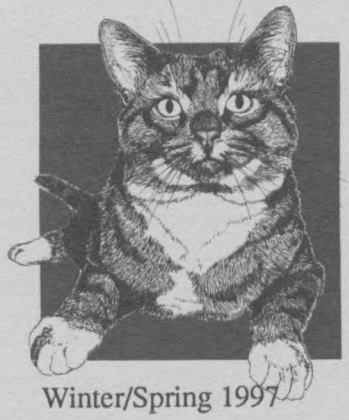


# Feline Health Topics

*for veterinarians*

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## Dr. Fred W. Scott Retires

**James R. Richards, D.V.M.**

On December 31, 1996, my colleague, mentor, and friend Dr. Fred W. Scott retired as the director of the Cornell Feline Health Center and as professor of virology at the College of Veterinary Medicine. Dr. Scott has achieved many milestones in his career. He has written or co-authored 197 research papers and four textbooks. Hundreds of veterinary students have learned the nuances of infectious diseases while under his tutelage as professor of virology. He also has been the advisor for 15 graduate students in the field of veterinary microbiology, and 5 postdoctoral fellows/research associates. Millions of cats have lived longer and healthier lives as a result of his dedication to studying feline diseases over the past two decades. He has received several prestigious awards in recognition of his advancement of feline medicine, including the Distinguished Scholar in Veterinary Medicine, National Academies of Practice; Carnation Award for Outstanding Achievement in Feline Medicine (1990); Academy of Feline Medicine Honorary First Fellow (1990); and the American Association of Feline Practitioners Research Award (1975).

Dr. Scott was named the director of the newly founded Cornell Feline Health Center in 1974. He started with no more than an idea—to create a veterinary medical specialty center whose sole purpose was to improve the health and welfare of cats everywhere. Dr. Scott ultimately led the Center to its present position as the most recognized and respected feline specialty center in the world.

My task as the new director is a daunting one. I hope to continue in the tradition of Dr. Scott's leadership, and build on the strong foundation that he laid. If I can infuse the Center with a fraction of his compassion, and guide it with a portion of his insight and skill, then the present and future of the Center are bright indeed. ■

### Inside this issue ...



### Cornell's Companion Animal Hospital Offers Radioiodine Therapy for Feline Hyperthyroidism

Radioiodine ($^{131}I$) provides a simple, effective and safe therapy for cats with hyperthyroidism. The Companion Animal Hospital at Cornell University's College of Veterinary Medicine has the necessary isolation facility to board treated cats for the 16- to 21-day required stay. Veterinarians can refer their clients' cats to obtain treatment by contacting the CAH (607-253-3060). The cost ranges from $850-$1,000 for the entire stay. ■

# Task Force Recommends Vaccine Protocol

The Vaccine-Associated Feline Sarcoma Task Force (VAFSTF) has been formed in response to the increased incidence of soft tissue sarcomas occurring at vaccine sites. The task force is facilitating the investigation of the epidemiology, etiopathogenesis, treatment, and prevention of sarcomas, as well as disseminate information to veterinarians and cat owners. The VAFSTF is a collaborative effort of the American Veterinary Medical Association (AVMA), the American Animal Hospital Association (AAHA), the American Association of Feline Practitioners (AAFP), and the Veterinary Cancer Society (VCS), and includes representatives from the U.S. Department of Agriculture's Animal and Plant Health Inspection Service (USDA/APHIS) and the Animal Health Institute (AHI).

Current recommendations and guidelines from the task force include:

1. The manufacturer's label recommendation is the only official item a veterinarian currently has to demonstrate the basis for vaccination.

2. Alternate vaccination routes (e.g., nasal, topical) should be considered if and when available.

3. The use of vaccines packaged in single-dose vials is encouraged.

4. Vaccination is a medical procedure and protocols should be individualized to the patient. Administration of any vaccine should proceed only after considering the medical significance and zoonotic potential of the infectious agent, the patient's risk of exposure, and relevant legal requirements.

5. Any occurrences of vaccine-associated sarcomas or other adverse reactions should be reported directly to the vaccine manufacturer and to the United States Pharmacopeia (U.S.P.). Information about the U.S.P. Practitioners' Reporting Program and a sample submission form is in the Journal of the American Veterinary Medical Association, Vol. 208, No. 3, February 1, 1996, pp. 361-363. Additional reporting forms can be obtained by calling 1-800-4-USP-PRN. Submission of the form can be facilitated by diagnostic laboratories if they include a report form with each diagnosis of vaccine-associated sarcoma. The record should include vaccine type, lot number and vaccination site; this information should also be incorporated into the patient's permanent medical file.

6. To further characterize the causal link and facilitate therapy of vaccine-associated sarcomas, the following general guidelines for vaccine (and other injectable product) administration are suggested:

a. Veterinarians should standardize vaccination (and other injection) protocols within their practice and document the location of the injection, the

*(continued on page 8)*

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.

*Director:* **James R. Richards, D.V.M.**
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# Part II: Strategies for Managing Persistent Vomiting

**Kenneth W. Simpson, BVM&S, Ph.D., MRCVS, D.A.C.V.I.M., D.E.C.V.I.M.**

## Uremia

The effects of uremic toxins on the CRTZ and afferent inputs from the inflamed stomach cause vomiting associated with uremia. Ameliorating uremia with fluid therapy, antagonizing the effects of uremic toxins on the CRTZ, and limiting afferent input from the inflamed gut are strategies to control vomiting. Uremic vomiting in cats is often most amenable to fluid therapy and treatment aimed at controlling the peripheral effects of uremia with an $H_2$ antagonist (e.g. famotidine 0.5-1.0 mg/kg SID-BID) and mucosal protectants (sucralfate 0.25-1.0 g PO TID). This contrasts with dogs where reducing CRTZ stimulation with a $D_2$-dopaminergic antagonist is necessary to control vomiting; however, $D_2$-dopaminergic anatgonists may be ineffective in reducing CRTZ stimulation in cats. Metoclopramide should not be given to patients receiving dopamine to promote diuresis; furthermore, it is not considered an effective central antiemetic in the cat. Cats with vomiting that are refractory to initial therapy for uremic gastritis may benefit from an $\alpha_2$-adrenergic antagonist such as chlorpromazine (0.2-0.4 mg/kg TID SC) or prochlorperazine (0.5 mkg/kg TID SC IM) to ensure adequate hydration.

## Gastritis and Gastric Ulceration

Vomiting in patients with acute gastritis or gastric ulceration is managed by providing adequate fluid therapy, restricting oral intake, and limiting afferent input from the inflamed gut by deceasing gastric acid secretion (e.g. $H_2$ antagonists) and providing mucosal protection (e.g. sucralfate). Where ulceration is severe and vomiting is not adequately controlled, chlorpromazine/prochlorperazine can be used as an adjunct therapy in the short term.

In patients with severe or persistent ulceration more complete inhibition of gastric acid secretion can be achieved with the H/K ATPase inhibitor, omeprazole (0.2-0.7 mg/kg SID PO). The safety and efficacy of omeprazole in cats has not been widely reported; however, it seems to be effective when given as part of combination therapy to cats with *Helicobacter* infection.

Mast cell tumors may cause vomiting via the central effects of histamine on the CRTZ (dogs) and the peripheral effects of histamine on gastric acid secretion, with resultant hyperacidity and ulceration. Treatment of mastocytosis with $H_1$ and $H_2$ histamine antagonists (e.g. diphenhydramine and famotidine) should reduce the effects of histamine. Omeprazole may be useful to limit acid secretion in patients refractory to $H_2$ antagonists. Corticosteroids are used to decrease tumor size and release of histamine.

Where chronic intermittent vomiting is associated with gastritis, a high-carbohydrate diet that is restricted in fat and moderate in protein may facilitate gastric emptying and digestion. Limited antigen diets can also be used in patients where gastritis and vomiting may be due to food intolerance or sensitivity.

The role of *Helicobacter spp.* in chronic vomiting in the cat is the subject of much speculation and debate. Where chronic intermittent vomiting and gastritis are associated with the presence of *Helicobacter spp.*, the combination of antibiotics (tetracycline or amoxicillin, and metronidazole), omeprazole,and bismuth subsalicylate may be effective in eliminating these bacteria and ameliorating vomiting. Care should be taken when administering bismuth subsalicylate to cats.

*(continued on next page)*

### Delayed gastric emptying

Delayed gastric emptying is caused by outflow obstruction or defective propulsion and is usually suspected by the vomiting of food 12 to 16 hours following ingestion. Other signs include abdominal discomfort, distention, bloating and intermittent anorexia. Outflow obstruction can be caused by polyps, foreign bodies, tumors, pyloric hypertrophy or stenosis, granulomata and extraluminal masses such as pancreatic tumors. Defective propulsion may result from primary gastric diseases such as gastritis, ulceration, neoplasia, and parasitism or non-gastric disorders such as stress, trauma, peritonitis, pancreatitis, infectious enteritis, electrolyte (especially hypokalemia) and metabolic derangements, drugs and surgery. The finding of hypochloremia, hypokalemia, metabolic alkalosis, and aciduria should raise the suspicion of an upper gastrointestinal obstruction or gastrinoma.

Radiography is used to investigate vomiting and to confirm delayed gastric emptying (retention of food or fluid after 16 to 17 hours following a meal) or delayed gastric emptying of liquid barium (30%w/v, 12-16 ml/kg via stomach tube; the stomach should empty within 15-60 minutes in cats), barium meal (normal range is within 10 to 15 hours) or barium polyspheres. Endoscopy is extremely useful for confirming gastric outflow obstruction and gastric and duodenal causes of decreased propulsion (e.g., ulcers, gastritis). Measurement of gastric pH and serum gastrin may help to determine the cause of gastric ulceration or mucosal abnormalities.

Treatment of gastric emptying disorders is directed at the underlying cause (e.g., surgery for pyloric outflow obstruction; antacids, mucosal protectants and/or antibiotics for gastritis). In non-obstructive situations gastric emptying can be enhanced by dietary modification. Diet modifications may include feeding small amounts of semi-liquid, protein- and fat- restricted diets at frequent intervals, or intestinal diets blended with water and mixed with an equal volume of boiled rice. Also administering prokinetic agents such as metoclopramide (0.2-0.5 mg/kg PO SC TID), or cisapride (0.1-0.5 mg/kg PO TID) may be beneficial.

### Idiopathic Inflammatory Bowel Disease

Idiopathic inflammatory bowel disease (IBD) describes a diverse group of intestinal disorders which are characterized by cellular infiltrates of the intestinal mucosa. Diagnosis is based on histological findings in intestinal biopsies and the exclusion of known causes of intestinal inflammation, such as endoparasites and dietary sensitivity. The most common inflammatory infiltrates are lymphocytes and plasma cells or eosinophils. Neutrophils or granulomatous inflammation are encountered less commonly.

*Lymphoplasmacytic Enteritis:*

Lymphoplasmacytic enteritis (LPE) is the most common type of inflammatory bowel disease in cats and dogs. It is characterized by the accumulation of excessive numbers of lymphocytes and plasma cells in the lamina propria of the intestine. The degree of cellular accumulation is variable and is subjectively categorized as mild, moderate, and severe. Moderate to severe lymphoplasmacytic enteritis can be associated with a protein-losing enteropathy, although this is rare in cats. The extent of inflammation appears variable, and ranges from the duodenum to the small and large bowels.

Vomiting, diarrhea, and weight loss are common clinical signs in cats with LPE. Vomitus often contains bile. Hairballs are frequent in LPE cats. Other findings include changes in appetite, excessive borborygmi, and abdominal discomfort. Physical findings range from normal to thickened intestines, mesenteric lymphadenopathy, marked weight loss, and ascites or edema in animals with severe protein-losing enteropathy.

A diagnosis of idiopathic lymphoplasmacytic enteritis is made by excluding systemic, parasitic, infectious, pancreatic and structural causes of chronic diarrhea and demonstrating excessive numbers of

lymphocytes and plasma cells in intestinal biopsies. Where vomiting is the presenting complaint, gastric disorders must also be excluded. There are no specific clinicopathological changes associated with lymphoplasmacytic enteritis. Hypoproteinemia associated with low albumin and globulin may be apparent in dogs, but is rare in cats. Leukocytosis may be present in some cats. Survey radiographs are normal but ultrasound may reveal diffuse thickening, mesenteric lymphadenopathy or small abdominal effusions in severe cases. Intestinal function tests are usually bypassed in patients with localizing signs, such as intestinal thickening or mesenteric lymphadenopathy, and in hypoproteinemic patients, in favor of endoscopic or surgical biopsy. The degree of lymphoplasmacytic infiltrate in intestinal biopsies is classified as mild, moderate, or severe. Villus atrophy is usually minimal. Severe villus atrophy and lymphoplasmacytic infiltrate is common with lymphosarcoma.

Treatment is based on the degree of cellular infiltrate and associated clinical and clinicopathological findings. Mild to moderate intestinal inflammation may be associated with dietary sensitivity or intolerance. A therapeutic trial with a highly digestible diet, which is restricted in fat, gluten-free and limited to a single protein source, may be undertaken to determine if dietary sensitivity or intolerance are present. Up to six weeks may be required to see a response. In patients who fail this trial and those with moderate to severe infiltrates, or hypoproteinemia, the administration of immunosuppressive agents is often required to achieve a response. Oral prednisolone (1.0-2.0 mg/kg PO BID) is the initial drug of choice. It is usually administered at an immunosuppressive dose for 2 to 3 weeks and then decreased by 50% every 2 to 3 weeks and then continued on an alternate day basis for 2 to 3 months. If clinical response is poor or the adverse effects of prednisolone predominate, azathioprine can be added to the regimen. Cats are more sensitive to azathioprine than dogs, but dosing at 0.3 mg/kg PO SID-EOD should be safe. The white cell count should be monitored every 2 to 4 weeks when azathioprine is being given. Metronidazole (15 mg/kg PO BID 10-14 days, then SID 10-14 days) can also be used in conjunction with corticosteroids to effect existing bacteria and the immune system. Successful treatment is accompanied by a decrease in clinical signs and an increase in plasma proteins. Once a patient has had 2 to 3 months remission from signs it may be possible to gradually withdraw immunosuppressive therapy. If signs recur, daily medication is continued until signs resolve then the dosage is gradually reduced. In patients that respond poorly to therapy or relapse after an initial response, lymphosarcoma should be ruled out.

The prognosis for lymphoplasmacytic enteritis is variable and depends on its severity. Many patients require prolonged treatment with glucocorticoids and diet. Since no accurate criteria exist for predicting response, it is wise to give a guarded prognosis.

*Eosinophilic Enteritis:*

Eosinophilic enteritis is characterized by the excessive accumulation of eosinophils in the lamina propria. It is speculated that it may result from an immunologic reaction to parasites or diet. The disease may also involve other areas of the gastrointestinal tract.

Chronic small bowel diarrhea accompanied by vomiting or weight loss are the principal clinical signs. Large bowel signs or vomiting predominate in some cases. Physical findings range from normal to focally or diffusely thickened intestines and marked weight loss.

The diagnosis of eosinophillic enteritis is achieved by adopting a similar approach to that described for lymphoplasmacytic enteritis (see above). Clinicopathologic abnormalities may include peripheral eosinophilia. Mast cell neoplasms, hypoadrenocorticism, and endoparasites can produce a similar spectrum of clinical signs and should be ruled out. The degree of eosinophilia can be extreme in cats and may be associated with eosinophilic infiltrates in the spleen,

liver, lymph nodes and bone marrow. Intestinal protein loss is less common than with lymphoplasmacytic enteritis.

Some patients may respond to a strict exclusion diet, though prednisolone (2.0 mg/kg PO SID) is usually required. Feeding an easily digestible diet that is restricted to a single novel protein source may help to maintain clinical remission. Prophylactic administration of an anthelminthic such as fenbendazole (50mg/kg PO SID 3 days) is warranted to treat potential visceral larval migrans that has been associated with eosinophilic gastroenteritis. Cats with hypereosinophilic syndrome often respond very poorly to treatment with immunosuppressive agents, diet and anthelminthics.

The prognosis is guarded because relapse is common. The prognosis in cats with hypereosinophilic syndrome is poor.

## Pancreatitis

Vomiting, lethargy, inappetence with elevated hepatic enzymes, hyperbilirubinemia, hyperglycemia, and glucosuria are frequently encountered in cats with acute pancreatitis. Pancreatitis may be the cause of diabetes mellitus in some cats, but the true association between these diseases is unclear. One study suggests that cats with pancreatitis and diabetes mellitus are very sensitive to insulin. Transient euglycemia and reduced insulin requirements after removal of a pancreatic abscess suggest that pancreatic inflammation or infection can exacerbate diabetes in cats. Transient diabetes mellitus has also been reported in a cat which was suspected of having pancreatitis. The antemortem diagnosis of feline pancreatitis is however rarely reported.

The major difficulty in detecting pancreatitis clinically is that the clinical signs, laboratory findings and abdominal radiographic abnormalities of cats with acute or chronic pancreatitis are non-specific and can easily be attributed to organ dysfunction in the absence of pancreatitis. For example, findings consistent with diabetes mellitus and hepatic lipidosis are indistinguishable in cats with and without pancreatitis. Potentially more specific indicators of pancreatic inflammation—serum amylase and lipase—appear to be of little utility in the cat. Antemortem diagnosis has hinged on a high index of clinical suspicion (especially cats with hepatic or biliary disease) and abdominal ultrasonography which enables pancreatitis to be detected. The development of more specific tests of pancreatic inflammation, such as the assay of feline trypsin-like immunoreactivity (fTLI), shows great promise in further facilitating the diagnosis of acute pancreatitis. The clinician is cautioned against basing a diagnosis of acute pancreatitis solely on the results of a single test. Diagnosis should be based on a combination of compatible clinical, clinicopathologic, and diagnostic imaging findings.

Vomiting in pancreatitis is probably due to direct afferent input to the vomiting center and ileus secondary to intestinal inflammation. However, anorexia is often more of a problem than chronic vomiting in cats with pancreatitis. Chlorpromazine or prochlorperazine may be useful in cats with persistent vomiting and pancreatitis. Ondansetron ($5HT_3$ receptor antagonist) may also have a role in limiting vomiting due to pancreatic or visceral stimulation; however, it requires evaluation in cats. The use of analgesia may also have a beneficial effect by decreasing afferent stimulation of the vomiting center.

## Cholangiohepatitis

Vomiting, anorexia, depression, weight loss, hepatomegaly and jaundice are common in cats with cholangiohepatitis. Suppurative cholangiohepatitis may be associated with fever, whereas lymphocytic cholangiohepatitis may present with ascites. Increased liver enzymes and serum bile acids are typically present along with hyperbilirubinemia or hyperbilirubinuria. Low BUN and albumin, and a bleeding tendency (vitamin K deficiency or synthetic failure) may be encounterd where liver dysfunction is severe. A high serum globulin concentration is often associated wth lymphocytic cholangiohepatitis.

Diagnosis requires liver biopsy and examination by a skilled pathologist. Care should be taken to distinguish other hepatopathies (e.g.hepatic lipidosis, hepatic lymphosarcoma, FIP) and to detect concommitant IBD and/or pancreatitis.

Treatment is aimed at decreasing hepatic inflammation and ameliorating the effects of decreased hepatic function. Ampicillin (10-20 mg/kg PO TID) and metronidazole (10-15 mg/kg PO BID) are used for suppurative cholangiohepatitis. Prednisolone is used for lymphocytic cholangiohepatitis. Vitamin K (5-10 mg), ursodiol (10 mg/kg PO BID), and a diet supplemented with vitamins and trace elements may be beneficial in both types of cholangiohepatitis. Where vomiting persists, the patient should be critically re-evaluated for pancreatitis and IBD.

### Motion Sickness

Stimuli from the vestibular system are thought to be the cause of motion sickness. Motion sickness in the cat does not appear to be controlled by histamine antagonists, but may be controlled with chlorpromazine.

### Idiopathic Vomiting

Symptomatic fluid therapy, diet restriction or modification, and antiemetics to control vomiting are indicated where vomiting is frequent or severe enough to cause derangements of fluid, electrolyte and acid-base balance. Antiemetics should not be given if intestinal obstruction or ingestion of a toxic substance is suspected. Antiemetic selection in patients with unknown causes of vomiting is based on a best guess and least harmful approach. Alpha-2 adrenergic antagonists (prochlorperazine, chlorpromazine) and $D_2$-dopaminergic antagonists (metoclopramide) are suggested as first and second choices. ■

### Further Reading:

Washabau RJ and Elie MS: Antiemetic Therapy. In:: Current Veterinary Therapy XII. pp. 679-684, 1995.

King GL. Animal Models in the Study of Vomiting. Can J Physiol Pharmacol 68: 260, 1990.

## Research Briefs

### Effects of Oral Ursodeoxycholic Acid in Healthy Cats on Clinicopathological Parameters, Serum Bile Acids and Light-microscopic and Ultrastructural Features of the Liver

(Authors— B.T. Nicholson, S.A. Center, J.F. Randolph, P.J. Rowland et al.)— A blind, placebo-controlled study evaluated the effects of ursodeoxycholic acid (UDCA) given orally at a dose of 15 mg/kg per day for eight weeks on the physical condition, hematological and serum biochemical profiles, urinalysis, total serum bile acids (TSBA) and hepatic histology of four healthy cats. There were no clinically important significant differences between the groups or within the treatment groups in clinicopathological parameters, TSBA concentrations or histology. A significant lower concentration/proportion of taurochenodeoxycholic acid was observed in the treated cats (P=0.05). Only one treated cat accumulated measurable quantities of UDCA, and the compound appeared to be non-toxic. It did not increase the concentration of TSBA, and accumulated minimally in the serum. It should be investigated for thereapeutic use in cats with hepatobiliary disease. (Resource: Res Vet Sci 61(3):258-262, 1996)

### Related Strains of Feline Infectious Peritonitis Virus Isolated from Immunocompromised Cats Infected with a Feline Enteric Coronavirus

(Authors—A.M. Poland, H. Vennema, J.E. Foley and N.C. Pedersen)—Two groups of cats were experimentally infected orally with the cat-passaged RM strain of feline enteric coronavirus (FECV-RM). One group of cats (n=19) had been chronically infected with feline immunodeficiency virus (FIV) for

## Research Briefs *(continued from page 7)*

over 6 years, while a second control group (n=20) consisted of FIV-naive siblings. Fecal virus shedding of FECV occurred in both groups starting on day 3 postinfection, nearly ceasing by 4 weeks in FIV-uninfected cats, but remaining at high levels in FIV-infected animals. FIV-infected cats shed virus for a longer period of time and at levels 10 to 100 times greater than those for FIV-uninfected cats. The coronavirus antibody response of the FIV-infected cats was delayed and of reduced titer compared with that of the FIV-uninfected animals. Cats in both groups remained asymptomatic for the first months following FECV-RM infection; however, 8 to 10 weeks postinfection two cats in the FIV-infected group developed feline infectious peritonitis (FIP). The FIP viruses (designated FIPV-UCD9 and UCD10) isolated from these two cats had almost complete genetic homology to each other and to the infecting FECV-RM. However, unlike FECV-RM, they readily induced FIP when inoculated intraperitoneally into specific-pathogen-free cats. This study confirms that FIPVs are frequently and rapidly arising mutants of FECV. Immunosuppression caused by chronic FIV infection may have enhanced the creation and selection of FIPV mutants by increasing the rate of FECV replication in the bowel and inhibiting the host's ability to combat the mutant viruses once they occurred. *(Resource: J Clin Microbiol 34(12):3180-3184, 1996)* ■

## Task Force Recommends Vaccine Protocol *(continued from page 2)*

type of vaccine or other injectable product administered, and the manufacturer and serial number of the vaccine in the patient's permanent medical record.

b. *Recommended injection sites:*

- Vaccines containing antigens limited to panleukopenia, feline herpesvirus type-1, and feline calicivirus (+/- chlamydia) be administered on the right shoulder according to the manufacturer's recommendations.
- Vaccines containing rabies antigen (and any other antigen) be administered on the right rear limb as distally as possible according to the manufacturer's recommendations.
- Vaccines containing feline leukemia virus antigen (+/- any other antigen except rabies) be administered on the left rear limb as distally as possible according to the manufacturer's recommendations.

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A client brochure, *Vaccines and Sarcomas: A Concern for Cat Owners,* has been developed by the task force. Veterinarians can obtain copies by contacting AAHA, AVMA, VCS, or the Cornell Feline Health Center. ■

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