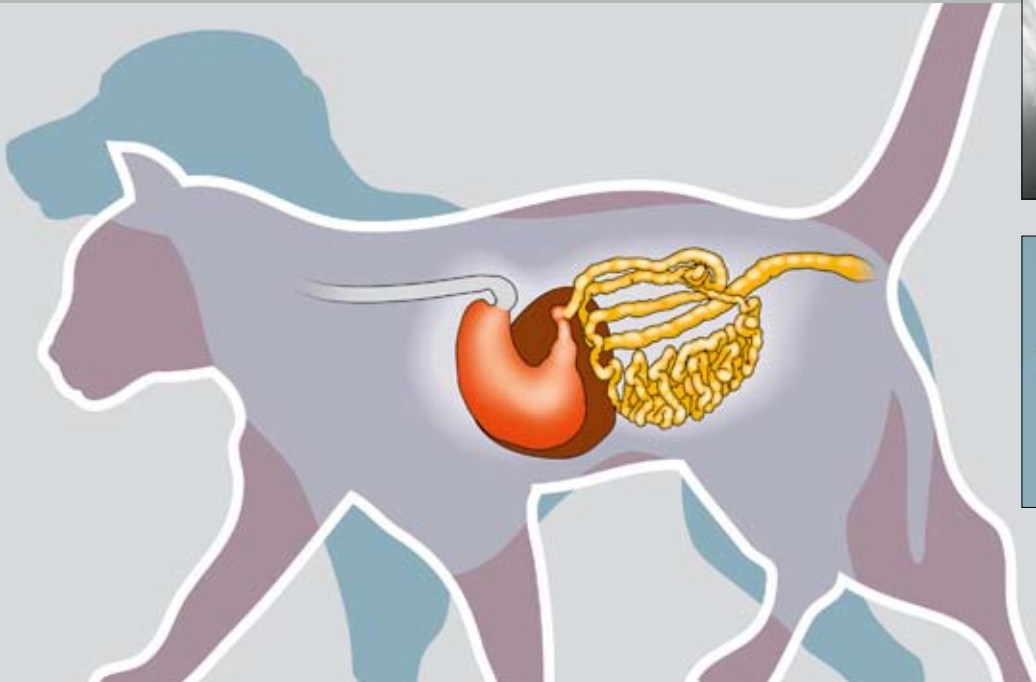


Jörg M. Steiner (ed.)

# Small Animal Gastroenterology

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Gastrointestinal disorders are common in canine and feline patients and diagnosis and treatment require up-to-date knowledge of this expanding field. This book is designed to be both a practical companion for veterinarians in general practice and a concise, yet comprehensive, resource for veterinary students, interns, and residents.

The book is structured into different sections. The first section covers diagnostic tools and provides a rational approach to common clinical problems. In the second section, diseases affecting each portion of the gastrointestinal tract are discussed in detail.



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with

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## Abbreviations

<sup>13</sup> C-OBT	<sup>13</sup> C–octanoic acid breath test	cTLI	canine trypsin-like immunoreactivity
5-ASA	5-aminosalicylic acid	CVP	cyclophosphamide, vincristine, and prednisone
5-HT <sub>3</sub>	5-hydroxytryptamine		
6MP	6-mercaptopurine		
		Da	Dalton
α <sub>1</sub> -PI	alpha <sub>1</sub> -proteinase inhibitor	DDAVP	desmopressin acetate
AC	adenocarcinoma	DIC	disseminated intravascular coagulation
ACh	acetylcholine	DOB	dose over baseline
ACTH	adrenocorticotrophic hormone	DSH	domestic shorthair cat
AgNOR	argyrophilic nucleolar organizer region		
ALP	alkaline phosphatase	EBDO	extrahepatic bile duct obstruction
ALT	alanine aminotransferase	ECG	electrocardiogram
APUDoma	tumors of cells of the amine precursor uptake and decarboxylation system	ECL	enterochromaffin-like
ARD	antibiotic-responsive diarrhea	ED	equilibrium dialysis
AST	aspartate aminotransferase	EE	eosinophilic enteritis
AT-III	antithrombin-III	EEG	electroencephalogram
		EGE	eosinophilic gastroenteritis
BIPS	barium-impregnated polyethylene spheres	EGEC	eosinophilic gastroenterocolitis
BUN	blood urea nitrogen	EGF	epidermal growth factor
BW	body weight	ELISA	enzyme-linked immunosorbent assay
BZ	benzodiazepine	EPEC	enteropathogenic <i>E. coli</i>
		EPI	exocrine pancreatic insufficiency
CAV1	canine adenovirus-1	ERCp	endoscopic retrograde cholangio-pancreatography
CBC	complete blood count	ETEC	enterotoxigenic <i>E. coli</i>
CCK	cholecystokinin		
CCNU	lomustine	FeCoV	feline coronavirus
CD	Crohn's disease	FeLV	feline leukemia virus
cDNA	complementary DNA	FIP	feline infectious peritonitis
CDV	canine distemper virus	FIV	feline immunodeficiency virus
CFU	colony-forming units	FNA	fine needle aspiration
CIBDAI	canine inflammatory bowel disease activity index	FO	foreign object
CK	creatinine kinase	FOS	fructo-oligosaccharides
CLO	<i>Campylobacter-like organism</i>	FPA	fecal proteolytic activity
CNS	central nervous system	fPLI	feline pancreatic lipase immunoreactivity
COX	cyclooxygenase	FPV	feline parvovirus
CPE	<i>Clostridium perfringens enterotoxin</i>	FRLBD	fiber-responsive large bowel diarrhea
cPL	canine pancreatic lipase	fTLI	feline trypsin-like immunoreactivity
cPLI	canine pancreatic lipase immunoreactivity		
CPSS	congenital portosystemic shunt	GABA	gamma-aminobutyric acid
CPV	canine parvovirus	GALT	gut-associated lymphoid tissue
CRI	constant rate infusion	G-CSF	granulocyte colony stimulating factor
CRT	capillary refill time	GDV	gastric dilatation-volvulus
CRTZ	chemoreceptor trigger zone	GER	gastroesophageal reflux
CSF	cerebrospinal fluid	GERD	gastroesophageal reflux disease
CT	computed tomography	GES	gastroesophageal sphincter
		GGT	γ-glutamyl transferase
		GHLO	gastric <i>Helicobacter</i> -like organisms
		GhRH	growth hormone releasing hormone

GI	gastrointestinal	PAA	pancreatic acinar atrophy
GIT	gastrointestinal tract	PABA	para-aminobenzoic acid
GN	glomerulonephritis	PAFANT	platelet activating factor antagonist
GSE	gluten-sensitive enteropathy	pANCA	perinuclear antineutrophilic antibodies
		PAS	periodic acid Schiff
H&E	hematoxylin and eosin	PCR	polymerase chain reaction
H <sub>2</sub> -RA	histamin <sub>2</sub> -receptor antagonists	PCV	packed cell volume
HAS	hemangiosarcoma	PEG	percutaneous endoscopic gastrostomy
HE	hepatic encephalopathy	PGE <sub>1</sub>	prostaglandin E <sub>1</sub>
HGE	hemorrhagic gastroenteritis	P-gp	P-glycoprotein
HGF	hepatocyte growth factor	PI	pulsatility index
HLA	human leukocyte antigen	PIVKA	proteins induced by vitamin K antagonism
Htc	hematocrit		
HUC	histiocytic ulcerative colitis	PLE	protein-losing enteropathy
		PLI	pancreatic lipase immunoreactivity
IBD	inflammatory bowel disease	PLN	protein-losing nephropathy
IBS	irritable bowel syndrome	PO	per os
IEL	intraepithelial lymphocyte	PPI	proton pump inhibitor
IF	intrinsic factor	PP	Peyer's patch
IFA	immunofluorescence assay	PSS	portosystemic shunt
IFCR	intrinsic factor-cobalamin receptor	PSTI	pancreatic secretory trypsin inhibitor
IFN- $\gamma$	interferon $\gamma$	PT	prothrombin time (i.e., one-stage prothrombin time)
IGF	insulin-like growth factor	PTT	partial thromboplastin time (i.e., activated partial thromboplastin time)
IHC	immunohistochemistry		
IL	interleukin	PU/PD	polydipsia
KCS	keratoconjunctivitis sicca		
KIT	CD117	q	( <i>quodque</i> ) every
		Q-PCR	quantitative polymerase chain reaction
L/R ratio	lactulose/rhamnose ratio		
LES	lower esophageal sphincter	RAST	radio-allergosorbent test
LI	large intestinal	RBC	red blood cell
LP	lamina propria	RI	resistive indice
LPC	lymphocytic-plasmacytic colitis	RIA	radioimmunoassay
LPE	lymphocytic-plasmacytic enteritis	ROS	reactive oxygen species
LSA	lymphosarcoma	RT-PCR	reverse transcriptase polymerase chain reaction
MAb	monoclonal antibody	SAF	sodium acetate/acetic acid/ formaldehyde
MALT	mucosal associated lymphoid tissue		
MCT	mast cell tumors	SAME	S-adenosyl methionine
MCT	medium-chain triglyceride	SBA	serum bile acids
MEN	multiple endocrine neoplasia	SI	small intestinal
MER	maintenance energy requirement	SIBO	small intestinal bacterial overgrowth
MHC	major histocompatibility complex	sIgA	secretory IgA
MRI	magnetic resonance imaging	SLE	systemic lupus erythematosus
MST	median survival time	SND	superficial necrolytic dermatitis
MVD	microvascular dysplasia	SNP	single nucleotide polymorphisms
		SPF	specific pathogen free
NET	neuroendocrine tumor	spp.	species
NK <sub>1</sub>	neurokinin <sub>1</sub>	SRS	somatostatin receptor scintigraphy
NME	necrolytic migratory erythema	sst2	somatostatin receptor subtype 2
NO	nitric oxide	STEC	Shiga-toxin-producing <i>E. coli</i>
NPO	nothing per os	SUCA	serum unconjugated cholic acid
NSAID	non-steroidal anti-inflammatory drug		
NTZ	nitazoxanide		

T4	thyroxine	UA	urinalysis
TAP	trypsinogen activation peptide	UC	ulcerative colitis
TFF	trefoil factor		
TGF	transforming growth factor	VIPoma	vasoactive intestinal polypeptidoma
Th1	T-helper cell, type 1	VLDL	very low density lipoproteins
Th2	T-helper cell, type 2	vWF	von Willebrand's factor
THV	terminal hepatic veins		
TLI	trypsin-like immunoreactivity	WSAVA	World Small Animal Veterinary Association
TNF	tumor necrosis factor		
TPMT	thiopurine methyltransferase	X/M ratio	xylose /3-O-methylglucose ratio
TRD	tylosin-responsive diarrhea		
TS	total solids	ZSC	zinc sulfate concentration centrifugation
		ZSFC	zinc sulfate fecal centrifugation

*Dedication*

This book is dedicated to Edward Anthony Kane.

## Preface

While much still remains to be discovered, our understanding of gastrointestinal diseases in dogs and cats is steadily progressing. This book aims to highlight and consolidate recent advances.

It has taken several years to complete this project and I feel very fortunate to have worked with such a fantastic group of authors from around the globe. Our goal was a textbook that is both scientific and practical. To that end, in addition to discussing gastrointestinal diseases, diagnostic modalities and

common clinical problems are discussed separately. We have tried to keep the material brief, but have provided pertinent references for readers wishing to evaluate the scientific evidence behind our observations.

We hope that this book helps you care for your patients, as ultimately, it was written for their benefit.

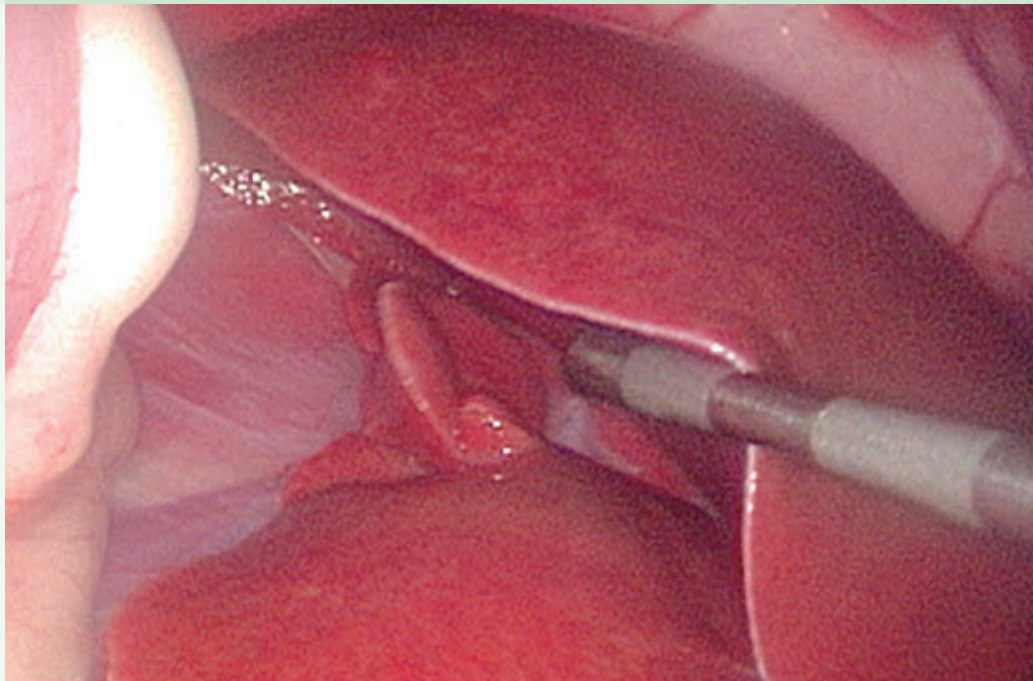
*College Station, January 2008*

*Jörg M. Steiner*



# Part I

## Diagnosis of Gastrointestinal Disorders





# 1 Diagnostic Tools

## 1.1 Clinical History

OLIVIER DOSSIN

### 1.1.1 Introduction

For most clinical problems, acquiring an accurate history can be as important as the clinical findings on physical examination. This is especially true for gastrointestinal disorders since they are usually expressed by clinical signs that cannot be observed during the clinical examination but are only reported by the owner. Therefore, the clinician must be skilled in obtaining the most accurate information by questioning the owner, which needs to be adapted to each case.

The steps and general guidelines for a good history are shown in the Tables 1.1 and 1.2. The observations of the owner, which are valuable, must be differentiated from his or her conclusion or interpretation, which may be misleading. For example, the terms vomiting and regurgitation may be used synonymously by the owner. In order to avoid confusion, it is essential to ask the owner to describe the patient's symptoms with his or her own words.

The signalment can be helpful as some age or breed predispositions (Tables 1.3 and 1.4) have been proposed for various gastrointestinal disorders. Also, a complete vaccination and drug history is important. Many drugs can induce gastrointestinal disturbances (e.g., NSAIDs can cause gastric ulcers and some antibiotic agents can be associated with diarrhea). Intolerance of anesthetic agents has been reported in patients with hepatic disorders, especially in those with portosystemic vascular anomalies.<sup>1</sup>

Written records of the history are essential for the follow-up. As a general rule, everything that can be quantified during history taking should be, as this could be beneficial for the assessment of the severity of the problem or for the follow-up.

### 1.1.2 History of specific gastrointestinal signs

This section focuses on the main specific gastrointestinal signs, but other, less specific signs, such as anorexia, weight loss, or polyuria/polydipsia should also not be overlooked.

**Table 1.1: Steps for taking a history in patients with gastrointestinal signs<sup>2,7,8</sup>**

#### Parameters addressed

- Signalment
- Chief complaint
- Present medical history (including chronology and treatments)
- Past medical history
- Systems review
- Current health status (including environment and dietary history)

**Table 1.2: Guidelines for history acquisition in patients with gastrointestinal signs<sup>10,11</sup>**

#### Guidelines for history acquisition

- always begin with the chief complaint
- start by asking broad questions
- ask narrow questions (questions that can be answered with few words such as "yes", "no", or "I do not know") to confirm previous answers
- try to use the owner's words to avoid confusion about what the owner means
- do not bombard the owner with questions
- avoid combining questions
- try to be reassuring when necessary
- try to focus owner on the main points of the history
- avoid categorical answers or judgments at the time of history collection
- avoid external interruptions

#### 1.1.2.1 Dysphagia and regurgitation

Dysphagia, which is defined as difficult or painful swallowing, can be classified as oral, pharyngeal, or esophageal. A good history can help the clinician to characterize the type of dysphagia. An owner questionnaire has been evaluated for characterization of dysphagia and was shown to be useful for exclusion of oral dysphagia and for the detection of pharyngeal dysphagia; however, it was less sensitive and specific for the assessment of esophageal dysphagia.<sup>2</sup>

Oral dysphagia is characterized by the dropping of food, leaking of water, or abnormally interrupted chewing followed by rejection of food during the meal. Oral dysphagia is associated with disorders of the oral cavity, or with neuromuscular or osteo-articular disorders that impair normal chewing.

Abnormal, repeated swallowing efforts followed by periprandial regurgitation of an alimentary bolus mixed with saliva are

**Table 1.3: Suspected or confirmed breed predispositions for gastrointestinal diseases in dogs<sup>3,6,12</sup>**

Breed	Disease predisposition
Australian Cattle Dog	Portosystemic vascular anomalies
Basenji	Immunoproliferative lymphoplasmacytic enteritis
Bedlington Terrier	Copper-associated chronic hepatitis
Belgian Shepherd	Gastric carcinoma
Brachycephalic breeds	Hiatal hernia, pyloric stenosis
Border Collie	Selective cobalamin malabsorption
Boston Terrier	Pyloric muscular stenosis, vascular ring anomalies
Bouvier des Flandres	Muscular dystrophy associated dysphagia
Boxer	Histiocytic ulcerative colitis, eosinophilic enteritis, lymphoplasmacytic colitis, pyloric muscular stenosis
Cairn Terrier	Portosystemic vascular anomalies
Cocker Spaniel	Chronic hepatitis and cirrhosis
Dalmatian	Copper-associated chronic hepatitis
Doberman Pinscher	Parvoviral enteritis, eosinophilic enteritis, chronic hepatitis
English Bulldog	Vascular ring anomaly, constipation, fecal incontinence
German Shepherd	Exocrine pancreatic insufficiency, megaesophagus, parvoviral enteritis, lymphoplasmacytic enteritis, eosinophilic enteritis, idiopathic hepatic fibrosis, vascular ring anomaly, perianal fistula, small intestinal bacterial overgrowth
Giant Schnauzer	Selective cobalamin malabsorption
Great Dane	Gastric dilation volvulus
Irish Setter	Megaesophagus, gastric dilation volvulus, gluten-sensitive enteropathy, vascular ring anomaly
Irish Wolfhound	Portosystemic vascular anomalies (intrahepatic shunt)
Labrador Retriever	Megaesophagus, portosystemic vascular anomalies, chronic hepatitis
Lhasa Apso	Hypertrophic pyloric gastropathy
Maltese	Hypertrophic pyloric gastropathy, portosystemic vascular anomalies
Miniature Schnauzer	Pancreatitis, portosystemic vascular anomalies
Norwegian Lundehund	Protein-losing enteropathy, lymphangiectasia
Pekingese	Hypertrophic pyloric gastropathy
Rottweiler	Parvoviral enteritis, eosinophilic enteritis
Rough Collie	Exocrine pancreatic insufficiency, gastric carcinoma
Shar Pei	Hepatic amyloidosis, hiatal hernia, protein-losing enteropathy, lymphoplasmacytic enteritis, eosinophilic enteritis, cobalamin deficiency
Shi Tzu	Hypertrophic pyloric gastropathy
Skye Terrier	Copper-associated chronic hepatitis
Soft-coated Wheaten Terrier	Protein-losing enteropathy and/or nephropathy
Standard Poodle	Lobular dissecting hepatitis
West Highland White Terrier	Copper-associated chronic hepatitis
Yorkshire Terrier	Acute pancreatitis, portosystemic vascular anomalies, intestinal lymphangiectasia, hypertrophic pyloric gastropathy

the hallmarks of pharyngeal dysphagia. The swallowing efforts are frequently associated with coughing or choking, and also with gagging at rest.

Esophageal dysphagia is associated with regurgitation, which is a passive process during which an undigested food bolus is thrown up. Sometimes, the food bolus has a cylindrical sausage shape and is covered with mucus (Figure 1.1). The time elapsed between the swallowing of the food and regurgitation of the

bolus is variable but can be quite long, especially when severe esophageal dilation is present. The time period is usually shorter in patients with esophagitis, esophageal stenosis, or esophageal obstruction. In contrast to pharyngeal dysphagia, esophageal dysphagia is usually not associated with swallowing attempts.<sup>3</sup> The history may also reveal respiratory signs such as coughing or nasal discharge, even as the primary complaint. A main task of history taking is to differentiate regurgitation from vomiting (Table 1.5).

**Table 1.4: Suspected or confirmed breed predispositions for gastrointestinal diseases in cats<sup>3,6,13</sup>**

Breed	Disease predisposition
Abyssinian	Hepatic amyloidosis
Manx	Fecal incontinence, constipation
Oriental Shorthair	Hepatic amyloidosis
Persian	Portosystemic vascular anomalies
Siamese	Megaesophagus, hepatic amyloidosis, pyloric stenosis, intestinal tumors

### 1.1.2.2 Gagging

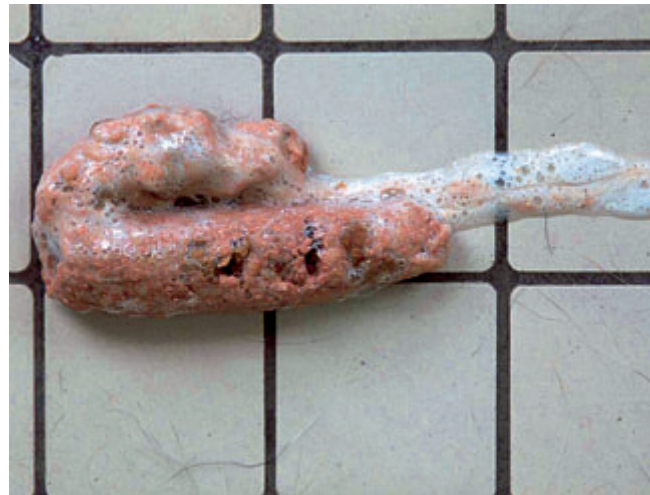
Gagging is defined as swallowing attempts without the presence of an alimentary bolus. Gagging can be a clinical manifestation of dysphagia in patients with pharyngeal disease, but it can also be associated with certain respiratory disorders of the nasal passages, larynx, trachea, or bronchi. Gagging can be associated with ptyalism or retching due to activation of the pharyngeal vomiting receptors.

### 1.1.2.3 Vomiting

Vomiting is not always linked to a primary gastrointestinal tract disorder. It must be differentiated from regurgitation and coughing as discussed before (Table 1.5). Vomiting is an active process with prodromal clinical signs and has three phases. The first phase, nausea, is frequently associated with hypersalivation, agitation or depression, yawning, lip licking, or repeated swallowing attempts. The second phase, retching, appears as a vomiting effort with abdominal wall contractions but without ejection of vomitus and can be associated with belching. The last phase, vomiting, is the forceful ejection of gastric contents, which is associated with very repetitive strong abdominal wall contractions.

Vomiting can be classified as acute or chronic, with chronic being defined as lasting for more than 3 weeks. Also, the content of the vomitus should be described, especially concerning the presence of food, parasites, or foreign bodies. Hematemesis is blood-tinged vomitus containing either fresh blood or digested blood, resembling coffee grounds. Hematemesis is associated with gastric or duodenal erosions and should always be considered as a sign of severe disease. However, small amounts of fresh blood are sometimes related to capillary rupture linked to the rise in venous pressure during vomiting.<sup>4</sup>

Gastritis may be associated with the vomiting of bile on an empty stomach or vomiting of food usually shortly (30 minutes to a few hours) after a meal.<sup>4,5</sup> Vomitus consisting of large volumes of fluid can be associated with ileus, small intestinal obstruction, or hypersecretory states such as gastrinoma.<sup>4</sup>



**Figure 1.1:** Regurgitated food. This figure shows regurgitated food from a dog with esophageal stenosis following general anesthesia for spaying. Note the mucus surrounding a sausage-like bolus of food.

**Table 1.5: Differentiation of regurgitation and vomiting<sup>3,5,6</sup>**

Clinical sign	Regurgitation	Vomiting
<b>Abdominal effort</b>	Absent	Present
<b>Bolus in the neck</b>	Possible	Absent
<b>Prodromal signs</b> (nausea, retching)	Absent (except rare salivation)	Present
<b>Character of ejected material</b>	Undigested	Can be partially digested (depending on time between ingestion and vomiting)
	Bile absent	Bile can be present
	pH variable	pH <5
	Can be tubular shape	Variable shape
<b>Time of ejection</b>	Not reliable	Not reliable
<b>Pain on swallowing</b>	Can be present	Absent

Vomitus that smells like fecal material can sometimes be observed in patients with intestinal obstruction or chronic small intestinal bacterial overgrowth. Vomiting associated with bile that occurs in the morning can be due to duodenal reflux gastritis in small breeds. Vomitus containing food that is produced more than 8 to 12 hours after a meal is highly suggestive of a gastric emptying disorder.<sup>3,5</sup> Projectile vomiting can be observed in patients with a gastric outlet obstruction, but may also simply reflect violent vomiting due to any cause.<sup>6</sup>

1



**Figure 1.2:** Mucoid stool. This figure shows mucoid stools from a dog with severe colitis due to whipworm infestation. Note that the stool is almost exclusively composed of mucus and blood.



**Figure 1.3:** Exocrine pancreatic insufficiency (EPI). Cow-patty yellowish stools from a dog with EPI.

Whenever possible, the number of vomiting episodes should be quantified. This is helpful to plan fluid therapy in patients with acute vomiting and also for the assessment of disease severity (canine IBD activity index; CIBDAI) in patients with chronic vomiting due to inflammatory bowel disease.<sup>7</sup>

#### 1.1.2.4 Retching

Retching is defined by repetitive efforts to vomit without any expulsion of vomitus. Diagnostically, retching should be approached like vomiting. However, in some instances, even a good history can not differentiate retching or the regurgitation of saliva or mucus from coughing and/or expectorations. In patients that present with acute retching and abdominal distension, gastric dilation/volvulus (GDV) should be ruled out immediately.<sup>4</sup>

#### 1.1.2.5 Diarrhea

Diarrhea is defined as the passage of feces that contain more water than normal, which increases daily fecal weight. As for vomiting, the first step is to classify whether the diarrhea is acute or chronic. The next step is to describe the fecal character by asking questions about any blood, foreign material, undigested food particles, or mucus in the feces (Figure 1.2). The color, volume, odor, and consistency of the feces might also be helpful, narrowing the list of differential diagnoses. For example, rancid smelling, large volume, grayish brown, and cow-

patty stools may suggest exocrine pancreatic insufficiency or another condition associated with maldigestion (Figure 1.3). The number of bowel movements per day should also be quantified. The main concern is to localize the origin of the diarrhea either to the small or to the large intestine. The characteristics that may be helpful for localization of the cause of diarrheal disease are shown in Table 1.6. However, these characteristics are not absolute by any means and patients with clinical signs of large bowel disease may have more significant small bowel disease. The use of photographic fecal scoring charts may be helpful for the characterization of the diarrhea (Figure 2.4).

#### 1.1.2.6 Other stool abnormalities

Melena is characterized by black and tarry stools (Figure 1.4) and is sometimes associated with diarrhea. Melena is a sign of the presence of blood in the GI tract. The blood usually comes from the GI tract itself, but it can be swallowed in patients that are bleeding into the respiratory tract. Melena is mostly associated with bleeding of the upper gastrointestinal tract, but the black color and tarry consistency are due to the transit of the blood through the intestinal tract.<sup>3</sup> Therefore, blood from the upper parts of the large intestine can also be black and tarry, and blood coming from the small intestine can be fresh if the transit time does not allow for the digestion of the blood.<sup>3</sup> Some medications, such as metronidazole, ferrous sulfate, or bismuth or some food ingredients (liver, spinach) can also produce dark stools that can sometimes be confused with melena. Asking about ulcerogenic medications, especially NSAIDs,



**Figure 1.4:**  
Melena. This figure shows melena in a dog with severe gastric bleeding.



**Figure 1.5:**  
Perineal herniation. Round stools that look like “horse manure” from a dog with perineal herniation.

anticoagulant exposure (e.g., rodenticide), or recent trauma is also important.

Fresh blood that is adherent to the feces is called hematochezia and is consistent with large bowel or recto-anal bleeding. Hematochezia can be associated with local disorders (colitis, proctitis, foreign body, or neoplasia) or coagulopathy. It is not always associated with dyschezia or straining to defecate and is therefore sometimes not observed by the owner.

Ribbon-like stools are observed in conditions associated with a narrowing of the colonic, rectal, or anal passage and frequently lead to dyschezia or constipation. Sometimes, round or ball-like feces looking like horse manure (Figure 1.5) are observed with perineal herniation or anal diverticula.

Acholic feces are clay-like feces that can be observed in patients with extra-hepatic bile duct obstruction or destructive cholangiolitis.<sup>1</sup>

### 1.1.2.7 Flatulence and borborygmus

Flatulence and borborygmus (rumbling of the gastrointestinal tract) are ultimately due to the presence of large amounts of intestinal gas that can occur in association with many digestive disorders. Excessive gas can also be associated with abdominal discomfort. A dietary history is important in these patients since certain types of foods commonly lead to the formation of excessive gas in the intestinal lumen (e.g., legumes, soy-beans, or excess fat).

**Table 1.6: Characterization of small and large bowel diarrhea**<sup>3,5,6,11</sup>  
Please note that none of these parameters is absolute.

Parameter	Small intestine	Large intestine
<b>Feces</b>		
Volume per defecation	Increased	Decreased or normal
Mucus	Absent (except in ileitis)	Frequently present
Melena	May be present	Rarely present
Hematochezia	Absent except in acute hemorrhagic diarrhea	Frequently present
Steatorrhea	Present in patients with maldigestion or malabsorption	Absent
<b>Defecation</b>		
Frequency	Usually slightly increased up to 4 times per day but can be severely increased in severe acute enteritis	Increased (many defecations with small volume)
Dyschezia	Absent	Present
Tenesmus	Absent	Frequently present
Urgency	Absent; except in severe cases	Usually present; sometimes with indoor soiling
<b>Other signs</b>		
Flatulence / borborygmus	May be present	May be present
Weight loss	May be present	Rare
Anal pruritus	Absent	May be present
Vomiting	May be present	May be present in acute colitis

### 1.1.2.8 Dyschezia

Dyschezia is defined as difficult or painful defecation characterized by straining to defecate with or without defecation. Dyschezia can be observed in association with large bowel diarrhea but also with some other conditions such as constipation, rectal disease, anal disorders such as anal furunculosis or anal gland sacculitis, or prostatomegaly in male dogs.

Tenesmus is straining to defecate and is a clinical manifestation of either dyschezia or colonic and recto-anal discomfort.

### 1.1.2.9 Constipation

Constipation is defined by the passage of dry, hard feces and is associated with a decreased number of bowel movements and straining to defecate. In cases of suspected constipation, it is important to ask about urination behavior as urinary straining can be confused with straining to defecate in some patients, especially in cats with lower urinary tract obstruction. Straining before defecation is usually associated with obstructive or functional disorders inducing constipation. In contrast, straining throughout defecation or persisting after defecation has been completed is frequently associated with diarrhea of inflammatory disorders of the colon and/or rectum.<sup>3</sup>

Sometimes, the constipated patient is presented for diarrhea. The diarrhea perceived by the owner is due to a small quantity of liquid fecal material passing around the fecal bolus lodged in the colon and/or rectum.

### 1.1.2.10 Fecal incontinence

Fecal incontinence is characterized by uncontrolled loss of feces without crouching to defecate. It must be differentiated from urgency to defecate by carefully questioning the owner as these two causes of fecal soiling are frequently confused. In patients with true fecal incontinence, the stools are usually normal and loss of feces frequently occurs during excitation or coughing. The owner should be questioned about the presence of controlled and normal defecation to assess whether the neuro-muscular control of defecation is functional. A history of trauma (especially tail trauma in cats) as well as medical or surgical conditions of the perianal area are frequently associated with fecal incontinence.

### 1.1.2.11 Anal pruritus

Anal pruritus is manifested by perineal licking or biting, anal scratching, or scooting. It is usually associated with recto-anal diseases such as anal furunculosis, anal gland disorders, or *Dipylidium* infestation, but can also be a manifestation of food allergy, constipation, or inflammatory bowel disease.

### 1.1.2.12 Abdominal pain

The owner may report certain behaviors such as a saw horse stance or prayer position (stretching out the forelimbs with the sternum in contact with the floor and standing on the hindlimbs), which are strongly suggestive of cranial abdominal pain.<sup>4</sup> Bruxism (teeth grinding) or restlessness can also sometimes be associated with severe abdominal pain in dogs and cats. Restlessness can sometimes be observed during the first stages of gastric dilation/volvulus.<sup>8</sup> In cases of extreme abdominal pain, the pet may be severely depressed or even aggressive.

## 1.1.3 Dietary history

A complete dietary history can be very important in the diagnosis of gastrointestinal disease and should include specific commercial foods that are being fed, commercial snacks or treats, supplements and chewable medications, chewable toys, human foods (especially leftover foods from human meals), access to other sources of food (ask about other pets in the household or about people who may give extra food to the pet).<sup>9</sup> Many medical supplements or certain drugs contain proteins or additives that may elicit allergic or other adverse reactions (especially vitamins, fatty acids, or dermatological supplements). It is also important to try to identify any possible correlation between clinical signs and certain types of food, supplements, or medications.

Abnormal feeding patterns should be documented. Coprophagia can be observed in any disorders causing polyphagia such as exocrine pancreatic insufficiency or high-dose corticosteroid treatment. Also, coprophagia or pica can be observed in patients with nutritional deficiencies or those with behavioral problems. Eating grass is frequently reported in dogs and cats, and is of concern when it is followed by nausea or vomiting.<sup>5</sup> Eating grass could either be the cause or more commonly a consequence of a gastrointestinal problem.

### Key Facts

- Acquiring an accurate history is essential to further characterize the clinical signs related to digestive diseases.
- Differentiation between regurgitation and vomiting is necessary to determine an optimal workup for each patient.
- In cats constipation must not be mistaken for urinary straining.
- Dietary history is a cornerstone in the diagnosis and management of digestive diseases.

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## 1.2 Physical Examination

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### 1.2.1 Introduction

By far the most important diagnostic tool that veterinarians can utilize is their ability to obtain a complete history and conduct a thorough physical examination. The objective of the physical examination is to recognize and describe gross deviations of the patient's physical appearance and behavior from those recognized as normal for the animal's species, breed, age, sex, and sexual status.

The trend toward an increased use of laboratory tests and instrumentation has added diagnostic capabilities, but such techniques are useful adjuncts for diagnosis only when a careful physical examination has been carried out. Thus, information gathered from laboratory and diagnostic imaging procedures must be considered a supplement to, but not a substitute for, hands-on examination. Inspection of the patient, palpation,

percussion, and auscultation, all have a place in every examination. Only acute life-threatening situations require a shorter initial examination, until the animal's condition can be stabilized. In fact, if the patient is in shock or hemorrhaging, or has gastric dilation/volvulus, it is essential to initiate supportive care immediately and obtain a complete history with careful examination afterwards.

The physical examination of the gastrointestinal tract, as described in this chapter, is part of the methodical and standardized physical examination first developed at the School of Veterinary Medicine of Bologna University.<sup>1</sup>

In this chapter, the discussion is mainly limited to parameters of the physical examination that are specific and directly related to the gastrointestinal system, but it is important to note that the clinician should examine all body systems when presented with a patient that manifests signs of gastrointestinal disease. Ophthalmic and neurological examination, not included in this chapter, should not be forgotten because they can sometimes provide invaluable clues to the cause of a gastrointestinal dysfunction.

1



**Figure 1.6:** Exocrine pancreatic insufficiency. This picture shows the failure to grow in two puppies with exocrine pancreatic insufficiency compared with a healthy littermate shown in the middle (these dogs are described in detail in: Boari A. et al. Observations on exocrine pancreatic insufficiency in a family of English setter dogs. *J Small Animal Practice* 1994, 35: 247–250).



**Figure 1.7:** “Prayer” position. This picture shows a dog with acute abdominal pain due to acute pancreatitis. This dog has assumed the “prayer” position with his front legs and sternum on the floor and his hind legs standing.

## 1.2.2 General physical examination

### 1.2.2.1 Skeletal growth and development

A failure to grow in dogs and cats, often seen with endocrinopathies such as hyposomatotropism and hypothyroidism, can also result from vascular ring or other esophageal anomalies, malabsorption, or portosystemic shunts (Figure 1.6).

### 1.2.2.2 Body condition

Measurement of body weight is inexpensive, easy, and very useful. Weight loss can be caused by inadequate nutrient assimilation (anorexia, regurgitation, vomiting, maldigestion, or malabsorption), increased loss of nutrients (protein-losing nephropathy [PLN] and/or protein-losing enteropathy [PLE]), and/or increased energy demand, as it occurs in hyperthyroid cats or febrile patients. Cachexia associated with fever can be due to infectious (e.g., FIP, FeLV), inflammatory (e.g., pancreatitis and others), or neoplastic (e.g., gastrointestinal neoplasia, lymphoma) causes.

Weight loss is unusual in patients with diarrhea due to large bowel disease, but it can be seen in patients with severe, long-standing colitis such as histiocytic ulcerative colitis (HUC), cecocolic intussusception, or diffuse colorectal neoplasia.<sup>2</sup>

However, it could also be caused by the tendency of owners to fast a pet with diarrhea.

It is very important to remember that an acute decrease in body weight can be due to water loss (i.e., vomiting and/or diarrhea) and that measurement of body weight will allow an accurate assessment of the level of dehydration.

### 1.2.2.3 Mental status

Depression or stupor can be related to abnormal brain function caused by metabolic (e.g., hepatic encephalopathy or acid-base and osmolality imbalances), inflammatory (e.g., canine distemper, FIP, sepsis), and vascular (e.g., coagulopathies, hypertension) disorders.

### 1.2.2.4 Abnormalities in posture and locomotion

In the cat, neck ventroflexion can be a sign of hypokalemia that can be caused by gastrointestinal loss of potassium due to vomiting, diarrhea, or anorexia. Abnormal posture, such as arching of the back or adoption of the “prayer” position (Figure 1.7), is a characteristic sign of abdominal pain, which needs to be differentiated from back pain.<sup>3</sup>

### 1.2.2.5 Mucous membranes

Mucous membrane color and capillary refill time (CRT) are used to estimate peripheral perfusion. A slower CRT suggests either dehydration or high peripheral sympathetic tone and vasoconstriction. Both are associated with low cardiac output. In a patient presented for vomiting, diarrhea, and/or anorexia, it is very important to address the patient's overall hydration status. To that end, the clinician should assess body weight, skin turgor or pliability, moistness and color of the mucous membranes, CRT, position of the eyes in their orbits, and the pulse and respiratory rates and their characteristics.

Pale mucous membranes are a manifestation of either decreased red blood cell mass or decreased peripheral perfusion. In the latter case, shock (i.e., hypovolemic, cardiogenic, or vasomotor shock) can cause hypovolemia, cardiac insufficiency, and vasoconstriction. As a result pallor is a hallmark of shock. In patients with endotoxemic shock, the mucous membranes may become cool to the touch.

Pale mucous membranes can also be associated with hepatobiliary disease due to increased consumption and/or as an effect of non-regenerative anemia due to chronic disease. Peptic and neoplastic gastric ulceration may also lead to anemia. Acute blood loss into the abdomen due to visceral hemangiosarcoma (HSA), which occurs much more frequently in dogs than cats, can also cause pale mucous membranes, weakness, abdominal distension, and increased pulse and respiration rates. The CRT is normal for anemic patients unless hypoperfusion is also present.

Also, the oral and ocular mucous membranes are often the first sites where jaundice can be seen. Yellow oral mucus membranes are almost always observed in cases of severe icterus and can be caused by immune-mediated hemolytic anemia or hepatobiliary disease.

A septic patient often has injected, highly vascular mucous membranes (brick-red) as can also occur in polycythemic patients, or those with acute hepatic and pancreatic disease, or severe azotemia. Congestion of mucous membranes may occur in patients with gastrointestinal disease, usually as a manifestation of dehydration. Mucous membranes should also be checked for signs of hemorrhage. Superficial bleeding into the skin or mucous membranes, and scleral and vitreal hemorrhage are often common manifestations of abnormalities of primary hemostasis. In addition to petechiae or ecchymoses, the animal may also present with hematemesis and melena as well as hematuria. Although infrequent, patients with severe hepatic disease can show bleeding diathesis due to coagulation factor deficiency, disseminated intravascular coagulation (DIC), or portal hypertension. Because of the multiple hemostatic defects present in patients with DIC, they may experience hemorrhage of any type and in any location (cavitary or superficial).

### 1.2.2.6 Peripheral lymph nodes

Mandibular, prescapular, and popliteal lymph nodes are usually palpable and they should be evaluated for size, shape, and consistency. The axillary and superficial inguinal lymph nodes are not always discernible. Given the close proximity of the mandibular lymph nodes and submaxillary salivary glands, it is essential that the clinician be able to distinguish between the two. Especially in cats, the surrounding subcutaneous fat may make the popliteal lymph nodes seem larger than their actual size. On the other hand, emaciated adult animals may have normal-sized popliteal lymph nodes that appear more prominent because of loss of fat and muscle mass.

Generalized lymphadenopathy usually indicates a systemic disease (e.g., immune-mediated disease, systemic infection, or more commonly neoplasia). Especially in the dog, the presence of markedly enlarged external lymph nodes that are firm and non-painful, is highly suggestive of lymphoma.<sup>4</sup>

### 1.2.2.7 Skin and subcutaneous tissue

The skin should be carefully inspected for areas of alopecia, inflammation, nodularity, and crustiness. Also, examination of the mucocutaneous junctions may reveal evidence for systemic immune-mediated diseases. Canine systemic lupus erythematosus has occasionally been reported to cause megaesophagus, chronic small bowel diarrhea with PLE, and chronic hepatitis.<sup>5-7</sup>

Non-seasonal pruritus, erythema, and papules can be attributed to food hypersensitivity or "intolerance" in both dogs and cats. Concurrent presence of gastrointestinal and dermatological signs in dogs and cats is strongly suggestive of food hypersensitivity.<sup>8-12</sup>

Dramatic skin lesions (i.e., erythema, crusts, erosions, ulcers, alopecia, or shiny skin) can be seen in high-contact areas and on the ventral thorax and abdomen in patients with superficial necrolytic dermatitis, which can be caused by liver disease or glucagonoma. Also, pancreatic tumors have been reported as a cause of alopecia in dogs and cats.<sup>13</sup>

The integument should also be evaluated for changes in skin turgor, which is assessed in a consistent manner and location, usually on the lateral thorax. In evaluating skin turgor, the clinician should take into account the fact that skin turgor is dependent on the amount of subcutaneous fat and elastin, as well as on the interstitial volume. Thus, emaciated and older animals may appear more dehydrated than they actually are. On the other hand, obese animals may appear falsely well-hydrated based on their skin tenting.

Generalized soft tissue swelling or enlargement involving the four limbs, often associated with ascites, may be due to edema. Edema can be easily distinguished from other subcutaneous fluid accumulations or masses because pressure applied to an edematous area results in an indentation that stays for a short period of time. Subcutaneous edema can occasionally be seen in hypoalbuminemic dogs with PLE, PLN, or severe hepatic insufficiency.

### 1.2.2.8 Body temperature

At the time the temperature is taken, the clinician should also note if the rectal area is clean or soiled from diarrhea or matting, and whether the anal sacs are distended. The perineum is also observed for the presence of tapeworm proglottides. At the end of the examination, the thermometer should be examined for any blood, melena, or mucous. The appearance of blood in the feces can vary according to the site of bleeding, the transit time through the gastrointestinal tract, and the volume of blood lost.<sup>14</sup>

Hematochezia (i.e., fresh blood in the stool) is strongly suggestive of large bowel disease, in particular colitis. However, enterocolic and cecocolic intussusception, colorectal neoplasia and coagulopathies, especially platelet disorders, must also be considered, even though these are uncommon. Melena describes tar-, coal-, or asphalt-colored stools, resulting from digested blood and can be observed due to bleeding into the pharynx, esophagus, stomach, or upper small bowel. When melena is present, a complete physical examination should include careful inspection of the nares, oropharynx, and lungs for evidence of the source of bleeding. It is important to remember that a life-threatening volume of blood can accumulate within the gastrointestinal tract, with little or no visible signs of external blood loss. If gastrointestinal blood loss is associated with acute diarrhea, regardless of its cause, this signals a loss of normal intestinal mucosal integrity. With the loss of this barrier, the normal enteric flora can cross into the bloodstream, leading to septicemia. In this case, the clinician needs to address this life-threatening complication of diarrhea, while determining its cause.

There are many causes of fever in patients with gastrointestinal disease, ranging from infectious diseases (e.g., FeLV, FIV, FIP, canine distemper, feline panleukopenia, canine parvovirus, leptospirosis, salmonellosis, toxoplasmosis, leishmaniasis, histoplasmosis, blastomycosis, cryptococcosis, coccidioidomycosis, or rickettsial infections) to disorders involving the liver, exocrine pancreas, and the peritoneum, or neoplasia such as lymphoma or carcinoma.

In contrast, severely uremic patients, patients with sepsis and shock, or patients in the final stages of several severe systemic diseases can be hypothermic.

### 1.2.2.9 Pulse rate

The effects of many systemic and metabolic diseases on cardiac structure and function are well recognized. In some cases these may constitute the major clinical concern, while in others these effects may be subtle or of minimal importance. Common causes of tachycardia are excitement, fever, anemia, hemorrhage, shock, hypotension, significant alterations in the concentration of electrolytes or acid-base balance, congestive heart failure, and some infections.

Patients with GDV often show cardiac dysfunction, especially after surgical decompression. This is often associated with tachyarrhythmias or, less commonly, bradyarrhythmias.

Septic shock, often caused by gram-negative bacteria, can result in brick-red mucus membranes with a strong pulse during the initial stages, or pale mucus membranes and a weak pulse during the latter stages.

Electrolyte and acid-base abnormalities can produce significant alterations in cardiac function. These can often be documented during examination of the pulse; but are better recorded using an ECG. Severe hyperkalemia (usually  $>8$  mEq/L) causes severe cardiac dysfunction. In addition to hypoadrenocorticism, hyperkalemia with hyponatremia (Na/K ratios  $<27:1$ ) may also be found in dogs with gastrointestinal disease due to trichuriasis, salmonellosis, or a perforated duodenal ulcer and, although rare, in patients with peritoneal effusion.<sup>15</sup> Hyperkalemia without hyponatremia is almost always associated with impaired renal excretion due to oliguric or anuric renal failure.

Arrhythmias can also be observed in patients with hypokalemia. Hypokalemia is sometimes associated with alkalemia but more often is due to gastrointestinal loss of potassium or renal loss in patients with polyuric renal failure.

### 1.2.2.10 Respiratory rate

Respiratory rate and quality should be recorded before beginning the specific examination. It is important to differentiate between an increased respiratory rate that is due to a physiological response to exercise, hyperthermia, and anxiety, and one that is due to disease. Inspiratory dyspnea can be observed in patients with an elongated or edematous soft palate or feline patients with a pharyngeal polyp.

Animals with esophageal disease or vomiting can be presented to a veterinarian for respiratory distress due to aspiration pneumonia. Suspicion of megaesophagus or other esophageal diseases should be high if the owner reports that recurrent regurgitation or vomiting took place before the respiratory signs developed.

Severe and life-threatening dyspnea is often present in patients with pleural effusion and ascites associated with ascites due to hypoproteinemia or non-septic inflammation (i.e., FIP).

Rapid breathing can also be attributed to cranial displacement of the diaphragm by abdominal masses, fluid, or gas (e.g., gastric torsion) and/or acid-base imbalances. A compensatory hyperventilation is frequently seen in animals with metabolic acidosis caused by severe diarrhea, chronic renal disease, diabetic ketoacidosis, or hypoadrenocorticism.

### 1.2.3 Examination of the gastrointestinal tract

The examination of the gastrointestinal or digestive tract begins at the head (i.e., oral cavity), passes through the neck, down to the abdomen, and finishes with a rectal examination. Examination of the mouth and pharyngeal structures often provides important clues as to the pathogenesis of anorexia, vomiting, regurgitation, or drooling. In particular, drooling, which is best defined either as ptyalism (i.e., overproduction of saliva) or pseudoptyalism (i.e., dribbling or drooling of saliva that has accumulated in the oral cavity), occurs because animals are unable to swallow or are in too much pain to do so, as occurs during nausea, hepatic encephalopathy (especially in the cat), seizures, stomatitis, glossitis, gingivitis, pharyngitis, tonsillitis, and oral or pharyngeal dysphagia. Nasal discharge can also be associated with dysphagia, gagging and sometimes vomiting.

**Swallowing and the gag reflex** are assessed by placing an index finger at the base of the tongue or by observing the animal while it is drinking or eating. Tonsillitis or enlargement of the tonsils, mainly in the dog, can cause anorexia, vomiting, or dysphagia, and can occasionally be a sign of systemic disease (e.g., lymphoma). The tongue is examined for color and movement. The underside of the tongue is checked for masses, string foreign bodies (in the cat), or a laceration of the frenulum as a result of a string.

The **odor** of the breath can be indicative of dental or periodontal disease, but also of uremia or ketonemia.

The high frequency of **hyperthyroidism** in geriatric cats obliges the clinician to carefully palpate the paratracheal area, extending from the caudal larynx to the thoracic inlet, in order to detect nodular swelling suggestive of thyroid gland enlargement. The normal feline thyroid glands cannot be palpated.

**Abdominal distension** may be due to gas, fluid, organomegaly, or a poor abdominal muscle tone. Association with other clinical signs such as vomiting, diarrhea, abdominal pain, polydipsia, polyuria, polyphagia, or edema, may serve as a clue to the underlying etiology. In the dog, gastric tympany results in a flaring of the posterior rib cage and the hypochondrium. As gastric distension increases, the posterior abdomen also becomes visibly distended. If the enlarged abdomen is due to abdominal fluid, ballottement should be carefully performed to determine if a fluid wave is present. With experience, false positive results are infrequent.

**Abdominal effusion** is usually caused by hypoalbuminemia, portal hypertension, or peritoneal inflammation. Effusion due to gastrointestinal disease is primarily caused by PLE, hepatic failure, rupture of the alimentary tract, or leakage following anastomosis.<sup>16,17</sup> PLE in a young dog with chronic intermittent diarrhea, without hookworms, should prompt suspicion of a chronic intussusception and an abdominal ultrasound should be performed.<sup>18</sup>

Pyogranulomatous inflammation of the abdominal or thoracic cavity that is associated with a characteristic effusion is typical for the effusive form of FIP.

Malignant abdominal **tumors** may lead to obstruction of lymphatic flow, increased vascular permeability, accumulation of a modified transudate, or development of non-septic peritonitis. Modified transudates can also result from hepatic or cardiac disease. Hepatobiliary malignancies or other intra-abdominal malignant forms of neoplasia that have spread to the peritoneum can elicit an inflammatory reaction, with subsequent exudation of lymph, fibrin, and blood. This fluid may be serosanguinous, hemorrhagic, or pseudochylous in appearance.

**Enlarged organs** that most often account for increased abdominal size are the liver, spleen, and occasionally, the kidneys. Alternatively, single neoplastic masses of other organs also frequently lead to abdominal distension.

Abdominal palpation is the cornerstone of the physical examination in dogs and cats with clinical signs of gastrointestinal disease. This can be a most informative procedure for the cat, because of the ease with which most of the viscera can be palpated in this species.

If a gas-distended abdomen is suspected, digital abdominal percussion should be performed, listening for a tympanic sound. A sudden onset of a gas-distended abdomen, shock, and even death are often observed in dogs with intestinal volvulus.

Some animals tense their abdominal muscles in response to palpation. It is essential to determine whether this is caused by pain, anxiety, or the exertion of too much pressure during palpation. More significance is attributed to the pain response elicited in stoic animals and to pain that is localizable, repeatable, and evident after minimal manipulation. It is necessary to determine whether the painful area is superficial, located in the cranial or caudal abdomen, and whether it originates from a specific viscus. Cranial abdominal pain is commonly observed in dogs with pancreatitis, but less commonly observed in cats. Generalized abdominal pain with rigidity of the abdominal musculature suggests generalized peritonitis.

It is worth noting that if an animal arches its back during abdominal palpation the primary problem may be in the spinal cord. The empty stomach in cats and dogs is usually only palpable in thin patients, although if distended with food, it can be palpated in the upper left abdominal quadrant in most patients. The gallbladder and pancreas are not normally palpable. However, in patients with pancreatitis, pancreatic neoplasia, or a pancreatic pseudocyst, an abdominal mass and pain associated with palpation of the mass may be observed in the right cranial quadrant of the abdomen.

The **small intestine** is easily palpated as a thin-walled and smooth object that fills much of the mid-abdomen, and slides through the fingers. In the cat, the ileocecal area can often be palpated as a firm, knot-like structure in the mid-cranial abdomen, and should not be confused with an abdominal mass. The bowel should be carefully evaluated for thickness, rigidity, and irregular masses. A thickened intestinal wall may be felt in patients with intestinal infiltration by inflammatory or neoplastic cells and in patients with intestinal smooth muscle hypertrophy.<sup>19</sup>

**Masses** (e.g., lymph node enlargements, foreign bodies, omental steatitis, neoplasia, intussusception, or focal granulomatous lesions, such as those that can be seen in patients with a non-effusive form of FIP) can cause partial or total intestinal obstruction, but may go undetected during physical examination because of their small size.<sup>20</sup> Fluid-distended small bowel loops are often palpable in patients with acute enteritis. Aggregated or accordion-like small bowel loops are characteristic of a linear foreign body obstruction in cats. Palpable mesenteric lymph node enlargements are often associated with tumors, granulomas, or intestinal inflammation (with or without foreign body obstruction). A massive mesenteric lymphadenopathy is often typical of dogs and cats with alimentary tract lymphoma, but modest lymphadenopathy can also be found in patients with IBD or other chronic intestinal diseases.

A sick animal that vomits shortly after abdominal palpation should be suspected of having a GI obstruction, severe GI tract inflammation, or pancreatitis.

The **transverse and descending colon** are often full of fecal material and can easily be identified by palpation of the mid-posterior abdomen, just ventral to the spine. An impacted colon (the colon must be at least twice its normal diameter before megacolon can be considered), is caused by intestinal obstruction or dysmotilities.

The **liver** can be palpated routinely just caudal to the costal arch along the ventral body wall in both canine and feline patients, but may not be palpable in some cases. If the liver is not palpable, it does not automatically mean that it is abnormally small. Microhepatia is mainly seen in patients with a congenital portosystemic shunt or those with chronic hepatic disease with progressive loss of hepatocytes. However, hepatic size is better evaluated by radiography. In lean cats, it is possible to palpate the diaphragmatic surface of the liver. In animals with pleural effusion or other diseases that expand the thoracic volume, the liver may appear enlarged due to caudal displacement. The pattern of hepatic enlargement may be generalized or focal depending on its cause. Infiltrative and congestive diseases tend to result in smooth, firm, and diffuse hepatomegaly. Primary or metastatic neoplasia, nodular hyperplasia, and some chronic hepatic diseases associated with nodular regeneration can cause focal or asymmetric hepatic enlargement.

In icteric dogs and cats, hepatosplenomegaly may be attributable to mononuclear-phagocytic cell hyperplasia and extramedullary hematopoiesis secondary to immune-mediated hemolytic anemia or to infiltrative processes such as systemic mast cell disease, lymphoma, or myeloid leukemia.

Palpation of the spleen is not always possible, but sometimes the free distal portion is palpable on the floor of the mid-abdomen. The spleen is palpated for identification of an increase in size and for nodules or larger masses. In patients with severe splenomegaly, the spleen may occupy the entire ventral abdominal floor.<sup>21</sup> When the enlarged spleen is folded over, it may be mistaken for a mass. With experience, the clinician can sometimes unfold the spleen with his or her thumb, and thus the “real” shape of the organ can be evaluated.

The **kidneys** can only be easily palpated in cats because they are more loosely attached than they are in dogs. The kidneys are normally located in the retroperitoneal area, and the right kidney lies slightly more cranial than the left. The kidneys are evaluated for size, shape, location, firmness, pain, and surface irregularities. The left kidney (the only one that can be palpated in some dogs) is especially movable and can easily be mistaken for an abdominal mass. Enlarged, abnormally-shaped kidneys may be caused by acute renal failure, renal neoplasia, renal cysts, abscesses, granulomatous nephritis due to FIP, hydronephrosis, or hematoma. In contrast, small renal size is often associated with chronic renal disease.

During abdominal palpation of **intact female animals**, the normal non-gravid uterus is usually non-palpable. Massive uteromegaly caused by pregnancy, pyometra, mucometra, or hydrometra can sometimes give the mistaken impression of ascites and must be carefully differentiated.

Lastly, abdominal auscultation may sometimes be helpful. Failure to detect intestinal sounds after two or three minutes of auscultation is suggestive of ileus.

The **perineal area** should be examined for evidence of diarrhea caked in the hair coat, masses, or herniations. **Rectal examination** must always be performed and the clinician should be able to identify and evaluate the colonic mucosa, anal sphincter, anal sacs, pelvic canal bones, urogenital tract, and luminal contents. Mucosal polyps can easily be misinterpreted as mucosal folds, and it is possible to miss partial strictures that are large enough to allow a single finger to pass through.

Pelvic canal obstruction due to congenital and acquired causes can lead to constipation and megacolon, especially in cats.

Rectal discomfort, hematochezia, and mucous can be found in patients with colitis, proctitis, or large bowel neoplasia as described previously.

During the rectal examination, the prostate of all mature male dogs must be evaluated for size, symmetry, surface structure, and pain. If enlarged, the prostate may extend slightly over the brim of the pelvis or fall into the abdomen. In this latter situation, the prostate can be palpated in the caudal abdomen ventral to the colon and caudal to the urinary bladder. To assist in rectal palpation, the other hand of the examiner should be used to gently push the prostate into a more dorsal and caudal position via abdominal palpation.

Also, in order to complete the physical examination, the clinician should observe the act of defecation whenever possible, especially if there is a history of dyschezia or tenesmus.

Whether tenesmus occurs before or after defecation can also aid in differentiation of the underlying disease process. Obstructive disorders are more commonly associated with tenesmus before evacuation of feces, whereas inflammatory disorders are often associated with persistent tenesmus after the evacuation of feces.

### Key Facts

- A life-threatening volume of blood can accumulate within the gastrointestinal tract, with little or no visible signs of external blood loss.
- Abdominal distension may be due to gas, fluid, organomegaly, or poor abdominal muscle tone.
- PLE in a young dog with chronic intermittent diarrhea, without hookworms, should prompt a suspicion of chronic intussusception.
- Aggregated (accordion-like) small bowel loops on abdominal palpation are characteristic of a linear foreign body obstruction in cats.
- A sick animal that vomits shortly after abdominal palpation should be suspected of having a GI obstruction, severe GI tract inflammation, or pancreatitis.
- Rectal examination should be performed in every patient presenting for evaluation of gastrointestinal disease.

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