fundus and corpus (▶Fig. 6-3, ▶Fig. 6-4). This low gastric luminal pH also helps control the microbiota of the stomach, activates pepsinogen, and helps digest protein. Secretion of hydrochloric acid is stimulated by acetylcholine, gastrin, and histamine.[2] Activation of acetylcholine (m_3), gastrin (G), and histamine (H_2) receptors increase the accumulation of cyclic adenosine monophosphate (cAMP) and intracellular Ca^{2+} in parietal cells. The accumulation of cAMP activates protein kinase A, which increases protein phosphorylation. Protein phosphorylation activates H^+, K^+, and ATPases (proton pumps), which hydrolyze ATP leading to the active transport of H^+ into the gastric lumen.

There are three phases of gastric acid secretion: cephalic, gastric, and intestinal. The **cephalic phase**, which accounts for 15–30% of total gastric secretion, is stimulated by vagal neurons and neuropeptides in response to taste, smell, or the sight of food. Acetylcholine released from cholinergic neurons of the vagal nerve stimulate muscarinic receptors (m_3) on the apical surface of parietal cells, activating their proton pumps and acid secretion. Acetylcholine also activates histamine secretion from enterochromaffin-like cells

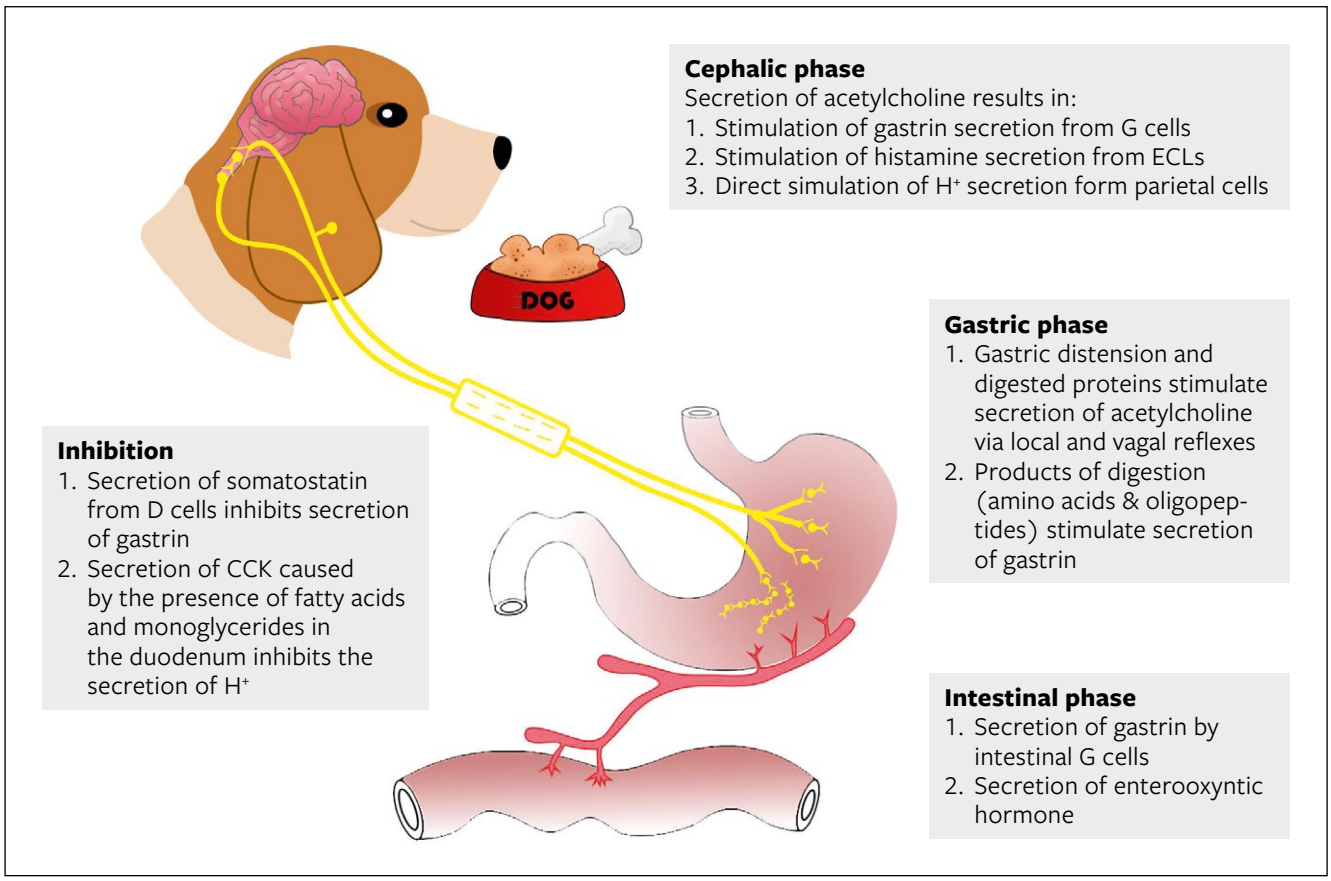

Fig. 6-3 Regulation of gastric acid secretion

Gastric acid secretion consists of three digestive phases (cephalic, gastric, and intestinal phase).

ECLs = enterochromaffin-like cells, **CCK** = cholecystokinin

in the gastric fundus. Histamine enhances the acid secretory response of parietal cells to gastrin and acetylcholine. Acetylcholine also stimulates gastrin secetion via bombesin and inhibits somatostatin secretion. The **gastric phase** accounts for 50–60% of total gastric secretion. This phase is initiated by gastric distention and gastrin-producing endocrine cell (G cell) stimulation by amino acids and peptides in the gastric lumen. Stretching of the stomach wall activates local reflexes (mediated by acetylcholine) and vasovagal reflexes (mediated by acetylcholine and bombesin). Both acetylcholine and bombesin activate G cells to secrete gastrin. Both acetylcholine and gastrin activate H^+ secretion from parietal cells. The **intestinal phase** accounts for 5% of total gastric secretion. Stimulation of gastric secretion occurs when the duodenal pH is >3. Gastrin from intestinal G cells and enterooxyntic hormone from the duodenum are released and stimulate H^+ secretion from parietal cells.

When the gastric pH is <3, somatostatin is released by D cells, initiating a **negative feedback** mechanism on G cells that inhibits gastrin secretion. At a gastric pH <2, the secretion of gastrin is completely inhibited. Cholecystokinin (CCK) is released by enteroendocrine cells (I cells) when amino acids, fatty acids, and peptides are present in the duodenum. CCK inhibits parietal cell secretion and activates exocrine pancreatic secretion and gall bladder contraction.

Gastric acid secretion also occurs during the interdigestive phase, which is a phase between meals. Gastric acid secretion in the **interdigestive phase**, which is important for cleaning the empty stomach in preparation for the next meal, is regulated by acetylcholine and histamine. This phase accounts for 15% of total gastric acid secretion.

Canine coronavirus has been reported as a cause of diarrhea in dogs, but its clinical importance is unknown. Co-infections with distemper and parvovirus and coronavirus with parvovirus have been reported to result in more severe clinical signs. There are occasional reports of other diarrhea-causing viral co-infections in dogs, such as rotavirus,[22] kobuvirus,[23] and bufavirus.[24] These are very rare and of minimal clinical significance.

Feline leukemia virus (FeLV) and **feline immunodeficiency virus** (FIV) can be associated with gastrointestinal signs, particularly diarrhea, due to secondary bacterial, protozoal, viral, or fungal infections resulting from the immunosuppressive state. Mild, transient, and self-limiting diarrhea can also be caused by an infection with **feline coronavirus** (FeCoV).

This chapter will focus on parvovirus infection because of its importance as a cause of diarrhea in dogs.

7.3.1.1.2 Clinical features

Clinical presentation and disease pathogenesis

Parvovirus is unable to induce mitosis in the cells it infects, and thus it relies on the rapidly multiplying cells of the body for its replication. Thus, the virus shows a **tropism** for the **thymus, bone marrow, spleen**, and **crypt cells of the gut epithelium**. Stress factors, in particular parasitic and other factors such as weaning, may predispose dogs to infection by increasing mucosal cell activity.[25] A robust antibody response is protective against parvovirus infection. Consequently, it is almost exclusively a disease of unvaccinated or inadequately vaccinated animals less than a year old that have passed the age of maternally-derived antibody protection. Therefore, vaccination status plays an important role in disease susceptibility.

Following oral infection, the virus disseminates to the regional lymph nodes of the pharynx and the tonsils. In an experimental study in dogs, fecal shedding was present from day 3 after oral infection, peaked on days 3 and 4, and was greatly reduced by day 7.[26] Shedding may occur a day or two before clinical signs are apparent. The incubation period of the disease varies from 4 days (under experimental conditions) to the more typical 1–2 weeks (under natural conditions). Following infection of lymphoid tissues of the upper GI tract (including the tongue), a cell-free viremia ensues. Under experimental conditions, this lasts from day 1–5 post-infection, with serum antibodies developing on day 5 and peaking from day 7 onwards.[26] Thymic and lymphoid tissue infection leads to thymic atrophy, lymphoid depletion, lymphopenia, and immunosuppression. The parvoviral anti-

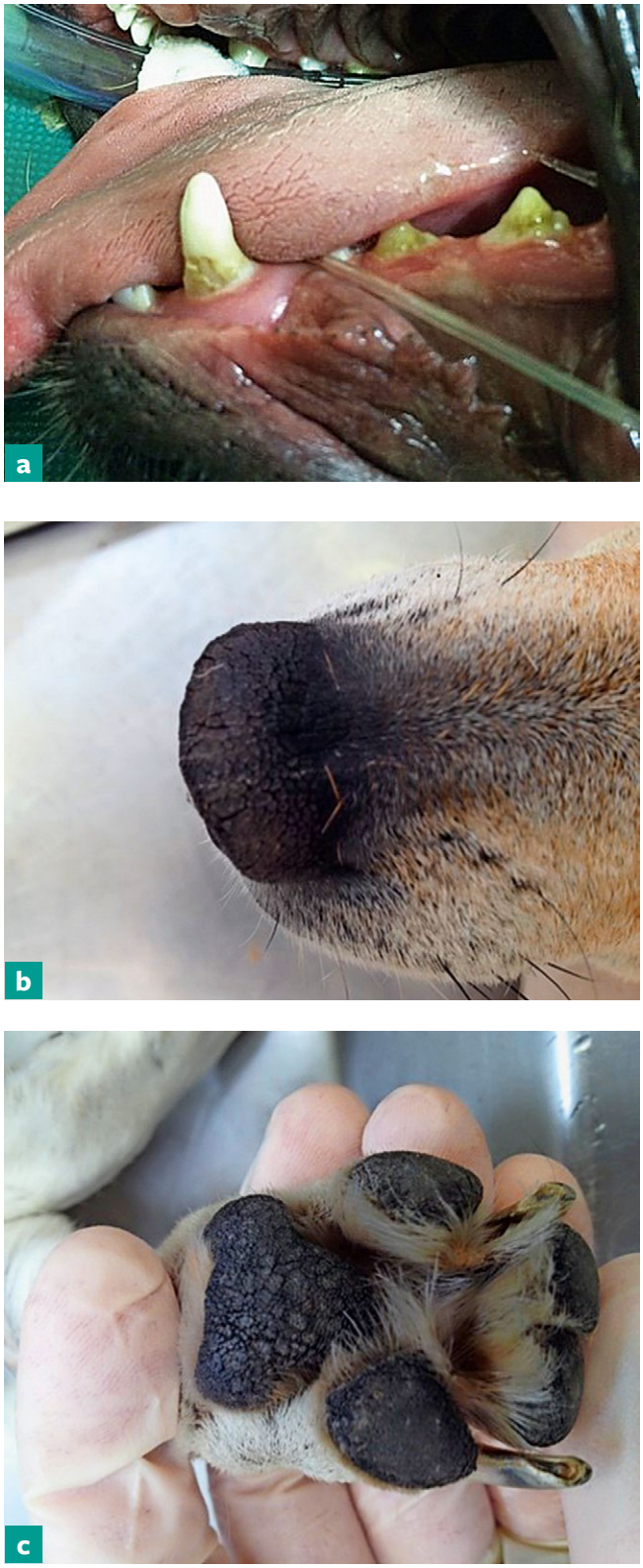

Fig. 7-5 Dog after survival of acute distemper virus

Dogs with distemper that survive the acute disease may go on to demonstrate (**a**) dental hypoplasia or (**b** and **c**) naso-digital hyperkeratosis.

7.3.1.3.5 Treatment

T. canis presents a zoonotic risk, and it is considered that almost 100% of dogs have been in contact with this parasite. Thus, a systematic deworming program must be implemented against this parasite.

To prevent transmission of *Toxocara canis* to fetuses during gestation, the European Specialist Counsel Companion Animal Parasites (ESCCAP) recommends treating pregnant female dogs with macrocyclic lactones on the 40th and 55th day of pregnancy or fenbendazole daily (q24h) from the 40th day of pregnancy until the 14th day postpartum.

Puppies need to be treated starting at 2 weeks of age, then every 2 weeks until 2 months, with fenbendazole/febantel, pyrantel, flubendazole, or nitroscanate.

Transplacental transmission does not occur in cats during gestation. Kittens need to be treated starting at 3 weeks of age, then every 2 weeks until 2 months, with fenbendazole/febantel, pyrantel, flubendazole, or nitroscanate.

Lactating bitches and queens must be treated at the same time as their puppies/kittens to prevent transmission by milk or feces (▶Tab. 7-3).

Although *Cystoisospora* coccidiosis may be self-limiting, anticoccidial therapy can speed up the resolution of clinical signs and may lessen environmental contamination. Several anticoccidial drugs (furazolidone, amprolium, diclazuril, ponazuril, toltrazuril, sulfadimethoxine, and trimethoprim-sulfonamide) can be used to treat *Cystoisospora* infections in dogs and cats. In clinical coccidiosis cases due to *C. canis* or *C. ohioensis*-complex, emodepside plus toltrazuril suspension is effective (reducing oocyst excretion and the number of days with diarrhea).[6] The number of oocysts excreted is also reduced markedly by administering emodepside plus toltrazuril suspension to cats during the prepatent period.

In catteries, breeding kennels, and shelters, these treatments need to be aligned with global hygiene management (▶Clinical pearls).

Tab. 7-3 Treatment options for main endoparasite infections
This table shows commonly used antiparasitic agents to treat helminth, Giardia, and coccidian infections in dogs and cats.

Agent	Commonly used dosage
Ascarids and hookworms	
Fenbendazole	50 mg/kg PO q24h for 3 consecutive days
Pyrantel pamoate	5–10 mg/kg PO once; consider repeating after 2–4 weeks
Febantel	10–20 mg/kg PO q24h for 3 days
Moxidectin (to prevent reactivation of dormant *Toxocara canis* larvae and their transmission to puppies)	1 mg/kg PO administered on days 40 and 55 of pregnancy
Milbemycin oxime	0.5–1 mg/kg PO once
Taenia, echinococcus, and dipylidium	
Praziquantel	5–10 mg/kg PO or SC; repeat 3 weeks later
***Giardia* spp.**	
Metronidazole	25–30 mg/kg PO q12h for 5–8 days (D)
Metronidazole benzoate	10–15 mg/kg PO q12h for 7 days (C)
Fenbendazole	50 mg/kg PO q24h for 3 days (D)
Coccidia	
Toltrazuril plus emodepside	9 mg/0.45 mg/kg once

PO = per os; **SC** = subcutaneous; **D** = applies for dogs; **C** = applies for cats

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Management of parasite contamination in kennels and catteries

Breeding kennels and catteries are specific environments with a high density of animals, particularly animals of young age. In these environments, parasite prevention and control measures are very important. These measures are divided into two main categories:

1. Preventing the introduction of parasites
2. Limiting parasite transmission.

Quarantine plays an important role in preventing the introduction of some endoparasites. All new dogs need to be housed in a specific quarantine ward, separated from other dogs, prior to being introduced in the kennel. During this period, dogs should be dewormed and groomed (9% of owned dogs carry eggs of *Toxocara canis* on their fur or hair).[5]

Decreasing the risk of parasitic transmissions requires the excretion, persistence in the environment, and exposition to parasites to be limited. Parasite excretion can be reduced by **anthelminthic treatment** adapted to the parasites observed in the kennel. Environmental conditions also affect the survival of parasites. Surface porosity, environmental temperature, and the presence of organic matter are important determinants of parasite survivability. This emphasizes the importance of using adequate surfaces in kennels, which should be highly resistant and of low porosity. To limit the persistence of parasites in kennels, good cleaning and disinfection procedures are mandatory. **Cleaning and disinfection** are to be distinguished; cleaning involves removing visible organic matter with soap or detergent, whereas disinfection is the application of chemicals or other procedures to kill the remaining microbes. The disinfectant should be selected based on the detected parasite. For example, sodium hypochlorite is highly effective against infective *T. canis* eggs, but it enhances the excystation of *Cystoisospora* spp. oocysts. Limiting the exposition to parasites, kennels are required to separate wards or sectors based on the vulnerability of the animals as determined by their physiological status.

An optimum solution is to separate the following areas within the kennel: maternity ward, adult dogs, quarantine ward, and medical ward. Specific cleaning equipment should be available in each sector.

The design should allow movement through the facility to proceed from areas housing animals most susceptible to disease and/or healthiest animals to those likely to be a source of infectious disease (puppies, adult dogs, dogs in quarantine, medical ward).

References

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7.3.1.4 Canine schistosomiasis

Johanna Heseltine

7.3.1.4.1 Etiology and clinical presentation

Canine schistosomiasis is caused by infection with the trematode *Heterobilharzia americana*. *H americana* is endemic to the Gulf coast and south Atlantic region of the United States, but canine infections have been reported more widely (Texas, Louisiana, Florida, North Carolina, South Carolina, Georgia, Oklahoma, Alabama, Virginia, Mississippi, Kansas, and Indiana).[1–4] Raccoons and nutria are definitive hosts, but infections also occur in other mammals. Eggs are shed in host feces. When exposed to freshwater, a ciliated miracidium hatches and infects the intermediate host, a lymnaeid snail. Cercariae form within sporocysts in the snail and then emerge in freshwater, where they can penetrate the skin of a dog in the water. Cercariae then migrate through the dog's lungs to the liver, where they mature into adults. Adults travel via portal veins to the mesenteric veins, where they reproduce and lay eggs. Proteolytic enzymes produced by the eggs allow penetration of the mesenteric vein and intestinal mucosa, with entry into the intestinal lumen.[2] Patent infection occurs approximately 68 days post-infection.[2] Young, large breed dogs with outdoor access have an increased incidence of infection.[5] Eggs are deposited in the small and large intestines and can migrate to other organs, including the liver, pancreas, lymph nodes, spleen, and kidneys, where they elicit granulomatous inflammation. The broad tissue distribution and variable degrees of inflammation and fibrosis result in variable clinical presentations. Clinical signs can be acute or chronic. Large intestinal diarrhea, small intestinal diarrhea, or a protein-losing enteropathy (▶Chap. 7.3.8.2) may develop. Weight loss, lethargy, vomiting, hyporexia, abdominal discomfort, and fever may occur. Hypercalcemia is seen in up to 50% of dogs, which may result in polyuria and polydipsia

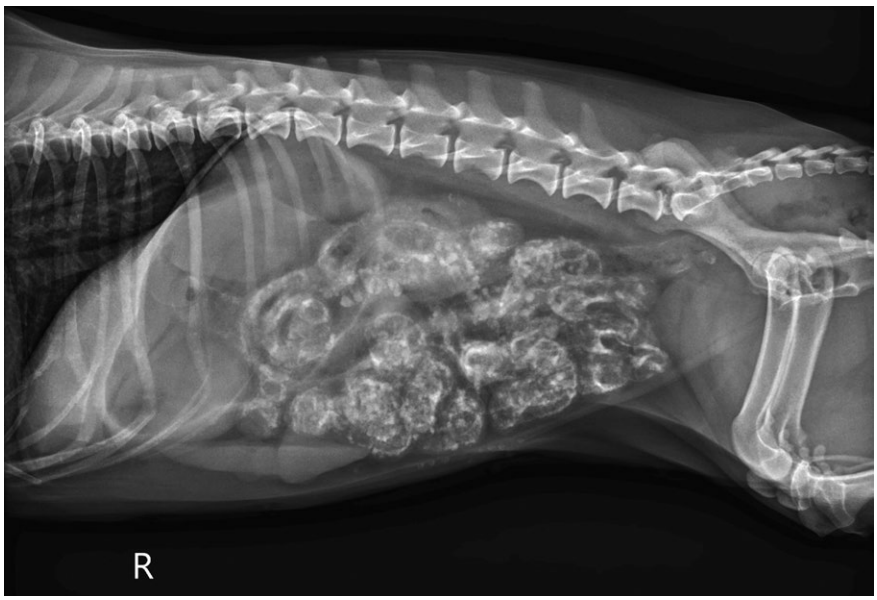

Fig. 7-13 Radiographic image showing mineralization of the intestinal wall from a dog infected with *H. americana*

(Courtesy of Texas A&M Radiology Department, College Station, TX.)

(PU/PD).[3] Some infected dogs are asymptomatic.[2,4,5] *H. americana* infection in humans results in cercarial dermatitis ("swimmer's itch").[5]

7.3.1.4.2 Diagnosis

Diagnosis of schistosomiasis requires fecal PCR testing, fecal saline sedimentation, or histopathology of affected tissues. No studies have compared the performance of these tests. Fecal PCR is commercially available (Gastrointestinal Laboratory, Texas A&M University, College Station, TX) and can detect >1–2 eggs/gram of feces.[6] Eggs may be found on fecal saline sedimentation or a direct fecal smear, whereas routine fecal flotation is usually negative. Shedding eggs into feces is intermittent, and testing several samples obtained on consecutive days or using a combination of tests may improve the diagnostic yield.[8] Granulomatous inflammation and fibrosis, sometimes containing eggs, can be found on biopsies of the liver, intestines, or other organs.[4] Less commonly, adult parasites are found in tissues.[8] Confirmation by PCR detection of parasite DNA in formalin-fixed, paraffin-embedded tissues has been described.[7] There is also a case report of eggs identified on cytology obtained via a hepatic fine needle aspirate.[8]

Abnormalities on other diagnostic tests may prompt testing for *H. americana*. Laboratory findings include anemia, eosinophilia, hypoalbuminemia, hyperglobulinemia, increased liver enzymes, hypercalcemia, and secondary renal azotemia. Glomerulonephritis has been reported secondary to *H. americana* infection.[4,9] Abdominal radiographs may be normal, but occasionally show linear mineralizations of the gastric or intestinal wall (▶Fig. 7-13), splenomegaly, or lack of serosal detail. Abdominal ultrasound may show thickening of the intestinal wall, sometimes with linear areas of mineralization in the submucosa or muscularis layers (▶Fig. 7-14).[1] The liver may appear coarse, heterogeneous, mottled, hyperechoic, or hypoechoic.[1,7,8] Granulomas and mineralized eggs may appear hyperechoic pinpoint foci in the liver, pancreas, and intestines.[7,8] Ascites secondary to severe liver disease and portal hypertension has been reported.[1,5] Abdominal lymphadenopathy, splenomegaly, and hyperechoic kidneys may also be seen. During endoscopy (▶Chap. 1.5), the intestinal mucosa may appear thickened and irregular.[1,5] The most common intraoperative or necropsy-derived gross findings are granulomas on the serosa of the intestines and liver.[4]

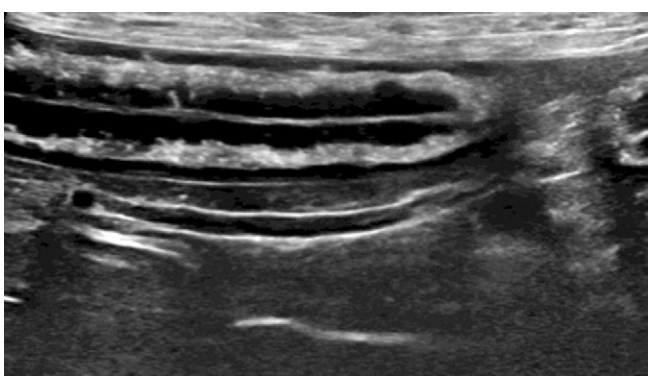

Fig. 7-14 Ultrasonographic image showing thickening of the submucosal layer of the small intestinal wall with pinpoint hyperechogenicities from a dog with *H. americana* infection

(Courtesy of Texas A&M Radiology Department, College Station, TX.)

7.3.1.4.3 Treatment and prognosis

Successful off-label treatment is reported with praziquantel 25 mg/kg PO q8h for 2–3 days, fenbendazole 40 mg/kg PO q24h for 10 days, or a combination of both drugs.[5] No clinical trials have compared the efficacy of these treatments. Comorbidities such as acute kidney injury should also be managed, although calcium may not normalize until an anthelminthic drug is given.[5] A protocol combining low-dose praziquantel (5 mg/kg PO q8h for 2 days) and fenbendazole (24 mg/kg PO q24h for 7 days) can effectively treat asymptomatic schistosomiasis in dogs.[10]

With treatment, the prognosis is good for acute cases but guarded for chronic cases and depends on the severity of the resulting granulomatous reaction and fibrosis. Treatment is not always successful and may need to be repeated in some cases.[1] Re-testing 2 weeks post-treatment is recommended. However, this may be too early in some cases, as residual eggs can still be shed.[4] Some dogs have had positive tests years apart, and it is unclear if this represents treatment failure or reinfection. Because dogs can be re-infected, the environment should be changed, if possible. Testing of other dogs sharing the same environment is recommended.[1,4] Testing may also be warranted during annual examinations for dogs from endemic regions, especially those exposed to freshwater sources that could be contaminated with *H. americana*.[4]

References

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7.3.1.5 Fungal and algae infections

Margaux Marclay

7.3.1.5.1 Pythiosis

Pythium insidiosum is an aquatic oomycete that causes severe gastrointestinal (GI) or cutaneous lesions in dogs and less commonly in cats. Large breed young to middle-aged outdoor male dogs are predisposed.[1–5] Repeated exposure to freshwater lakes, swamps, and ponds that may contain *P. insidiosum* zoospores increase the likelihood of contracting the disease, but even animals without this history may become infected.[6] Pythiosis is most common in tropical and subtropical regions such as the southeastern United States, Southeast Asia, Central and South America, and eastern Australia.

Clinical presentation

Gastrointestinal pythiosis typically causes chronic weight loss associated with clinical signs of GI disease corresponding to the affected sites. Signs of systemic illness, such as lethargy are typically absent, and physical examination often reveals poor body condition in an alert patient. A palpable abdominal mass or thickened colonic mucosa are common findings. Pythiosis causes marked eosinophilic pyogranulomatous inflammation leading to severe segmental, transmural thickening of the GI wall. Mesenteric lymph nodes are often enlarged and reactive but are usually not infected. Acute hemoabdomen and sudden death is an uncommon complication that may result from an invasion of the mesenteric vasculature. Diagnosis of GI pythiosis is suspected based on patient history, clinical signs, and the presence of an abdominal mass. Eosinophilia is often detected.[4,6]

Diagnosis

Abdominal ultrasound can confirm the presence of typical lesions and assess their extent and location to determine whether surgical resection is possible. Segmental loss of the normal GI wall layering associated with moderate to severe wall thickening is typically seen.[7] Cytological examination (▶ Chap. 1.7) of ultrasound-guided fine-needle aspirates of GI lesions or rectal scrapings may show eosinophilic pyogranulomatous inflammation and occasionally *P. insidiosum* hyphae. Histopathology is better assessed on surgical than endoscopic biopsies, as the hyphae are usually found in the deep layers of the intestinal wall. Hyphae are broad (2–9 µm in diameter with a mean diameter/width of 5 µm), rarely septate, have non-parallel walls, and stain well with Gomori's methenamine silver. Culture can be successful if appropriate tissue samples are obtained at the time of biopsy. A technique for the extraction, amplification, and sequencing, of fungal and oomycete DNA from formalin-fixed paraffin-embedded tissues is also routinely available.[8] Assessment of anti-*P. insidiosum* antibodies has often been used to support the diagnosis of pythiosis, but false-positive results may occur.[9]

Treatment

Complete surgical resection is the treatment of choice, if possible. Otherwise, medical therapy with a combination of itraconazole, terbinafine, and an anti-inflammatory dose of prednisone is recommended.[10]

9.2 Physiology

Randi Gold

9.2.1 Metabolism

The liver plays a central role in the body's metabolism. Most of the metabolic and detoxifying functions of the liver occur during the passage of blood along the sinusoids.[1]

9.2.1.1 Carbohydrate metabolism

Glucose is the primary energy source for most mammals. Its metabolism is tightly regulated, and availability is dependent upon two sources: dietary absorption from the intestinal tract and release of glucose from the liver. The final products of carbohydrate digestion in the intestinal tract are almost entirely glucose, fructose, and galactose. After absorption from the intestinal lumen, much of the fructose and almost all of the galactose are rapidly converted to glucose in the liver by **glucose phosphatase**.[2] Once absorbed into a cell, glucose can either be used immediately or stored in the form of glycogen. Liver cells can store 5–8% of their weight as glycogen.[2] When the body's stores of carbohydrates fall below normal levels, glucose is generated from amino acids and the glycerol portion of triglycerides in a process called gluconeogenesis.[2]

Cats are obligate carnivores and show several differences in carbohydrate metabolism compared with dogs. For example, they have minimal to absent hepatic glucokinase, an enzyme that catalyzes the first step of glycolysis.[3]

9.2.1.2 Lipid metabolism

Lipids are water-insoluble organic compounds. Of the many groups of lipids, the most important from a clinical standpoint are fatty acids, sterols, and acylglycerols (mainly triglycerides).[4] **Cholesterol** is the main sterol in animals, with dietary intake being the primary source (exogenous transport). However, the liver and other tissues can also synthesize it endogenously (▶Fig. 9-5).

9.2.1.2.1 Exogenous transport

Dietary lipids that reach the duodenum undergo emulsification by bile salts followed by hydrolysis by pancreatic lipase supported by the actions of colipase.[5] Hydrolysis products are then transferred to intestinal microvilli at the epithelial brush border, where they undergo solubilization of fatty acids and monoglycerides into mixed micelles.[6] The mixed micelles then diffuse through the epithelial cell membranes into the intestinal mucosal cells. Within the epithelial cells, free fatty acids and monoglycerides reassemble to form triglycerides, which combine with phospholipids, cholesterol, and apolipoprotein (apo) B48, forming chylomicrons.[5,17] Chylomicrons are then released from enterocytes by way of exocytosis, traverse the interstitial space, and enter lacteals through gaps between endothelial cells. From there, they are transported into intestinal lymphatics and later the systemic circulation, where they acquire apo C and E from circulating high-density lipoproteins (HDLs).[4] Apolipoprotein C–II on the chylomicron surface then activates lipoprotein lipase on the capillary surface, allowing hydrolysis of free fatty acids, which in turn can enter muscle and fat cells to be utilized for energy generation.[4] The chylomicron remnants with attached cholesterol can then be taken up by the liver to be stored or used to create very low-density lipoproteins (VLDLs).

9.2.1.2.2 Endogenous transport

VLDLs, intermediate-density lipoproteins, low-density lipoproteins (LDLs), and HDLs are involved in the metabolism of endogenously produced lipids.[5] VLDLs are secreted from the hepatocytes and acquire apo C and E from HDLs in the bloodstream. Apo C-II on the VLDL surface activates lipoprotein lipase on the capillary surface allowing hydrolysis of free fatty acids, which then can enter muscle and fat cells to be utilized for energy generation.[4] The VLDLs are then either removed from circulation by the liver or undergo further transformation by lipoprotein lipase and/or hepatic lipase into intermediate-density lipoproteins and LDLs.[4] LDLs circulate until they are absorbed by the liver or other peripheral tissues, where they are hydrolyzed within lysosomes, releasing cholesterol.[5]

9.2.1.3 Protein metabolism

The liver deaminates amino acids and converts them to carbohydrates and lipids. This occurs mainly by transamination, which is the transfer of one amino group to some acceptor substance.[8] An example of amino transamination is:

α-ketoglutaric acid + alanine → glutamic acid + pyruvic acid

Also, the liver is an important site for ammonia removal. In the liver, ammonia is converted to urea by the enzymes of the urea cycle or is used during the conversion of glutamate to glutamine. Urea leaves the liver through the venous blood

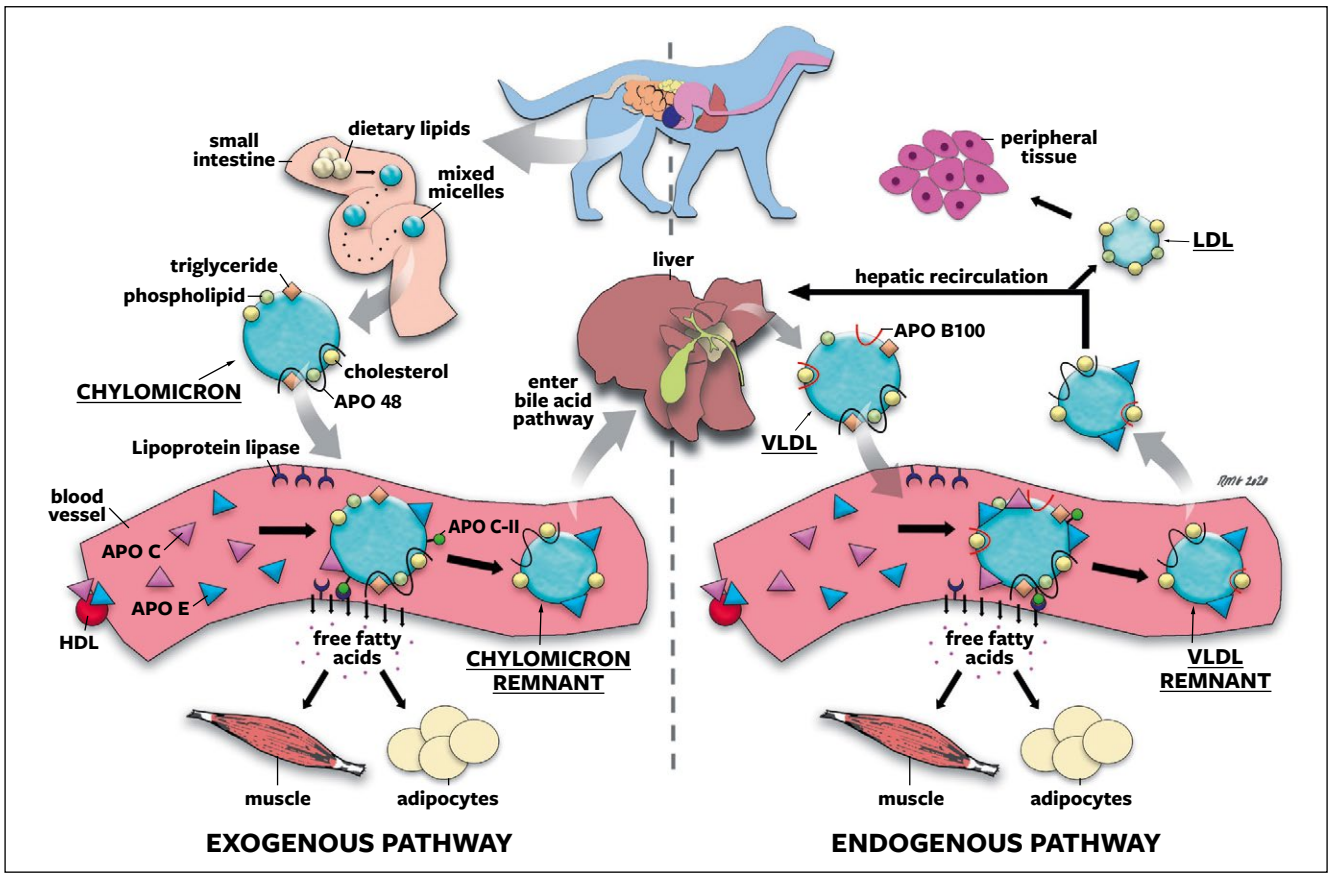

Fig. 9-5 Lipid metabolism

Left panel: In the exogenous pathway, dietary lipids undergo conversion into mixed micelles within the small intestine. They diffuse into the epithelial cells, where they combine with triglycerides, phospholipids, cholesterol, and apo B48, forming chylomicrons. Once they reach the lacteals, they are transported via the lymphatic system to the systemic circulation, where they acquire apo C and E from circulating high-density lipoproteins (HDLs). Apoprotein (Apo) C-II on the chylomicron surface then activates lipoprotein lipase on the capillary surface, allowing hydrolysis of free fatty acids that muscle cells and adipocytes utilize for energy. Chylomicron remnants with attached cholesterol can then be stored or used to create VLDLs.

Right panel: In the endogenous pathway, very low-density lipoproteins (VLDLs) are secreted from the liver and acquire apo C and E from HDLs in the bloodstream. Apo C-II on the VLDL surface activates lipoprotein lipase on the capillary surface, allowing hydrolysis of free fatty acids that muscle and fat cells utilize for energy. VLDLs are then either removed from circulation by the liver or undergo further transformation by lipoprotein lipase and/or hepatic lipase into intermediate-density lipoproteins and low-density lipoproteins (LDLs). LDLs circulate until they are absorbed by the liver or other peripheral tissues.

Apo = apolipoprotein; **HDL** = high-density lipoprotein; **VLDL** = very-low density lipoproteins; **LDL** = low-density lipoprotein

and is excreted by the body via the kidneys. Any ammonia not removed by the liver enters into the systemic circulation.[9,10] The liver also synthesizes many proteins, including albumin and fibrinogen, most α-globulins and some β-globulins, prothrombin, and a variety of clotting factors, including factors V, VII, VIII, IX, and X.[5]

9.2.2 Bile secretion

The primary secretory product of the liver is bile, which serves as a means for the excretion of waste products into the small intestines. Bile acids are an important component of bile and are important for:

- Helping to emulsify large fat particles from the diet into smaller particles, which can then be hydrolyzed by pancreatic lipase
- Aiding in the absorption of digested fat end products through the intestinal mucosal membrane[20]

More recently, bile acids have been shown to also have an endocrine function and thus play an important role in regulating metabolism.[12] To produce bile acids, cholesterol is

Although the physical examination may be unremarkable, some distinct signs may raise suspicion of a hepatobiliary disorder. Icterus is one of the most common manifestations of hepatobiliary disease and is often observed on mucus membranes, sclera, third eyelid, soft palate, and below the tongue.[21] ▶Fig. 9-7 illustrates an approach to the icteric patient. Abdominal palpation may reveal abnormalities, such as hepatomegaly or ascites. Ascites can occur with hepatic diseases that cause portal hypertension, such as advanced chronic hepatitis, cirrhosis, portal vein hypoplasia, or lobular dissecting hepatitis.[23]

Hepatomegaly in dogs can occur with primary hepatic tumors or secondary hepatic involvement with venous congestion or glycogen accumulation.

In cats, hepatomegaly is common and is suggestive of hepatic steatosis.

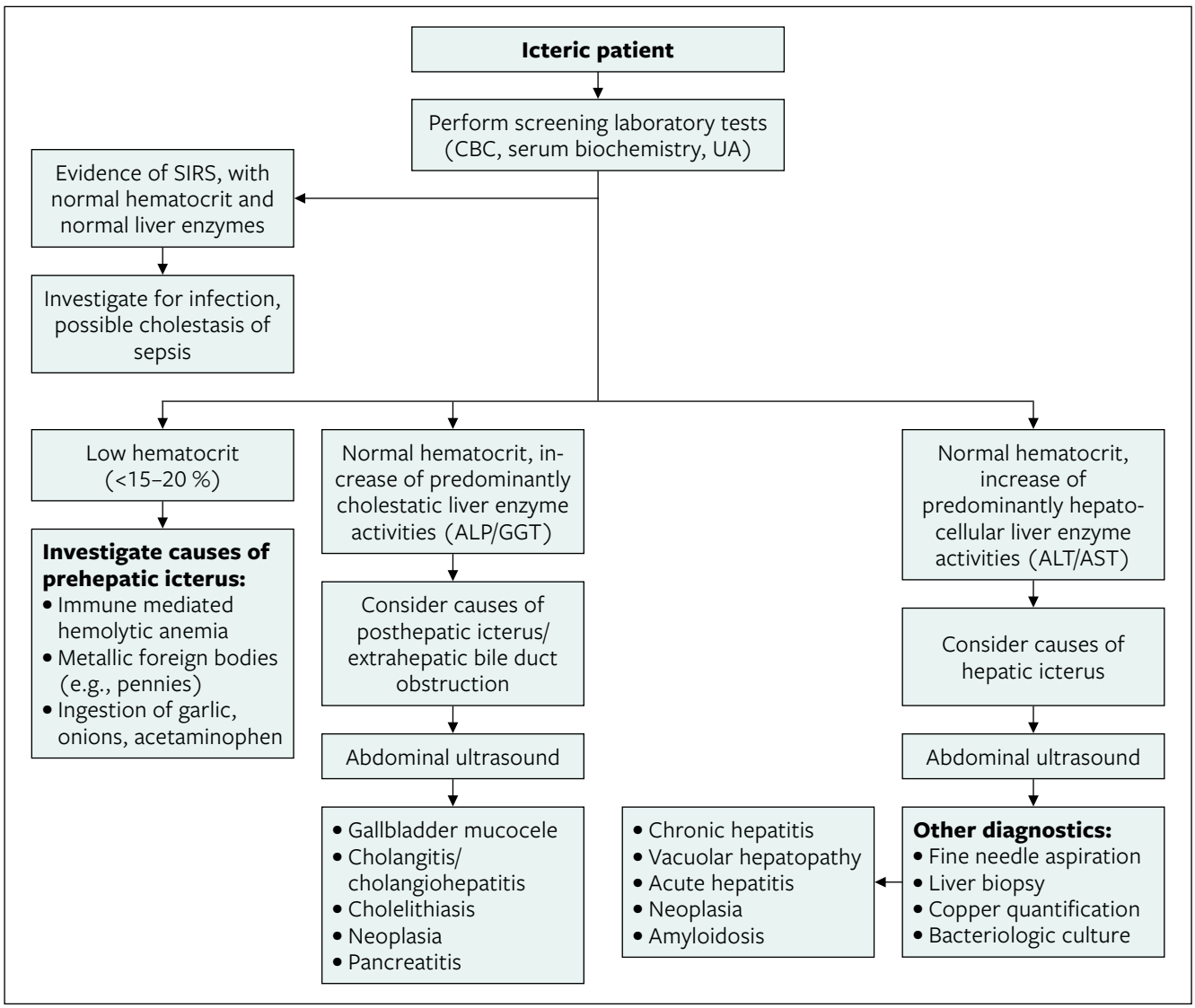

Fig. 9-7 Approach to an icteric patient

CBC = complete blood count; **UA** = urinalysis; **SIRS** = systemic inflammatory response; **ALP** = serum alkaline phosphatase; **GGT** = γ-glutamyl transferase; **ALT** = serum alanine transaminase; **AST** = serum aspartate transaminase

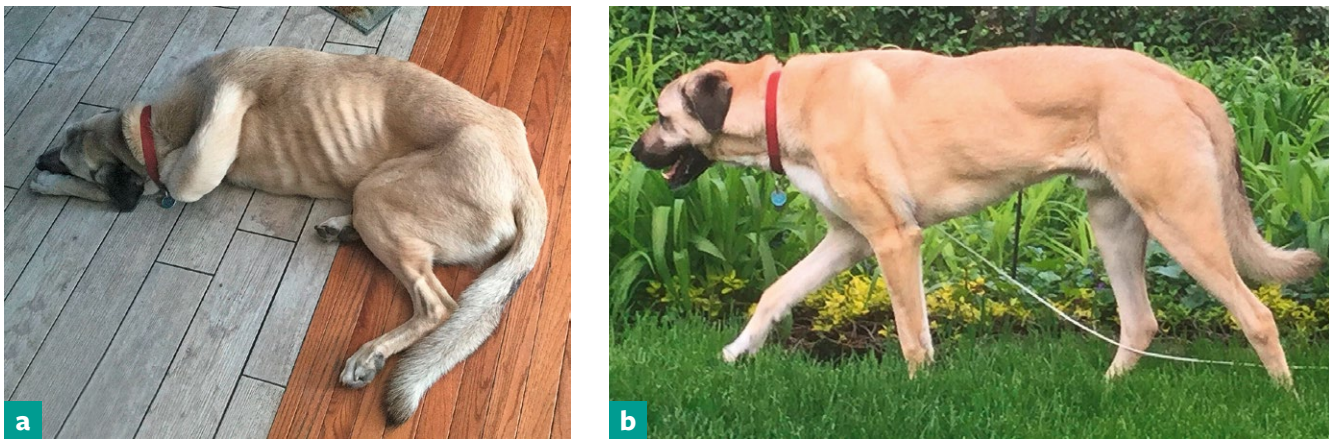

Fig. 10-15 Exocrine pancreatic insufficiency in a dog

a shows the dog before initiation of therapy. The dog has lost a significant amount of weight and has a poor hair coat.

b shows the same dog after treatment with pancreatic enzyme replacement therapy

(Courtesy of Roxene and James Moloney, Palos Hills, Illinois, USA.)

10.3.2.3 Pathogenesis

Pancreatic secretory products and, most importantly, pancreatic enzymes are crucial for the digestion and absorption of macronutrients. When pancreatic acinar cells are lacking, regardless of the cause, maldigestion occurs. It is important to note that the GI tract is a highly redundant system, and for most pancreatic digestive enzymes, there are other enzymes with the same function that are synthesized and secreted by other organs. For example, pancreatic lipase is crucial for fat digestion, but the stomach also synthesizes and secretes a lipase, gastric lipase, which is responsible for a significant portion of normal fat digestion in dogs.[14] Also, the exocrine pancreas has a huge reserve capacity. In humans, it has been estimated that clinical signs of EPI only ensue when more than 90% of exocrine pancreatic function has been lost.[15]

Maldigestion leads to undigested food components in the intestinal lumen, which can lead to diarrhea, alteration of the small intestinal microbiota, and weight loss.[15–18] Interestingly, though the dysbiosis improves with pancreatic enzyme replacement therapy, it does not normalize.[16] It is important to note that the clinical signs observed may not solely be due to maldigestion. For example, the pancreas secretes large quantities of bicarbonate, which is necessary to buffer gastric acid. A lack of bicarbonate leads to a decrease in duodenal pH, which can have an effect on brush border and pancreatic enzyme activities and also the intestinal microbiota. In addition, the pancreas is believed to synthesize and secrete trophic factors that help maintain a normal GI mucosa, and a lack of these factors may lead to malabsorption in addition to maldigestion. Also, rarely patients have been reported to be presented for neurologic signs. One cat was ultimately diagnosed with D-lactic acidosis, which was hypothesized to be due to intestinal dysbiosis. This cat responded well to pancreatic enzyme replacement therapy (PERT, ▶ Chap. 10.3.2.6.1).[19] In another cat, the neurologic signs were hypothesized to be due to secondary cobalamin deficiency.[20]

In sharp contrast to humans, where intrinsic factor is mainly secreted by the gastric mucosa, the exocrine pancreas is the major source of intrinsic factor in both dogs and cats.[21,22] As a result, the majority of dogs and cats with EPI are cobalamin deficient.[17,18]

10.3.2.4 Clinical presentation

EPI can be subclinical.[23] In two large case series of German shepherd dogs, severely decreased serum TLI concentrations were identified in several dogs without any clinical signs.[9,23] Some of these dogs underwent exploratory laparotomy, and their pancreatic mass was found to be severely decreased.[23] This, once again, underscores the overall high degree of redundancy in the process of digestion.

The most consistent clinical sign in dogs and cats with EPI is weight loss (▶ Fig. 10-15).[18,24–26] If the patient is very young, a failure to thrive may be reported instead of weight loss.[27] Loose stools are also commonly observed, but watery diarrhea is rather uncommon.[18,24] Often, affected patients have a poor hair coat, and dogs with EPI are commonly reported to have borborygmus and increased flatulence.[18,24,25] Many dogs and cats with EPI show an increased appetite, and some dogs show coprophagia or even pica.[24] However,

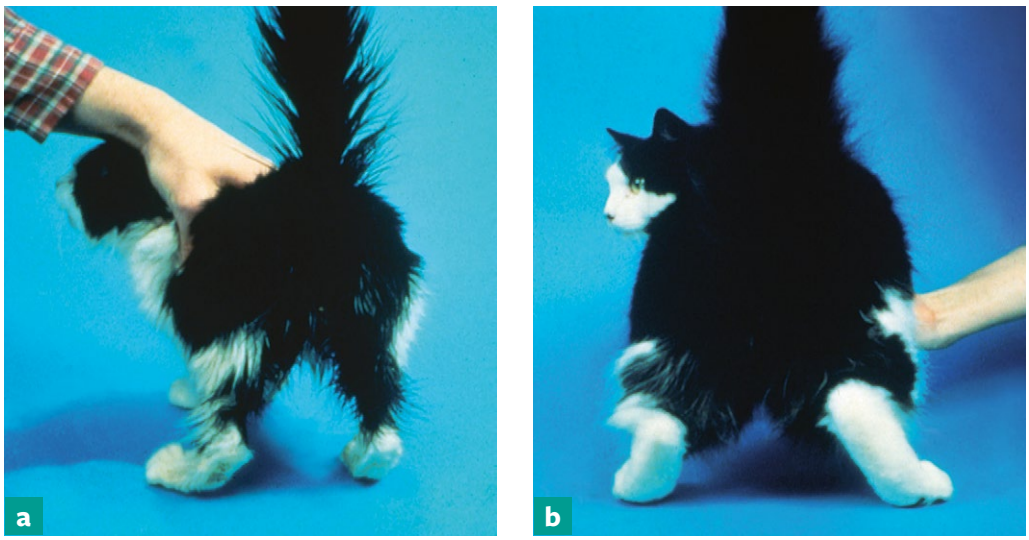

Fig. 10-16 Exocrine pancreatic insufficiency in a cat

a shows the cat before initiation of therapy. The cat has lost a significant amount of weight and has a poor hair coat. In addition, there is greasy soiling of its hair coat in the perineal region.

b shows the same cat after treatment with pancreatic enzyme replacement therapy.

(Courtesy of Dr. David A. Williams, University of Illinois, USA.)

some animals have a decreased appetite, which is most likely due to underlying chronic pancreatitis or comorbid disease, such as IBD. In some cats, greasy soiling of the hair coat in the perineal region can be observed (▶Fig. 10-16).[28] Finally, some patients with EPI, mostly those that have EPI as a long-term consequence of chronic pancreatitis, may show clinical signs of comorbid diabetes mellitus.[27]

10.3.2.5 Diagnosis

A diagnosis of EPI is made based on the demonstration of a lack of exocrine pancreatic function. Historically, several function tests have been described, including the plasma turbidity test, the para-aminobenzoic acid test, the fecal test for undigested starch and muscle fibers, and the fecal proteolytic activity test, but none of these should be used for the diagnosis of EPI in either dogs or cats.

CLINICAL PEARLS

The fecal proteolytic activity test can be used for the diagnosis of EPI in exotic species for which a species-specific trypsin-like immunoreactivity (TLI) test is not available.

Serum canine and feline trypsin-like immunoreactivity (**cTLI and fTLI**) are the gold standard tests for the diagnosis of EPI in dogs and cats, respectively.[28,29] The TLI assay is highly species-specific and measures the mass concentration of cationic trypsinogen, cationic trypsin, and some cationic trypsin molecules bound to proteinase inhibitor molecules. Under physiological conditions, only a small amount of the trypsinogen synthesized by pancreatic acinar cells is released into the vascular space. Trypsinogen and trypsin are relatively small molecules and thus get quickly excreted by the kidney. Therefore, only if the pancreas is functioning normally can a small amount of trypsinogen be detected in the serum. In contrast, in patients with EPI, regardless of the cause, the amount of trypsinogen released into the serum, and in turn, serum TLI concentration is severely decreased to undetectable.[28] In general, serum TLI is highly sensitive and specific for the diagnosis of EPI in both dogs and cats.[28,29] However, there are two scenarios where serum TLI can be normal despite the patient having EPI. The first is isolated pancreatic lipase deficiency.[30] It has long been recognized that the rate-limiting enzyme of pancreatic digestion is pancreatic lipase. Thus, patients with an isolated lipase deficiency may have clinical signs of EPI but have a normal serum TLI concentration. Another scenario where serum TLI concentration could be normal in a patient with EPI is a patient with an obstructed

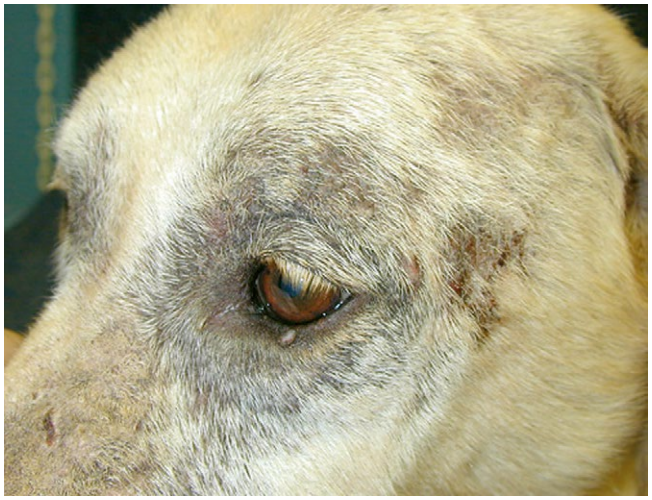

Fig. 11-2 Adverse food reaction

This figure shows periocular alopecia, hyperpigmentation, and excoriations secondary to intense pruritus resulting from an adverse cutaneous reaction to beef protein.

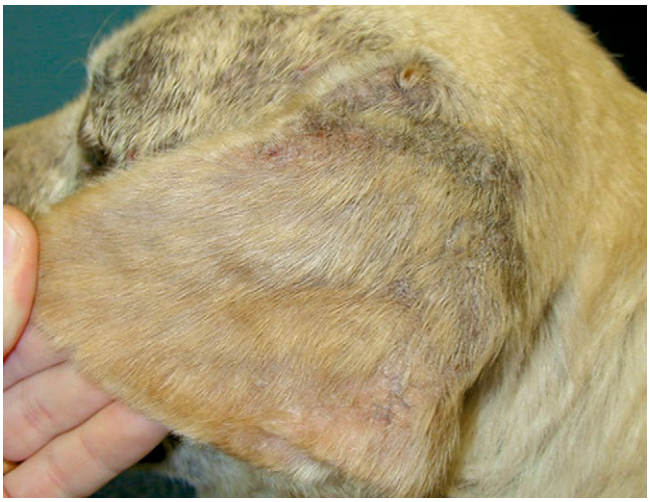

Fig. 11-3 Adverse food reaction

This figure shows erythema, alopecia, and excoriations on the ear lobe secondary to intense pruritus resulting from an adverse cutaneous reaction to beef protein.

defecation, were reported less commonly in dogs. In cats, uncommon non-cutaneous signs of AFR include conjunctivitis, salivation, flatulence, and respiratory signs.

There is no age, gender, or breed predisposition for CAFR in dogs. Several different breeds (German shepherd dogs, Labrador retrievers, golden retrievers, and West Highland white terriers) appear overrepresented for the development of AFR.[17] The reported age of onset may vary, but approximately 40% of dogs develop cutaneous manifestations of AFR by one year of age. Dermatological manifestations typically occur as generalized nonseasonal moderate to severe pruritus, or the patient may present with otitis externa as the only clinical sign. Other cutaneous manifestations, including recurrent bacterial (presumed staphylococcal) and yeast skin infections, otitis externa, and atopic dermatitis, may occur in the same canine patient (▶ Fig. 11-2, ▶ Fig. 11-3, ▶ Fig. 11-4). Regarding non-cutaneous signs, a high number of dogs (70–88%) with AFR exhibit vomiting and/or diarrhea.[17] In most cases, the diarrhea is of large bowel origin and is accompanied by an increased frequency of defecation, tenesmus, or both. In some studies, clinical improvement of GI signs was observed following a change in diet, but re-challenge with the original diet was not performed in all instances.

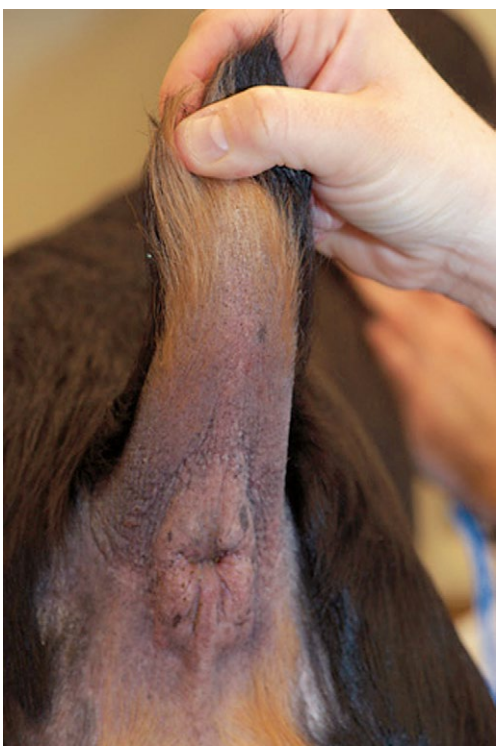

Fig. 11-4 Adverse food reaction

This figure shows perianal alopecia, erythema, lichenification, and hyperpigmentation secondary to intense pruritus resulting from an adverse cutaneous reaction to chicken protein.

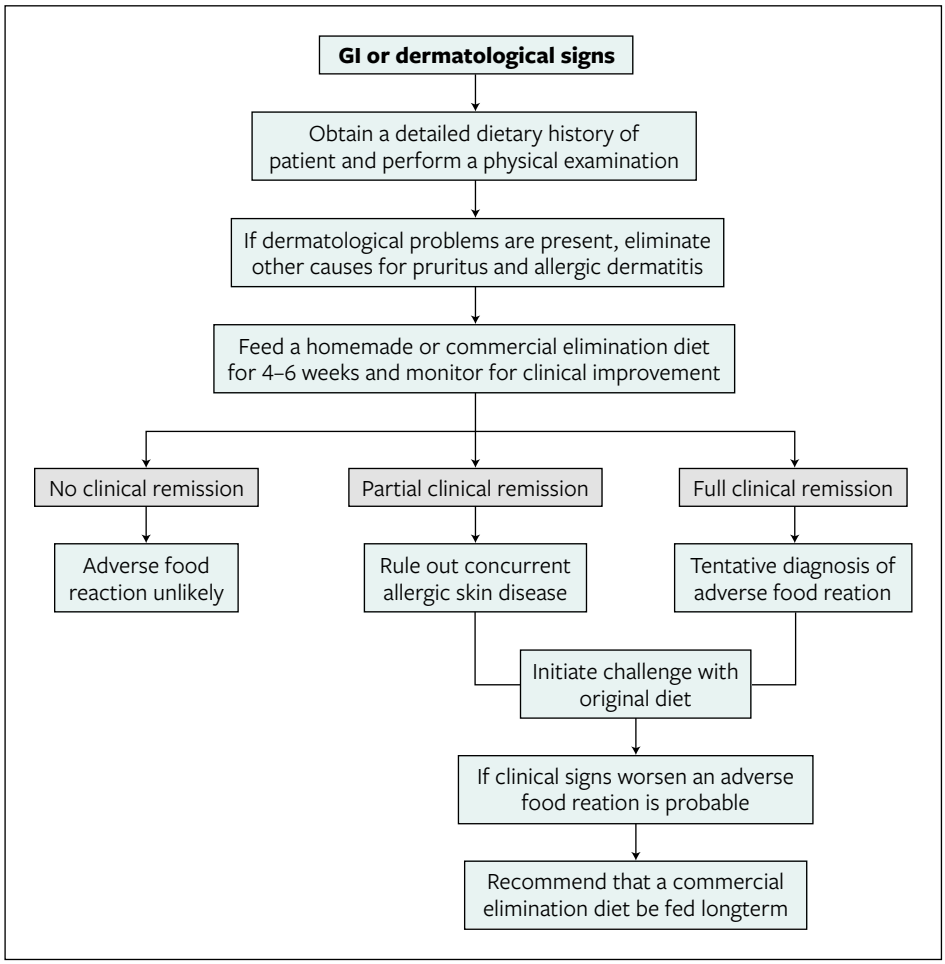

Fig. 11-5 Practical approach to elimination trials for the diagnosis of adverse food reactions (modified from Gaschen et al. 2011[22])

GI = gastrointestinal

A home-cooked elimination diet should consist of a single protein source and single carbohydrate source, with both ingredients not present in previously fed diets.

Suggested food components in dogs include fish, rabbit, venison, rice, potatoes, and tofu.

Cats, being obligate carnivores, do well on all-meat diets.

Home-cooked diets are often advocated as the initial test food for dogs and cats with suspected AFR. While it is generally best to gradually introduce the new diet over 3–4 days, many patients, especially dogs, will immediately eat the new diet. Caution is advised when feeding homemade diets for longer than 3 weeks, as they may be nutritionally unbalanced.[23] Ideally, consultation with a veterinary nutritionist is recommended for optimal formulation if long-term use is anticipated.

Most commercial selected protein diets are indicated for the long-term management of dogs and cats with AFR. These products are attractive because they are convenient, balanced, and nutritionally complete for either dogs or cats. However, different studies have shown that proteins not listed on the label were also included in the prescription formulation.[24] Importantly, the clinical significance of extra-label proteins in AFR was not evaluated through properly conducted feeding provocation trials. In animals failing to respond to a single elimination diet, a second diet may be indicated to confirm a diagnosis.

Protein hydrolysate diets contain proteins that have been hydrolyzed into smaller peptides and amino acids, which reduces the potential allergenicity of the diet. These diets are useful in reducing clinical signs of CAFR but also in

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Romy M. Heilmann, Dr. med. vet., PhD, DACVIM-SAIM, DECVIM-CA, MANZCVS, AGAF, Professor and Chair of Small Animal Internal Medicine, University of Leipzig, Germany; Romy Heilmann leads and participates in several clinical and basic science projects in the fields of small animal gastroenterology, hepatology, immunopathology, and translational medicine.

Jonathan A. Lidbury, BVMS, PhD, MRCVS, DACVIM, DECVIM-CA, Associate Professor of Small Animal Internal Medicine and Director of Clinical Services, Gastrointestinal Laboratory at Texas A&M University; Rob and Roxann Bilger Chair in Feline Hepatology; Jonathan Lidbury is interested in all areas of small animal gastroenterology and is working to develop new non-invasive tests for liver disease in cats and dogs.

Jörg M. Steiner, Dr. med. vet., PhD, DACVIM-SAIM, DECVIM-CA, AGAF, Professor of Small Animal Internal Medicine & Director of the Gastrointestinal Laboratory at Texas A&M University; Dr. Mark Morris Chair in Small Animal Gastroenterology & Nutrition; Jörg Steiner received the AVMA Career Achievement Award in Canine Research (2018) and was selected as a University Distinguished Professor (2019) and Regents Professor (2021).

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