

Feline Health Topics

for veterinarians

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Part I: Management of Persistent Vomiting in the Cat

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Vomiting is clinically important because of its association with various diseases and the potentially life-threatening consequences such as aspiration pneumonia, fluid and electrolyte depletion, acid-base imbalances, and esophagitis. Patient management includes determining the medical significance of vomiting, and detecting and treating the cause of vomiting. If the cause is undetermined, it is necessary to adopt a rational approach to controlling emesis.

Physiology of Vomiting

Vomiting is a reflex act that is initiated by stimulating the vomiting center in the medulla. The vomiting center can be stimulated directly or indirectly via the chemoreceptor trigger zone (CRTZ), which is located in the area postrema. The blood-brain barrier may be limited at this point enabling blood-borne substances such as toxins or drugs to stimulate the

CRTZ. Neurological input from the vestibular nucleus can also stimulate the vomiting center. Disease or irritation of the gastrointestinal tract (GIT), abdominal organs or peritoneum, and cerebral diseases can directly stimulate the vomiting center via visceral receptors and vagal afferents.

Once the vomiting center is adequately stimulated a set of visceral events is initiated. These include the sequential inhibition of proximal gastrointestinal motility, a retrograde power contraction in the small intestine, and antral relaxation that enables transfer of intestinal contents to the stomach. These events are followed by moderate amplitude contractions in the antrum and intestine, and shortening of the intra-abdominal esophagus. Dilatation of the cardia and lower esophageal sphincter enables transfer of gastric contents to the esophagus during retching and vomiting. Retching often precedes vomiting and is characterized by rhythmic inspiratory movements against a closed glottis. Negative intrathoracic pressure during retching prevents expulsion of esoph-

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ageal contents. During vomiting the abdominal muscles contract and the intrathoracic and intra-abdominal pressures are positive which results in the forceful expulsion of gastric contents from the mouth.

Causes of Vomiting

There are so many potential causes of vomiting that it is often easiest to think in broad terms initially (i.e., gastric, intestinal, intra-abdominal non-GIT, metabolic-endocrine, drugs, dietary, neurologic, infectious diseases). After categorizing the vomiting into one of the following groups, then consider more specific causes.

Gastric—gastritis, ulceration, neoplasia, outflow obstruction, foreign bodies, motility/functional disorders

Intestinal—inflammatory bowel disease, neoplasia, foreign bodies, intussusception, functional disorders

Intra-abdominal (non -GIT)—pancreatitis, pancreatic neoplasia, hepatitis, cholangiohepatitis, biliary obstruction, nephritis, pyelonephritis, nephrolithiasis, urinary obstruction, pyometra, peritonitis

Metabolic/Endocrine—uremia, hyperthyroidism, diabetic ketoacidosis, hepatic encephalopathy, hypoadrenocorticism, hypercalcemia, septicemia

Drugs—xylazine, digoxin, erythromycin (?), chemotherapy

Toxins—strychnine, ethylene glycol, lead

Dietary—indiscretion, intolerance, allergy

Neurologic—vestibular disease, encephalitis, neoplasia, raised intracranial pressure

Infectious—panleukopenia, feline infectious peritonitis, Salmonella, Helicobacter

Parasitic—Ollulanus, Physaloptera, heartworm

Patient Evaluation and Diagnostic Approach

The initial diagnostic plan is to determine acute and self-limiting problems versus those that require more thorough investigation and treatment. If vomiting is acute and the cat is systemically well, the packed cell volume and total protein can be measured to evaluate hydration status and a fecal examination performed to detect endoparasites. In these patients further diagnostic testing is usually not warranted as vomiting often resolves on its own or after short-term symptomatic therapy.

If the animal is systemically unhealthy, has been vomiting for more than a week, or has hematemesis, bloody diarrhea, or abdominal pain, a more aggressive work-up is necessary to define the nature of the problem. (See figure 1 for a basic diagnostic procedure.)

Most causes of vomiting, including foreign body or intussusception, are usually detected or ruled out by taking a detailed history, performing a thorough physical examination, routine laboratory tests (CBC, serum chemistry profile, urinalysis, fecal; and T₄, FeLV, FIV, lipase/TLI where indicated), and abdominal radiographs. Abdominal ultrasound is useful for detecting pancreatic lesions, confirming gastrointestinal thickening, and sampling masses and parenchymal abnormalities. If these tests are negative or show abnormalities compatible with primary gastric or intestinal disease, endoscopic examination of the stomach and upper duodenum or contrast radiography are the principal noninvasive diagnostic options. Endoscopy enables detailed examination

Feline Health Topics

A publication for veterinary professionals

The ultimate purpose of the Cornell Feline Health Center is to improve the health of cats everywhere by developing methods to prevent or cure feline diseases, and by providing continuing education to veterinarians and cat owners. All contributions are tax-deductible.

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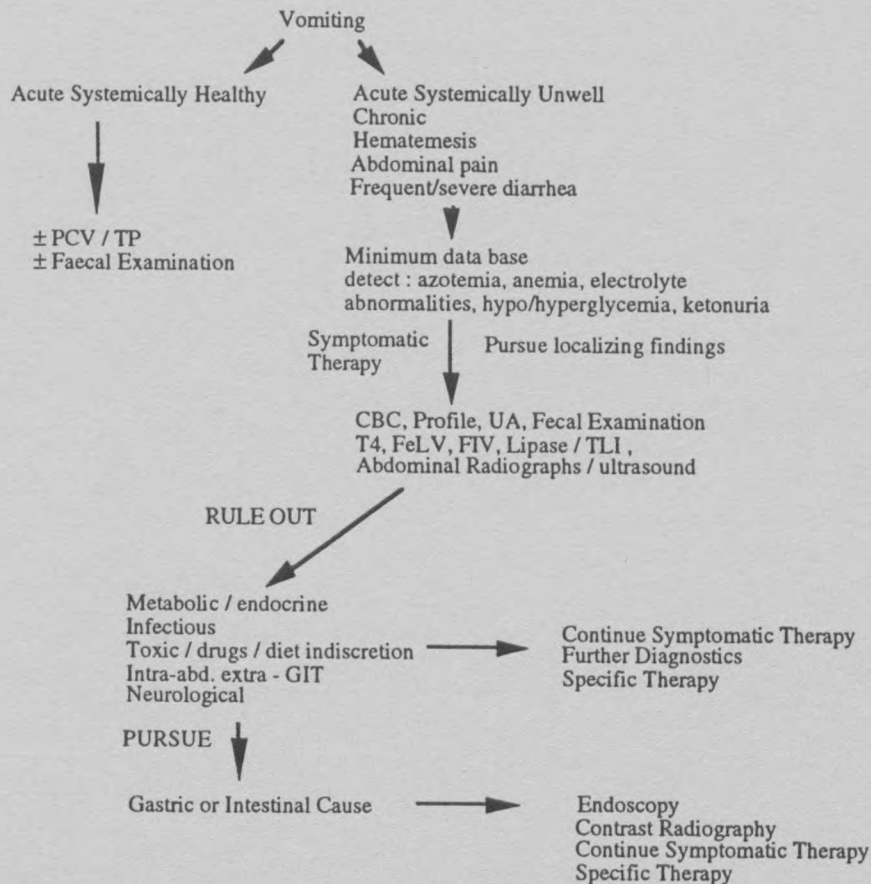
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Figure 1. Diagnostic Approach



which is high in carbohydrate, restricted in fat, and moderate in protein as this facilitates gastric emptying and digestion. Limited antigen diets can be employed in patients with chronic intermittent vomiting which is thought to be due to food intolerance/sensitivity. Gastric protectants (e.g., sucralfate) can be used to bind toxins and protect the gastrointestinal mucosa where vomiting is associated with gastritis or gastric ulceration.

Inhibitors of gastric acid secretion (usually H₂ antagonists) are used to limit gastric erosion/ulceration in vomiting patients with gastritis/ulceration and those con-

and sampling of the gastric and duodenal mucosa with minimal patient discomfort and is generally accepted as the best method of evaluating mucosal abnormalities. Radiographic contrast studies (fluoroscopy) are a good way of examining functional (emptying) disorders of the stomach and the anatomy and patency of the intestinal tract distal to the duodenum. Some patients require both endoscopy and radiography to adequately evaluate their disorder.

Therapy for Vomiting

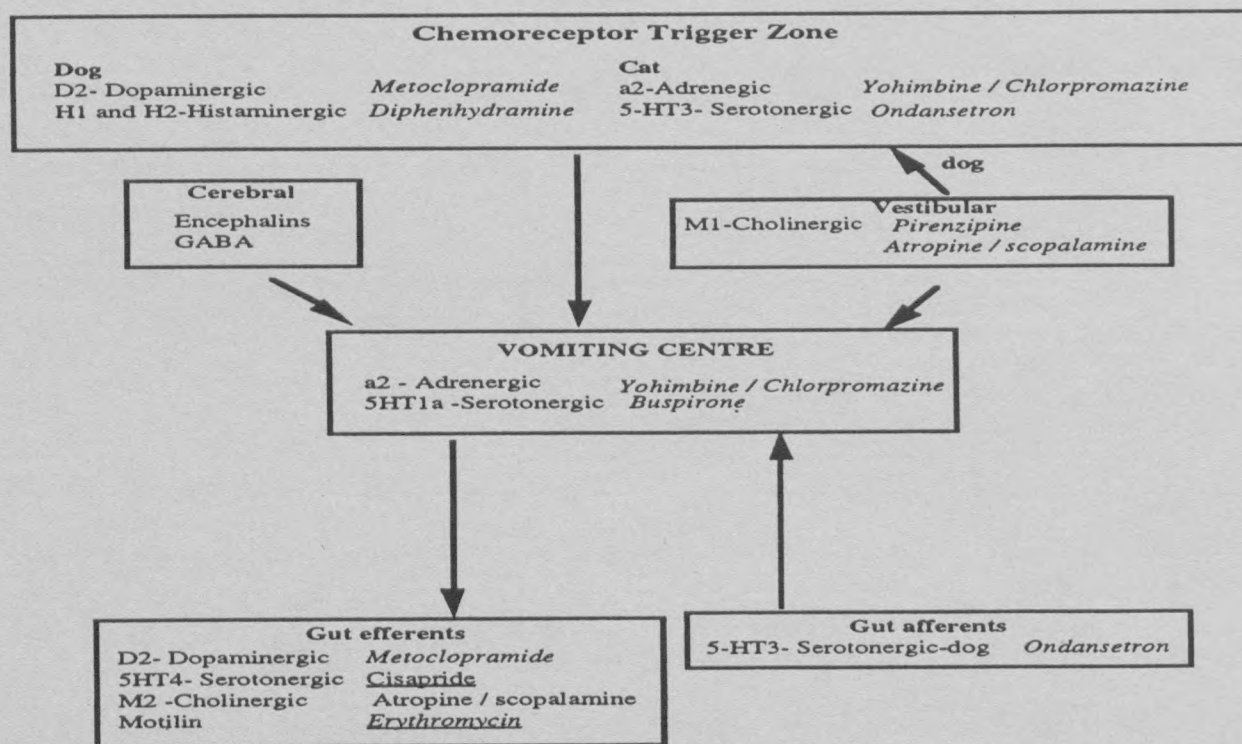
Parenteral fluid therapy (usually intravenous) should be tailored to correct dehydration and electrolyte and acid-base abnormalities. Patients with persistent vomiting are held NPO for 24 to 48 hours where vomiting is acute and severe followed by a gradual transition to a bland diet when vomiting decreases. Patients with chronic intermittent vomiting may benefit from a diet

considered at risk of developing gastrointestinal ulceration (e.g. shock) or esophagitis. Inhibition of gastric acid may also limit the hypochloremia and alkalosis that is associated with gastric outflow obstruction.

Antiemetics are indicated in patients with vomiting that compromises hydration status and affects electrolyte and acid-base balances; patients that are high risk for esophagitis or aspiration pneumonia; and patients that are distressed by repeated vomiting. Chemotherapy (cyclophosphamide-oncovin-prednisone protocol) has successfully induced remission (7 to 8 month median survival) in cats with gastric lymphoma (most cats are FeLV-positive). Surgery is indicated to remove large foreign bodies, treat some causes of pyloric outflow obstruction and to obtain full thickness gastrointestinal biopsies.

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Figure 2. Pharmacological Control of Vomiting



The pharmacological control of vomiting involves antagonizing the central and peripheral receptors that cause emesis and stimulating receptors that promote ordered gastrointestinal motility. The receptor subtypes involved in vomiting and examples of drugs that are commonly employed in the management of vomiting are summarized in figure 2. Drugs that act at these receptors may limit or effect emesis. Some drugs have more than one mechanism of action, such as phenothiazines (e.g. chlorpromazine) which are antagonists of α 1- and α 2- adrenergic, H1- and H2-histaminergic, and D2-dopaminergic receptors. Metoclopramide hydrochloride antagonizes D2-dopaminergic and 5HT3-serotonergic receptors, and has cholinergic effects on smooth muscles.

Antiemetics are contraindicated in patients with gastrointestinal infection or toxicity as the drugs may limit expulsion of the infectious or toxic agent. The side effects of some drugs may also limit their appli-

cation. Phenothiazines may cause hypotension and sedation and decrease the seizure threshold in animals with epilepsy. Nonselective cholinergic receptor antagonists (other than pirenzepine), such as atropine, scopolamine, aminopentamide, and isopropamide may cause ileus, delayed gastric emptying, and dry mouth. Prokinetic agents (e.g. metoclopramide, cisapride, erythromycin) are contraindicated where there is a suspicion of intestinal obstruction. Certain antiemetics (ondansetron, metoclopramide, scopolamine, erythromycin, and pirenzepine) are not recommended or require caution or ineffective when used in the cat. ■

(Part II will present strategies for managing persistent vomiting.)

Dr. Kenneth Simpson is assistant professor of medicine in the Department of Clinical Sciences at the College of Veterinary Medicine at Cornell University.



Research Briefs

Fulminant Hepatic Failure Associated with Oral Administration of Diazepam in 11 Cats

(Authors: S.A. Center, T.H. Elston, P.H. Rowland, D.K. Rosen, et al.)—Eleven nonpurebred cats developed signs of hepatotoxicosis 5 to 11 days after initial oral treatment with diazepam. Cats ranged 2 to 8 years of age, and nearly equal numbers of males and females (all neutered) were affected. Dosage of diazepam ranged from 1.0 mg PO every 24 hours, to 2.5 mg PO every 12 hours. Treatment with diazepam was recommended for inappropriate urination, suspected urethral spasm, or aggression.

Common clinical signs included anorexia, vomiting, dehydration, lethargy, hypothermia, and jaundice. Notable clinicopathologic features included no specific hematologic changes; relatively low to low concentrations of serum glucose, serum cholesterol, and BUN; high serum creatinine concentration; markedly high serum alanine transaminase and aspartate transaminase activity; modestly high serum alkaline phosphatase and γ -glutamyltransferase activity; moderate hyperbilirubinemia; and moderately to markedly high serum creatine kinase activity.

The cases reported in this study represent circumstantial evidence that oral administration of diazepam can result in severe diffuse hepatocellular injury in cats. The authors believe that this represented idiosyncratic hepatotoxicosis, because 10 of 11 cats were treated within the established dosage range and most cats (10/11) had never before been treated with a benzodiazepine drug, based on their medical records.

The findings of this study suggest a likely relationship between the repeated oral administration of diazepam and development of fulminant hepatic ne-

crosis in some cats. Therefore, careful consideration of diagnosis and treatment options in cats with suspected behavioral problems seem prudent. This implies that hematologic and biochemical evaluations should be completed prior to recommended drug treatment.

The authors believe that diazepam treatment should be discontinued if a cat becomes unduly sedated, lethargic, or anorectic, or begins vomiting during the first week of treatment. A baseline biochemical analysis should be performed prior to treatment, then 3 to 5 days after treatment is initiated. Also suggested is that serum alanine transaminase and aspartate transaminase activity be evaluated at this early treatment interval, to possibly identify the need for drug discontinuation at a state where recovery may be possible. (Resource: *J Amer Vet Med Assn* 209(3):618-625, 1996)

A Fatal Case of Intrathoracic Cuterebriasis in a Cat

(Authors: S.D. Fitzgerald, C.A. Johnson, E.J. Peck)—A seven-year-old, castrated male, domestic short-hair cat was presented with lethargy, fever, and sneezing. In spite of intensive treatment, the cat's condition progressively worsened to severe dyspnea and death. At necropsy, a single, second instar larval stage of *Cuterebra* sp. was found in the trachea. This represents an unusual site for *Cuterebra* migration in an aberrant host. The cause of death was attributed to a combination of local tissue damage and anaphylaxis. Veterinarians should include *Cuterebra* migration in their differential diagnosis list for dyspnea. (Resource: *J Amer Anim Hosp Assn* 32(4): 353-357, 1996)

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Pulmonary and Mediastinal Metastases of a Vaccination-site Sarcoma in a Cat

(Authors: D.G. Rudmann, W.G. Vanalstine, F. Doddy, G.E. Sandusky, et al.)—Sarcomas at vaccination sites in cats were first reported in 1992. Recent retrospective studies have confirmed an association between these vaccination-site sarcomas (VSS) and feline leukemia virus (FeLV) and/or rabies vaccines. In most cases, VSS are locally invasive fibrosarcomas that tend to recur but rarely metastasize. The authors report the mediastinal and pulmonary metastases of a VSS in a FeLV- and feline-immunodeficiency-virus- negative, 8-year-old, domestic short-hair cat. The primary sarcoma was removed from an interscapular vaccination site and diagnosed as a VSS three months prior to radiographic lesions suggestive of pulmonary and mediastinal metastases. At necropsy, there were multiple pulmonary and mediastinal nodules that histologically and ultrastructurally were fibrosarcomas, cytomorphologically similar to the VSS. In addition, immunohistochemical staining patterns of the VSS and metastatic sites were consistent with that described for VSS. Recent reports of pulmonary and mediastinal metastases of interscapular VSS emphasize the importance of early diagnosis and treatment of these tumors. (Resource: Vet Pathol 33(4):466-469, 1996)

Relationship Between Inflammatory Hepatic Disease and Inflammatory Bowel Disease, Pancreatitis, and Nephritis in Cats

(Authors: D.J. Weiss, J.M. Gagne, P.J. Armstrong)—This study tries to determine whether cats with inflammatory hepatic disease had concurrent inflammatory bowel disease (IBD), pancreatitis, or chronic interstitial nephritis. This was a prospective case study that reviewed histologic sections of liver, intestine, pancreas, and kidney from 78 cats that had previous necropsy examinations. From the

tissue specimens, researchers tried to determine the prevalence of lymphocytic portal hepatitis, cholangiohepatitis, IBD, pancreatitis, and chronic interstitial nephritis, and the relationship among them. Results showed that 36 cats had lymphocytic portal hepatitis, 18 had cholangiohepatitis, and 24 did not have any inflammatory hepatic disease. The prevalence of IBD (10/36; 28%) and pancreatitis (5/36; 14%) in cats with lymphocytic portal hepatitis was not significantly different from cats without inflammatory hepatic disease. The prevalence of IBD (15/18; 83%) and pancreatitis (9/18; 50%) was greater ($P < 0.05$) for cats with cholangiohepatitis, compared with cats without inflammatory hepatic disease. Thirty-nine percent of cats (7/18) with cholangiohepatitis had IBD and pancreatitis. Evidence of IBD in association with cholangiohepatitis was characterized by infiltration of lymphocytes and plasma cells into the lamina propria; however, neutrophilic infiltrates also were found in 6 of 15 (40%) cats with cholangiohepatitis. Pancreatitis was mild in all cats. The clinical implication of this study is that cats with a diagnosis of cholangiohepatitis should be evaluated for IBD and pancreatitis. (Resource: J Amer Vet Med Assn 209(6):1114-1116, 1996.)

Other Research Articles of Interest

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Buffington CA and Chew DJ: Intermittent alkaline urine in a cat fed an acidifying diet. J Amer Vet Med Assn 209(1):103-104, 1996.

Crenshaw KL and Peterson ME: Pretreatment clinical and laboratory evaluation of cats with diabetes mellitus: 104 cases (1992-1994). J Amer Vet Med Assn 209(5):943, 1996.

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Feline Nutrition

April 5-6, 1997 Ithaca, New York

Program

This intensive course is designed for cat owners, cat breeders, veterinary technicians, and other people with a serious interest in cats. It will be taught by faculty of the Cornell University College of Veterinary Medicine and the College of Agriculture and Life Sciences.

Topics to be covered include:

- Anatomy of the Feline Digestive System
- How the Feline Digestive System Works
- Function of Nutrients
- Nutrient Requirements
- Sources of Nutrients
- Characteristics of Commercial Foods
- Feeding Programs for Growing Kittens and Breeding Animals

Faculty

Dr. Harold F. Hintz, Professor of Animal Nutrition; Chair of the Department of Animal Science in the College of Agriculture and Life Sciences;
Dr. Francis A. Kallfelz, the Mark L. Morris Professor of Veterinary Nutrition; Director of the Veterinary Medical Teaching Hospital;
Dr. Arleigh J. Reynolds, Assistant Professor of Veterinary Nutrition, Department of Clinical Sciences.

Accommodations

Rooms have been reserved at the following locations: Best Western University Inn, (607) 272-6100, \$60 single/\$65 double; Ramada Inn, (607) 257-3100, \$70 single/double; Holiday Inn, (607) 272-1000, \$67 single/double. The above reduced rates are available if, when you make your reservations, you mention the program by name; please make your reservations early. For additional housing options, please call the Special Programs office at (607) 255-7259.

Program Charge

The program charge is \$285 and includes tuition; course materials; a certificate of completion; and meals throughout the program including continental breakfasts, lunch, and refreshment breaks on Saturday and Sunday. Persons whose cancellations are received in writing by March 21 will receive a full refund. Cancellations received after March 21 are subject to a \$100 cancellation fee. Substitutions may be made at any time before the program begins. Program costs may be tax deductible.

Travel Planning

Participants should arrive by 8:30 a.m., Saturday, April 5. The program will conclude by 4:30 p.m. on Sunday, April 6.

Further Information

Feline Nutrition, Cornell University, B20 Day Hall, Ithaca, NY 14853-2801; telephone: (607) 255-7259; fax: (607) 255-8942; e-mail: sp@sce.cornell.edu

Registration Form

Please print or type:

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Registration should be submitted as soon as possible since enrollment will be limited. Send this form, along with payment or charge authorization, to: **Feline Nutrition**, Cornell University, Box 251, B20 Day Hall, Ithaca, NY 14853-2801; fax (607) 255-8942.

Research Briefs *(continued from page 6)*

Harvey CJ, Lopez JW, Hendrick MJ: An uncommon intestinal manifestation of feline infectious peritonitis: 26 cases (1986-1993). *J Amer Vet Med Assn* 209(6):1117, 1996.

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Photocopies of the above articles are available by making your request via mail to the Flower-Sprecher Library, College of Veterinary Medicine, Ithaca, NY 14853; or by telephone at (607) 253-3510; or by fax at (607) 253-3080.



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