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Small Animal Internal Medicine

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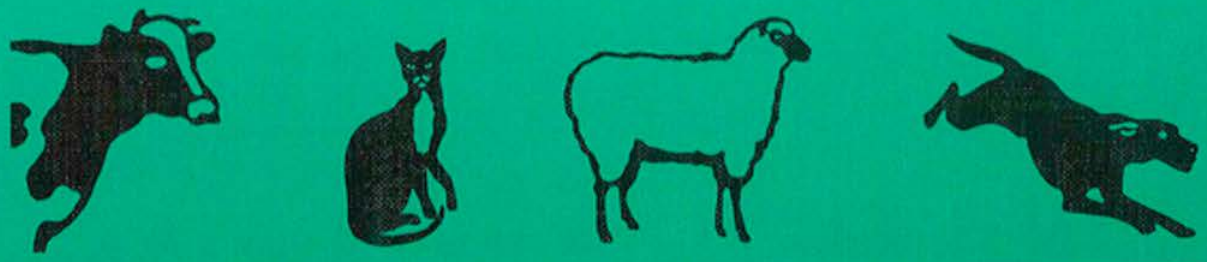

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Blackwell
Publishing

Blackwell Publishing Professional
2121 State Avenue, Ames, Iowa 50014, USA

Orders: 1-800-862-6657
Office: 1-515-292-0140
Fax: 1-515-292-3348
Web site: www.blackwellprofessional.com

Blackwell Publishing Ltd
9600 Garsington Road, Oxford OX4 2DQ, UK
Tel.: +44 (0)1865 776868

Blackwell Publishing Asia
550 Swanston Street, Carlton, Victoria 3053, Australia
Tel.: +61 (0)3 8359 1011

First edition, 1997

Library of Congress Cataloging-in-Publication Data
Shaw, Darcy H.
Small animal internal medicine / Darcy H. Shaw, Sherri L. Ihle. — 1st ed.
p. cm — (The national veterinary medical series for independent study)
Includes index.
ISBN-13: 978-0-6830-7670-7
ISBN-10: 0-6830-7670-1
1. Dogs—Diseases. 2. Cats—Diseases. 3. Veterinary internal medicine. I. Ihle, Sherri L. II. Title. III. Series.
SF991.S53 1997
636.7'0896—dc21 97-3958

The last digit is the print number: 9

Dedication

To Tao, Ted, Wally, Henry, and Norm, truly wonderful cats who have enriched my life immensely with their antics, eccentricities, and unconditional affection.

To all of my canine and feline patients who through my mistakes and successes have taught me so much about medicine and life.

To my past students who have kept me honest and enthused.

To my wife, Shelly Burton, who is my best friend and who makes it all worthwhile.

D. H. S.

Contents

Preface

The objectives of *NVMS Small Animal Internal Medicine* are to provide students with a concise, well-organized, and up-to-date overview of the discipline and to offer the opportunity to test comprehension of the material. In our effort to be concise and emphasize the key points regarding clinical signs, diagnosis, and treatment, information relating to pathophysiologic mechanisms and detailed treatment strategies is decidedly brief. Consequently, other textbooks and scientific publications should be sought for this information.

The main audience for *NVMS Small Animal Internal Medicine* is third- and fourth-year veterinary students, but interns, residents, and private practitioners will also find the book useful. *NVMS Small Animal Internal Medicine* is organized into three general sections. The first section, Chapters 1 through 38, deals with clinical problems and their causes. The next section, Chapters 39 through 50, covers diseases associated with organ systems. A self-assessment section containing a 100-question multiple choice examination concludes the book.

We are confident that readers will find this a current, accurate, and complete overview of small animal internal medicine and will be challenged by the self-assessment activities. A companion volume, *NVMS Small Animal Internal Medicine Case Management Test Booklet,* is also available to readers who wish to practice working through cases similar to those encountered in clinical practice and on national board examinations.

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Sherri L. Ihle

Acknowledgments

The authors would like to thank Williams & Wilkins for providing us with the opportunity to write this book, and Melanie Cann, our development editor, for her patience, timely prodding, and editorial contributions. We would also like to thank Lisa Kiesel and Lynne Stockton for their editorial contributions.

PART I

CLINICAL PROBLEMS

Chapter 1

Halitosis

I. DEFINITION. Halitosis is offensive or foul-smelling breath.

II. CAUSES of halitosis are listed in Table 1-1.

A. In many cases, necrotic tissue, bacterial proliferation in retained food particles, or both are responsible for the odor.

B. Consumption of a foul-smelling substance can cause transient halitosis.

III. CLINICAL FINDINGS vary with the underlying disease.

A. **Drooling** may be seen with any of the oral or pharyngeal disorders listed in Table 1-1.

B. **Oral pain** may indicate periodontal disease, neoplasia, or inflammation.

C. **Dysphagia** in the presence of normal food prehension may indicate pharyngeal or esophageal disease.

IV. DIAGNOSTIC APPROACHES

A. **History and physical examination,** including a full **oral examination,** will usually narrow the list of differential diagnoses.

B. **Viral serology** or **biopsy** may be useful if oral ulceration or a mass is found.

TABLE 1-1. Causes of Halitosis

Oral disease
Dental tartar or periodontal disease
Neoplasia
Granuloma
Stomatitis or pharyngitis
Food retention
Esophageal disease
Neuromuscular disease with retention of food
Neoplasia
Granuloma
Miscellaneous causes
Gastritis (rare)

C. **Complete blood count (CBC), serum biochemical profile,** and **urinalysis** may help rule out systemic diseases that may cause oral lesions.

D. **Observation of the animal eating,** to assess food prehension and swallowing, may be helpful in the absence of dental tartar, oral ulceration, or oral masses (see Chapter 2).

V. TREATMENT is aimed at the primary disease.

Chapter 2

Dysphagia and Regurgitation

I. DEFINITION

A. **Dysphagia** is difficulty in prehending or swallowing food.

B. **Regurgitation** is the passive expulsion of undigested food from the esophagus.

II. CAUSES

A. **Dysphagia.** The causes of dysphagia are listed in Table 2-1.

B. **Regurgitation.** The causes of regurgitation are listed in Table 2-2.

III. CLINICAL FINDINGS

A. **Dysphagia.** A dysphagic animal may attempt to eat but is either unable to prehend the food, chew the food, or move the food to or beyond the pharyngeal region. The extent to which the animal is able to proceed with food consumption is determined by the site and type of disease. The clinical findings vary according to the underlying disorder.

1. **Drooling** may result from an inability or reluctance to swallow.
2. **Pain**
   a. **Oral pain** may occur with oral or pharyngeal trauma or foreign bodies.
   b. **Oral pain** accompanied by **halitosis** may occur with periodontitis, stomatitis or pharyngitis, neoplasia, or granuloma.
3. **Neurologic abnormalities**
   a. A **"dropped jaw"** will be found with trigeminal nerve dysfunction.
   b. **Facial muscle pain** or **atrophy** may occur with facial myositis.
   c. The presence of **other neurologic abnormalities** (e.g., **weakness, abnormal mentation, ataxia**) suggests the presence of a central nervous system (CNS) disorder or a neuromuscular disease.

B. **Regurgitation.** Food prehension, mastication, tongue movement, and pharyngeal motility are usually normal, but esophageal structure or function is abnormal. The ejected material is often tubular in shape and alkaline.

IV. DIAGNOSTIC APPROACHES. If differentiation between dysphagia and regurgitation is not possible from the history, watching the animal eat and drink may be helpful.

A. **Dysphagia** (see Table 2-1). A thorough neurologic and oral examination should be performed on all dogs and cats with dysphagia.

1. If **neurologic abnormalities** are present, useful tests include a cerebrospinal fluid (CSF) tap, electrodiagnostic tests, and nerve or muscle biopsy.
2. If **oral lesions** are present, a biopsy or further dental evaluation may be needed.

TABLE 2-1. Causes of and Diagnostic Tests for Dysphagia

Cause	Diagnostic Test
Neuromuscular disease	
Oral	
Myositis	Serum creatine kinase, electromyography, biopsy
Trigeminal nerve dysfunction	Physical examination, biopsy
Neuromuscular trauma	. . .
Pharyngeal	
Cricopharyngeal achalasia	Contrast radiographs with fluoroscopy
Myasthenia gravis	Acetylcholine receptor antibody titer
Myositis	Serum creatine kinase, electromyography, biopsy
Rabies	Histopathologic studies
Idiopathic	. . .
Obstructive disease	
Tumor	Biopsy
Granuloma	Biopsy
Foreign bodies	Oral examination, radiography
Sialocoele	Oral examination
Infectious and inflammatory disease	
Periodontitis	Oral examination
Stomatitis or pharyngitis	Oral examination, biopsy
Abscess (tooth root, retrobulbar)	Oral examination, radiography
Osteomyelitis	Radiography, biopsy, culture
Miscellaneous causes	
Trauma (e.g., fracture, laceration, hematoma)	Oral examination, radiography
Temporomandibular joint problems	Physical examination, radiography

TABLE 2-2. Causes of Regurgitation

Esophageal obstruction
- Foreign body
- Granuloma
- Periesophageal mass or fibrosis
- Persistent right aortic arch (PRAA) and other vascular ring anomalies
- Stricture

Megaesophagus
- Idiopathic megaesophagus
  - Congenital
  - Acquired
- Secondary megaesophagus

Miscellaneous causes
- Esophagitis
- Esophageal diverticulum
- Esophageal fistula
- Hiatal hernia

3. If **no abnormalities** are found, then contrast radiographs with fluoroscopy may delineate a pharyngeal or upper esophageal sphincter problem.

B. **Regurgitation** must be differentiated from vomiting (see Chapter 3). Diagnostic approaches to esophageal disease are discussed in Chapter 41 II A 4.

V. TREATMENT

A. **Dysphagia.** Treatment is aimed at the primary disorder. Parenteral fluid administration may be necessary.

B. **Regurgitation.** Treatment is aimed at the primary disorder. Retention of ingesta in the esophagus should be minimized by elevating the food and water supply and feeding the animal multiple small meals, consisting of a food of optimal consistency (the optimal consistency varies with the individual).

Chapter 3

Vomiting

I. DEFINITION

A. **Vomiting** is a reflex act characterized by forceful expulsion of gastric or small intestinal contents from the stomach, coordinated by the vomiting center in the medulla.

1. The vomiting center can be stimulated directly by drugs and toxins (endogenous and exogenous).
2. It can be triggered by afferent nerves from the viscera (especially the abdominal viscera), the chemoreceptor trigger zone, the vestibular apparatus, or the cerebrum.

B. **Hematemesis** is the vomiting of blood.

II. CAUSES of vomiting are summarized in Tables 3-1 through 3-4.

A. Vomiting soon after eating is most commonly due to overeating, dietary indiscretion, or gastritis.

B. Vomiting more than 8 hours after eating is more suggestive of gastric outflow obstruction or a motility disorder.

III. CLINICAL FINDINGS

A. Vomiting is usually preceded by nausea (evidenced by **hypersalivation, frequent swallowing,** and **restlessness**) and **anxiety** and is accompanied by repeated contractions of the diaphragm and abdomen. The ejected material may be digested or undigested and often contains bile.

B. Clinical findings that may accompany vomiting vary according to the cause of the vomiting (see Tables 3-1 through 3-4).

TABLE 3-1. Causes of Acute Vomiting without Systemic Signs of Illness

Cause	Common Concurrent Clinical Findings
Change in diet	Diarrhea
Dietary intolerance	Diarrhea
Dietary indiscretion	Diarrhea
Gastric foreign body	Abdominal discomfort
Motion sickness	Usually none
Medication	Variable, depending on the medication
Parasitic infection	Diarrhea
Psychogenic	Usually none
Early stage of a more chronic or serious disorder	Variable

TABLE 3-2. Causes of Acute Vomiting with Systemic Signs of Illness

Cause	Possible Concurrent Clinical Findings
Extra-gastrointestinal disorders	
Central nervous system (CNS) disease	Abnormal mentation, neurologic deficits
Diabetic ketoacidosis	Polyuria, polydipsia, anorexia, depression, dehydration, weight loss, polyphagia
Hepatic disease	Anorexia, diarrhea, icterus, ascites, neurologic abnormalities
Hypercalcemia	Weakness, anorexia, polyuria, polydipsia
Hypoadrenocorticism	Anorexia, diarrhea, dehydration, weakness, bradycardia
Hypocalcemia	Anorexia, muscle twitches, tetany
Hypokalemia	Weakness, polyuria
Pancreatitis	Anorexia, cranial abdominal discomfort, fever, dehydration, diarrhea, icterus
Peritonitis	Anorexia, depression, dehydration, abdominal pain, shock
Prostatitis	Anorexia, fever, hematuria, palpable prostatomegaly, prostatic pain
Pyometra	Anorexia, polyuria, polydipsia, vaginal discharge, depression, fever
Renal disease	Anorexia, depression, weight loss, polyuria or oliguria
Sepsis	Anorexia, fever, depression, dehydration
Urinary obstruction	Anorexia, abdominal discomfort
Vestibular disease	Head tilt, nystagmus
Primary gastrointestinal disorders	
Gastric dilatation	Anorexia, cranial abdominal distention
Gastric dilatation/volvulus (GDV)	Cranial abdominal distention, nonproductive retching, shock
Gastritis or enteritis	Anorexia, diarrhea, dehydration
Hemorrhagic gastroenteritis (HGE)	Hematemesis, hemorrhagic diarrhea, dehydration
Intestinal volvulus	Abdominal pain, shock
Neoplasia	Variable, depending on the type and site of neoplasia
Obstipation	Anorexia; dehydration; palpable, distended, firm colon
Obstruction (gastric or intestinal)	Diarrhea, abdominal discomfort, dehydration, shock
Parasitic infection	Diarrhea
Ulcers	Hematemesis, melena, abdominal discomfort, pale mucous membranes
Viral infection	Diarrhea, fever
Early stage of a more chronic disorder	Variable
Miscellaneous causes	
Diaphragmatic hernia	Anorexia, respiratory distress, history of trauma
Hyperthermia	Hyperthermia, depression, shock
Medications	Variable, depending on the medication
Toxins	Variable, depending on the toxin

TABLE 3-3. Causes of Chronic or Intermittent Vomiting

Cause	Possible Concurrent Clinical Findings
Extra-gastrointestinal disorders	
Diabetes mellitus	Polyuria, polydipsia, weight loss, polyphagia
Heartworm disease (cats)	Anorexia, coughing, dyspnea
Hepatic disease	Anorexia, diarrhea, icterus, ascites
Hyperthyroidism (cats)	Polyuria, polydipsia, polyphagia, weight loss, diarrhea, hyperactivity, palpable cervical mass
Hypoadrenocorticism	Anorexia, diarrhea, weight loss, weakness, bradycardia
Hypocalcemia	Anorexia, muscle twitching, tetany
Pancreatitis	Anorexia, cranial abdominal discomfort, fever, diarrhea
Renal disease	Anorexia, depression, polyuria and polydipsia or oliguria, weight loss
Primary gastrointestinal disorders	
Colitis	Large bowel diarrhea
Chronic inflammatory gastritis	Anorexia, weight loss, diarrhea
Enterogastric reflux (bilious vomiting syndrome)	Usually none
Fungal infection	Anorexia, fever, diarrhea, lymphadenopathy, other organ involvement
Idiopathic gastric hypomotility	Anorexia
Inflammatory bowel disease	Anorexia, diarrhea, weight loss
Irritable bowel syndrome	Diarrhea
Neoplasia	Variable, depending on the type and site of neoplasia
Obstruction	
Gastric antral mucosal hypertrophy	Usually none
Pyloric stenosis	Usually none
Upper intestinal (partial)	Diarrhea, anorexia, weight loss
Parasitic infection	Diarrhea
Ulcers (usually secondary to another disorder)	Anorexia, hematemesis, melena, abdominal discomfort, pale mucous membranes, other signs specific to the underlying disease
Miscellaneous causes	
Diaphragmatic hernia	Anorexia, history of trauma
Abdominal neoplasia	Variable, depending on the type and site of neoplasia

TABLE 3-4. Causes of Hematemesis

Cause	Possible Concurrent Clinical Findings
Gastrointestinal disorders	
Gastritis or enteritis	Anorexia, diarrhea, dehydration
Hemorrhagic gastroenteritis (HGE)	Depression, hemorrhagic diarrhea, dehydration
Neoplasia	Variable, depending on the type and site of neoplasia
Ulcers (usually secondary to another disorder)	Anorexia, melena, abdominal discomfort, pale mucous membranes, other signs specific to the underlying disorder
Other causes	
Coagulopathy	Petechiae, ecchymoses, other types of hemorrhage
Swallowed blood from hemoptysis	Cough, hemoptysis, tachypnea
Swallowed blood from oral hemorrhage	Oral lesions

IV. DIAGNOSTIC APPROACHES

A. Acute vomiting without systemic signs of illness

1. The diagnosis is often one of exclusion based on the **history** and the **physical examination.** A lack of response to conservative medical therapy indicates a need for additional testing.
2. Ascariasis is detected by fecal examination or response to treatment with pyrantel pamoate.

B. Acute vomiting with systemic signs of illness or chronic intermittent vomiting

1. **Complete blood count (CBC)**
   a. **Leukocytosis** may be seen with pancreatitis, peritonitis, pyometra, sepsis, or severe gastrointestinal (GI) inflammation.
   b. **An increased hematocrit but normal serum protein concentration** in a dog with hematemesis and bloody diarrhea is highly suggestive of hemorrhagic gastroenteritis (HGE).
   c. **Nonregenerative anemia** may be seen with chronic disease, peracute or chronic blood loss, or malnutrition.
   d. **Regenerative anemia** may be seen with subacute gastric hemorrhage.
   e. **Eosinophilia** may be seen with some parasitic infections, eosinophilic gastroenteritis, or hypoadrenocorticism.
2. **Serum biochemical profile**
   a. **Hypochloremic metabolic alkalosis** suggests loss of gastric acid because of gastric or upper duodenal vomiting.
   b. **Hyper- or hypocalcemia** may be the cause of the vomiting.
   c. **Hypoproteinemia** may result from blood loss, severe inflammation, or hepatic failure (hypoalbuminemia).
   d. **Hyperglycemia** is consistent with diabetes mellitus if concurrent glucosuria is present.
   e. **Increased serum hepatic enzyme concentrations** may be seen with hepatic disease.
   f. **Hypoalbuminemia** may be seen with hepatic failure, severe inflammatory disease, or severe GI hemorrhage.

g. **Hypoglycemia** may be seen with hypoadrenocorticism, pancreatitis, hepatic failure, and sepsis.
h. **Hyperkalemia, hyponatremia,** and **hypochloremia** may be seen with hypoadrenocorticism.
i. **Increased serum amylase** and **lipase concentrations** are suggestive of pancreatitis.
j. **Azotemia** with concurrent **isosthenuria** is most consistent with renal failure but can also be seen with acute hypoadrenocorticism.
k. **Hyperbilirubinemia** may be seen with hepatic disease or biliary obstruction caused by pancreatitis.

3. **Urinalysis**
   a. **Glucosuria** is consistent with diabetes mellitus if hyperglycemia is also present. **Concurrent ketonuria** suggests diabetic ketoacidosis.
   b. **Isosthenuria** may be seen with diabetes mellitus, hepatic failure, hypercalcemia, hypoadrenocorticism, hypokalemia, pyometra, and renal failure.
4. **Fecal flotation** may reveal parasitic infection.
5. **Radiology**
   a. **Survey radiographs**
      (1) Hepatomegaly or microhepatica may be seen with hepatic disease.
      (2) Loss of cranial abdominal detail may be seen with pancreatitis.
      (3) A generalized loss of abdominal detail may be seen with ascites (e.g., hepatic failure) or peritonitis.
      (4) A large, fluid-filled tubular structure (i.e., an enlarged uterus) may be seen in the caudal abdomen with pyometra.
      (5) Renomegaly or small kidneys may be seen with renal disease.
      (6) Radiographic findings suggestive of gastric disease are discussed in Chapter 41 III A 5 a (1).
      (7) A mass, lymphadenopathy, or other organomegaly may also be diagnostic.
   b. **Contrast radiographs** (with fluoroscopy if possible) may be helpful [see Chapter 41 III A 5 a (2)].
6. An **adrenocorticotrophic hormone (ACTH) stimulation test** is indicated if the history, clinical findings, or laboratory results suggest hyperadrenocorticism.
7. **Serum bile acid concentrations** should be assessed if the history, clinical findings, or laboratory results suggest hepatic failure.
8. **Ultrasound**
   a. A gastric foreign body, mass lesion, or gastric wall thickening may be visible.
   b. Ultrasound can better assess any mass or change in organ size seen on survey radiographs.
9. **Endoscopic evaluation** (see Chapter 41 III A 5 c) may help with the diagnosis.
10. **Exploratory laparotomy**
    a. Full-thickness gastrointestinal biopsies and biopsies of multiple organs can be obtained.
    b. Surgery may be diagnostic as well as therapeutic in some situations (e.g., foreign bodies, neoplasms, obstructive lesions, peritonitis).

C. **Hematemesis** is usually an indication for a diagnostic evaluation.

1. **History.** The owner should be questioned about any current medications or the presence of a cough.
2. **Physical examination**
   a. The mouth and nose should be examined for hemorrhage.
   b. The skin should be evaluated for any masses (i.e., possible mast cell tumors).
3. **Laboratory tests** can be assessed to rule out extra-GI causes of GI ulceration or hemorrhage (see Chapter 41 III B 4) if no abnormalities are found on physical examination. Useful tests include the following:

a. **CBC**
b. **Serum biochemical profile**
c. **Urinalysis**
d. **Clotting tests** [i.e., activated clotting time or prothrombin time (PT) and partial thromboplastin time (PTT)]

4. **Upper GI endoscopy** should be performed if laboratory test results are within normal limits. Endoscopy may be used to look for erosions or ulcers and to obtain biopsies for histopathology.

V. TREATMENT

A. Acute vomiting without systemic signs of illness. Food and water should be withheld for at least 12 hours to rest the GI tract, progressing to small amounts of water for 12–24 hours and later small meals of a bland, low-fat diet (e.g., cottage cheese or boiled meat mixed with rice or potatoes).

B. Acute vomiting with systemic signs of illness

1. The primary disorder should be treated.
2. Food and water should be withheld for at least 12–24 hours to rest the GI tract. Parenteral fluids are often needed during this time to correct or prevent dehydration and to correct electrolyte imbalances.
3. Antiemetics (e.g., chlorpromazine, prochlorperazine, metoclopramide) can be considered if vomiting is excessive; however, it should be remembered that these agents do not resolve the main problem. Metoclopramide is contraindicated in the presence of an obstruction. Phenothiazines are contraindicated in animals with severe seizure disorders.

C. Chronic intermittent vomiting

1. The primary disorder should be treated.
2. Parenteral fluid administration is not often needed but should be instituted if dehydration or electrolyte imbalances are present.

D. Hematemesis

1. Food and water should be withheld and parenteral fluid administered to correct and maintain hydration and to correct any electrolyte imbalances.
2. A transfusion may also be needed if the hemorrhage is severe.
3. Because gastrointestinal ulceration is the most common cause of hematemesis, treatment with sucralfate and a histamine-2 (H_2) antagonist may be instituted while awaiting the results of diagnostic tests.