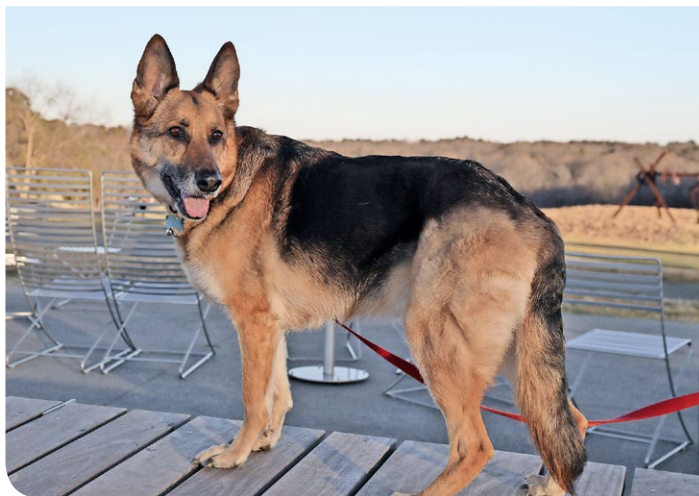
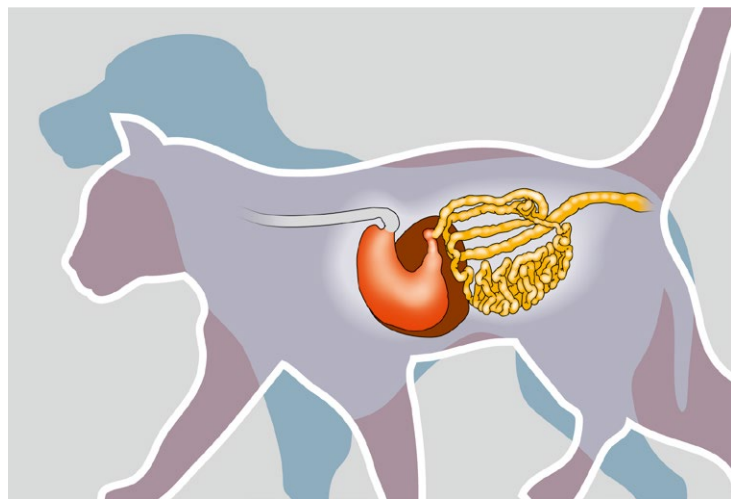


Romy M. Heilmann | Jonathan A. Lidbury |
Jörg M. Steiner (eds.)

Small Animal Gastroenterology

Second, revised and expanded edition



VET EXPERTISE

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Contents

Editors	X	1.4.7	Liver and biliary tract	81
Authors	XI	1.4.8	Pancreas	91
Abbreviations	XX	1.5	Endoscopy	94
Preface	XXV		<i>Michael D. Willard</i>	
		1.5.1	Introduction	94
		1.5.2	Endoscopic procedures	95
		1.5.3	Collection of biopsies	98
		1.5.4	Endoscopical findings	102
		1.5.5	Interventional procedures	106
		1.5.6	Capsule endoscopy	110
		1.6	Diagnostic laparoscopy	111
			<i>David C. Twedt</i>	
		1.6.1	Introduction	111
		1.6.2	Indications	111
		1.6.3	Laparoscopic equipment	111
		1.6.4	Biopsy techniques	112
		1.6.5	Ancillary procedures	116
		1.6.6	Complications of laparoscopy	118
		1.7	Cytology	118
			<i>Jelena Palić, Johannes Hirschberger</i>	
		1.7.1	Introduction and technique	118
		1.7.2	Stomach and intestines	119
		1.7.3	Liver	122
		1.7.4	Bile	128
		1.7.5	Pancreas	128
		1.8	Histopathology	130
		1.8.1	Gastrointestinal tract	130
			<i>Mark R. Ackermann, Paula Giaretta</i>	
		1.8.2	Liver and the gallbladder	147
			<i>John Cullen</i>	
		1.8.3	Pancreas	156
			<i>Katja Steiger</i>	
		1.9	Assessment of gastrointestinal (GI) motility	162
			<i>Frédéric P. Gaschen</i>	
		1.9.1	Introduction and physiology of GI motility ..	162
		1.9.2	Indications for assessment of GI motility ...	162
		1.9.3	Available methods for evaluation of GI motility	162
		1.9.4	Clinical evaluation of GI motility	165

2	Clinical evaluation of dogs and cats with specific clinical signs	167			
2.1	Clinical evaluation of patients with acute signs of gastrointestinal disease	167			
	<i>Emily N. Gould</i>				
2.1.1	Introduction	167			
2.1.2	Clinical signs consistent with acute gastrointestinal disease	167			
2.1.3	Differentials for acute gastrointestinal disease	168			
2.1.4	Patient history	170			
2.1.5	Physical examination	171			
2.1.6	Diagnostic testing	171			
2.2	Clinical evaluation of patients with chronic vomiting	173			
	<i>Panagiotis G. Xenoulis</i>				
2.2.1	Introduction	173			
2.2.2	Causes of chronic vomiting	175			
2.2.3	Initial database	177			
2.2.4	Diagnostic approach	179			
2.3	Clinical evaluation of patients with chronic diarrhea	181			
	<i>Barbara Kohn, Christiane Weingart</i>				
2.3.1	Definition and differentials	181			
2.3.2	Signalment	183			
2.3.3	Medical history	183			
2.3.4	Clinical examination	184			
2.3.5	Laboratory tests	185			
2.3.6	Imaging techniques	186			
2.3.7	Additional testing	187			
2.4	Clinical evaluation of patients with chronic weight loss	188			
	<i>Aarti Kathrani</i>				
2.4.1	Definition	188			
2.4.2	Epidemiology	189			
2.4.3	Pathophysiology	189			
2.4.4	Historical assessment	189			
2.4.5	Clinical evaluation	189			
2.4.6	Physical evaluation	189			
2.4.7	Differential diagnoses	190			
2.4.8	Diagnostic investigations	191			
2.4.9	Treatment	192			
2.5	Clinical evaluation of patients with constipation	192			
	<i>Silke Salavati Schmitz</i>				
2.5.1	Definition	192			
2.5.2	Pathophysiology and differential diagnoses	192			
2.5.3	Diagnostic approach	193			
2.5.4	Treatment and management	194			
2.5.5	Prognosis	197			
2.6	Clinical evaluation of patients with jaundice/icterus	198			
	<i>Micah A. Bishop</i>				
2.6.1	Introduction and definitions	198			
2.6.2	Historical assessment and signalment	198			
2.6.3	Physical assessment	199			
2.6.4	Diagnostic approach	199			
II	Clinical nutrition				
3	Nutritional management of gastrointestinal diseases	203			
3.1	Nutritional principles	203			
	<i>Aarti Kathrani</i>				
3.1.1	Assessment of nutritional status	203			
3.1.2	Assessment of diets, feeding plans, and environmental influences	204			
3.1.3	Reassessment and monitoring	207			
3.2	Nutritional management of patients with gastrointestinal disease	208			
3.2.1	Enteral feeding	208			
	<i>Aarti Kathrani</i>				
3.2.2	Placement and management of feeding tubes	213			
	<i>Natalie Hughes, James Howard, Valerie J. Parker</i>				
III	Diseases of the gastrointestinal tract				
4	Oral cavity	227			
4.1	Anatomy and physiology	227			
	<i>Ana C. Castejon-Gonzalez, Alexander M. Reiter</i>				
4.2	Diseases of the oral cavity	228			
4.2.1	Gingivostomatitis	228			
	<i>Lara Boland</i>				
4.2.2	Eosinophilic granuloma complex	231			
	<i>Lara Boland</i>				
4.2.3	Salivary disorders	232			
	<i>Ana C. Castejon-Gonzalez, Alexander M. Reiter</i>				
4.2.4	Neoplastic conditions of the oral cavity	235			
	<i>Heather Wilson-Robles</i>				

5	Esophagus	239	7	Small intestine	299
5.1	Anatomy	239	7.1	Anatomy	299
	<i>Joao Felipe de Brito Galvao</i>			<i>Craig G. Ruaux</i>	
5.2	Physiology	240	7.1.1	Introduction	299
	<i>Joao Felipe de Brito Galvao</i>		7.1.2	Gross anatomy of the intestinal tract	299
5.3	Diseases of the esophagus	241	7.1.3	Anatomical features of the small intestine ..	300
5.3.1	Cricopharyngeus muscle dysfunction	241	7.2	Physiology	301
	<i>Romy M. Heilmann, Stanley L. Marks</i>			<i>Craig G. Ruaux</i>	
5.3.2	Esophagitis	243	7.2.1	Introduction	301
	<i>Joao Felipe de Brito Galvao</i>		7.2.2	Function of the villus	301
5.3.3	Gastroesophageal reflux	245	7.2.3	Gastrointestinal hormones	302
	<i>Joao Felipe de Brito Galvao</i>		7.2.4	Gut-associated lymphoid tissue and immune system	303
5.3.4	Esophageal foreign bodies	246	7.2.5	Intestinal bacteria	304
	<i>Joao Felipe de Brito Galvao</i>		7.3	Small intestinal disease	305
5.3.5	Esophageal strictures	250	7.3.1	Infectious causes of small intestinal disease	305
	<i>Joao Felipe de Brito Galvao</i>		7.3.2	Dietary indiscretion (garbage can intoxication)	327
5.3.6	Esophageal diverticula	255		<i>Melanie Werner, Stefan Unterer</i>	
	<i>Joao Felipe de Brito Galvao</i>		7.3.3	Acute hemorrhagic diarrhea syndrome	327
5.3.7	Airway-esophageal fistula	257		<i>Kathrin Busch, Stefan Unterer</i>	
	<i>Joao Felipe de Brito Galvao</i>		7.3.4	Alterations in the intestinal microbiota (dysbiosis)	330
5.3.8	Esophageal hypomotility/megaesophagus ..	257		<i>Jan S. Suchodolski</i>	
	<i>Joao Felipe de Brito Galvao</i>		7.3.5	Non-obstructive motility disorders	336
5.3.9	Hiatal hernia	259		<i>Frédéric Gaschen</i>	
	<i>Harry Cridge</i>		7.3.6	Intestinal obstruction	337
5.3.10	Gastroesophageal intussusception	262	7.3.7	Short bowel syndrome	340
	<i>Joao Felipe de Brito Galvao,</i> <i>Lars F.H. Theyse</i>			<i>Anna-Lena Ziese, Stefan Unterer</i>	
5.3.11	Vascular ring anomalies	263	7.3.8	Protein-losing enteropathy	341
	<i>Lars F.H. Theyse</i>			<i>Romy M. Heilmann</i>	
5.3.12	Neoplastic conditions of the esophagus	263	7.3.9	Neoplasia of the small intestine	348
	<i>Katie Z. Wright, Ann E. Hohenhaus</i>			<i>Emma Warry</i>	
6	Stomach	267	8	Large intestine and anorectum	353
6.1	Anatomy	267	8.1	Anatomy	353
	<i>Panpicha Sattasathuchana</i>			<i>Timothy Bolton, Michael S. Leib</i>	
6.2	Physiology	268	8.2	Physiology	355
	<i>Panpicha Sattasathuchana</i>			<i>Michael S. Leib, Timothy Bolton</i>	
6.2.1	Gastric emptying and motility	268	8.2.1	Motility	355
6.2.2	Gastric acid secretion	268	8.2.2	Water and electrolyte transport	356
6.2.3	Gastric mucosal barrier	270	8.2.3	Mucus secretion	356
6.3	Diseases of the stomach	271	8.2.4	Colonic microbiome	356
6.3.1	Acute and chronic gastritis	271	8.2.5	Immune function	358
	<i>M. Katherine Tolbert</i>		8.3	Diseases of the large intestine	359
6.3.2	Motility disorders	280	8.3.1	Acute and chronic colitis	359
	<i>Silke Salavati Schmitz</i>			<i>Michael S. Leib, Timothy Bolton</i>	
6.3.3	Gastric dilatation-volvulus	290	8.3.2	<i>Clostridium perfringens</i> enterotoxigenesis	365
	<i>Lars F.H. Theyse</i>			<i>Michael S. Leib, Timothy Bolton</i>	
6.3.4	Neoplastic conditions of the stomach	291			
	<i>Katie Z. Wright, Ann E. Hohenhaus</i>				

8.3.3	<i>Tritrichomonas blagburni</i> infection	367	9.4.5	Dermatologic signs	408
	<i>M. Katherine Tolbert</i>		9.4.6	Gastrointestinal signs	408
8.3.4	Irritable bowel syndrome	369	9.4.7	Bleeding disorders	408
	<i>Michael S. Leib, Timothy Bolton</i>		9.4.8	Management of main complications of	
8.3.5	Fiber-responsive large bowel diarrhea	371		liver disease	409
	<i>Michael S. Leib, Timothy Bolton</i>		9.5	Diseases of the liver	410
8.3.6	Feline megacolon	372	9.5.1	Hepatotoxins and drug-induced liver injury ..	410
	<i>Andreas H. Hasler</i>			<i>Adam Gow</i>	
8.3.7	Neoplastic diseases of the large intestine ...	379	9.5.2	Infectious diseases of the liver	414
	<i>Emma Warry</i>			<i>Penny Watson</i>	
8.4	Diseases of the anorectum	382	9.5.3	Hepatic vascular anomalies	421
8.4.1	Proctitis and rectal prolapse	382		<i>M. S. Tivers</i>	
	<i>Lars F.H. Theyse, Romy M. Heilmann</i>		9.5.4	Metabolic diseases of the liver	429
8.4.2	Rectoanal malformations	384		<i>Floris C Dröes and Jonathan A Lidbury</i>	
	<i>Anna-Lena Ziese, Stefan Unterer</i>		9.5.5	Inflammatory diseases of the liver	451
8.4.3	Perianal fistula	386		<i>Emma O'Neill and Myles McKenna</i>	
	<i>Adam P. Patterson</i>		9.5.6	Developmental disorders of the liver	461
8.4.4	Rectal and perianal neoplasia	390		<i>Penny Watson</i>	
	<i>Emma Warry</i>		9.5.7	Neoplasms of the liver	467
				<i>Brandan Wustefeld-Janssens, Emma Warry</i>	
9	Liver and biliary tract	395	9.6	Diseases of the biliary tract	471
9.1	Anatomy	395	9.6.1	Extrahepatic biliary obstruction	471
	<i>Randi Gold</i>			<i>Vanna Dickerson, Kelley Thieman Mankin</i>	
9.1.1	Liver structure	395	9.6.2	Gallbladder mucocele	478
9.1.2	Cells of the liver	396		<i>Kelley M. Thieman Mankin,</i>	
9.1.3	Hepatic pigments	397		<i>Vanna M. Dickerson</i>	
9.1.4	Biliary tract structure	398	9.6.3	Cholecystitis/cholangitis	482
9.1.5	Portal circulation	399		<i>Jared A. Jaffey</i>	
9.2	Physiology	400	9.6.4	Cholelithiasis/choleductolithiasis	490
	<i>Randi Gold</i>			<i>Jared Jaffey</i>	
9.2.1	Metabolism	400	9.6.5	Parasitic diseases of the biliary system	492
9.2.2	Bile secretion	401		<i>Jenny Stiller</i>	
9.2.3	Vitamin storage	402	9.6.6	Neoplasms of the biliary system	495
9.2.4	Hemostasis	403		<i>Brandan Wustefeld-Janssens</i>	
9.2.5	Detoxification	403			
9.2.6	Hematologic and immunologic functions ...	403	10	Exocrine pancreas	497
9.3	Diagnostic approach to liver disease	403	10.1	Anatomy	497
	<i>Adrian Tinoco-Najera, Jonathan Lidbury</i>			<i>Jörg M. Steiner</i>	
9.3.1	Introduction	403	10.2	Physiology	498
9.3.2	Prevalence and breed predispositions	404		<i>Jörg M. Steiner</i>	
9.3.3	Clinical presentation	404	10.3	Diseases of the exocrine pancreas	499
9.3.4	Approach to a patient with suspected liver		10.3.1	Pancreatitis	499
	disease	404		<i>Jörg M. Steiner</i>	
9.4	Complications of liver disease	407	10.3.2	Exocrine pancreatic insufficiency	516
	<i>Adrian Tinoco-Najera, Jonathan Lidbury</i>			<i>Jörg M. Steiner</i>	
9.4.1	Icterus	407	10.3.3	Exocrine pancreatic neoplasia	522
9.4.2	Polyuria and polydipsia	407		<i>Katja Steiger</i>	
9.4.3	Portal hypertension and ascites	407	10.3.4	Rare diseases of the exocrine pancreas	526
9.4.4	Hepatic encephalopathy	408		<i>Jörg M. Steiner</i>	

11	Diseases that affect more than one organ of the gastrointestinal tract	531		
11.1	Adverse reactions to food – allergy versus intolerance	531		
	<i>Albert E. Jergens, Elizabeth R. Drake</i>			
11.1.1	Introduction	531		
11.1.2	Terminology	531		
11.1.3	Etiopathogenesis of food allergy	532		
11.1.4	Adverse food reactions	532		
11.2	Chronic inflammatory enteropathies	536		
	<i>Edward Hall and Karin Allenspach</i>			
11.2.1	Introduction and definitions	536		
11.2.2	Etiopathogenesis	537		
11.2.3	Classification	540		
11.2.4	Clinical presentation	543		
11.2.5	Diagnosis	544		
11.2.6	Treatment	547		
11.2.7	Prognosis	550		
11.3	Gastrointestinal lymphoma	551		
	<i>Sina Marsilio</i>			
11.3.1	Introduction	551		
11.3.2	Feline gastrointestinal lymphoma	551		
11.3.3	Canine gastrointestinal lymphoma	555		
11.4	Neuroendocrine tumors of the gastrointestinal tract	557		
	<i>Jörg M. Steiner, Cynthia Ward</i>			
11.4.1	Introduction	557		
11.4.2	Insulinoma	560		
11.4.3	Gastrinoma	566		
11.4.4	Glucagonoma	570		
11.4.5	Gastrointestinal carcinoids	572		
11.4.6	Somatostatinoma	572		
11.4.7	Pancreatic polypeptidoma	573		
11.4.8	Other neuroendocrine tumors of the gastrointestinal tract	573		
11.5	Adipositas/Obesity	573		
	<i>Alexander J. German</i>			
11.5.1	Definition	573		
11.5.2	Is obesity a disease?	574		
11.5.3	Prevalence	574		
11.5.4	Etiology and pathogenesis	575		
11.5.5	Clinical presentation	581		
11.5.6	Differential diagnoses	582		
11.5.7	Diagnosis	582		
11.5.8	Management	588		
11.5.9	Prevention of obesity	594		
	Appendix			
	Index	599		

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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

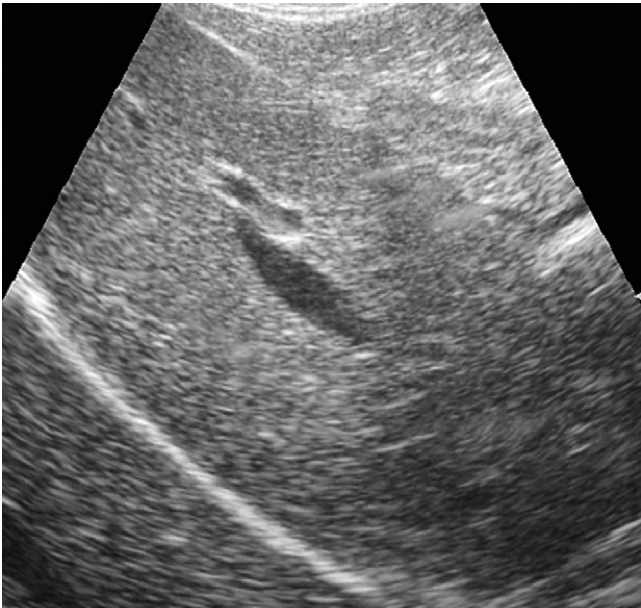


Fig. 1-47 Normal canine liver

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

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 hyper-

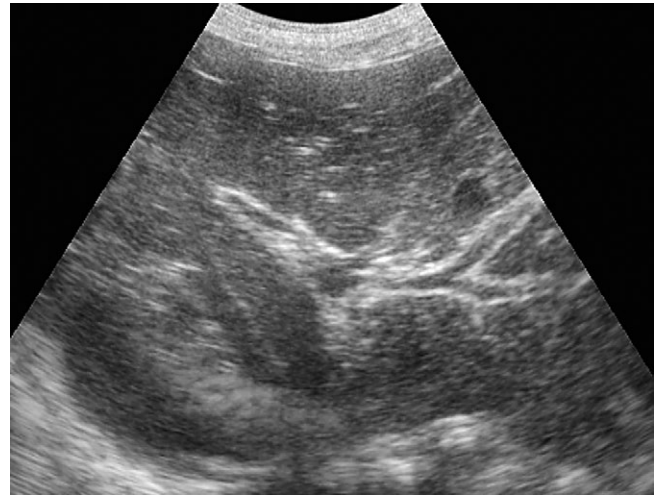


Fig. 1-48 Cholangiohepatitis

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.

echoic 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.

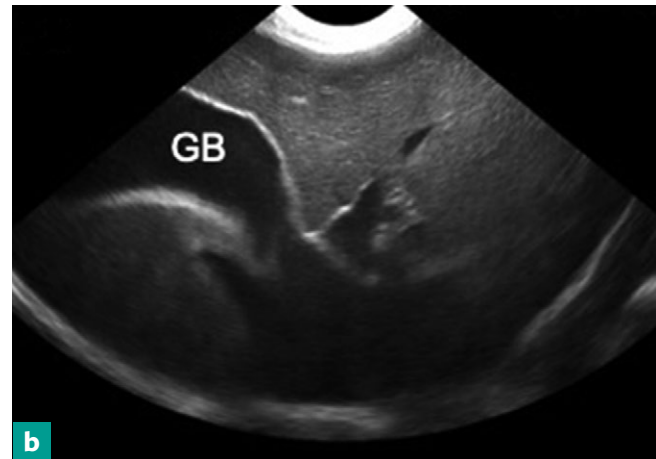
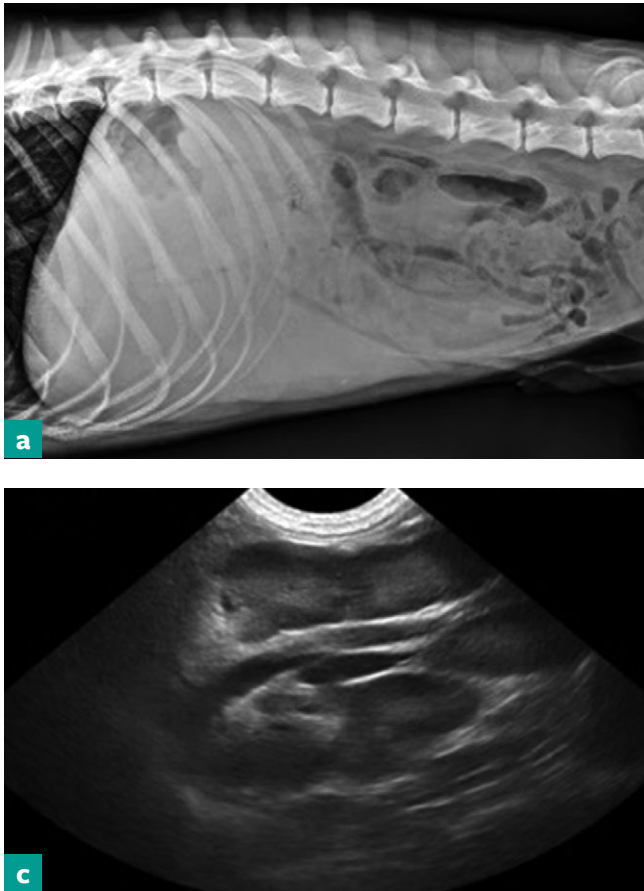


Fig. 1-49 Lymphoma

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

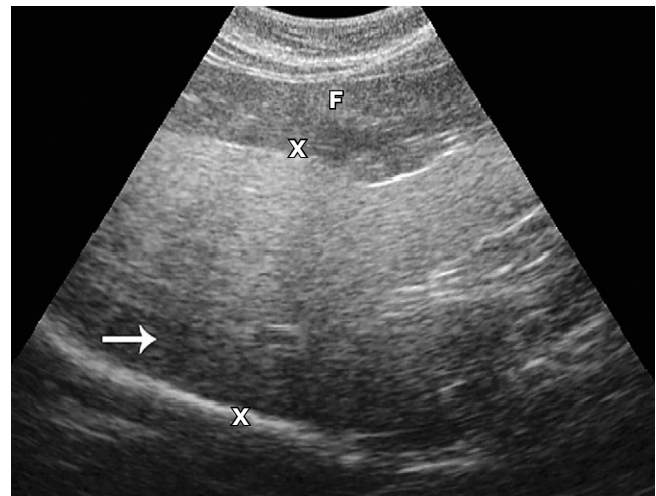


Fig. 1-50 Hepatic lipidosis

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

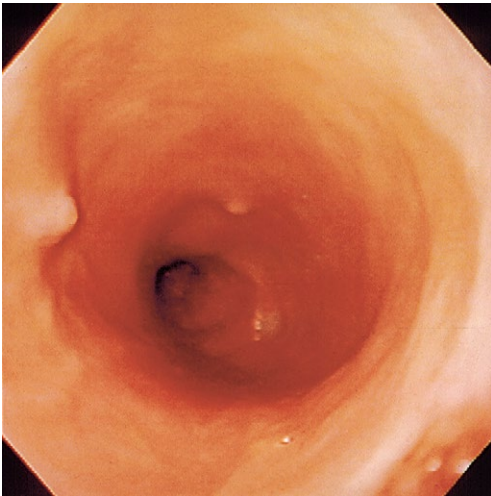


Fig. 1-71 Duodenal papilla
Endoscopic view of a duodenum showing the duodenal papilla at the 9 o'clock position. (Used with permission from Fossum T, 2002³³)



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 Endoscopic 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 sphincter (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

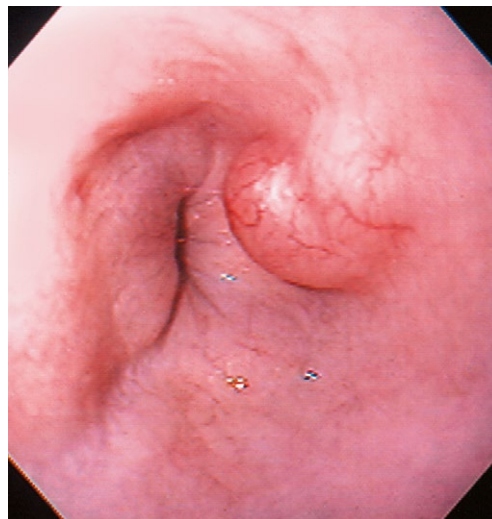


Fig. 1-73 Submucosal leiomyoma in a dog
(Used with permission from Nelson R, 2003²²)

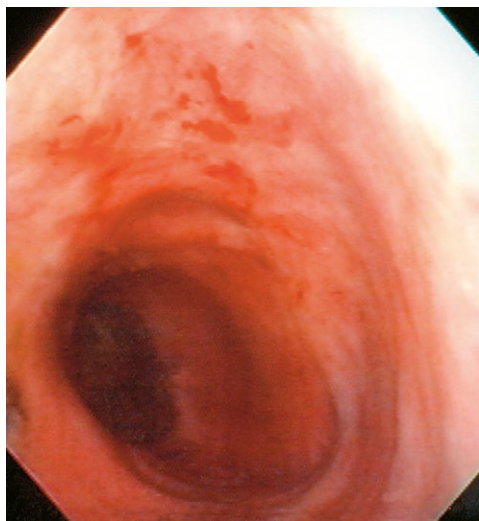


Fig. 1-74 Esophagitis
Note distinct areas of hyperemia.

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).

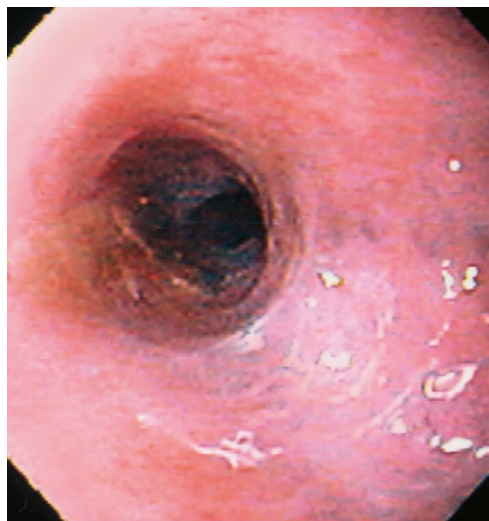


Fig. 1-75 Esophageal stricture
Note the narrowing of the lumen and the white tissue (scar tissue) at the 4 o'clock position.

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 CO₂ 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

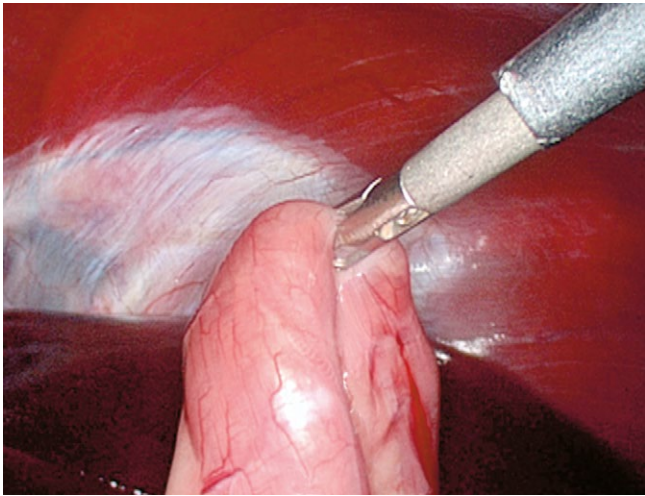


Fig. 1-91 Small intestinal biopsy

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

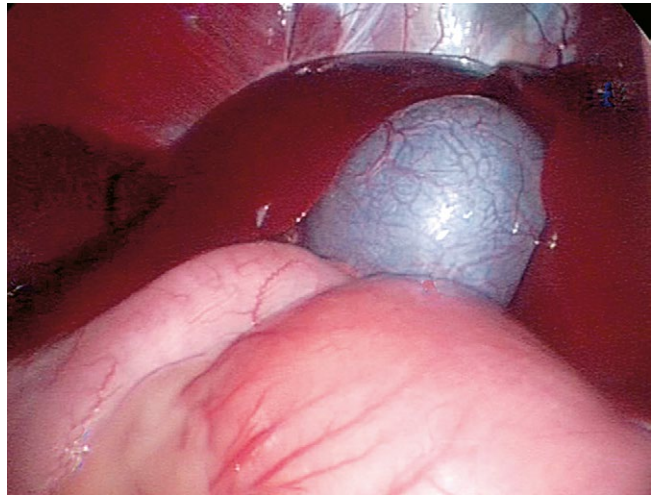


Fig. 1-92 Liver, gallbladder, and intestine

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

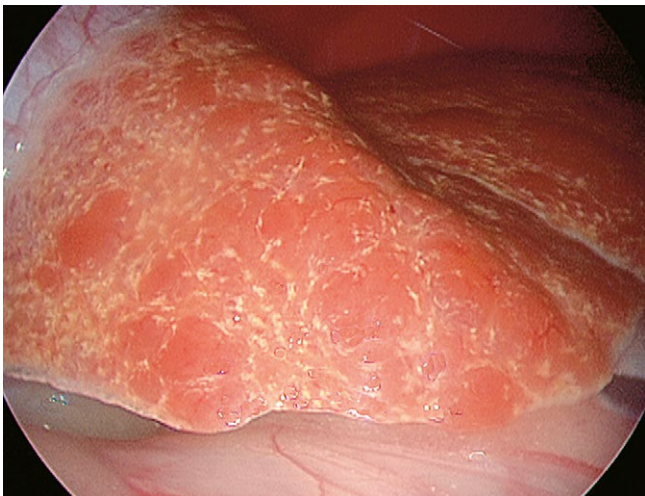


Fig. 1-93 Nodular liver

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

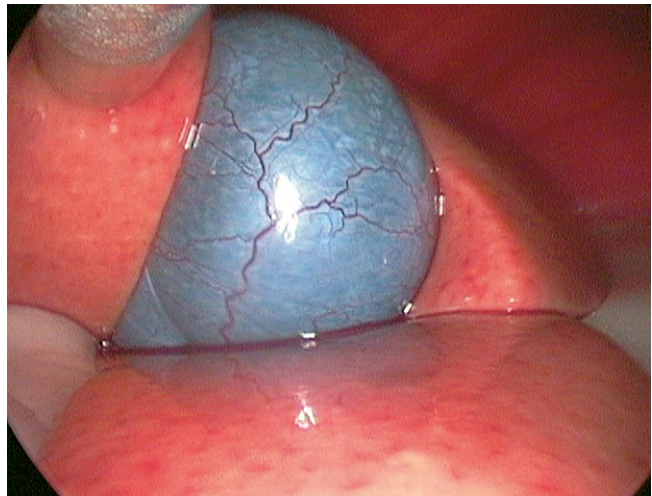


Fig. 1-94 Hepatic lipidosis

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 infor-

mation 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

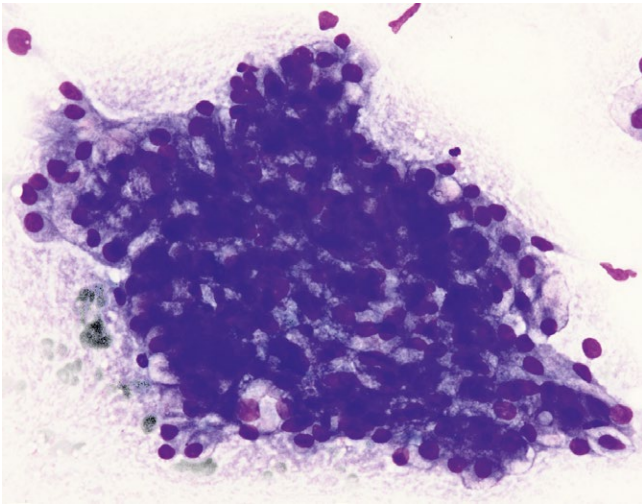


Fig. 1-122 Well-differentiated hepatocellular carcinoma (confirmed by histopathology) in a 13-year-old mixed-breed dog with a surgically removed massive (10 cm diameter) liver mass 1 year prior to fine-needle aspiration (FNA)

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

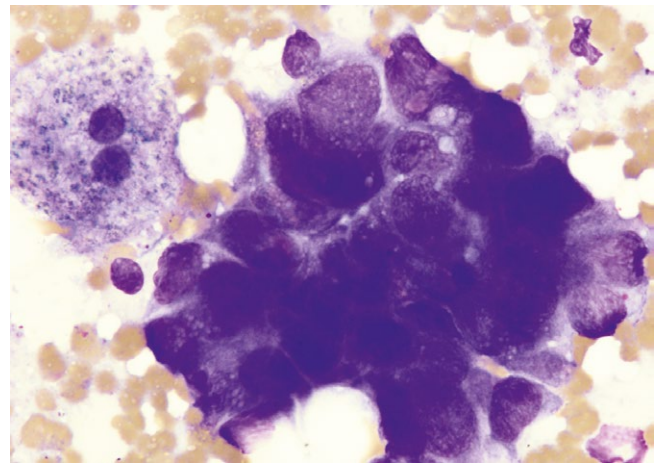


Fig. 1-123 Hepatocellular carcinoma in a 13-year-old female mixed breed dog with multiple hypoechogenic liver masses noted during ultrasonographic examination

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

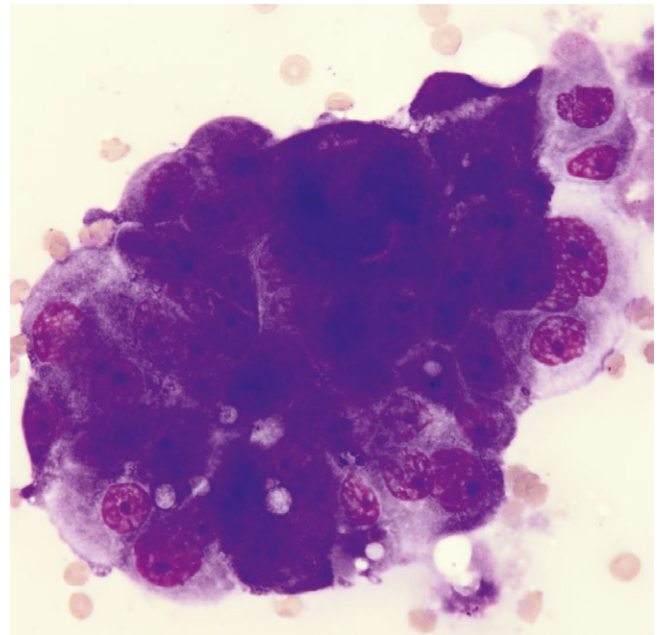


Fig. 1-124 Cholangiocellular carcinoma in a 16-year-old male Persian cat with ascites and marked ultrasonographic changes of the liver parenchyma

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

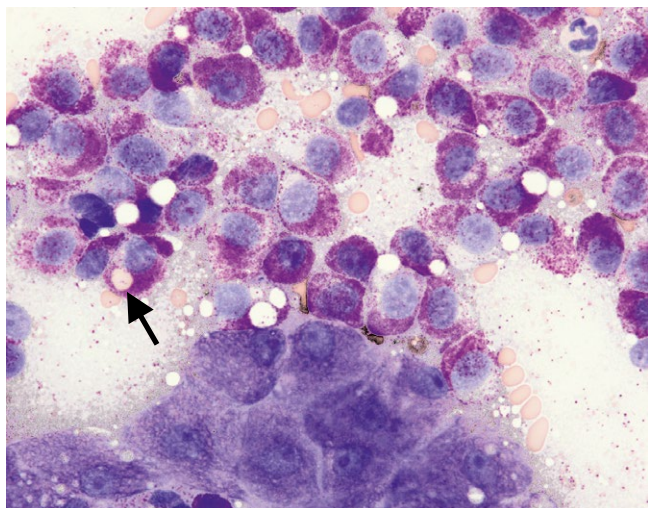


Fig. 1-125 Mast cell tumor in the liver of a 9-year-old male cat with multiple cutaneous mast cell tumors, splenomegaly, and hepatomegaly

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).

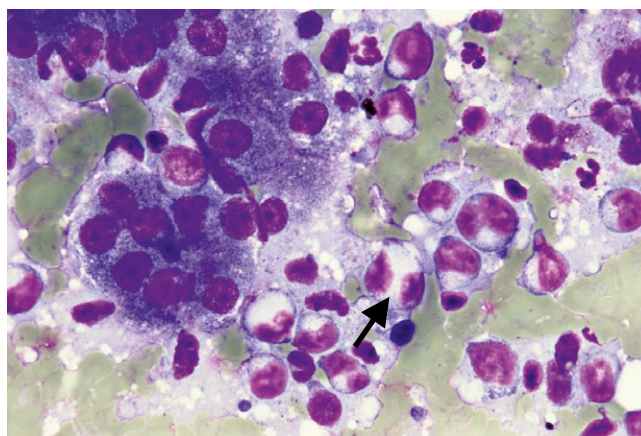


Fig. 1-126 Lymphoma in an 8-year-old male mixed breed dog with hepatomegaly and increased liver enzyme activities

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).

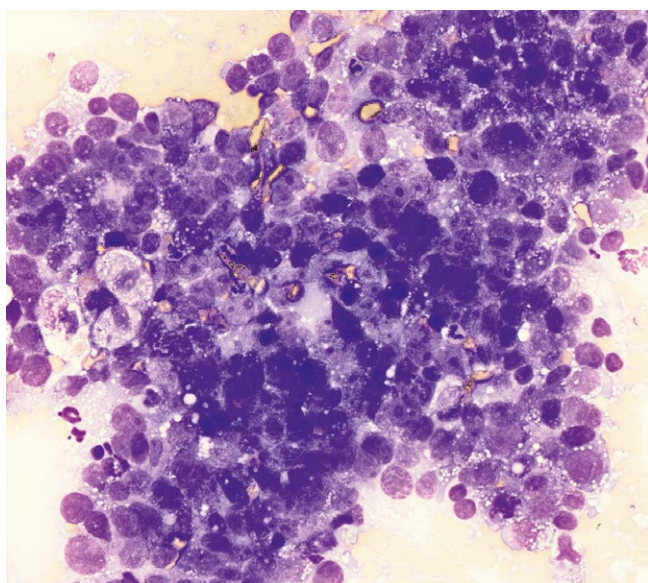


Fig. 1-127 Metastatic pancreatic adenocarcinoma in the liver of an 8-year-old male Labrador retriever with increased liver enzyme activities, hyperbilirubinemia, hypoproteinemia, and hypoalbuminemia. Pancreatic adenocarcinoma with hepatic, intestinal, lymph node, and mesenteric metastases was diagnosed 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).

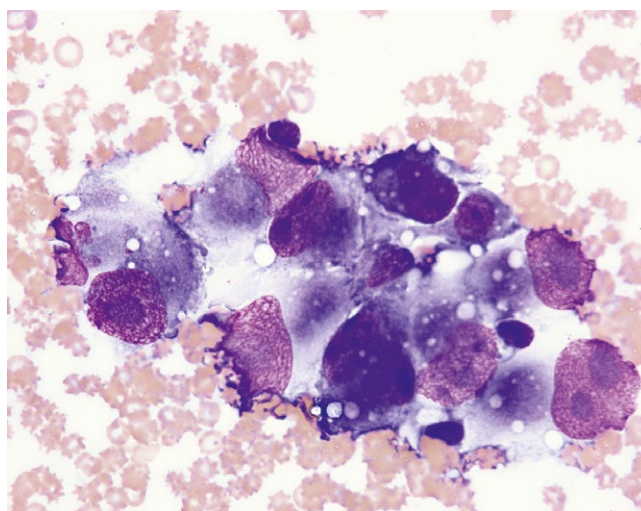


Fig. 1-128 Hemangiosarcoma in the liver of a 10-year-old male German shepherd dog with a splenic mass, enlarged and ultrasonographically heterogeneous liver, and increased liver enzyme activities

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

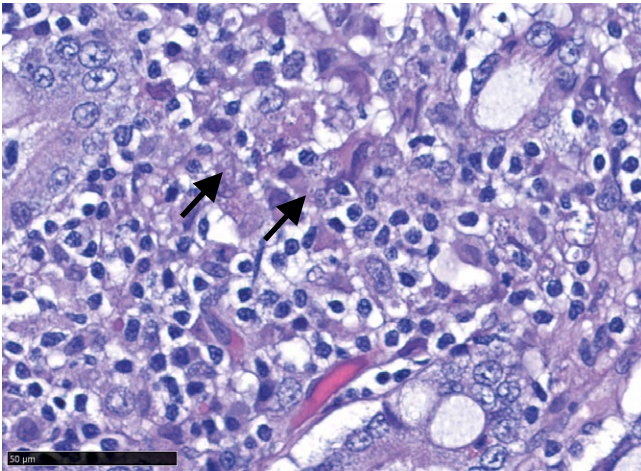


Fig. 1-148 *Histoplasma* spp. infection in the colon of a dog. Macrophages and lymphocytes infiltrate the lamina propria. The cytoplasm of macrophages occasionally contains round, 2 µm-diameter yeasts surrounded by a clear halo (arrows) (H&E stain).

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}

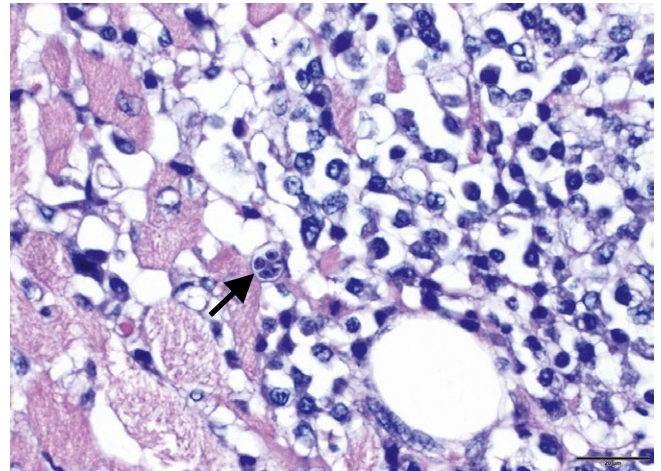


Fig. 1-149 *Prototheca* spp. infection in the colon of a dog. Macrophages, plasma cells, and lymphocytes infiltrate the external muscularis layer. A 7 µm-diameter, oval alga with endospores (arrow) is seen within the inflammation (H&E stain).



In endemic areas 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

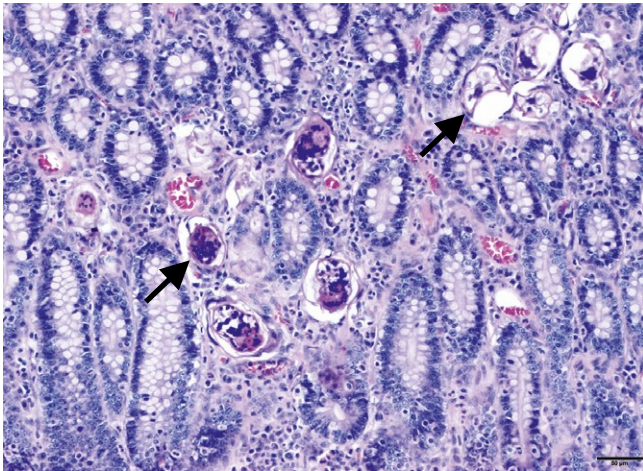


Fig. 1-150 *Heterobilharzia americana* infection in the jejunum of a dog

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 μm 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 approxi-

mately 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* O157: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).

Tyzzler'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	<ul style="list-style-type: none"> Equipment widely available Survey and contrast studies allow the identification of mechanical GI obstructions. Survey films may allow diagnosis of severe adynamic ileus 	<ul style="list-style-type: none"> 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	<ul style="list-style-type: none"> Equipment widely available Qualitative: rapid assessment of gastric and small intestinal motility Quantitative: accurate, real-time assessment of actual GE and gastric motility 	<ul style="list-style-type: none"> 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	<ul style="list-style-type: none"> 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	<ul style="list-style-type: none"> 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	<ul style="list-style-type: none"> Can be used in the home environment WMC: Pressure curves and motility index provide information on motility VEC: easily available 	<ul style="list-style-type: none"> 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 dogs¹¹ 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, ¹¹¹Indium 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

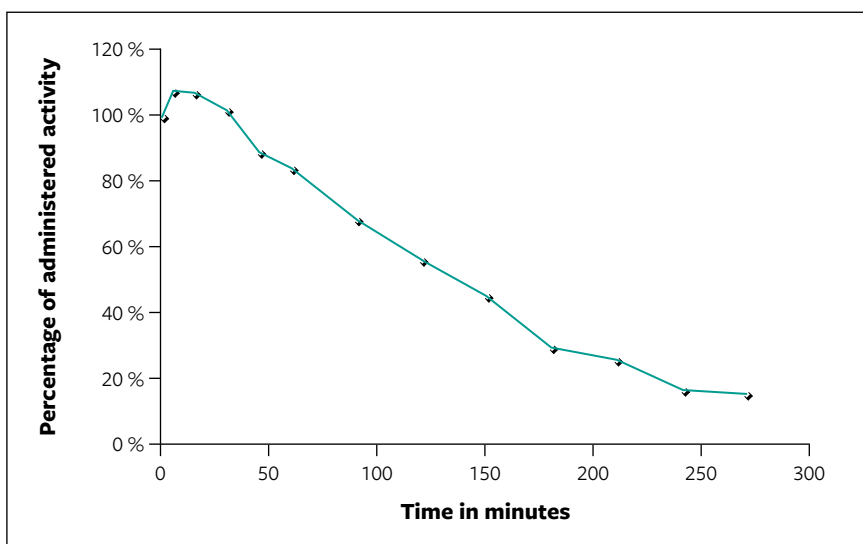


Fig. 1-178 Gastric emptying curve obtained by radionuclide scintigraphy in a healthy dog

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 B₁₂) and folate (vitamin B₉), 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.

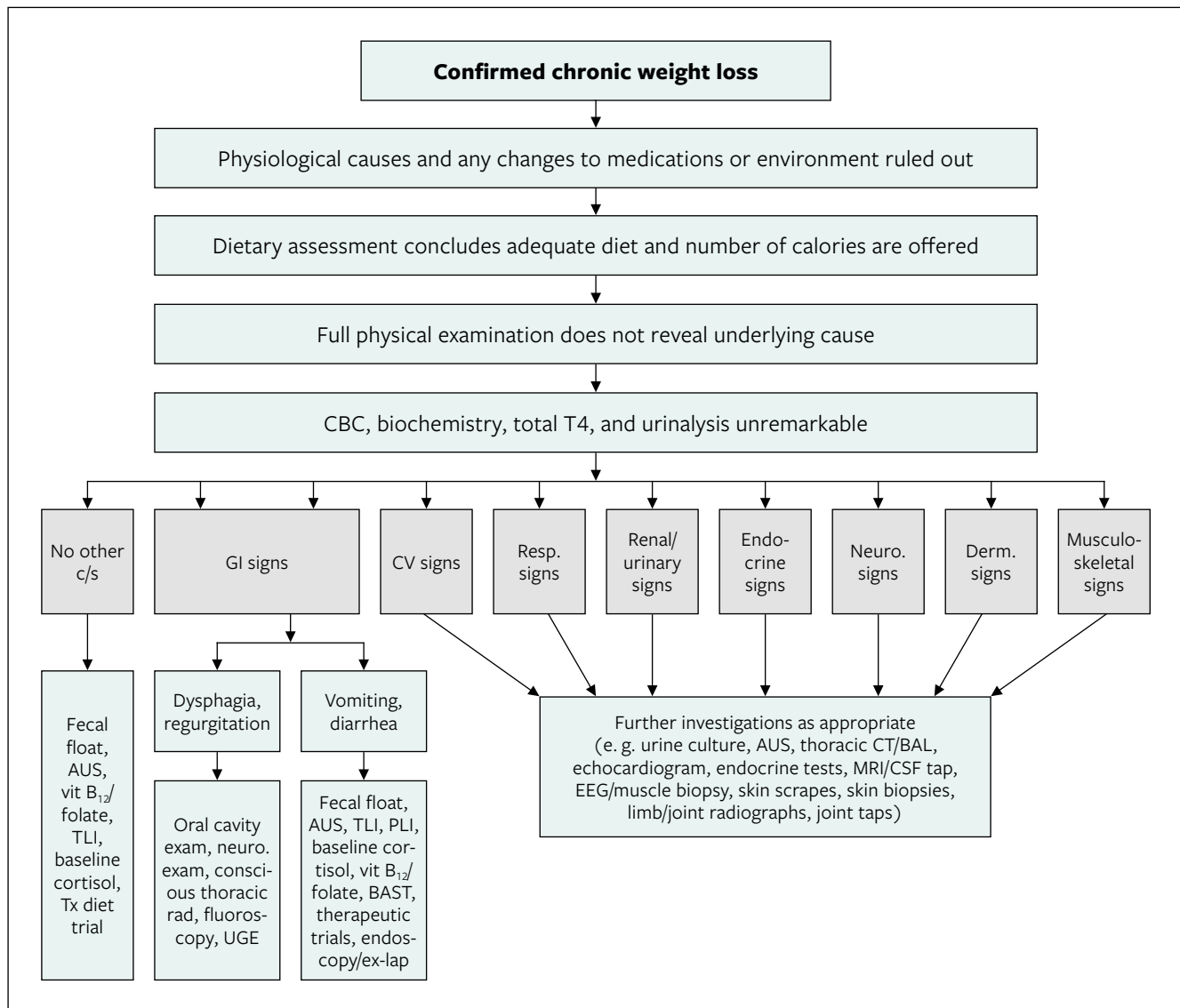


Fig. 2-6 An algorithm for the diagnostic investigation of chronic weight loss in dogs and cats

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 (\pm 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:8080/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)			Dog = cat
Lower motor neuron/spinal cord disease L4-S2: <ul style="list-style-type: none"> • Cauda equina, intervertebral disc herniation • Sacral spinal cord deformity • Neoplasm 			<ul style="list-style-type: none"> • Dog >> cat • Cat (Manx) • Dog >> cat
Dysautonomia (Key-Gaskell syndrome)			Cat >> dog
Colic ganglioneuritis		Dog = cat	
Endocrine		<ul style="list-style-type: none"> • Hypothyroidism • (post-treatment for hyperthyroidism) 	<ul style="list-style-type: none"> • 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.



CLINICAL PEARLS

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 post-mortem 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.

4 Oral cavity

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 H₁-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](https://vetline.de/svg.to/9842-04-02-02):
[svg.to/9842-04-02-02](https://vetline.de/svg.to/9842-04-02-02)



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.

Sialoceles, 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

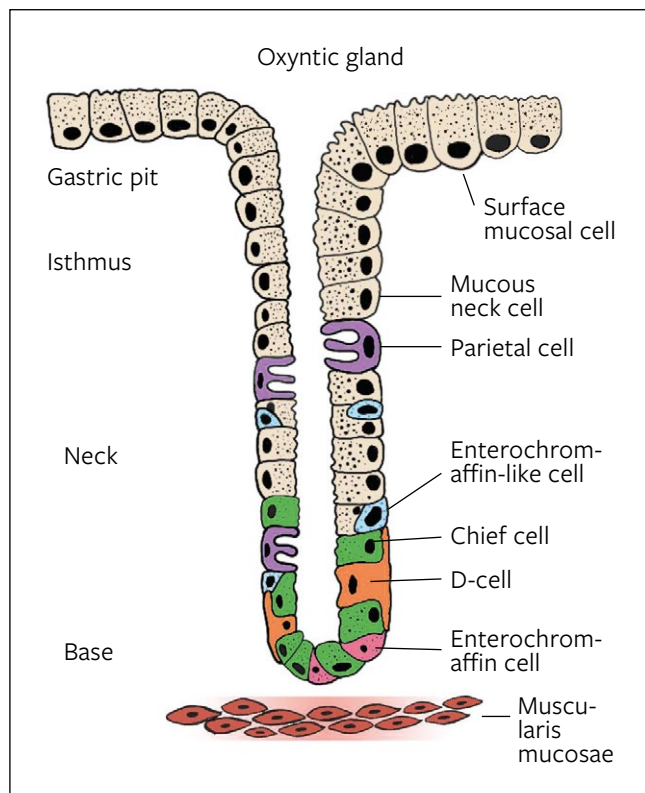


Fig. 6-2 Structure of a gastric fundic gland

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

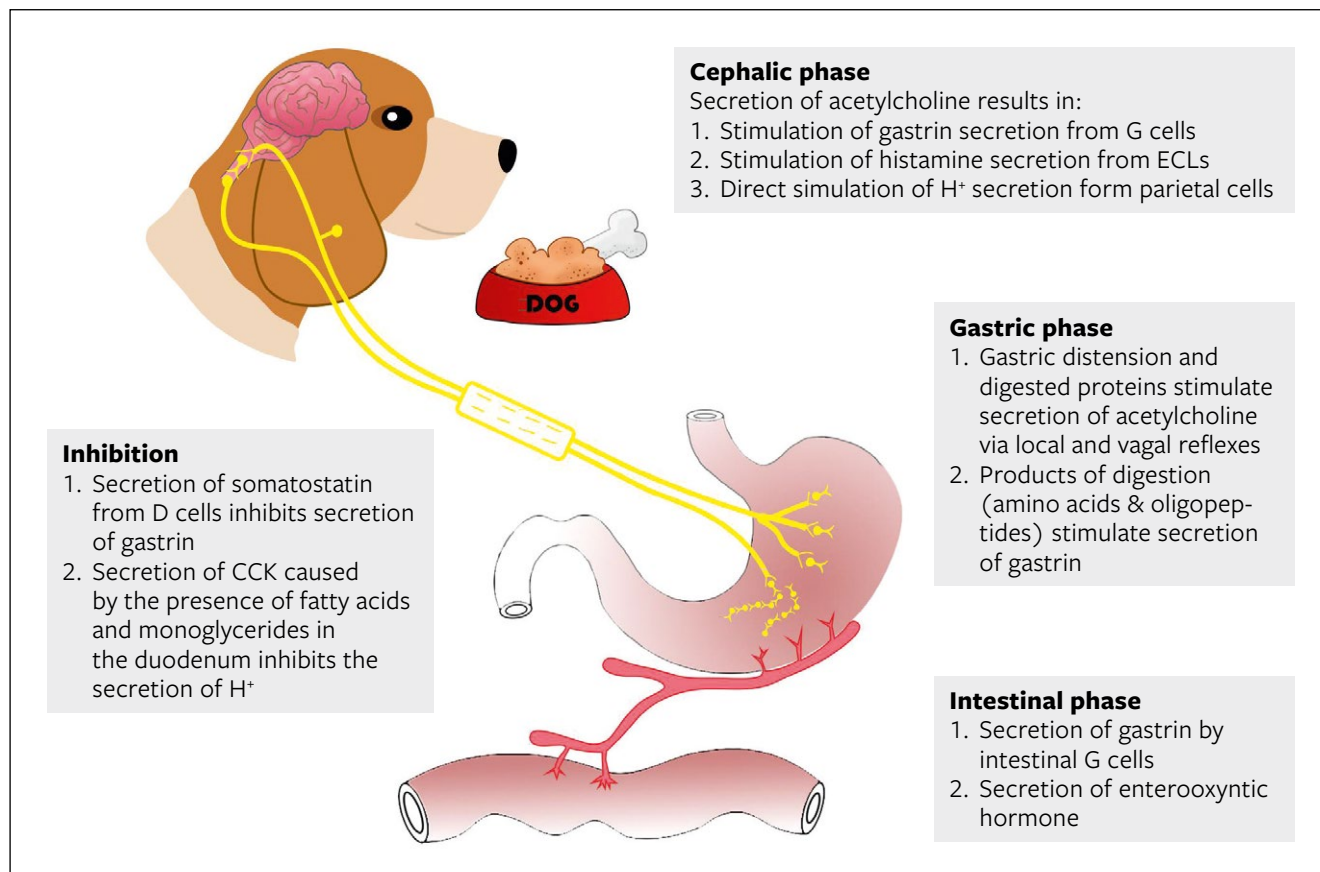


Fig. 6-3 Regulation of gastric acid secretion

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 secretion 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 enteroxyntic 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

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 <i>Toxocara canis</i> 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
<i>Giardia</i> 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

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).



CLINICAL PEARLS

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 **anthelmintic 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

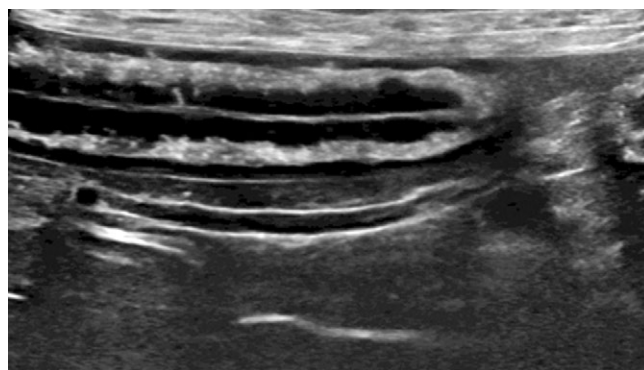


Fig. 7-14 Ultrasonographic image showing thickening of the submucosal layer of the small intestinal wall with pinpoint hyperechoicities from a dog with *H. americana* infection (Courtesy of Texas A&M Radiology Department, College Station, TX.)

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 anthelmintic 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

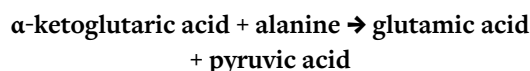
brush border, where they undergo solubilization of fatty acids and monoglycerides into mixed micelles.⁶ The mixed micelles then diffuse through the epithelial cell membranes into the intestinal mucosal cells. Within the epithelial cells, free fatty acids and monoglycerides reassemble to form triglycerides, 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:



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

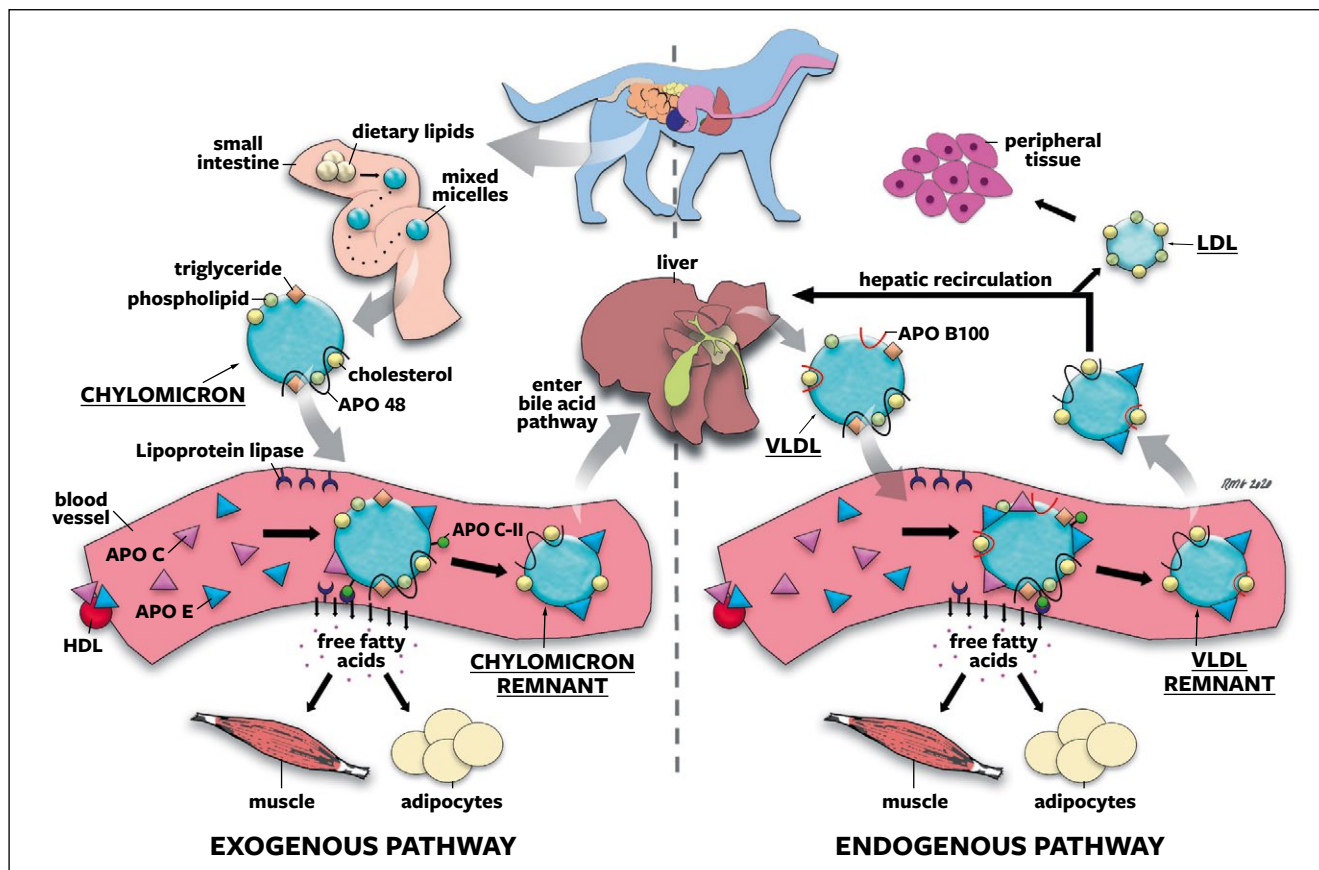


Fig. 9-5 Lipid metabolism

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 membrane²⁰

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.

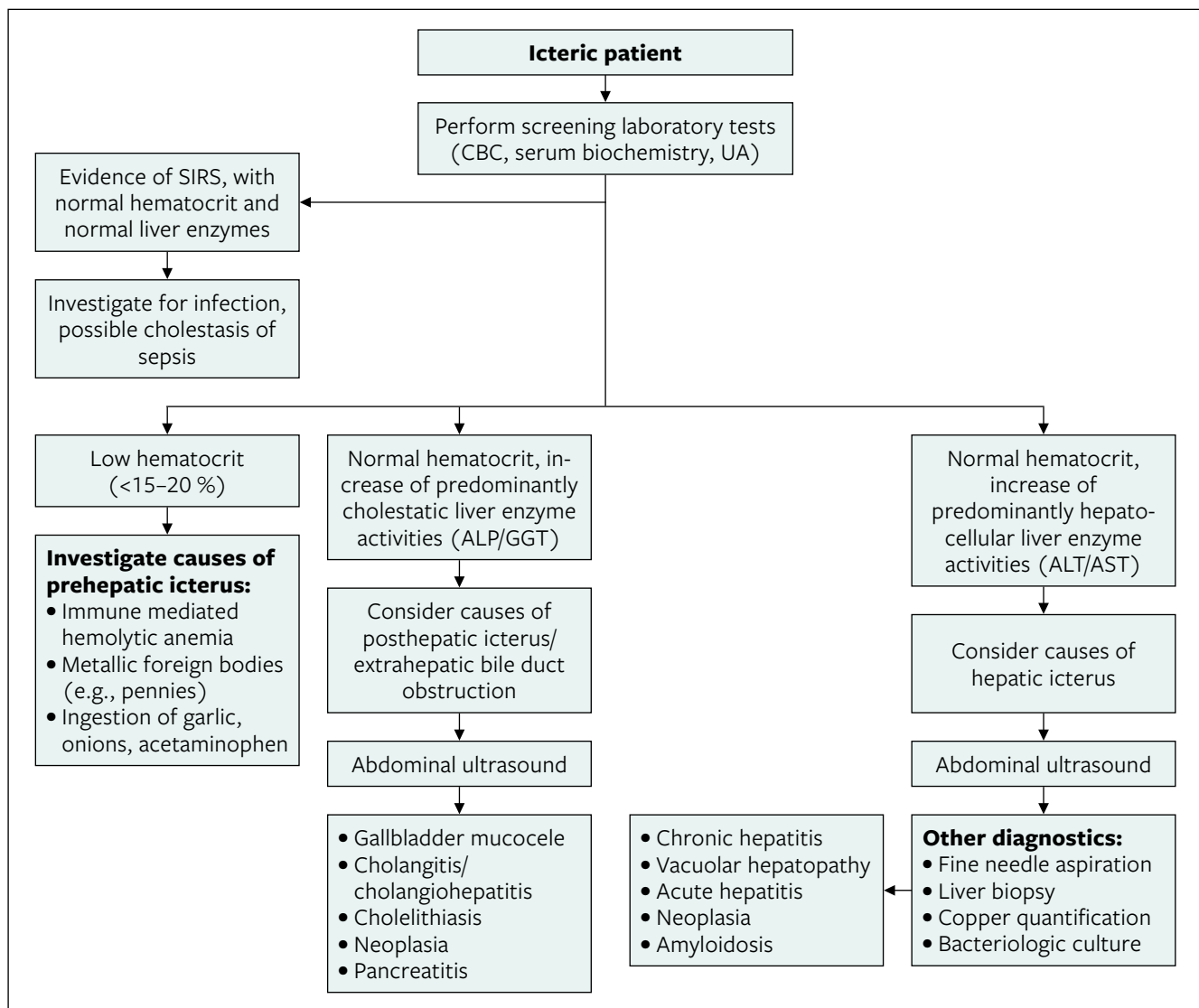


Fig. 9-7 Approach to an icteric patient

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

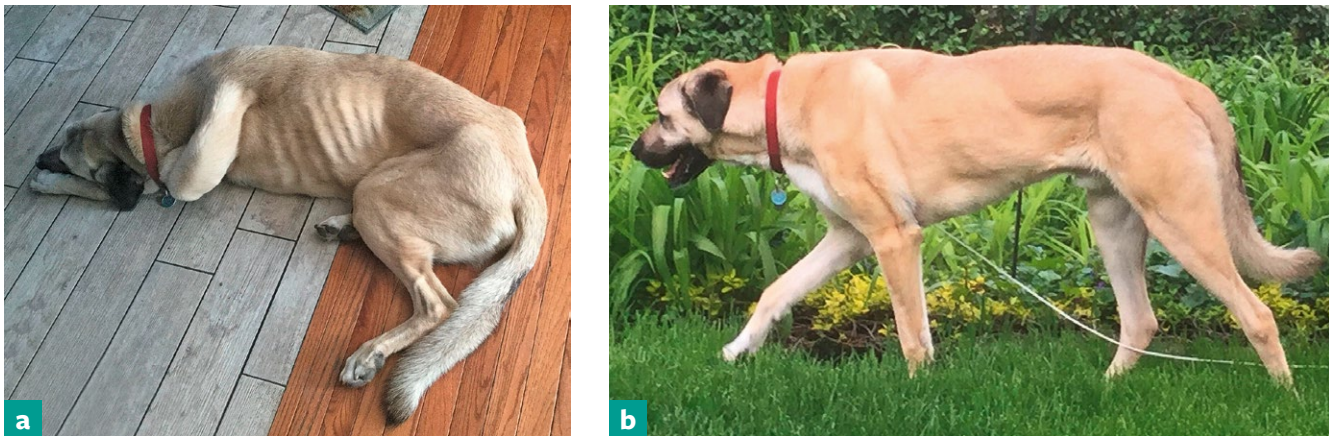


Fig. 10-15 Exocrine pancreatic insufficiency in a dog

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,

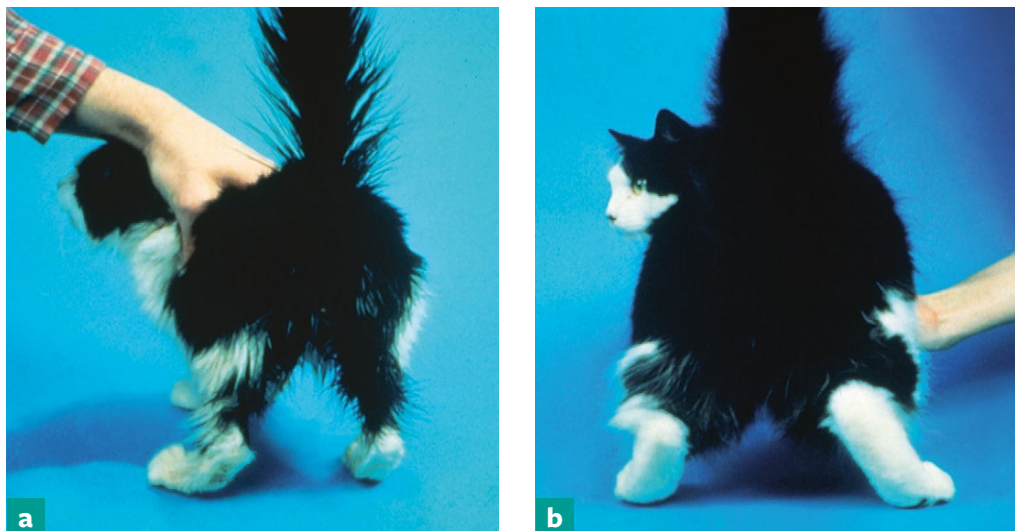


Fig. 10-16 Exocrine pancreatic insufficiency in a cat

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.

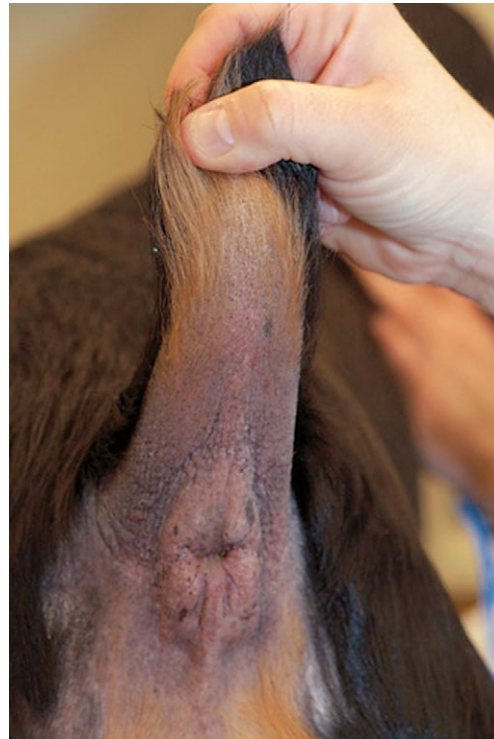
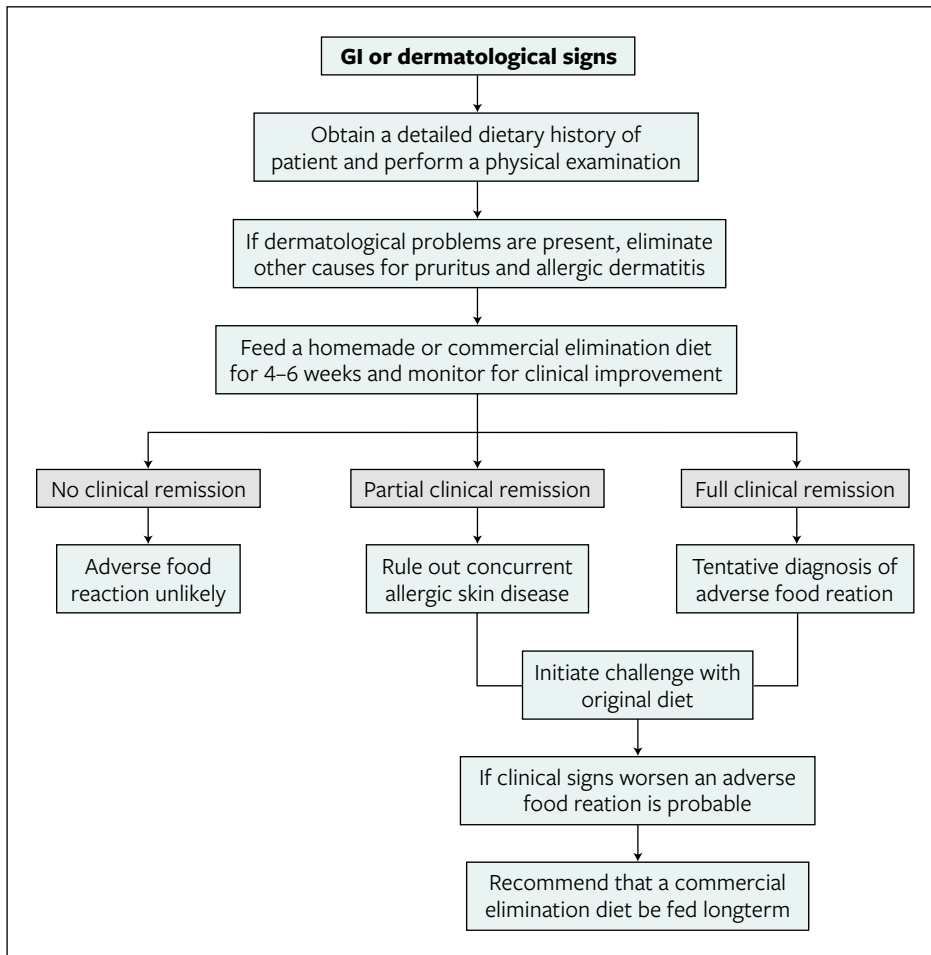


Fig. 11-4 Adverse food reaction

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. 2011²²)

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

Index

1,4- α -glucan 6-glucanotransferase defect 437
5-aminosalicylic acid (5-ASA) products 549
5-HT4 serotonergic agonists 197

A

AAFCO see Association of American Feed Control Officials 216
ABCB4 gene defect 478
Abdomen/Abdominal
– acute 22
– auscultation 23
– distension 21
– protein-losing enteropathy (PLE) 342
– effusion 21
– examination 21
– fluid 21
– malignant tumors 21
– pain 15
– cranial 22
– exocrine pancreatic neoplasia 524
– palpation 22
– pancreatitis 504, 510
– posture 17
– palpation 21
– ultrasonography
– aminoaciduric canine hypoaminoacidemic hepatopathy syndrome (ACHES) 449
– congenital portosystemic shunt (CPSS) 423
– gallbladder mucocele 479
– gastrointestinal motility 163
– ileus 75
– jaundice 200
– pancreatitis 505
– protein-losing enteropathy (PLE) 344
Abdominocentesis 350, 381
Aberrant vessels 84
Abscess
– oropharyngeal 59
– pancreatic 116, 502, 527

Absorption of water and electrolytes 356
Abyssinian
– disease predisposition 5
ACA see Adenocarcinoma 292
Acanthocytes 36
Acanthomatous ameloblastoma (AA) 235
Accessory pancreatic duct 497
Accordion-like small bowel loops 22
Acetaminophen, hepatotoxicity 411
Acetylcholinesterase inhibitors 287, 288
Achalasia
– cricopharyngeal 62
– lower esophageal sphincter 65
Acholic feces 14
Acid alpha-glucosidase defect 437
Acid-base abnormalities 18, 311
Acidophil cell hepatitis 419
Acinar cell(s)
– carcinoma 523
– pancreatic 498
Acotiamide 289
ACTH stimulation test 173
Activated partial thromboplastin time (APTT) 36
Activity-based lipase assay
– cats 39
– dogs 38
Acute abdomen 22
Acute gastrointestinal disease
– clinical signs 167
– differentials 168
Acute hemorrhagic diarrhea syndrome (AHDS) 18, 136, 282, 327
– bloody diarrhea 17
– clinical presentation 328
– clostridium 365
– complications 329
– diagnosis 328
– pathogenesis 327
– prognosis 329
– treatment 328
Acute hepatitis (AH) 452, 453
– diagnosis 453
– treatment 454
Acute kidney injury, pancreatitis 505

Acute phase protein (APP) 30
– chronic inflammatory enteropathy (CIE) 545
Acute signs
– clinical evaluation 167
– diagnostic testing 171
– differentials 168
– patient history 170
– physical examination 171
Acylglycerols 400
Adenocarcinoma (ACA) 144
– gastric 292, 293
– intestinal 348, 351
– rectal 390
Adenomas 143
Adenomatous polyps 379
Adenosine monophosphate kinase activator (AMP kinase activator) 433
Adherent-invasive *E. coli* (AIEC) 318
Adipositas 573
Adverse food reactions (AFR) 210, 212, 327, 531, 532
– clinical signs 532
– diagnosis 534
– elimination diet 534
– etiopathogenesis 532
– prognosis 536
– terminology 531
– treatment 534
– types 531
Aflatoxins 413
Aganglionic megacolon 375
AGASACA see Apocrine gland anal sac adenocarcinoma 391, 393
AHDS see Acute hemorrhagic diarrhea syndrome 18
Air biting 17
Airway-esophageal fistula 257
Alanine aminotransferase (ALT) 32
– elevation 32
– plasma half-life 33
Albumin 36
– pancreatitis 505, 512
Algae infections 325
ALICAM[®] capsule 165

- Alimentary lymphoma (AL) 536
- Alkaline phosphatase (ALKP) 33
- elevation 32
 - plasma half-live 33
- Alopecia 20
- adverse food reaction 533
 - hepatic neoplasia 468
 - paraneoplastic 20, 524
- Alosetron 370
- Alpha-1-antitrypsin 446
- Alpha-1-protease inhibitor (α_1 -PI)
- deficiency 446
- Alpha-1-proteinase inhibitor (α_1 -PI) 29
- Alpha-1-proteinase inhibitor (α_1 -PI) 499
- Alpha-2-macroglobulin (α_2 -macro-globulin) 499
- ALT see alanine aminotransferase 32
- Alveolar bone 227
- Amatoxins 413
- Ameloblastoma 235
- Ameroid constrictor 426
- Amino acids, decreased 449
- Aminoaciduric canine hypoaminoacidemic hepatopathy syndrome (ACHES) 448
- clinical examination 449
 - diagnosis 449
 - prognosis 450
 - treatment 450
- Amino transamination 400
- Ammonia 35, 36
- hepatic encephalopathy (HE) 408
 - removal 400
 - sample requirements 35
 - tolerance test 36
- Ammonium urate uroliths/crystals 36
- Amoebiasis 141
- Amoxicillin-clavulanate 458, 459, 460
- Amphimerus pseudofelineus 492
- pancreas 502, 528
- Ampicillin, hepatic encephalopathy (HE) 409, 410
- Amylin 503
- Amylo-1,6-glucosidase defect 437
- Amyloidosis 125, 146, 444
- diagnosis 444
 - hepatic 150, 444
 - hereditary 52
 - pathophysiology 445
 - prognosis 446
 - treatment 445
- Anaerobiospirillum 319
- Anal furunculosis 386
- Analgesia
- pancreatitis 510
- Anal pruritus 15
- Anal sacculectomy 387
- Anal sac tumors 390
- Anaphylactoid food reaction 531
- Ancillary procedures 116
- Anemia
- chronic hepatitis 456
- Angiodysplasia 146
- Anorectum 353
- diseases 382
- Anorexia
- exocrine pancreas neoplasia 524
 - pancreatitis 504
- Antacids
- pancreatitis 513
- Anthelmintic treatment
- gastritis 273
 - small intestinal disease 323
- Antibiotic-responsive diarr-rhea (ARD) 537, 542
- Antibiotic-responsive enteropathy (ARE) 188
- vomiting 177
- Antibiotics
- cholangitis 488
 - chronic inflammatory enteropathy (CIE) 545, 549
 - dysbiosis 334
 - pancreatitis 512
 - parvovirus enteritis 313
- Anticholinergics 370
- Anticoagulant factors 403
- Antiemetics 274
- gastritis 273
 - pancreatitis 511
 - parvovirus enteritis 313
- Antigen exposure list 205
- Antigen-presenting cells (APCs) 358
- Antigens 304
- Antihormonal therapy, insulinoma 566
- Antihypoglycemic therapy, insulinoma 565
- Antimicrobial therapy
- chronic hepatitis 458, 459, 460
- Antimicrobial therapy see also anti-biotics 458, 459, 460
- Antioxidants 513
- Antiparasitic agents 322
- Antiperistalsis 355
- Antithrombin 185
- Antithrombotics 550
- Anti-TNF- α monoclonal antibody therapy 550
- Antral mucosal hypertrophy 104
- Apocrine gland anal sac adenocarcinoma (AGASACA) 390
- clinical presentation 391
 - diagnosis 391
 - incidence 390
 - prognosis 393
 - treatment 392
- Apolipoprotein C (APO-C) 400
- Apoprotein (Apo) C 401
- Apoptosis 150, 452
- Appetite stimulants 511
- Aprotinin 512
- Arching of the back 17
- Arginine deficiency 448
- Arteriovenous fistula/malformation, hepatic 461, 465
- Ascarids 136
- Ascending colon 353
- Ascites 407
- chronic hepatitis 458, 459, 460
 - drugs 409, 410
 - hepatitis 453
 - imaging 83
 - medication 458, 459, 460
- Ascomycota 357
- Aspartate aminotransferase (AST) 33
- elevation 32
 - plasma half-live 33
- Aspergillus 357
- Aspiration pneumonia 18
- Aspirin 550
- Association of American Feed Control Officials (AAFCO)
- nutrient minimums 216
 - nutritional adequacy statement 204
- ATP-binding cassette transport proteins (ABC transporters) 411
- Atresia
- ani 193, 385
 - types 385
 - coli 52
- Atrophic gastritis 276
- Auscultation, abdominal 23
- Australian cattle dog, disease predisposi-tion 4, 5
- Autodigestion 499
- Autonomic nervous system 75, 354
- Azathioprine 502
- chronic inflammatory enteropathy (CIE) 549
 - hepatotoxicity 412
 - immune-mediated hepatitis 458, 459, 460
- Azithromycin 289
- motility disorders 287, 288
- Azotemia
- diarrhea 185

- B**
- Babesia canis* 502
- Bacteria(l) 330
- aerobic 357
 - anaerobic 357
 - culture 333
 - folic acid 333
 - gastrointestinal 356
 - infection 315
 - cholecystitis 483
 - hepatitis 418, 419
 - pancreas 502
 - intestinal 304
- Bacteroides 357
- Bacteroidetes 356
- Bactibilia 128, 455, 488
- Balloon-assisted foreign body removal 246, 248
- Balloon catheter 109
- Balloon dilatation
- cricopharyngeus muscle dysfunction (CPMD) 243
 - esophageal stricture 250, 253, 254
 - esophagostomy tube 254
- Balsalazide 549
- Barium contrast studies 58
- Barium enema 354
- Barium esophagram 66
- Barium-impregnated polyethylene spheres (BIPS) 25, 284
- Barium swallow 61
- Barrett's esophagus 264
- Bartonella 418
- Basenji
- disease predisposition 4, 5
 - lymphoproliferative enteropathy 540
- Beagle, disease predisposition 4, 5
- Bedlington terrier
- copper-associated hepatopathy 404, 439
 - disease predisposition 4, 5
- Beef protein, adverse reaction 533
- Behavioral alterations, obesity 576
- Behavioral modification, irritable bowel syndrome (IBS) 370
- Belgian shepherd, disease predisposition 4, 5
- Bentiromide absorption (BT-PABA) test 44
- Betanechol, motility disorders 287, 288
- Bezafibrate 436
- Bicarbonate deficiency 518
- Biclonal profile 55
- Bifidobacteria* 304, 371
- Bile 398
- cytology 128, 485, 488
 - drainage 489
 - pigment 397
 - salts 400, 402
 - secretion 401
 - vomiting 7
- Bile acid 34, 330, 401
- binding drugs 340
 - conversion 357
 - metabolism 402
 - primary 402
 - secondary 402
 - stimulation test 35
- Bile duct 399
- hyperplasia 452
 - obstruction 117
- Biliary adenoma 155
- Biliary carcinoma 155, 495
- clinical signs 468
- Biliary cysts 467
- Biliary disorders 152
- Biliary neoplasms 495
- diagnostic evaluation 496
 - prognosis 496
 - staging 496
 - treatment 496
- Biliary obstruction 89
- Biliary sludge 87, 478
- Biliary system
- histology 152
 - inflammation 153
 - parasites 492
- Biliary tract 395
- anatomy 398
 - dilation 488
 - disease 471
 - non-obstructive 85
 - obstructive 89
 - imaging 81
 - interventional procedures 91
 - physiology 400
 - structure 398
- Bilirubin 34, 200
- cholelithiasis 491
 - conjugated 34
 - metabolic alterations 407
 - mucous membranes 19
 - non-conjugated 34
 - urinary 36
- Bioimpedance 585
- Biomarkers of inflammatory cell activity 30
- Biopsy 98
- colonic 100
 - constipation 194
 - duodenal 99
 - forceps 100, 101
 - gastric 99
 - tumors 296
 - handling tissue samples 101
 - hepatic 91, 113
 - ileal 100, 546
 - intestinal 112
 - chronic inflammatory enteropathy (CIE) 546
 - laparoscopic 112
 - pancreatic 91, 114, 157, 508
 - rectal 100
- Bisacodyl 377
- Black-pigment choleliths 490
- Bleeding
- diathesis 19
 - disorders 408
 - superficial 19
- Blended canned diet 220
- Blood cultures 172
- Blood glucose concentration
- hypoglycemia 560
 - insulinoma 561, 563
- Blood loss
- gastrointestinal 17
 - pale mucous membranes 19
- Blood products, parvovirus enteritis 312
- Blood stool 14
- Blue-green algae (BGA) 413
- Body composition assessment 585
- Body condition score (BCS) 190, 203, 583
- cats 204
 - dogs 204
 - ideal 204
- Body mass index (BMI) 573
- Body temperature 17
- Body weight
- measuring 16, 190, 203, 582
 - obesity 574
- Bone marrow infection, parvoviral 308
- Borborygmus 14, 23
- Border collie, disease predisposition 4, 5
- Boston terrier, disease predisposition 4, 5
- Botulinum toxin injection, cricopharyngeus muscle 243
- Bouvier des Flandres, disease predisposition 4, 5
- Bowel
- movements, diarrhea 7
 - palpation 22
 - wall infiltration, diffuse 78
- Bowel see also small/large intestine 353
- Boxer, disease predisposition 4, 5
- Brachycephalic breeds, disease predisposition 4, 5
- Bradycardia 18

- Breath
- odor 21
 - tracer 165
- Breed predispositions
- cats 5
 - dogs 4, 5
 - obesity 575
- Bromotyrosine (3-BrY) 30
- Bronchoesophageal artery 240
- Brown-pigment choleliths 490
- Bruxism 15
- Buccal carcinoma 236
- Budesonide
- chronic inflammatory enteropathy (CIE) 548
 - gastritis 279
- Bulk-forming laxatives 377
- B vitamin supplementation 548
- C**
- Cachexia 16, 188
- Cairn terrier
- congenital esophageal fistulae 257
 - disease predisposition 4, 5
- Calcineurin inhibitors 388
- Calcium
- pancreatitis 502, 505
 - soaps 503
- Calcium bilirubinate 490
- Calgranulin C 30
- Caloric intake, estimating 205
- Calprotectin 30
- fecal 545
- Campylobacter 317
- Candida 357
- Canine acute pancreatitis severity (CAPS) 515
- Canine adenovirus type 1 (CAV-1), hepatitis 414, 415
- Canine chronic enteropathy clinical activity index (CCECAI) 9, 185
- Canine coronavirus 307
- Canine distemper virus (CDV) 306
- Canine enteric coronavirus 136
- Canine granulocyte-colony stimulating factor (G-CSF) 313
- Canine histiocytic ulcerative colitis (HUC) 138
- Canine inflammatory bowel disease activity index (CIBDAI) 8, 185
- Canine inherited disorders database 45
- Canine pancreatic lipase immunoreactivity (cPLI) 38, 39
- Canine pancreatic lipase, parvovirus infection 311
- Canine parvovirus (CPV) 305, 308
- Canine trypsin-like immunoreactivity (cTLI)
- EPI 519
 - pancreatic neoplasia 524
- Cannabinoid type 2 receptor agonists 289
- Canned blended diet 220
- Capillary refill time (CRT) 19
- Capromorelin 289, 303, 511
- Capsule endoscopy 110
- GI motility 165
 - vomiting 181
- Carbapenems 364
- Carbimazole, hepatotoxicity 412
- Carbohydrate metabolism 400
- Carboplatin 525
- Carcinoid 145, 156
- gastrointestinal 572
- Carcinoma
- gastric 291, 292
 - in situ 144, 292, 379
 - intestinal 348
- Cardiac glands 267
- Caroli's disease 465, 466
- Cathepsin B 503
- Cavalier King Charles spaniel, disease predisposition 4, 5
- Cecocolic orifice 353
- Cecum 98, 353
- Celiotomy, exploratory 546
- Cell-mediated immunity 358
- Cementum 227
- Cestodes 137
- CEUS see Contrast-enhanced ultrasonography 58
- Chemotherapy 381
- exocrine pancreatic neoplasia 525
 - hepatic neoplasia 471
 - insulinoma 565
 - intestinal neoplasia 350
 - lymphoma 554, 556
 - oral tumors 237
 - rectal tumors 392
- Chicken protein, adverse reaction 533
- Chlorambucil
- chronic inflammatory enteropathy (CIE) 549
 - lymphoma 554
- Chlordiazepoxide 370
- Cholangiocellular adenoma 155, 495
- Cholangiocellular carcinoma 126, 155, 495
- staging 496
- Cholangiocytes 397
- Cholangiohepatitis 451
- ascending 477
 - imaging 82, 85
- Cholangitis 451, 482, 485
- chronic 486
 - clinical findings 487
 - destructive 155, 486, 487
 - diagnostic evaluation 488
 - etiopathogenesis 486
 - imaging 85
 - liver flukes 489, 492
 - lymphocytic 154, 486, 487, 489
 - neutrophilic 154, 486, 488
 - parasitic 154
 - prognosis 488, 489
 - therapy 488
 - types 486
- Cholecystectomy
- cholecystitis 484
 - extrahepatic bile duct obstruction (EHBO) 475
- Cholecystitis 155, 482
- chronic 91, 483, 484
 - clinical findings 483
 - diagnostic evaluation 483
 - emphysematous 86
 - etiopathogenesis 482
 - imaging 85
 - medical management 485
 - sonographic appearance 484
 - surgery 484
- Cholecystocentesis 116, 117
- Cholecystoduodenostomy 475, 477
- Cholecystoenterostomy 472, 475, 476
- complications 477
- Cholecystography 116
- Cholecystojejunostomy 474, 475
- Cholecystokinin (CCK) 34, 269, 302, 402, 498, 499
- function 558, 559
 - pancreatitis 513
- Cholecystolithiasis 483
- Cholecystostomy tube 472
- complications 474
 - laparoscopic placement 473
 - techniques 472
- Cholecystotomy 475
- choledocholithiasis 491
 - cholelithiasis 491
 - gallbladder mucocele 480, 481
- Choledochal cysts 467
- Choledochal stenting 474
- Choledocholithiasis 86, 200, 490
- clinical findings 491
 - diagnostic evaluation 491
 - etiopathogenesis 490
 - medical management 491
 - surgery 491

- Choledochotomy 475
- Cholelithiasis 86, 89, 91, 200, 475, 490
 - cholecystitis 482
 - clinical findings 491
 - diagnostic evaluation 491
 - etiopathogenesis 490
 - medical management 491
 - surgery 491
- Cholescintigraphy 89
- Cholestasis 34, 36, 124, 453
 - extrahepatic 89, 153
 - intrahepatic 152
- Cholestatic disease, diarrhea 408
- Cholestatic markers 33
- Cholesterol 34, 400
 - sample requirements 35
- Cholestyramine 340
- Chow chow, disease predisposition 4, 5
- Chronic enteropathy (CE) 282, 536
- Chronic hepatitis (CH) 452, **454**
 - bacterial causes 418
 - breed predispositions 404
 - clinical pathology 455
 - clinical signs 455
 - complications 458, 459, 460
 - copper-associated **454**
 - diagnostic imaging 456
 - drug-induced 455
 - histopathology 456
 - immune-mediated 455
 - infectious 455
 - leptospirosis 417
 - medications 458, 459, 460
 - nutrition 458, 459, 460
 - prognosis 458
 - signalment 455
 - treatment 458
 - viral causes 419
- Chronic hypertrophic gastritis 132
- Chronic hypertrophic pyloric gastropathy 133
- Chronic inflammatory enteropathy (CIE) 30, 304, 536
 - adjunctive treatment 550
 - antibacterial therapy 549
 - canine 539
 - classification 540
 - clinical presentation 543
 - clinical signs 543
 - diagnosis 544
 - diagnostic imaging 545
 - diet 547
 - differentiation of LGAL 56, 57
 - duodenal 135
 - empirical treatment trials 545
 - endoscopic appearance 546
 - etiopathogenesis 537
 - fecal examination 544
 - genetic predisposition 538
 - granulomatous colitis 361
 - idiopathic 540
 - intestinal biopsy 546
 - intestinal microbiota 537
 - intestinal mucosal immune system 538
 - laboratory tests 544
 - novel therapies 550
 - pancreatitis 514
 - physical examination 544
 - prognosis 550
 - signalment 543
 - supplements 547
 - surgery 550
 - treatment 547
- Chronic inflammatory hepatic disease 83
- Chronic kidney disease (CKD)
 - classification 574
 - vomiting 175
- Chronic pyloric obstruction 69
- Chylomicrons 400, 401
- Ciclosporin see cyclosporine 549
- Cimetidine 340, 413
- CIRMD critical illness related motility disorder 336
- Cirrhosis 83, 152, 453
- Cisapride 197, 286, 378
 - esophagitis 244
 - GERD 245
 - motility disorders 287, 288
- Cleft lip/palate 20
- Clidinium bromide 370
- Clinical evaluation/examination
 - acute signs 167
 - constipation 192
 - diarrhea 184
 - jaundice 198
 - weight loss 188
- Clinical history 3
- Clonorchis sinensis 495
- Clopidogrel 550
- Clostridioides*
 - *difficile* 136, 315
- Clostridium* 357
 - *difficile* 315, 357
 - *hiranonis* 330, 332
 - *perfringens* 316, 365
 - enterotoxigenic 359, 365, 366
 - type A 142, 327
 - *piliforme* 141, 319
- Coagulation factors 36, 403
- Cobalamin 26, 552
 - absorption 27
 - deficiency 26, 28, 521
 - malabsorption 548
 - small intestinal microbiota 333
 - supplementation 335
- Coccidia 137
- Coccidiosis 141
 - biliary 492
- Cocker spaniel, disease predisposition 4, 5
- Colectomy
 - constipation 197
 - megacolon 378
- Colipase 498
- Colitis
 - acute 137, 359
 - chronic 359
 - clinical signs 543
 - diarrhea 81
 - eosinophilic 138
 - granulomatous 318, 361
 - histiocytic ulcerative 362
 - idiopathic 137
 - nervous 369
 - perianal fistula 387
 - pyogranulomatous 138
- Colloid therapy 312
- Colon
 - abdominal palpation 22
 - anatomy 353
 - biopsy 100
 - blood supply 353
 - cell-mediated immunity 358
 - constipation 192
 - dilatation 80
 - endoscopic findings 106
 - enlarged 372
 - epithelial cells 356
 - immune function 358
 - impaction 22, 374
 - inertia 373
 - innervation 354
 - microbiome 356
 - mucosa 356, 369
 - neoplasia 81
 - spastic 369
 - torsion 80
- Colonoileoscopy 94, 97
- Colonoscopy
 - constipation 194
 - flexible 98
 - large intestinal neoplasia 381
 - rigid 97
 - with biopsy 187
- Colopexy, rectal prolapse 384
- Colorectal cancer (CRC)
 - clinical presentation 380
 - diagnostic evaluation 381
 - incidence 380
 - prognosis 382
 - staging 381
 - treatment 381
- Commercial foods 15

- Common bile duct 399
 - Common mucosal immune system 303
 - Compensatory hyperventilation 19
 - Computed tomography angiography (CTA)
 - congenital portosystemic shunt (CPSS) 423
 - Computed tomography (CT) 58
 - hepatic neoplasia 468
 - Congenital hepatic fibrosis (CHF) 465
 - Congenital portosystemic shunt (CPSS) 421, 461
 - breed predispositions 404
 - clinical signs 421, 422
 - complications of surgery 427
 - diagnosis 422
 - diagnostic imaging 423
 - differential diagnosis 423
 - extrahepatic 421
 - intrahepatic 421
 - laboratory investigations 422
 - liver function tests 422
 - outcome 428
 - postoperative mortality 428
 - prognosis 428
 - treatment 425
 - Congenital vascular disorders 461
 - Constipation 15, 372
 - causes 193
 - clinical evaluation 192
 - definition 192
 - diagnostic imaging 194
 - diet 209, 211
 - differential diagnoses 192
 - feeding plan 212
 - history 193
 - laboratory testing 194
 - pathophysiology 192
 - physical examination 193
 - prevention 376
 - prognosis 197
 - radiography 374, 375
 - recurrent 195
 - signalment 193
 - tissue biopsy 194
 - treatment 194, 375
 - Continuous rate infusion (CRI) 214
 - Contrast-enhanced abdominal CT
 - pancreatitis 506
 - Contrast-enhanced ultrasonography (CEUS) 58
 - pancreatitis 506
 - Copper
 - accumulation 151, 404
 - chelators 442
 - granules 398
 - hepatic 397
 - Copper-associated chronic hepatitis 454
 - breed predispositions 404
 - Copper-associated hepatopathy
 - cats 443
 - diagnosis 440
 - dogs 439
 - prognosis 443
 - treatment 441
 - Copper-restricted diet 442
 - Copper-storage hepatopathy 458, 459, 460
 - Coprophagia 15
 - Coronavirus
 - canine 307
 - diarrhea 301
 - feline 307
 - Corticosteroids
 - cholangitis 489
 - gastritis 279
 - serum liver enzyme activities 33
 - Cortisol 565
 - Cow-patty yellowish stools, EPI 11
 - COX-2 inhibiting therapy
 - esophageal tumors 266
 - CPMD (cricopharyngeus muscle dysfunction) 241
 - Cr-51-EDTA 30
 - Cranial abdominal pain 15
 - C-reactive protein (CRP) 25, 30
 - diarrhea 186
 - parvovirus infection 311
 - PLE 343
 - Cricopharyngeal achalasia 62, 241
 - Cricopharyngeal dysphagia 62
 - Cricopharyngeus muscle dysfunction (CPMD) 241
 - diagnosis 241
 - prognosis 243
 - treatment 242
 - Cricopharyngeus muscle dyssynchrony (CPD) 241
 - Critical illness related motility disorder (CIRMD) 282, 336
 - CRT see Capillary refill time 19
 - Crusts 20
 - Cryptosporidium* 134, 137, 320, 321
 - Crypts of Lieberkühn 353
 - CT see Computed tomography 58
 - Cutaneous adverse food reaction (CAFR) 532
 - Cyanobacteria 413
 - Cyanocobalamin 335, 340
 - feline hepatic lipidosis (FHL) 433
 - Cyathospirura* 133
 - Cycad 414
 - Cycasin 414
 - Cyclophosphamide
 - chronic inflammatory enteropathy (CIE) 549
 - Cyclosporine 383
 - chronic inflammatory enteropathy (CIE) 549
 - eosinophilic granuloma complex 232
 - gingivostomatitis 230
 - immune-mediated hepatitis 458, 459, 460
 - pancreatitis 515
 - serum liver enzyme activities 33
 - Cyclosporine A
 - perianal fistula 388, 389
 - Cylicospirura* 133
 - Cyniclomyces* 133
 - Cystic pancreatic tumors 523
 - Cystoisospora* 320, 322
 - Cytochrome P450 (CYP) enzymes 410
 - Cytokines 30
 - Cytology 118
 - bile 128
 - intestines 119
 - liver 122
 - pancreas 128
 - pancreatic 508
 - stomach 119
 - Cytotoxic agents 549
- D**
- D2 dopaminergic antagonists 286
 - Dalmatian
 - disease predisposition 4, 5
 - Damage-Associated Molecular Pattern (DAMP) 30
 - Defecation 23
 - reflex 355
 - Defense mechanisms, mechanical 358
 - Deficient acetaminophen glucuronidation 52
 - Dehydration
 - skin turgor 20
 - treatment 312
 - Delayed gastric emptying 72
 - Dendritic cells, hepatic 397
 - Dental formula
 - cat 227
 - dog 227
 - Dentin 227
 - Depression 17
 - Dermatitis
 - adverse food reaction 534
 - glucagonoma 570
 - necrolytic 408, 450
 - superficial necrolytic 20
 - Dermatological signs 20

- Descending colon 353
- Detoxification 403
- Dextrose
 - diarrhea 312
 - insulinoma 564
- DGGR-based lipase assays 507
- Diabetes insipidus, nephrogenic 349
- Diabetes mellitus 560
 - exocrine pancreas neoplasia 524
 - glucagonoma 570
 - histopathology 161
 - pancreatitis 503
- Diacylglycerol O-acyltransferase 1 inhibitor (DGAT1 inhibitor, T863) 433
- Diagnostic imaging 58
 - biliary tract 81
 - esophagus 63
 - large intestine 79
 - liver 81
 - oropharynx 59
 - pancreas 91
 - small intestines 72
 - stomach 66
 - vomiting 180
- Diagnostic laparoscopy 111
- Diagnostic testing
 - acute signs 171
 - diarrhea 172
 - regurgitation 171
 - vomiting 171
- Diagnostic tools
 - clinical history 3
 - cytology 118
 - diagnostic imaging 58
 - diagnostic laparoscopy 111
 - endoscopy 94
 - gastrointestinal motility assessment 162
 - histopathology 130
 - laboratory tests 24
 - physical examination 16
- Diarrhea 7
 - acute 7
 - AHDS 18
 - antibiotic-responsive 542
 - bloody 17
 - chronic 7, 181, 184
 - causes 183
 - imaging 78
 - chronic inflammatory enteropathy 544
 - clinical evaluation/examination 167, 181, 184
 - colitis 81, 359
 - coronaviral 301, 307
 - diagnostic approach 184
 - diagnostic testing 172
 - differentials 169, 181, 182
 - distemper virus 305
 - EPI 518
 - feline hepatic lipidosis (FHL) 432
 - hemorrhagic 327
 - hepatic encephalopathy (HE) 408
 - imaging techniques 186
 - laboratory tests 185
 - medical history 183
 - pancreatitis 504
 - parvoviral 305
 - patient history 170
 - physical examination 171
 - rotaviral 301
 - signalement 183
 - viral causes 305
- Diazepam, hepatotoxicity 412
- Diazoxide 566
- Dicrocoeliidae 492
- Dicyclomine D 370
- Didanosine 502
- Diet
 - allergens 532
 - assessment 204
 - canned
 - blended 220
 - veterinary 216
 - copper-restricted 442
 - digestibility 206
 - dysbiosis 334
 - environmental risk 206
 - feeding tube 216
 - gastritis 273, 279
 - history 204
 - home-prepared 205, 211, 535
 - hydrolyzed 209
 - hypoallergenic 346
 - indiscretion/intolerance
 - colitis 359
 - pancreatitis 502
 - insulinoma 565
 - limited-ingredient novel protein 210
 - low-fat 346
 - monitoring 207
 - obesity 576, 589
 - protein-losing enteropathy (PLE) 344
 - reassessment 207
 - therapeutic 216
 - unconventional 205
 - vegetarian 205
- Dietary history 15
- Dietary indiscretion 327
- Dietary trial 534
 - duration 536
 - interpreting 536
- Diffuse bowel wall infiltration 78
- Digestibility 206
 - high 208
- Digestive enzymes 498
 - exocrine pancreas 498
- Digestive functions 299
- Digestive tract, examination 20
- Diphenoxylate 370
- Dipylidium caninum* 137
- Dirlotapide 588
- Dirofilaria immitis* 176
- Disease predisposition of breeds 4, 5
- Distemper virus infection
 - enteritis 305
 - vomiting 168
- Diuretics
 - chronic inflammatory enteropathy (CIE) 550
 - insulinoma 566
- Diverticulectomy 257
- Diverticulum
 - acquired 255
 - congenital 255
 - esophageal 255, 256
- D-lactic acidosis 518
- Dobermann pinscher, disease predisposition 4, 5
- Dolasetron 274
 - pancreatitis 511
- Domperidone 286, 287, 288
- Dopamine, pancreatitis 512
- D-penicillamine 442, 458, 459, 460
- Drooling 20
- Drug history 3
- Drug-induced hepatotoxicity 411
- Drug-induced liver injury 410
- Dual-energy X-ray absorptiometry (DEXA) 585, 586
- Dual Phase CT 58
- Ductal plate abnormalities 465
- Ductus pancreaticus 497
 - accessorius 497
- Duodenotomy 474
- Duodenum/Duodenal
 - biopsy 99
 - chronic enteropathy 135
 - endoscopic findings 106
 - infiltrative disease 106
 - intestinal structure 300
 - lymphoma 143
 - masses 106
 - mucosa 101
 - abrasions 96
 - papilla 102
 - perforation 109
 - reflux 7
 - stricture 106
 - ulcer
 - endoscopy 106
 - gastrinoma 567
- Duplex gallbladder 467

- Dysautonomia 194, 336
- Dysbiosis 330
- antibiotics 334
 - clinical signs 331
 - cobalamin supplementation 335
 - diagnosis 331
 - dietary management 334
 - EPI 518
 - intestinal 358
 - pathophysiology 330
 - prognosis 335
 - treatment 333
- Dysbiosis index (DI) 332
- Dyschezia 14, 23
- Dysmotility, esophageal 257
- Dysphagia
- cricopharyngeal 62
 - esophageal 5
 - history 5
 - oral 5
 - pharyngeal 5
 - VFSS 61
- Dyspnea
- inspiratory 18
 - severe life-threatening 19
- ## E
- Eating grass 15
- Ecchymoses 19
- Echinococcus* 521
- *multilocularis* 125
- Echoendoscopy
- esophageal 63, 64
 - pancreatic 93
- Edema 20
- protein-losing enteropathy (PLE) 342
- EGC (eosinophilic granuloma complex) 231
- Electrocautery techniques 110
- Electrolyte
- abnormalities 18
 - imbalances 310
 - transport 356
- Electrophoresis results 54
- Elimination diet 534, 535
- Eluxadoline 370
- Emodepside 361
- Emollient laxatives 377
- Enamel 227
- Endocrine pancreatic tumors 129
- Endoparasites 320
- Endoscopic biopsy forceps 99
- Endoscopic capsules 110
- Endoscopic retrograde cholangio-pancreatography (ERCP) 89
- Endoscopy 94
- biopsy 98
 - choices of endoscopes 94
 - esophageal 64
 - indications 94
 - interventional procedures 106
 - physiologic findings 102
 - procedures 95
 - vomiting 180
- Enema 97, 354
- constipation 195
 - megacolon 375
- English bulldog, disease predisposition 4, 5
- English Springer spaniel, disease predisposition 4, 5
- Enrofloxacin
- *Campylobacter infection* 317
 - *Escherichia coli infection* 364
- Enteral feeding 208
- Enteral nutritional intervention 286
- Enteral products
- human 216
 - veterinary 216
- Enteric nervous system 301
- Enteritis
- acute 22
 - bacterial 315
 - coronaviral 301
 - distemper virus 305
 - eosinophilic 78, 541
 - granulomatous 137, 542
 - lymphoplasmacytic 134
 - neutrophilic 542
 - parvoviral 301, 305
 - rotaviral 301
- Enteroggregative *E. coli* (EAEC) 318
- Enterochromaffin-like cells (ECL) 269
- Enterococcus faecium* SF68 335
- Enterocytes 300, 301
- brush border 302
- Enterohepatic circulation 402
- Enteroinvasive *E. coli* (EIEC) 318
- Enteropathic bacteria 537
- Enteropathogenic *E. coli* (EPEC) 141, 318
- Enteropathogens 135, 315
- Enteropathy
- breed-related 540
 - chronic
 - adverse food reaction 534
 - diet 208, 209, 210, 211
 - dysbiosis 331
 - feeding plan 211
 - inflammatory 29
- Enteropeptidase 498
- deficiency 517
- Enterotoxigenic *E. coli* (ETEC) 318
- Enterotoxin, *Clostridium perfringens* 365
- Environmental risk factors 206
- Enzymes, digestive 498
- Eosinophilic colitis 138
- Eosinophilic enteritis (EE) 78, 537, 541, 546
- Eosinophilic gastritis 275
- Eosinophilic gastroenteritis (EGE) 541
- Eosinophilic granuloma complex (EGC) 231
- diagnosis 231
 - prognosis 232
 - treatment 232
- Epithelial cells
- gastric 270
 - gastrointestinal 302
- Epithelial necrosis 142
- Epulides 235
- Erosion 20
- Erythema 20
- adverse food reaction 533
- Erythrocyte alterations 36
- Erythromycin 289
- motility disorders 287, 288
- Escherichia coli* (*E. coli*) 318, 363
- granulomatous colitis 362
- esomeprazole 274
- gastrinoma 569
 - GERD 245
- Esophagitis 243
- clinical presentation 243
 - endoscopy 244
 - eosinophilic 244
 - PEG tube 219
 - prognosis 244
 - treatment 244
- Esophagogastroduodenoscopy 94, 95
- Esophagography 63
- Esophagoscopy 259
- hiatal hernia 261
- Esophagostomy tube 217
- balloon dilatation 254
- Esophagus
- abdominal 239
 - airway-fistula 257
 - anatomy 239
 - carcinoma 64
 - cervical 239
 - dilatation 65
 - generalized 65
 - segmental 66
 - diseases 241
 - radiographic signs 63
 - diverticulum 255
 - dysmotility 257
 - dysphagia 5

- foreign body 246, 247, 248
- removal 106
- hypomotility 257
- imaging 63
- masses 64, 66
- mucosal layer 240
- muscular layer 239
- neoplastic conditions 263
- nerves 240
- physiology 240
- stricture 250, 251, 255
- dilatation 109
- submuscular layer 239
- swallowing 240
- thoracic 239
- tumors 263
- clinical signs 264
- diagnosis 264
- histopathology 264
- prognosis 266
- systemic complications 266
- treatment 265
- ulceration
- gastrinoma 568
- wall 239
- E tube 217, 222
- E tube see esophagoscopy tube 217
- Eurytrema procyonis* 528
- Examination
 - abdominal 21
 - gastrointestinal tract 20
 - mouth 20
 - perineal area 23
 - pharyngeal area 20
 - rectal 23
- Excoriations
 - adverse food reaction 533
- Exocrine pancreas
 - adenocarcinoma 160
 - adenoma 160
 - anatomy 497
 - diseases 499, 526
 - neoplasia 522, 523
 - clinical signs 524
 - EPI 524
 - prognosis 525
 - therapy 525
 - nodular hyperplasia 160
 - physiology 498
 - secretory products 498
 - tumors 128
- Exocrine pancreatic insufficiency (EPI) 516
 - clinical presentation 518
 - cobalamin deficiency 27
 - diagnosis 519
 - diarrhea 7
 - diet 521
 - dysbiosis 331, 333
 - etiology 517
 - growth failure 16
 - histopathology 159
 - laboratory tests 42
 - non-responders 521
 - pathogenesis 518
 - prognosis 522
 - serum trypsin-like immunoreactivity (TLI) 28
 - stool 11
 - therapy 520
 - vitamin supplementation 521
- Extracellular matrix (ECM) 452
- Extrahepatic bile duct obstruction (EHBO) 471
 - acholic feces 14
 - cholecystoenterostomy 475
 - cholecystostomy tube 472
 - cholecystotomy 475
 - choledochal stenting 474
 - choledochotomy 475
 - management 472
 - pancreatitis 506, 513
 - postoperative management 477
 - surgery 472
- Exudation 21
- F**
- Famotidine
 - gastrinoma 569
 - hypergastrinemia 340
- Farnesoid X receptor agonists 289
- Fasting bile acid concentration 35
- Fat, dietary
 - assessment 206
 - low 209
- Fatty acids 400
- FCGS (feline chronic gingivostomatitis) 228
- Febantel 361
- Fecal alpha-1-proteinase inhibitor (α_1 PI), PLE 343, 545
- Fecal antigen test 172
- Fecal biomarkers 545
- Fecal calprotectin 545
- Fecal dysbiosis index 332
- Fecal enteropathogen testing 172
- Fecal incontinence 15, 374
- Fecal microbiota transplantation (FMT) 333, 335, 378, 548
- Fecal pancreatic elastase 43, 520
- Fecal proteolytic activity (FPA) 44
- Fecal scoring system 12
- Fecal α_1 -proteinase inhibitor concentration 26
- Fecal α_1 -proteinase inhibitor (α_1 -PI)
 - diarrhea 185
- Feces
 - acholic 14
 - bloody 14, 17
 - characterizing 7
 - consistency 12
 - diarrhea 7
 - examination
 - chronic inflammatory enteropathy (CIE) 544
 - diarrhea 172
 - parasites 186
 - smear 367
 - vomiting 179
 - microscopic examination 44
 - removal 375
- Feeding
 - abnormal patterns 15
 - enteral 208
 - monitoring 211
 - plan 206, 211
 - tube 213, 220
 - complications 221
 - maintenance 221
 - removing 223
- Feline chronic enteropathy activity index (FCEAI) 185
- Feline chronic gingivostomatitis (FCGS) 228
 - treatment 229
- Feline coronavirus (FeCoV) 307
- Feline enteric coronavirus 136
- Feline gastrointestinal eosinophilic sclerosing fibroplasia (FGESF) 132, 541, 542
- Feline hepatic lipidosis (FHL) 429
 - clinical examination 431
 - diagnosis 431
 - electrolyte abnormalities 431
 - nutritional supplements 433
 - prognosis 433
 - treatment 432
- Feline idiopathic megacolon (FIM) 194
- Feline immunodeficiency virus (FIV) 307
- Feline infectious peritonitis (FIP) 136
 - hepatitis 414, 419
 - pyogranulomatous inflammation 21
- Feline leukemia virus (FeLV) 307
- Feline pancreatic lipase immunoreactivity (fPLI) 39, 40
- Feline parvovirus (FPV) 305
- Feline trypsin-like immunoreactivity (fTLI)
 - EPI 519
 - pancreatic neoplasia 524

- Fenbendazole 361
 - chronic inflammatory enteropathy (CIE) 545
 - stomach worms 273
 - whipworms 361
 - Fenofibrate 436
 - Fenthion 502
 - Fever 18
 - Fiber
 - constipation 376
 - dietary
 - assessment 206
 - high 210
 - water-insoluble 371
 - water-soluble 371
 - Fiber-responsive large bowel diarrhea 187, 371
 - clinical presentation 371
 - diagnostic evaluation 371
 - pathophysiology 371
 - prognosis 372
 - treatment 371
 - Fiber-supplemented diets 377
 - Fibrinogen 36
 - Fibroma, odontogenic 235
 - Fibrosarcoma (FSA) 235
 - Fibrosis 452
 - Fine-needle aspiration (FNA) 91, 118
 - large intestinal neoplasia 381
 - pancreatic tumors 524
 - FIP see Feline infectious peritonitis 136
 - Firmicutes 356
 - Flatulence 14
 - Fluid therapy
 - chronic inflammatory enteropathy (CIE) 550
 - gastritis 273
 - pancreatitis 510
 - parvovirus infection 312
 - Fluorescence in situ hybridization (FISH)
 - granulomatous colitis 363
 - leptospires 418
 - Fly snapping 17
 - Focal granulomatous lesions 22
 - Focal intestinal lipogranulomatous lymphangitis (FLL) 345
 - Folate 26, 28
 - absorption 28
 - malabsorption 548
 - FOLFIRINOX 525
 - Folic acid
 - bacteria 333
 - Food
 - adverse reaction 210, 212
 - allergy 209, 210, 531, 532
 - antigens 358
 - history 15
 - hypersensitivity 209, 532
 - dermatological signs 20
 - idiosyncrasy 531
 - intolerance 327, 531
 - intoxication 327
 - poisoning 531
 - sensitivity 327
 - toxicity 531
 - Food-responsive enteropathy (FRE)
 - diarrhea 187
 - Foreign body
 - cervical 60
 - colitis 359
 - diagnosis 337
 - esophageal 64, 106, 246, 247, 248
 - gastric 69, 108
 - intestinal 76, 108, 337
 - linear 21, 76, 77, 108, 337
 - lingual 60
 - prognosis 337
 - removal 106, 246
 - retrieval devices 108
 - salivary duct 233
 - small intestinal 73
 - sublingual 21
 - treatment 337
 - vomiting 178
 - Frenulum laceration 21
 - Fresh-frozen plasma 512
 - Functional ileus 75
 - Functional swallowing disorders 60
 - Fundic glands 267, 268
 - Fungal infection 325, 357
 - Furosemide 409, 410
 - Furunculosis, anal 386
 - Fusobacterium 356
 - Fuzapladib 511
- G**
- Gabexate mesylate 512
 - Gagging 6
 - Gag reflex 20
 - Gallbladder 398, 402
 - adenoma 495
 - agenesis 467
 - carcinoma 495
 - disorders 155
 - dysmotility 478
 - histopathologic examination 483
 - hypomotility
 - cholecystitis 483
 - infarction 155
 - mobilisation 476
 - mucocele 87, 155, 436, 478
 - classification 479
 - clinical signs 479
 - diagnostic evaluation 479
 - etiology 478
 - prognosis 481
 - surgery 479
 - treatment 479
 - palpation 22
 - rupture 485
 - Gallbladder wall
 - edema 87
 - thickened 86
 - Gamma-glutamyl transferase (GGT) 33
 - plasma half-live 33
 - Ganglioneuromatosis 147
 - Garbage can intoxication 327
 - Gas, excessive 14
 - Gastric (...) see also Stomach 7
 - Gastric acid
 - secretion 268, 269
 - suppressants 25
 - suppression 244
 - Gastric adenocarcinoma 144
 - Gastric biopsy 99
 - Gastric bleeding
 - melena 14
 - Gastric brush preparation 119
 - Gastric carcinoma 71, 120
 - Gastric dilatation-volvulus (GDV) 282, 290
 - clinical signs 290
 - diagnostic approach 290
 - etiology 290
 - imaging 67
 - prognosis 291
 - pulse rate 18
 - treatment 291
 - Gastric disease
 - laboratory tests 24
 - Gastric distension 21
 - imaging 67
 - relieving 286
 - Gastric emptying 268, 283
 - disorder 282
 - vomitus 7, 177
 - patterns 285
 - time 284
 - evaluation 25, 165
 - Gastric erosion 105
 - endoscopy 105
 - Gastric foreign body 70
 - removal 108
 - Gastric glands 267
 - Gastric hemorrhage 105
 - Gastric inflammation 119

- Gastric inhibitory polypeptide (GIP) 558, 559
- Gastric juice 268
 - analysis 25
- Gastric lipase 518
- Gastric lymphoma 121
- Gastric motility 268
 - disorder
 - clinical signs 281
 - diagnostic approach 283
 - treatment 285
- Gastric motility disorder 280
- Gastric mucosal barrier 270
- Gastric neoplasia 294
 - imaging 69
 - vomiting 168
- Gastric parasites 24, 133
- Gastric permeability, increased 25
- Gastric pits 267
- Gastric torsion 290
- Gastric tumors 103, 291
 - biopsy 296
 - clinical pathology 294
 - clinical signs 294
 - diagnostic imaging 295
 - histology 292
 - invasion 296
 - metastasis 296
 - prognosis 297
 - systemic complications 296
 - treatment 296
- Gastric ulceration 69, 132, 279
 - biopsy 99
 - endoscopy 105
 - laboratory tests 24
 - neoplastic 104
 - NSAID-induced 280
 - treatment 280
- Gastric wall 267
 - edema 69
 - thickening, pancreatitis 505
- Gastrin 25, 302, 558, 559, 566
- Gastrinoma 25, 566, 567
 - clinical signs 567
 - diagnosis 568
 - immunohistochemistry 567
 - prognosis 570
 - staging 569
 - surgery 569
 - therapy 569
- Gastritis
 - acute 130, 271
 - clinical findings 272
 - diagnostic approach 272
 - history 272
 - treatment 273
 - atrophic 25, 132, 276
 - chronic 131, 275
 - classification 275
 - diagnostic approach 278
 - etiology 275
 - therapeutic approach 279
 - classification 271
 - eosinophilic 275
 - Helicobacter-associated 276
 - hypertrophic 276
 - infectious 277
 - lymphoplasmacytic 275
 - vomiting 7, 168
- Gastrocolic reflex 355
- Gastroduodenal ulceration
 - Hepatic encephalopathy (HE) 408
- Gastroduodenoscopy 95
 - intraoperative 97
- Gastroenteritis
 - acute 208, 209
 - bacterial 315
 - diet 208, 211
 - hemorrhagic 327
 - low-fat diet 209
- Gastroesophageal intussusception 262
- Gastroesophageal reflux disease (GERD) 245
 - clinical presentation 245
 - diagnosis 245
 - treatment 245
- Gastroesophageal sphincter (GES) 239
 - swallowing 240
- Gastrointestinal bacteria 356
- Gastrointestinal blood loss 17
- Gastrointestinal carcinoids 572
- Gastrointestinal diseases
 - hereditary 44, 46, 48, 50, 52
 - infiltrative 177
 - nutritional management 203, 208
- Gastrointestinal hormones 302
- Gastrointestinal lymphoma 551
 - classification 551, 555
 - clinical signs 552, 556
 - diagnosis 552, 556
 - dogs 555
 - feline 551
 - prognosis 554, 556
 - signalment 552, 556
 - treatment 554, 556
- Gastrointestinal motility
 - assessment 162
 - clinical evaluation 165
 - methods 162
- Gastrointestinal mucosal inflammation 29
- Gastrointestinal neoplasia 120
- Gastrointestinal neuroendocrine tumors 557, 560, 573
- Gastrointestinal protein loss, assessment 29
- Gastrointestinal regulatory peptides 558, 559
- Gastrointestinal signs
 - acute 171
 - chronic 208
- Gastrointestinal stromal tumor (GIST) 120, 143, 348, 351
- Gastrointestinal tract examination 20
- Gastrointestinal ulceration
 - chronic hepatitis 458, 459, 460
 - medication 458, 459, 460
- Gastrokinetics 289
- Gastroparesis
 - clinical signs 283
 - treatment 286
- Gastropathy
 - acute 271
 - causes 272
 - hypertrophic 276
 - uremic 146
- Gastropexy 291
- Gastroprotectants 274
- Gastroscopy 24
- Gastrostomy tube 217
 - cricopharyngeus muscle dysfunction (CPMD) 243
- Gemcitabine plus capecitabine 525
- Gemfibrozil 436
- General anaesthesia, gastroesophageal reflux disease (GERD) 245
- General physical examination 16
- GERD (gastroesophageal reflux disease) 245
- German shepherd dog
 - disease predisposition 4, 5
 - enteropathy 541
 - pancreatic acinar atrophy (PAA) 517
- Ghrelin 267, 303, 558, 559
 - antagonists 289
 - receptor agonist 303
- Giant migrating contractions 355
- Giant schnauzer, disease predisposition 4, 5
- Giardia* 320
 - diagnostic tests 321
 - *duodenalis* 119
- Giardiasis 137
- Gingiva 227
- Gingival sulcus 227
- Gingivostomatitis 228, 229
 - treatment 229
- Glands
 - circumanal 384
 - gastric 267

- Glucagon 497, 558, 559
 – insulinoma 564
 Glucagon-like peptide 1 (GLP-1) 357, 558, 559
 Glucagonoma 570
 – ACHES 450
 – diagnosis 571
 – superficial necrolytic dermatitis 20
 – treatment 572
 Glucocorticoids 356
 – insulinoma 565
 – pancreatitis 511
 – perianal fistula 387
 – steroid hepatopathy 438
 Glucocorticosteroids
 – eosinophilic granuloma complex 232
 Gluconeogenesis 400
 Glucose 400
 Glucose-6-phosphatase defect 437
 Glucose phosphatase 400
 Glucuronidation 411
 – lower capacity 403
 Glutathione deficiency 411
 Gluten-sensitive enteropathy 534
 Glycine 402
 Glycogen storage diseases 436
 – diagnosis 437
 – treatment 437
 Glycogen type vacuolar hepatopathy 438
 Gnathostoma 133
 Golden retriever
 – disease predisposition 4, 5
 Granulomatous colitis (GC) 361, 537, 542
 – bacterial isolation 364
 – diagnostic evaluation 362
 – histopathology 363
 – infectious causes 362
 – prognosis 364
 – treatment 364
 Granulomatous enteritis 137, 542
 Grass eating 15
 Great Dane, disease predisposition 4, 5
 Growth
 – charts 588
 – failure
 – EPI 16
 – stunted 16
 Growth hormone-releasing hormone (GhRH) 303
 G tube 217
 – cricopharyngeus muscle dysfunction (CPMD) 243
 – mushroom-tipped 219
 Gut-associated lymphoid tissue (GALT) 301, 303, 358
 Gut neuroendocrine system 557
- H**
 HDL see high-density lipoprotein 401
 Heartworm disease
 – gastritis 271
 – vomiting 176
Helicobacter 131
 – gastritis 275, 276
 – vomiting 177
Helicobacter-like organisms (HLO) 24, 119
 Hemangiosarcoma
 – hepatic 127
 – rupture 19
 – splenomegaly 23
 Hematemesis 6, 7
 – chronic inflammatory enteropathy 543
 Hematochezia 14, 17
 Hematology 36
 – sample requirements 35
 Hematopoiesis 403
 – extramedullary 125
 Hemochromatosis 443
 Hemolysis, laboratory interferences 32
 Hemoperitoneum 349
 Hemorrhagic gastroenteritis (HGE) 282
 Hemostasis 403
 Hepadnavirus 420
 Hepatic (...) see also Liver 397
 Hepatic abscess 152
 Hepatic amyloidosis 444
 Hepatic arteriovenous fistula 461
 Hepatic arteriovenous malformation (HAVM) 428, 461
 Hepatic artery 395
 Hepatic changes, diffuse 84
 Hepatic circulation 399
 Hepatic cirrhosis 114
 Hepatic copper accumulation 152
 Hepatic dendritic cells 397
 Hepatic disease
 – diagnostic approach 404
 – focal 85
 Hepatic encephalopathy (HE) 408
 – acute 408
 – chronic 408
 – congenital portosystemic shunt 421
 – dermatologic signs 408
 – drugs 409, 410
 – gastrointestinal signs 408
 – medication 458, 459, 460
 – polyuria/polydipsia 407
 Hepatic enlargement 22
 Hepatic fibrosis 83
 – congenital 465
 Hepatic functional mass, reduced 36
 Hepatic glutathione stores 411
 Hepatic glycogen accumulation 123
 Hepatic granuloma 152
 Hepatic hyperplasia 155
 Hepatic immune cells 397
 Hepatic impairment, bile acid stimulation tests 35
 Hepatic infections 415
 Hepatic Kupffer cells 414
 Hepatic lipidosis 113, 123
 – feline 429
 – icterus 407
 – imaging 82
 – secondary 436
 Hepatic lipids 36
 Hepatic lobules 396
 Hepatic metabolic storage disease 152
 Hepatic neoplasia 126, 155, 467
 – clinical signs 468
 – diagnostics 468
 – prognosis 469
 – staging 469
 – surgery 470
 – treatment 469
 Hepatic parenchymal disease 82
 Hepatic pigments 124, 397
 Hepatic proteins 36
 Hepatic sinusoids 395, 396
 Hepatic size 22
 Hepatic stellate cells 148
 Hepatic synthetic capacity 36
 Hepatic vascular anomalies 421
 Hepatitis 451
 – acute 151, 452, 453
 – chronic 151, 404, 452, 454
 – granulomatous 418
 – immune-mediated 458, 459, 460
 – infectious 415
 – lymphocytic 122
 – pathophysiology 451
 Hepatitis B-like virus 420
 Hepatobiliary disease
 – inflammatory 451
 – laboratory tests 31
 – vomiting 175
 Hepatobiliary laboratory biochemical testing 32
 Hepatobiliary malignancies 21
 Hepatocellular adenoma 155
 Hepatocellular carcinoma (HCC) 86, 126, 155
 – clinical signs 468
 – diagnostics 468
 Hepatocellular damage markers 32
 Hepatocutaneous-associated hepatopathy 448

- Hepatocutaneous hepatopathy 408
- Hepatocutaneous syndrome 20, 408
 - imaging 85
- Hepatocytes 148, 396
 - apoptosis 150
 - bile acids 402
 - cell death 452
 - cytoplasmic and nuclear changes 123
 - lipid vacuoles 150
 - necrosis 150
 - regeneration 452
- Hepatomegaly 405
- Hepatopathy
 - copper-associated 439
 - hepatocutaneous-associated 448
 - vacuolar 429
- Hepato-protectants 458, 459, 460
- Hepatosplenomegaly 23
- Hepatotoxicity
 - diagnosis 411
 - drug-induced 411
 - idiosyncratic 411
 - mechanisms 411
- Hepatotoxins 410
 - environmental 413
- Hereditary gastrointestinal diseases
 - cats 52
 - dogs 44, 46, 48, 50
- Hernia
 - diaphragmatic 118
 - hiatal 65, 259
 - perineal 23
 - peritoneopericardial 52
- Herringbone appearance, esophagus 239
- Heterobilharzia americana 141, 323, 419, 528
- Heterobilharziosis 169
- Hiatal hernia 259
 - acquired 259
 - clinical presentation 259
 - congenital 259
 - diagnosis 259
 - management 261
 - prognosis 261
 - radiographs 259
- High-density lipoprotein (HDL) 401
- High-throughput RNA sequencing 356
- Hind limb weakness 17
- Histamine (H₂) receptor antagonists 289
- Histiocytic colitis 139
- Histiocytic ulcerative colitis (HUC) 362, 537, 542
 - AIEC 318
- Histology-guided mass spectrometry (HGMS) 57
- Histopathology
 - gallbladder 147
 - gastrointestinal tract 130
 - liver 147
 - pancreas 156
- Histoplasma capsulatum 120, 326
- Histoplasmosis 139, 326
 - diarrhea 172
- History
 - acquisition 3
 - acute signs 170
 - diarrhea 183
 - dietary 15
 - dysphagia 5
 - specific gastrointestinal signs 5
- Hormones, gastrointestinal 302
- Human liquid enteral products 216
- Hydrochloric acid 268
- Hydroxocobalamin 548
- Hyoscyamine 370
- Hyperadrenocorticism 560
 - gallbladder mucocele 478
 - iatrogenic 548
 - obesity 576
 - pancreatitis 503
 - steroid hepatopathy 438
- Hyperammonemia 36
- Hyperbaric oxygen therapy 513
- Hyperbilirubinemia 34
 - hepatic 198
 - pancreatitis 505
 - post-hepatic 198
 - pre-hepatic 198
- Hypercalcemia
 - of malignancy 503
 - pancreatitis 502, 505
 - treatment 509
- Hypercholesterolemia 34
- Hypergastrinemia 340
- Hyperglycemia
 - glucagonoma 570
 - pancreatitis 503, 505
- Hyperinsulinism 560
- Hyperkalemia
 - pulse rate 18
- Hyperlipidemia
 - hereditary 434
 - postprandial 435
- Hyperlipoproteinemia
 - hereditary 434
- Hyperosmotic laxatives 377
- Hyperpigmentation
 - adverse food reaction 533
- Hypersensitivity reactions 327
- Hyperthermia 18
- Hyperthyroidism
 - constipation 375
 - examination 21
 - vomiting 176
- Hypertriglyceridemia 434
 - diet 514
 - management 509
 - pancreatitis 502
 - prognosis 436
 - treatment 436
- Hypertrophic gastropathy/gastritis 276
- Hypertrophic osteopathy 266
- Hyperventilation 19
- Hypoadrenocorticism 560
 - diagnostic testing 173
 - megaesophagus 65
 - protein-losing enteropathy (PLE) 343
 - vomiting 176
- Hypoalbuminemia 36
 - acute hemorrhagic diarrhea syndrome (AHDS) 329
 - edema 20
 - protein-losing enteropathy (PLE) 29, 343
 - treatment 312
- Hypoaminoacidemia 449
- Hypoantithrombinemia 515
- Hypochloremic metabolic alkalosis
 - gastric carcinoma 296
 - vomiting 171
- Hypocholesterolemia 34, 36
- Hypocobalaminemia
 - lymphoma 552
 - PLE 343
- Hypofolatemia
 - PLE 343
- Hypoglycemia
 - insulinoma 560, 561
 - paraneoplastic 296, 349
- Hypokalemia
 - arrhythmia 18
 - neck ventroflexion 17
- Hypomotility
 - esophageal 257
- Hypoproteinemia
 - parvovirus infection 311
- Hyporexia
 - pancreatitis 511
- Hypotension
 - pancreatitis 502
- Hypothermia 18
- Hypothyroidism
 - gallbladder mucocele 478
 - megaesophagus 65
 - obesity 576
 - pancreatitis 503

- Hypovitaminosis D
 – chronic inflammatory enteropathy (CIE) 545
 Hypoxic hepatocyte injury 34
- I**
- Icterus 34, 198
 – causes 198
 – hepatobiliary disorders 407
 – hepatosplenomegaly 23
 – laboratory interferences 32
 Ideal body weight 583, 588
 Idiopathic esophageal dysmotility (IED) 65
 Idiopathic inflammatory bowel disease
 – adverse food reaction 534
 Idiopathic lymphoplasmacytic enteritis 540
 IgA 304
 Ileocecal area
 – palpation 22
 Ileocolic valve 98, 107, 331
 Ileum 300
 – biopsy 100
 – resection 340
 Ileus
 – abdominal auscultation 23
 – complicated 77
 – functional 75
 – imaging 72
 – postoperative 336
 – ultrasonography 75
 Imerslund-Gräsbeck syndrome 186
 Imidazoles, hepatotoxicity 412
 Immune function of the GI tract 358
 Immune-mediated hepatitis
 – medication 458, 459, 460
 Immune-mediated hypersensitivity 411
 Immune response, pathogenic 358
 Immune system 303
 – cells 304
 Immunoglobulin 358
 – A (IgA) 358
 – E (IgE) 358
 Immunoglobulin heavy chain (IGH) V-J gene 53
 Immunohistochemistry (IHC) 130
 – intestinal neoplasia 350
 – LGAL 56
 Immunological food reactions 327
 Immunological lipase assay
 – cats 39
 – dogs 38
 Immunomodulatory therapy
 – perianal fistula 387
 Immunoreactive pepsinogen 25
 Immunosuppressant drugs 279, 548
 – pancreatitis 514
 Immunosuppressant-responsive enteropathy (IRE) 188, 537
 Incontinence, fecal 15
 Inertia, colonic 373
 Infection, chronic 537
 Infectious disease, hepatic 414
 Inflammatory bowel disease (IBD) 134, 537
 – differentiation to LGAL 56
 Inflammatory enteropathies 29
 Inguinal lymph nodes 19
 Injected membranes 19
 In situ carcinoma 144
 Inspiratory dyspnea 18
 Insulin 497, 558, 559
 – deficiency 560
 Insulin-like growth factor-1 499
 Insulinoma 129, 560
 – breed distribution 562
 – clinical signs 561, 562
 – diagnosis 561
 – imaging 93
 – metastases 563
 – pathology 561
 – prognosis 566
 – staging 563
 – surgery 564, 565
 – treatment 564
 Integument, examination 20
 Interferon 230
 Interventional procedures 106
 Intestinal adenocarcinoma 121, 144
 Intestinal bacteria 304
 Intestinal barrier function 26, 30
 Intestinal biopsy 112
 – chronic inflammatory enteropathy (CIE) 546
 Intestinal disorders
 – laboratory tests 26
 Intestinal dysbiosis 331, 358
 Intestinal foreign body
 – radiolucent 76
 – removal 108
 Intestinal gas 14
 Intestinal inflammation 119
 Intestinal intussusception 78, 338
 Intestinal lymphangiectasia 146
 Intestinal lymphoma
 – imaging 79
 Intestinal microbiota
 – function 330
 – pathophysiology 330
 Intestinal mucosa
 – functional assessment 26
 Intestinal mucosal cells 400
 Intestinal mucosal inflammation
 – non-invasive assessment 29
 Intestinal neoplasia 348
 – abdominal exploration 350
 – clinical signs 349
 – diagnostics 349
 – immunohistochemistry (IHC) 350
 – incidence 348
 – metastatic 349
 – prognosis 351
 – staging 350
 – surgery 350
 Intestinal obstruction 75, 337
 Intestinal parasites 136
 Intestinal permeability assessment 30
 Intestinal pseudo-obstruction 146
 Intestinal small-cell lymphoma 57
 Intestinal sounds 23
 – failure 23
 Intestinal tenesmus 14
 Intestinal torsion 339
 Intestinal tract
 – anatomy
 -- gross 299
 -- microscopy 300
 – digestive functions 299
 – major functions 26
 Intestinal vascular ectasia 146
 Intestinal volvulus 21
 Intestinal wall
 – layering 67
 – thickened 22
 Intoxication
 – garbage can 327
 – vomiting 272
 Intrahepatic arteriovenous fistula/malformation 465
 Intraoperative mesenteric portovenography (IOMP) 423, 424
 Intravenous fluid therapy (IVFT) 282
 – motility disorders 286
 Intrinsic factor 498
 Intrinsic nervous system 355
 Intussusception 78, 147
 – gastroesophageal 262
 – intestinal 338
 – prolapsed 383
 Iohexol 31
 Irish setter
 – disease predisposition 4, 5
 Irish wolfhound
 – disease predisposition 4, 5

- Iron
 - hepatic 398
 - hepatocytes 398
- Iron deficiency anemia
 - gastric tumors 296
- Iron hepatopathy 443
- Irritable bowel syndrome (IBS) 369
 - clinical presentation 369
 - diagnostic evaluation 369
 - pathophysiology 369
 - prognosis 370
 - treatment 370
- Islet hyperplasia 160
- Islets of Langerhans 497

- J**
- Jack Russel terrier, disease predisposition 4, 5
- Jaundice 34
 - clinical evaluation 198
 - definition 198
 - diagnostic approach 199
 - historical assessment 198
 - physical examination 199
 - signagelement 198
- Jejunum 300

- K**
- Key Gaskell syndrome 194
- Kidneys
 - abdominal palpation 23
 - abnormally-shaped 23
 - enlarged 23
- Klebsiella pneumoniae 502
- KRAS gene mutation 523
- Kupffer cells 148, 397, 414

- L**
- Laboratory accreditation 31
- Laboratory interferences 32
- Laboratory parameters, falsely increased/decreased 32
- Laboratory quality control 31
- Laboratory tests
 - chronic inflammatory enteropathy (CIE) 544
 - diagnosis of exocrine pancreatic disorders 37
 - diagnosis of gastric disease 24
 - diagnosis of intestinal disorders 26
 - diagnosis of liver and biliary tract disease 31
 - molecular-genetics-based 45
 - reference intervals 31
- Labrador retriever
 - disease predisposition 4, 5
- Lactobacillus* 304, 357
- Lactulose
 - CPSS 425
 - hepatic encephalopathy (HE) 409, 410
 - laxative 197, 377
- Laparoscopy
 - biopsy 112
 - complications 118
 - contraindications 111
 - diagnostic 111
 - equipment 111
 - procedures 111
- Laparotomy, exploratory 180
- Large bowel diarrhea 11
 - colitis 359
 - fiber-responsive 371
- Large bowel disease 7
- Large-cell lymphoma 142, 551
- Large granular lymphocyte (LGL) lymphoma 121, 551, 554
- Large intestine 353
 - absorption of water and electrolytes 356
 - anatomy 353
 - diarrhea 167
 - differentials 169
 - diseases 359
 - imaging 79
 - immune function 358
 - inflammation 137
 - microbiome 356
 - motility 355
 - mucus secretion 356
 - neoplasia 379
 - physiology 355
- L-arginine 289
- Laser therapy 230
 - gingivostomatitis 230
- L-asparaginase
 - hyperammonemia 36
- Lavage solutions 97
- Laxatives 196, 376
 - bulk-forming 196, 377
 - emollient 196, 377
 - hyperosmotic 377
 - lubricant 196, 377
 - osmotic 197
 - stimulant 197, 377
- L-carnitine
 - feline hepatic lipidosis (FHL) 433
- L-carnitine supplementation 433
- LDL see low-density lipoprotein 401
- Leiomyoma 143
 - gastric 292
 - intestinal 348
- Leiomyosarcoma
 - esophageal 264
 - intestinal 348
 - prognosis 351
- Leiomyositis 146
- Leishmania* 125
 - *infantis* 502
- Leishmaniasis 140
- Leptospirosis 416
- Lethargy, exocrine pancreas neoplasia 524
- Leukocyte antigens (DLA) class II gene 517
- Leukopenia, parvoviral 310
- Lexipafant 511
- L-glutamate 289
- Lhasa Apso, disease predisposition 4, 5
- Librax® 370
- Lichenification
 - adverse food reaction 533
- Lidocaine 289
- Linear foreign body 21, 77, 337
 - obstruction 22
 - removal 108
- Lingual foreign body 60
- Lingual molar glands 228
- Lipase 506, 518
- Lipase assays
 - cats 39
 - dogs 38
- Lipemia 435
 - laboratory interferences 32
- Lipid
 - metabolism 400
- Lipofuscin 397, 398
- Lipogranuloma 146
- Lipomatosis, pancreatic 161
- Lipoprotein lipase (LPL) deficiency, feline 434
- Lipoprotein X 34
- Liver 395
 - abdominal palpation 22
 - acute injury 82
 - anatomy 395
 - biopsy 115
 - chronic hepatitis 456
 - congenital hepatic fibrosis (CHF) 466
 - hepatic neoplasia 468
 - jaundice 200
 - laparoscopic 113
 - cells 396
 - cirrhosis 152, 453
 - copper accumulation 151
 - copper level 442

- cytology 122
- hematopoiesis 403
- homeostasis 403
- homeostatic functions 451
- imaging 81
- immunologic functions 403
- inflammation 124
- interventional procedures 91
- iron accumulation 444
- lobe agenesis 467
- lymphoma 83
- metabolism 400
- metastases 467
- insulinoma 563
- neoplasms 467
- nodular hyperplasia 122, 155
- normal histology 147
- parenchymal disorders 149
- physiology 400
- pigments 124
- regeneration 452
- regenerative nodules 155
- structure 395
- tumors 467
- vascular disorders 149, 421
- Liver see also hepatic ... 397
- Liver disease 410
 - breed predispositions 404
 - clinical findings 404
 - complications 407, 409
 - developmental 461
 - diagnostic approach 403
 - drug-induced 410
 - drugs 409, 410
 - infectious 414
 - inflammatory 451
 - metabolic 429
 - hereditary 446
 - prevalence 404
 - radiation-induced 471
- Liver enzymes 33
 - activities
 - hepatitis 454
 - activity
 - cholestatic 483
 - drug effects 33
 - elevations 32
 - plasma half-lives 33
- Liver fibrosis 124, 452
- Liver flukes
 - cats 419
 - cholangitis 487, 489, 492
 - clinical signs 493
 - diagnostic evaluation 493
 - dogs 419
 - pancreatic 528
 - prognosis 495
 - treatment 489, 494
- LMW heparin
 - acute portal vein thrombosis 409, 410
- Locomotion abnormalities 17
- Lomustine
 - hepatotoxicity 412
 - lymphoma 554
- Loperamide 340, 370
- Low-density lipoprotein (LDL) 401
- Lower esophageal sphincter achalasia (LES-AS) 65
- Low-fat diet 209
- Low-grade alimentary lymphoma (LGAL) 56
- Low-grade small-cell lymphoma 551
- Low-grade T-cell lymphoma 553
- Low residue diet 376
- Lubricants 377
- Lupus erythematosus 387
- Lymphadenomegaly
 - pancreatic neoplasia 92
- Lymphadenopathy
 - generalized 19
 - mesenteric 22
- Lymphangiectasia 542
 - duodenal 106
 - intestinal 146, 341
 - diet 209
 - feeding plan 212
- Lymphatic system 401
- Lymph nodes
 - metastatic 237
 - peripheral 19
- Lymphocytes 304
- Lymphocytic cholangitis 154
- Lymphocytic enteritis 119
- Lymphocytic hepatitis 122
- Lymphocytic-plasmacytic enteritis 56, 57
- Lymphoid follicles 304
- Lymphoma 142, 551
 - alimentary 57, 120, 551
 - cats 551
 - dogs 555
 - gastric 121, 292, 295
 - gastrointestinal 551
 - hepatic 127
 - imaging 83
 - intestinal 57, 79, 348
 - peripheral lymph nodes 19
- Lymphoplasmacytic enteritis (LPE) 134, 537, 546
 - idiopathic 540
- Lymphoplasmacytic gastritis 275
- Lymphoproliferative enteropathy of the basenji 540
- Lysosomal storage diseases 448
- M**
- Macrolide antibiotics 289
 - motility disorders 287, 288
- Magnetic resonance imaging (MRI) 58
 - pancreatitis 506
- Main pancreatic duct 497
- Maintenance energy requirement (MER) 205
- Maldigestion, EPI 518
- Malignant abdominal tumors 21
- Malignant melanoma (MM), oral 235
- Malnutrition 188, 203
- Maltese, disease predisposition 4, 5
- Mandibular lymph nodes 19
- Mandibular salivary glands 228
- Manx, disease predisposition 5
- Maropitant 274, 289
 - feline hepatic lipidosis (FHL) 433
 - liver disease 409, 410
 - pancreatitis 511
- Masses
 - intestinal neoplasia 349
 - palpation 22
 - polypoid 483
 - *Pythium insidiosum* 278
- Mast cells 304
- Mast cell tumor 145
 - hepatic 127
 - intestinal 348
- Median survival time (MST), hepatic neoplasia 469
- Medical history 183
- Medical supplements 15
- Megacolon 192
 - clinical history 373
 - clinical presentation 374
 - colectomy 378
 - definition 372
 - diagnosis 374
 - differential diagnoses 373
 - etiology 372
 - feline 372
 - idiopathic 373
 - pathophysiology 373
 - prognosis 379
- Megaesophagus 258
 - clinical presentation 258
 - diagnosis 258
 - generalized 65
 - imaging 61, 65
 - persistent right aortic arch 218
 - prognosis 258
 - treatment 258
- Melena 14, 17
- Mental status 17
- Mesalazine 549

- Mesenchymal stem cell therapy 230, 388
 Mesenteric lymph node enlargement 22
 Mesenteric venous portogram 84
 Mesocestoides 137
 Mesothelioma 145
 Metabolic food reactions 531
 Metamucil® 372
 Metastasis
 - exocrine pancreatic neoplasia 525
 - gastric tumors 296
 - intestinal neoplasia 349
 - large intestinal neoplasia 381
 - rectal tumors 391
 Metastatic lesions, liver 467
 Methimazole, hepatotoxicity 412
 Methotrexate 549
 Methylazoxymethanol 414
 Metoclopramide 274, 286
 - esophagitis 244
 - feline hepatic lipidosis (FHL) 433
 - motility disorders 287, 288
 - pancreatitis 511
 Metorchis bilis 493
 Metronidazole 313
 - Clostridium perfringens enterotoxigenic 366
 - dysbiosis 334
 - hepatic encephalopathy (HE) 409, 410
 - perianal fistula 388
 Micelles 400
 Microbe-associated molecular patterns (MAMPs) 358
 Microbiome 304
 - colonic 356
 - altering 378
 Microcystins 413
 Microhepatia 22
 MicroRNA (miR) testing 479
 Microsomal triglyceride transfer protein (MTP) inhibitors 588
 Microvascular dysplasia 461
 Microvilli 300
 - colon 353
 Midline defects 467
 Migrating motor complexes (MMC) 281
 Mineralocorticoids 356
 Mineral oil 377
 Miniature schnauzer
 - disease predisposition 4, 5
 - hypertriglyceridemia 434, 514
 - pancreatitis 502
 Minimal change enteropathy 537
 Minimally invasive markers for gastric disease 25
 Mirtazapine 289
 - motility disorders 287, 288
 Misoprostol 378
 Mitoxantrone 525
 Mitratapide 588
 Mixed bowel diarrhea 167
 Modified canine activity index (MCAI) 10
 - pancreatitis 515
 Modified transudates 21
 Molecular clonality testing 52
 Molecular-genetics-based laboratory tests
 - hereditary conditions 45
 - non-hereditary conditions 52
 Monoclonal profile 55
 Mosapride 286, 378
 - motility disorders 287, 288
 Motilin 303, 558, 559
 Motilin agonists 287, 288
 Motility disorders
 - critical illness-related 336
 - intestinal 336
 - non-obstructive 336
 Motility, large intestine 355
 Motility-modifying agents 370
 Mouth, examination 20
 MRI see Magnetic resonance imaging 58
 Mucocele, gallbladder 87, 88, 155, 478
 Mucocutaneous junctions 19
 Mucocutaneous lupus erythematosus 387
 Mucogingival junction (MCJ) 227
 Mucoïd stool 11
 Mucosa
 - colonic 369
 - esophageal 240
 - gastric 267
 - intestinal 299
 - oral 227
 Mucosal disease in the ileum 27
 Mucosal immune responses 357
 Mucosal polyps 23
 Mucous membranes
 - examination 19
 - hemorrhagic 19
 - injected 19
 - pale 19
 - yellow 19
 Mucus colitis 369
 Mucus secretion, large intestine 356
 Multifocal necrotizing steatitis 524
 Multiorgan failure 515
 Multiple endocrine neoplasia (MEN) 560
 Muscle condition 204
 - scoring 583
 Muscle wasting, PLE 342
 Muscularis 299
 Mycobacterium 140
 Mycophenolate mofetil 388, 549
 - immune-mediated hepatitis 458, 459, 460
 Mycotoxins 327
 Myenteric plexus 354
- N**
 N-Acetylcystein (NAC) 433
 Na/K ratio, decreased 18
Nanophyteus salmincola 137
 Nasal discharge 20
 Nasoesophageal tube (NE tube) 213
 Nasogastric tube (NG tube) 213
 - placement 213
 - security 214
 Natural killer cells 304, 397
 Nausea 6
 - delayed gastric emptying 281
 Neck ventroflexion in cats 17
 Necrolytic dermatitis 408
 - ACHES 450
 Necrolytic migratory erythema (NME) 570, 571
 Necrosectomy, pancreatitis 513
 Necrosis 150, 452
 - rectal prolapse 384
 Necrotic fluid collection 527
 Necrotizing sialometaplasia 233, 234
 Necrotoxicogenic *E. coli* (NTEC) 318
 Nematodes 136
 Neoplasia
 - colorectal 381
 - cytology 120
 - esophageal 264
 - exocrine pancreas 522
 - gastric 69
 - hepatic 126
 - large intestine 379
 - rectal 382
 - salivary glands 232
 - small intestines 348*Neorickettsia helminthoeca* 137
 Nervous system
 - autonomic 75
 - central 35
 - enteric 162, 301
 - intrinsic 355
 NET see Nasoesophageal tube 213
 Neuroendocrine carcinoma 145
 Neuroendocrine cells 497

- Neuroendocrine tumors (NET) 557, 560, 573
- carcinoids 572
 - gastrinoma 566
 - glucagonoma 570
 - insulinoma 560
 - pancreatic polypeptidoma 573
 - somatostinoma 572
- Neurotensin 558, 559
- Neurotransmitters 355
- Neutering 576
- Neutrophilic cholangitis 154
- Neutrophilic enteritis 542
- Neutrophilic extravascular traps (nETs) 511
- Neutrophilic inflammation 125
- NG tube see Nasogastric tube 213
- Nizatidine 289, 378
- motility disorders 287, 288
- N-methylhistamine (NMH) 30
- Non-steroidal anti-inflammatory drugs (NSAIDs)
- gastric ulceration 280
 - gastritis 271
 - gastrointestinal disturbances 3
 - hepatotoxicity 412
 - pancreatitis 511
- Normal range 31
- Norwegian lundehund
- disease predisposition 4, 5
- NPO (nothing per os) 510
- NSAIDs see Non-steroidal anti-inflammatory drugs 3, 271
- Nuclear factor-kappa B (NF- κ B) 503
- Nuclear scintigraphy 58
- Nutritional management
- gastrointestinal diseases 203, 208
- Nutritional status assessment 203
- O**
- Obesity
- clinical signs 581
 - definition 574
 - diagnosis 582
 - dietary management 589
 - disorders, associated 578
 - etiology 575
 - genetic causes 575
 - lifespan 578
 - management 588
 - pathogenesis 577
 - physical activity 591
 - prevalence 574
 - prevention 594
 - risk factors 575
 - sex associations 576
- Obesity see also adipositas 573
- Obstipation 372
- definition 192
- Obstruction
- esophageal 246
 - intestinal 337
 - mechanical 168
 - small intestines 72, 75
 - vomiting 168
- Occult blood 545
- Oclacitinib 232
- Octreotide 566
- gastrinoma 569
 - glucagonoma 572
 - hypergastrinemia 340
 - pancreatitis 513
 - PLE 346
- Odansetron 274
- Odontogenic fibroma 235
- Oligoclonal profile 54
- Ollulanus tricuspis* 24, 133, 277
- Olsalazine 550
- Omentalization 526, 528
- Omeprazole 274
- EPI 522
 - esophagitis 244
 - feline hepatic lipidosis (FHL) 433
 - gastrinoma 569
 - gastrointestinal ulceration 458, 459, 460
 - GERD 245
 - hypergastrinemia 340
 - liver disease 409, 410
- Ondansetron
- feline hepatic lipidosis (FHL) 433
 - liver disease 409, 410
 - pancreatitis 511
- Online mendelian inheritance in animals (OMIA) 45
- Opioids 558, 559
- Opisthorchiidae 492
- Opisthorchis felineus* 492, 493
- Oral cavity
- anatomy 227
 - diseases 228
 - neoplastic conditions 235
 - physiology 227
- Oral dysphagia 5
- Oral mucosa 227
- Oral tumors
- benign 235
 - biologic behavior 236
 - diagnosis 235
 - prognosis 237
 - staging 235
 - treatment 237
- Oriental shorthair, disease predisposition 5
- Oropharynx
- diagnostic imaging 59
 - examination 21
 - imaging 59
 - swallowing 240
- Osteosarcoma (OSA) 235
- Ovariohysterectomy 576
- Overall survival (OS), hepatic neoplasia 469
- Overweight 574, 575
- Oxygen therapy 513
- Oxyntic glands 267
- Oxyntomodulin 558, 559
- P**
- Packed cell volume 24
- Pain
- abdominal see abdominal pain 504
 - biliary 479
- Palate
- cleft 20
 - elongated/edematous 18
 - eosinophilic granuloma 231
 - hard 227
 - soft 227
- Pale mucous membranes 19
- Palpation
- abdominal 21
 - rectal 23
- Pancreas
- abscess 116, 502
 - adenocarcinoma 129
 - metastatic 127
 - anatomy 497
 - aplasia 516
 - atrophy 115
 - bacterial infection 502
 - biopsy 91, 114, 157, 508
 - cysts 161
 - cytology 128, 508
 - ducts 497
 - exocrine 497
 - fluid collection 526
 - necrotic 526
 - FNA 91
 - fungal infiltration 502
 - histology 157, 497
 - histopathology 156, 157, 500
 - hyperplasia 159
 - hypoperfusion 502
 - hypoplasia 516, 517
 - imaging 91
 - inflammation 501
 - insulinoma 561, 564
 - lipomatosis 161

- neoplasia 92, 159, 524
- palpation 22
- parasites 528
- surgery 564
- Pancreatectomy 513, 525
 - gastrinoma 569
 - insulinoma 565
- Pancreatic acinar atrophy (PAA) 516, 517
- Pancreatic acinar cells, lacking 518
- Pancreatic adenocarcinoma 522
- Pancreatic adenoma 522, 525
- Pancreatic bladder 528
- Pancreatic carcinoma (PaCa) 523, 525
 - breed disposition 524
- Pancreatic duct 399
 - obstruction 516
- Pancreatic enzyme replacement therapy (PERT) 520
- Pancreatic enzymes 498, 520
- Pancreatic fibrosis 505
- Pancreatic lipase 518
- Pancreatic lipase immunoreactivity (PLI)
 - assays 37
 - pancreatitis 507
- Pancreatic lipase-related proteins 1/2 (PLRP1/2) 498
- Pancreatic necrosis 505
 - prognosis 515
 - walled-off 527
- Pancreatic nodular hyperplasia 529
- Pancreaticoduodenectomy 525
- Pancreatic polypeptide (PP) 558, 559
- Pancreatic polypeptidoma 573
- Pancreatic pseudobladder 528
- Pancreatic pseudocyst 526
- Pancreatic sarcoma 523
- Pancreatic secretory trypsin inhibitor (PSTI) 498, 499
- Pancreatitis 158, 499
 - acute 92, 501
 - cytology 509
 - management 509
 - after pancreatic surgery 565
 - analgesia 510
 - autoimmune 514
 - biopsy 158
 - chronic 41, 159, 484, 501, 504, 517
 - cytology 509
 - EPI 516
 - fibrosing 116
 - management 513
 - vomiting 176
 - classification 501
 - clinical presentation 504
 - complications 510
 - contrast-enhanced abdominal CT 506
 - cytology 128
 - definition 499
 - diagnosis 504
 - diet 510, 514
 - drug-induced 502
 - endocrine disorders 503
 - etiology 501
 - hereditary 502
 - histopathological lesions 500
 - hypertriglyceridemia-induced 502, 510
 - imaging 91, 505
 - infectious 502
 - laboratory tests 37
 - magnetic resonance imaging (MRI) 506
 - markers 506
 - necrotizing 526
 - pathogenesis 503
 - prednisone 515
 - prognosis 515
 - surgery 513
- Pancreatolithiasis 528
- Panleukopenia, feline 305
- Pantoprazole 274
- Panzquin® 521
- Papules 20
- Paraneoplastic alopecia 20
- Paraneoplastic hypoglycemia 296
- Paraneoplastic syndrome 266, 349
- Paraplegia 17
- Parasitic disease
 - biliary system 492
 - cholangitis 154
 - chronic 138
 - contamination management 323
 - gastric 24, 133
 - gastritis 277
 - intestinal 136
 - pancreatic 528
 - small intestines 320
 - vomiting 176
- Paratracheal area
 - examination 21
- Parietal cells 270
- Parotis 228
- Parvovirus 135, 305
 - bone marrow infection 308
 - clinical pathology 310
 - clinical signs 307
 - diagnosis 310
 - diagnostic imaging 311
 - disease pathogenesis 307
 - enteritis 301
 - pathology 311
 - physical examination 309
 - prevention 314
 - prognosis 313
 - treatment 311
- tropism 307
- vaccination 314
- vomiting 168
- Pathogen-associated molecular patterns (PAMPs) 539
- Pathogens 358
- Patient history
 - acute signs 170
 - diarrhea 170
 - regurgitation 170
 - vomiting 170, 178
- Pattern recognition receptors (PRRs) 358, 539
- PEGtube 217, 219
 - low-profile balloon-tipped 219
 - placement 219
- Pekinese, disease predisposition 4, 5
- Peliosis hepatis 149
- Pelvic canal obstruction 23
- Pembroke Welsh corgi, disease predisposition 4, 5
- Peptide YY 558, 559
- Percutaneous endoscopic gastrostomy (PEG) 109
- Percutaneous ultrasound-guided cholecystocentesis 91, 485
 - liver flukes 494
- Perianal adenocarcinoma 390, 391
 - diagnostics 391
 - treatment 392
- Perianal adenoma
 - clinical presentation 391
 - incidence 390
 - treatment 392
- Perianal fistula 386
 - clinical presentation 386
 - diagnosis 387
 - monitoring 387
 - therapy 387
- Perianal neoplasia 390
- Perineal area examination 17, 23
- Perineal herniation
 - round stools 14
- Periodontal ligament 227
- Periodontium 227
- Peripancreatic fat necrosis 503
- Peripancreatic fluid collection 526
- Peripheral lymph nodes 19
 - enlarged 19
- Peritoneal effusion 21
- Peritoneopericardial hernia 52
- Persian, disease predisposition 5
- Persistent right aortic arch 52, 263
 - megaesophagus 218
- Petechiae 19
- Petrolatum 377

- Peyer's patches 304, 358
 Pharmacological food reactions 531
 Pharyngeal dysphagia 5
 Pharyngoesophageal sphincter 239
 Pharynx
 – examination 20
 – imaging 59
 Phenobarbital
 – hepatotoxicity 412
 – serum liver enzyme activities 33
 Phosphate-containing suppositories 196
 Phosphofructokinase defect 437
Physaloptera 133, 277
 – *rara* 24
 Physical activity 591
 Physical examination 16
 – acute signs 171
 – diarrhea 171
 – general 16
 – nutritional health 203
 – regurgitation 171
 – vomiting 171, 178
 Pigment stones 490
 Plasma
 – lactate concentration 25
 – pancreatitis 512
 – proteins 403
 – tracers 164
 Plasma cell tumors, extramedullary 145
 Platelet-activating factor antagonists (PAFANTs) 511
 Platynosomosis 489
Platynosomum fastosum 492
 PLE see Protein-losing enteropathy 207
 Poikilocytosis 36
 Polyacrylic acid-silicone gradual occlusion devices (PAS-OD) 425
 Polyclonal multicapillary electrophoresis 54
 Polydipsia 407
 Polyethylene glycol 375
 Polyethylene glycol solution 376
 Polymerase chain reaction for antigen receptor rearrangement (PARR) 52, 279
 Polypeptidoma, pancreatic 573
 Polypoid mass 107, 483
 Polyps 381
 – hyperplastic 143
 – rectal prolapse 383
 Polystomatic sublingual gland 234
 Polyuria 407
 Popliteal lymph nodes 19
 Portal blood flow 462
 Portal circulation 399
 Portal hypertension 407
 – after CPSS surgery 427
 – hepatitis 453
 – non-cirrhotic 461
 Portal triad 395
 – abnormal 466
 Portal vein 395, 399
 – hypoperfusion 149
 – hypoplasia 461, 462, 466
 -- cats 465
 -- dogs 463
 -- treatment 464
 Portography 117
 Porto-portal fibrosis 466
 Portosystemic shunt (PSS)
 – bile acids 35
 – extrahepatic 84
 – hereditary 52
 Portovenogram 424
 Post-attenuation neurological signs (PANS) 427
 Postoperative ileus (POI) 282, 336
 Posture abnormalities 17
 Potassium secretion 356
 Prayer position 15, 17
 Praziquantel 489
 – liver flukes 494
 Prebiotics 334, 371, 547
 Prednisolone 383
 – cholangitis 489
 – chronic inflammatory enteropathy (CIE) 548
 – gastritis 279
 – immune-mediated hepatitis 458, 459, 460
 – insulinoma 565
 – pancreatitis 515
 Prednisone 383
 – chronic inflammatory enteropathy (CIE) 548
 – gastritis 279
 – insulinoma 565
 – perianal fistula 387
 Pre-obesity 574
 Primary copper associated hepatopathy 52
 Primidone
 – hepatotoxicity 412
 – serum liver enzyme activities 33
 Probiotic-responsive enteropathy (PRE) 187
 Probiotics 197, 334, 547
 Proctitis 382
 Proctoscopy 98
 – large intestinal neoplasia 381
 Progression-free survival (PFS), hepatic neoplasia 469
 Projectile vomiting 7
 Prokinetic drugs 197, 286, 287, 288
 – colonic 378
 – esophagitis 244
 Pro-opiomelanocortin C (POMC) gene mutation 575
 Propantheline 370
 Prostaglandins 271
 Prostate examination 23
 Protease inhibitors 512
 Protein
 – hydrolyzed 209
 – limited-ingredient novel 210
 – loss, assessment 29
 – metabolism 400
 Protein hydrolysate diets 535
 Protein-losing enteropathy (PLE) 341
 – abdominal effusion 21
 – antithrombotics 550
 – body condition 207
 – clinical presentation 341
 – diagnostic approach 342
 – diagnostic evaluation 341
 – diagnostic imaging 343
 – duodenal 134
 – histopathology 344
 – imaging 79
 – laboratory parameters 341
 – medical management 346
 – nutritional management 344
 – prognosis 347
 – soft-coated wheaten terrier 534, 541
 – treatment 344
 – ultrasonography 344
Proteobacteria 356
 Prothrombin 36
 Prothrombin time (PT) 36
 Proton pump inhibitors (PPI) 274
 – esophagitis 244
 – gastric ulceration 280
 – gastrinoma 568
 – GERD 245
Prototheca 140, 326
 Protothecosis 326
 – diarrhea 170
 Prucalopride 286, 378
 – motility disorders 287, 288
 Pruritus 20
 – adverse food reaction 533
 – anal 15
 Pseudobladder, pancreatic 528
 Pseudoclonal pattern 54
 Pseudocyst, pancreatic 526
Pseudomonas aeruginosa 502
 Pseudoptyalism 20
 Psyllium 211, 371, 377

- Ptyalism 20
 Pulp 227
 Pulse rate 18
 Pulsion diverticulum 257
 Pumpkin 377
 Purse-string suture 384
 Pyloric glands 268
 Pyloric hypertrophy 276
 Pyloric obstruction, chronic 69
 Pyogranulomatous inflammation 21
 Pyrantel pamoate
 – stomach worms 273
 Pyridoxine deficiency 442
 Pythiosis 138
 Pythium insidiosum 278, 325
- Q**
- Questioning the owner 3
- R**
- Radiation-induced liver disease (RILD) 471
 Radiation therapy
 – hepatic neoplasia 471
 – intestinal neoplasia 350
 – oral tumors 237
 – rectal tumors 392
 Radiography 58
 – biliary tract 81
 – esophagus 63
 – gastrointestinal motility 163
 – large intestine 79
 – liver 81
 – megacolon 374
 – oropharynx 59
 – pancreatic neoplasia 524
 – stomach 66
 Radioisotope studies 26, 30
 Radionuclide scintigraphy 164
 Ranitidine 289, 378
 – motility disorders 287, 288
 Rapid breathing 19
 Raw meat-based diets (RMBD) 315
 Reactive oxygen species (ROS) 513
 Recombinant feline interferon omega (rFeIFN- ω) 230
 Rectal adenocarcinoma 144
 Rectal adenoma 144
 Rectal area examination 17
 Rectal biopsy 100
 Rectal constipation 192
 Rectal examination 23
 – perianal fistula 387
 Rectal neoplasia 390
 Rectal polyps 381
 Rectal prolapse 383
 – advanced 384
 – treatment 384
 Rectal stenting 382
 Rectal tumors 381
 Rectoanal malformations 384
 Refeeding syndrome 223
 Reference intervals 31
 Regional lymph node aspirates 237
 Regurgitation
 – clinical evaluation 167
 – diagnostic imaging 59
 – diagnostic testing 171
 – differentials 169
 – differentiation 6
 – esophageal dysphagia 5, 6
 – patient history 170
 – physical examination 171
 Respiratory rate 18
 Resting energy requirement (RER) 208, 432
 Restlessness, abdominal pain 15
 Restricted copper diet 458, 459, 460
 Restricted sodium diet 409, 410
 Retching 6, 7
 Rhythmic segmentation 355
 Ribbon-like stools 14
 Rifaximin 370
 Rivaroxaban 409, 410
 Ronidazole 368
 Rotavirus 301
 Rottweiler, disease predisposition 4, 5
 Rough collie, disease predisposition 4, 5
 Rough endoplasmatic reticulum (rER) 498
 Round cell tumor 292
 Routine clinical chemistry panels 24
 Ruga/Rugae 300
- S**
- Saccharomyces* 357
 – *boulardii* 335
 Sacculectomy, anal 387
 S-adenosyl-methionine (S-AdoMet) 442
 – chronic hepatitis 458, 459, 460
 – feline hepatic lipidosis (FHL) 433
 – gallbladder mucocele 479
 Sago palm 414
 Saliva 227
 Salivary disorders 232
 Salivary glands 227, 228
 – neoplasia 232, 233, 234
 Salivary overproduction 20
 Salmonella 136, 317
 – hepatitis 418
 Salmon poisoning 137, 169
 Sarcina-like bacteria 133
 Sarcoma
 – esophageal 264
 – gastric 292
 – intestinal 348
 Sarcopenia 188
 Saw horse stance 15
 Schistocytes 36
 Schistosomiasis 140
 – dogs 323
 Scintigraphy
 – gastric emptying 283
 – nuclear 164
 – somatostatin receptor 564
 – splenic portal 423
 Scirrhus reaction 292
 Scleral hemorrhage 19
 Scottish terrier
 – alkaline phosphatase (ALKP), increased 34
 – disease predisposition 4, 5
 Secretin 302, 557
 – gastrinoma 569
 Secretory IgA deficiency 304
 Selected protein diets 535
 Semaglutide 588
 Septic bilious effusion 485
 Septic mucous membranes 19
 Septic shock 18
 Serosa 299
 Serotonin (5-HT₄) receptor agonists 286
 Serum albumin, malnutrition 204
 Serum amylase activity 41
 Serum amyloid A 30
 Serum baseline cortisol
 – PLE 343
 Serum bile acids 35
 Serum biochemistry, sample requirements 35
 Serum cobalamin 26, 29
 – chronic inflammatory enteropathy (CIE) 545
 – diarrhea 186
 – dysbiosis 333
 Serum folate 26, 29
 – chronic inflammatory enteropathy (CIE) 545
 – dysbiosis 333
 Serum gastrin concentration 25
 Serum lipase activity 41
 – pancreatitis 506

Index

- Serum liver enzyme activities 33
 - increased 405
 - hepatic neoplasia 468
- Serum triglyceride concentration
 - hyperlipidemia 435
 - pancreatitis 502
- Serum trypsin-like immunoreactivity (TLI) 28
 - EPI 518
- Serum vitamin B₁₂ concentrations 279
- Shar-pei
 - disease predisposition 4, 5
 - enteropathy 541
- Sheather's sugar flotation solution 494
- Shetland sheepdog
 - disease predisposition 4, 5
 - gallbladder mucocele 478
- Shiga-toxin-producing E. coli (STEC) 318
- Shih tzu, disease predisposition 4, 5
- Shiny skin 20
- Short bowel syndrome (SBS) 340
- Short-chain fatty acids (SCFA) 304, 330, 357, 371, 376
- Sialadenectomy 234
- Sialadenitis 233
 - treatment 234
- Sialadenosis 233
 - diagnosis 234
- Sialocele 232
 - fine-needle aspiration 233
 - treatment 234
- Sialography 233
- Sialoliths 233
- Sialometaplasia, necrotizing 233
- Siamese, disease predisposition 5
- Signalment 3
- Signet ring adenocarcinoma 293
- Silibinin 413
- Silymarin 458, 459, 460
- Skeletal growth and development 16
- Skin
 - biopsy, perianal fistula 387
 - diseases, adverse food reaction 532
 - examination 19
 - lesions 20
 - glucagonoma 571
 - shiny 20
 - turgor 20
- Skye terrier
 - disease predisposition 4, 5
- Slow-wave activity 355
- Small bowel
 - diarrhea 11
 - disease 7
 - loops
 - aggregated 22
 - fluid-distended 22
- Small-cell lymphoma (SCL) 142, 143, 551, 553
- Small intestines/Small intestinal
 - anatomy 299
 - bacterial infection 315
 - bacterial overgrowth 537
 - biopsy 113
 - diarrhea 167
 - differentials 169
 - diseases 305
 - divisions 300
 - foreign body 73, 337
 - imaging 72
 - inflammation 134
 - chronic 537
 - intussusception 338
 - layers 299
 - motility disorders 336
 - neoplasia 74, 348
 - obstruction 337
 - complete 75
 - partial 72
 - palpation 22
 - parasitic diseases 320
 - physiology 301
 - surface area 300
 - torsion 339
- Small T-cell lymphoma 120
- SmartPill® capsule 165
- SNAP cPL 507, 508
- SNAP fPL 507
- Sodium absorption 356
- Soft-coated wheaten terrier
 - disease predisposition 4, 5
 - protein-losing enteropathy (PLE) 534, 541
 - and nephropathy 29
- Soft tissue swelling 20
- Soluble fibers 211
- Somatostatin 303, 558, 559, 566
- Somatostatinoma 572
- Somatostatin receptor scintigraphy 564
- Spastic colon 369
- Spec cPL/Spec fPL 507
- Specific canine pancreatic lipase 38, 343
- Specific feline pancreatic lipase 39
- Sphincter of Oddi 497
- SPINK1 gene mutation 499, 501
- Spirocerca lupi 133
 - esophageal sarcoma 264
- Spironolactone 409, 410, 458, 459, 460
- Spleen, abdominal palpation 23
- Splenomegaly 23
- Splenoportography 117
- Spleno systemic shunt 423
- Squamous cell carcinoma (SCC)
 - esophageal 264, 265
 - oral 235
- Standard poodle, disease predisposition 4, 5
- Star gazing 17
- Stellate cells 397, 452
- Stem cell therapy 550
 - gingivostomatitis 230
- Stereotactic body radiation therapy (SBRT) 471
- Steroid hepatopathy 438
- Steroid-induced hepatopathy 150
- Steroid-responsive enteropathy (SRE) 537
- Sterols 400
- Stimulant laxatives 377
- Stomach
 - anatomy 267
 - biopsy 99
 - cytology 119
 - decompressing 291
 - diseases 271
 - imaging 66
 - inflammation 130
 - motility disorders 280
 - neoplastic conditions 291
 - palpation 22
 - physiology 268
 - tumors 292
 - ulceration 279
 - worms 273
 - vomiting 168
- Stomach see also Gastric ... 7
- Stool
 - ribbon-like 14
 - round 14
- Stricture, esophageal 250, 251, 255
- Strongyloides 137, 141
- Stunted growth 16
- Stupor 17
- Subcutaneous tissue, examination 19
- Sublingual linear foreign body 21
- Sublingual region, examination 21
- Sublingual salivary glands 228
- Submucosa
 - gastric 267
 - intestinal 299

- Sucralfate
- esophagitis 244
 - gastrinoma 569
 - liver disease 409, 410
- Sucrose permeability testing 25
- Sugar probes 26, 30
- Sulfasalazine 383, 549
- Superficial necrolytic dermatitis 20
- Suppositories 195
- Swallowing 20, 61, 240
- disorders 60
 - esophageal phase 240
 - gastroesophageal sphincter relaxation 240
 - oropharyngeal phase 240
 - painful see Dysphagia 5
 - physiology 240
 - studies 61
- Systemic lupus erythematosus (SLE), megaesophagus 19
- T**
- Tachycardia 18
- Tachykinins 558, 559
- Target lesion 85
- Taurine 402
- feline hepatic lipidosis (FHL) 433
 - source 205
 - synthetic 205
- T-cell lymphoma 553
- intestinal 121
- T-cell receptor gamma-chain (TCRG) gene 53
- T cells 304, 358
- Teeth 227
- grinding see Bruxism 15
- Telescopes 111
- Temperature evaluation 17
- Tenesmus 14, 23
- Thermometer 17
- Thiazide diuretics 566
- Thin film bands (TFB) 425
- Thioureylenes, hepatotoxicity 412
- Thromboembolism 550
- Thyroid arteries 240
- Thyroid gland enlargement 21
- TLR5 mutation 539
- Toceranib 351, 392
- Toll-like receptors (TLRs) 539
- Tongue 228
- eosinophilic granuloma 231
 - examination 21
- Tonsillitis 21
- Tooth extraction 229
- Torsion
- gastric 290
 - intestinal 339
- Total protein concentration 24
- Toxocara canis* 320, 321
- Toxoplasma gondii* 128
- pancreas 502, 528
- Tracers 164
- Traction diverticulum 257
- Transamination 400
- Transarterial chemoembolization (TACE) 470
- Transverse colon 353
- Trefoil factor 1/2 (TFF 1/TFF 2) 267
- Trematodes 137
- eggs identification 494
- Triaditis 514
- Trichobezoar
- cats 177
 - esophageal 246
- Trichuriasis 141
- Trichuris vulpis* 359
- proctitis 382
- Trientine 458, 459, 460
- Triglycerides 400
- Trimethoprim-sulphonamides 412
- Tritrichomonas blagburni* 141, 367, 540
- diagnosis 367
 - infection 367
 - therapy 368
- Tritrichomoniasis, diagnostic testing 173
- Trituration 280
- Trypsin 498
- Trypsin-like immunoreactivity (TLI) 41, 42, 333
- pancreatitis 506
- Trypsinogen 498
- pancreatitis 503
- Tubes, feeding 220
- Tumor
- esophageal 263
 - gastric 294
 - intestinal 348
 - oral 235
- Two-hit model 504
- Tylosin
- *Clostridium perfringens* enterotoxigenesis 366
 - dysbiosis 334
 - EPI 522
- Tyrosine kinase inhibitors (TKI) 382
- Tyzzler's disease 141, 319
- U**
- Ulcer(ation)
- dermal 20
 - gastroduodenal 408
- Ultrasonography 58
- oropharynx 59, 60
 - (trans-)abdominal
 - biliary tract 81, 91
 - gastrointestinal motility 163
 - ileus 75
 - jaundice 200
 - large intestine 80
 - liver 81
 - pancreas 91
 - pancreatitis 505
 - protein-losing enteropathy (PLE) 344
 - stomach 67
- Ultrasound-guided fine-needle aspiration (FNA), pancreas 508
- Upper esophageal sphincter (UES) 239
- impairment of maximal opening diameter 241
- Urate urolithiasis 36
- Urea 400
- Urea cycle enzyme deficiency (UCED) 446
- Uremic gastropathy 146
- Urinalysis 36
- Ursodeoxycholic acid 458, 459, 460
- cholangitis 489
 - cholelithiasis 492
 - gallbladder mucocele 479
 - liver flukes 490
- Uteromegaly 23
- Uterus, abdominal palpation 23
- V**
- Vaccination
- history 3
 - parvovirus 305, 314
- Vagus nerve 240
- Vascular disorders, congenital 461
- Vascular ring anomalies 263
- Vasoactive intestinal polypeptide (VIP) 558, 559
- Vcheck® cPL/Vcheck® fPL 508
- Vena cava 399
- Veress needle 112
- Verocytotoxin-producing *E. coli* (VTEC) 318
- Very low-density lipoprotein (VLDL) 400, 401
- Veterinary canned diet 216

Index

- Veterinary liquid enteral products 216
 - VetScan® cPL rapid assay 508
 - Videofluoroscopy
 - balloon dilatation 254
 - cricopharyngeus muscle achalasia 242
 - hiatal hernia 259
 - swallow study 60, 242
 - Villus/Villi 300, 301
 - Vitamin A 402
 - Vitamin B₆
 - deficiency 442
 - Vitamin B₁₂ 26
 - feline hepatic lipidosis (FHL) 433
 - supplementation 340
 - Vitamin D
 - chronic inflammatory enteropathy (CIE) 545
 - Vitamin E 442
 - chronic hepatitis 458, 459, 460
 - Vitamin K 36
 - Vitamin K₁
 - cholestatic liver disease 409, 410
 - feline hepatic lipidosis (FHL) 433
 - Vitamin, storage 402
 - Vitrear hemorrhage 19
 - VLDL see very-low density lipoprotein 401
 - Volvulus
 - gastric see Gastric dilatation-volvulus 18
 - intestinal 21
 - Vomiting 6
 - acute 6
 - chronic 6, 24, 173
 - causes 175, 176
 - gastric causes 67
 - chronic inflammatory enteropathy 543
 - clinical evaluation 167, 173
 - diagnostic approach 179
 - diagnostic imaging 180
 - diagnostic testing 171
 - differentials 168
 - differentiation 6, 177
 - exocrine pancreatic neoplasia 524
 - exploratory laparotomy 180
 - extra-GI causes 168
 - feline hepatic lipidosis (FHL) 432
 - gastric emptying disorder 281
 - gastric tumors 294, 296
 - gastritis 272
 - hepatic encephalopathy (HE) 408
 - intoxication 272
 - material characteristics 177
 - patient history 170, 178
 - physical examination 171, 178
 - shortly after abdominal palpation 22
 - therapeutic trials 179
 - treatment 274
 - Walled-off pancreatic necrosis 527
 - Water absorption 356
 - Weight loss 16
 - chronic 188, 191
 - chronic inflammatory enteropathy 544
 - clinical assessment 189
 - definition 188
 - diagnostic investigations 191
 - differential diagnoses 190
 - EPI 518
 - epidemiology 189
 - exocrine pancreas neoplasia 524
 - historical assessment 189
 - involuntary 190
 - pathophysiology 189
 - physical assessment 189
 - treatment 192
 - Weight management 588
 - outcomes 593
 - West Highland white terrier
 - disease predisposition 4, 5
 - Wheat bran 377
 - Whipple's triad 561
 - Whipworm
 - eggs 360
 - infection
 - clinical presentation 360
 - diagnostic evaluation 360
 - pathophysiology 359
 - prognosis 361
 - treatment 361
 - Wilson's disease gene mutation 404
 - Wireless motility capsule (WMC) 165
 - World Small Animal Veterinary Association (WSAVA) Global Nutrition Committee 203
 - Wound healing, poor 203
 - WSAVA GI Standardization Group 546
- ## W
- Walled-off pancreatic necrosis 527
 - Water absorption 356
 - Weight loss 16
 - chronic 188, 191
 - chronic inflammatory enteropathy 544
 - clinical assessment 189
 - definition 188
 - diagnostic investigations 191
 - differential diagnoses 190
 - EPI 518
 - epidemiology 189
 - exocrine pancreas neoplasia 524
 - historical assessment 189
 - involuntary 190
 - pathophysiology 189
 - physical assessment 189
 - treatment 192
 - Weight management 588
 - outcomes 593
 - West Highland white terrier
 - disease predisposition 4, 5
- ## X
- Xylitol 412
- ## Y
- Yaw snapping 17
 - Yeast probiotic 335
 - Yersinia enterocolitica* 319
 - Yorkshire terrier, disease predisposition 4, 5
- ## Z
- Zinc (acetate/gluconate) 442, 458, 459, 460
 - Zoometry 585
 - Zygomatic salivary gland 228
 - Zymogens 498, 499
 - pancreatitis 503



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