Gastrointestinal Disorders of the Cat

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Few patients can compete with the chronically diarrheic cat in imparting such a sense of frustration in the clinician. When faced with such a cat, it often seems that logic and basic scientific principles go out the window and both the patient and client suffer because of the veterinarian's failure to properly investigate the pathophysiologic basis of the diarrhea.

In 1977, Gruffyd-Jones and colleagues at the University of Bristol, England, described an apparently new syndrome of cats consisting of chronic diarrhea and protrusion of the nictitating membrane.[22] Soon afterward, numerous letters appeared in the correspondence columns relating the experience of practicing veterinarians with similar cats. Viruses, pancreatic enzyme deficiency, and an association of the condition with the cat's molting cycle were all suggested as causes. One correspondent even noted an improvement of his patients by using herbal remedies containing the bark of *Berberis vulgaris*, the leaves of *Rubus idaeus*, the root of *Rheum*, and a small quantity of *Foeniculum vulgare*! The continuing use of such remedies serves to remind us that knowledge of feline gastrointestinal disease has lagged slightly behind the other rapidly developing areas of feline medicine.

It should be emphasized that this article is not a comprehensive review of feline gastroenterology, since some of the more common disorders such as panleukopenia have not been included. Many of the subjects discussed are, at present, of questionable clinical significance, but future studies will likely prove them to be important diagnostic considerations when evaluating the vomiting or diarrheic cat.

ESOPHAGEAL DISORDERS

Megaesophagus

In the absence of congenital vascular ring anomalies or pyloric dysfunction, megaesophagus is an uncommon disorder in cats.[2] Examination

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of pedigrees from related cats with this disorder suggests that megaesophagus is inherited as a recessive non–sex-linked trait.[14]

The feline esophagus is composed of circular and longitudinal layers of smooth muscle in its middle and caudal sections. There is no identifiable muscular thickening of the lower esophagus consistent with a distinct anatomic sphincter.[5] The smooth muscle of the feline esophagus is innervated by postganglionic parasympathetic fibers originating in the intrinsic autonomic plexuses within the smooth muscle. In comparison with the dog, where these plexuses are limited in number in the cranial esophagus, the plexuses of the cat are equally distributed throughout the length of the esophagus.[5] Regulation of plexus activity, and thus coordination of muscular contraction, is supplied through preganglionic fibers carried in the vagus and arising from neurons in the dorsal motor nucleus of the brainstem.[55]

Experimental destruction of the dorsal motor nuclei in cats results in esophageal dysfunction resembling megaesophagus.[14] Following bilateral cervical vagotomy, complete paralysis of the feline esophagus develops with eventual gradual return of peristalsis to the areas composed of smooth muscle. No loss of ganglion cells in the intrinsic plexuses, subsequent to this procedure, has been noticed.[8]

The cause of megaesophagus in the cat is not understood. There does not appear to be a congenital deficiency of cells in the dorsal motor nuclei[1] or intrinsic plexuses[12] of cats with megaesophagus. In fact a significant increase in the number of cells was noted in the dorsal motor nuclei of cats with megaesophagus when compared with normal cats. Although the observed changes may have been artifactual because of a delay in perfusion of brain tissue compared with the control group, the cells of the dorsal motor nucleus and the nucleus ambiguus of these cats were smaller and more pyknotic than those of the controls.[13]

Reports of histologic examination of esophageal tissues from cats with megaesophagus are conflicting. No evidence of neuronal degeneration or primary neurogenic atrophy of cells in the intrinsic plexuses has been noted.[2] However, a lack of definition of the nuclei and nucleoli of ganglion cells was observed in affected cats in another study. Occasional degenerative changes in the esophageal muscle with some monocytic infiltrate were also present.[12]

Two Siamese cats that had been vomiting since weaning were found to have megaesophagus with accompanying areas of heterotopic gastric mucosa in the esophagus.[2] It is speculated that the changes may have occurred as an incidental developmental abnormality or as a result of a metaplastic healing process secondary to an esophagitis caused by reflux of gastric contents into the flaccid esophagus.[2]

Esophagitis

Esophagitis of the cat and dog has been reported as a complication of anesthesia from minor surgical procedures. Pooling of gastric contents in the esophagus during anesthesia with a concurrent loss of the swallowing reflex and secondary esophageal peristalsis was considered to be the cause. Within a few days of the surgical procedure, affected cats lost weight

rapidly, became clinically dehydrated, and regurgitated food though remaining hungry.[47] In persons, esophagitis is considered to be a result of dysfunction of the lower esophageal sphincter with a subsequent failure of the antireflux barrier at the gastroesophageal junction.[18] The effectiveness of this barrier becomes further compromised during esophagitis as esophageal inflammation severely disrupts orderly esophageal motor activity and further decreases lower esophageal sphincter pressure.[29] Esophagitis in humans, and presumably in cats and dogs, may therefore become a self-perpetuating disorder that requires prompt recognition and therapy. Interestingly, under experimental conditions using dogs, neither acid nor bile alone produced significant esophagitis. When 1 per cent taurocholate was added to acid and instilled into the esophagus, severe inflammation was produced.[29]

Anterior Mediastinal Lymphosarcoma

Both esophageal and pulmonary dysfunction may occur when a rapidly expanding mass develops in the anterior thorax. In a recent series of 30 cats with this tumor, 9 cats (30 per cent) were presented because of recurrent regurgitation (in comparison with 43 per cent with the primary problem of dyspnea).[20] Anterior mediastinal lymphosarcoma is a disease of the young adult cat (peak incidence, 1 to 2 years), and oriental breeds are predisposed.[20] Feline leukemia virus was detected in 77 per cent of cats with this tumor in one study.[24]

Cats with anterior mediastinal lymphosarcoma are frequently presented with an acute onset of severe dyspnea. Tachypnea, intolerance to handling, and orthopnea are often noticed on the examination table. Lung sounds are usually muffled or absent, and the anterior thorax is frequently incompressible. Occasionally a mass may be noted at the thoracic inlet. Dilatation of the cervical esophagus and obstruction to passage of contrast material at the thoracic inlet have been noted.[20]

The site of primary neoplastic proliferation is the thymus. The neoplastic lymphoid cells induce rosette formation with guinea-pig erythrocytes[31] and metastasize to the paracortical or thymus-dependent areas of local lymph nodes, thus confirming their origin as T-cells.[39] Direct extension of the tumor into the lungs and pericardium usually occurs, and microscopic infiltration of the splenic white pulp is common.[39]

GASTRIC DISORDERS

Parasitic Gastritis

The clinical importance of *Ollulanus tricuspis*, the feline stomach worm, as a cause of chronic vomiting in cats in the United States remains to be clarified. The current prevalence of the parasite in the United States is unknown, but the worm is known to infest certain cat colonies in the Pacific Northwest.[26] Recently the parasite was isolated from 206 of 542 cats (38 per cent) in rural Germany.[27]

The parasite has a direct life cycle. The viviparous adult female parasite (which is larger than the male) measures 0.04 by 0.8 to 1 mm and is thus invisible to the naked eye.[26] Larvae develop to the third stage in utero and are then released into the gastric lumen.[10] Infective larvae leave the cat in vomited material, which is then ingested by other cats to complete the life cycle. Larvae are not found in the intestines or lungs.[10] It has been suggested that long-haired cats, owing to their predisposition to gastric trichobezoars causing recurrent vomiting, may be more efficient transmitters of the parasite.[25]

Many cats infected with *Ollulanus tricuspis* are asymptomatic[23] although more severe infections may cause unthriftiness, local gastric irritation, and vomiting.[26] The adult parasites either reside in a layer of mucus overlying the gastric mucosa or penetrate the gastric crypts.[25] Local mucosal erosions and increased production of mucus may occur in heavier infections.[10] Fibrous connective tissue may be deposited around the gastric crypts.[26] Recently severe chronic fibrosing gastritis has been reported in two cats infected with *Ollulanus tricuspis*.[23, 25]

It would therefore seem appropriate to include this parasite as a possible cause of chronic vomiting in pet cats, considering its prevalence abroad and the recent confirmation of its presence in the United States.[26] Detection of the parasite depends on examination of vomitus with the aid of a dissecting or compound microscope.[26] Since larvae do not enter the lower intestinal tract, fecal examinations are not helpful.

Pyloric Dysfunction

Chronic vomiting, corrected by pyloroplasty, was diagnosed as pyloric stenosis in five cats (four of which were Siamese).[59, 60] Examination of the pylorus of these cats, however, revealed no evidence of muscular hypertrophy or change in caliber of the pyloric region.

In a series of 13 cats with pyloric dysfunction (12 of which were Siamese), a number of the cats had concurrent megaesophagus.[48] Radiographic examination demonstrated retention of contrast material in the esophagus, increased gastric volume, marked reduction in the quantity of barium passing through the pylorus at each relaxation (in spite of normal peristaltic activity) and delayed gastric emptying time. The pylorus of all cats appeared normal at the time of surgery. Following pyloromyotomy, gastric abnormalities and clinical signs disappeared unless severe esophageal dilatation was initially present. It was suggested that this complex may be due to autonomic nervous system dysfunction and may be an inherited trait in the Siamese breed. Two cats in this series with megaesophagus were presented with signs of dyspnea rather than regurgitation.

INTESTINAL DISORDERS

BACTERIAL ENTERITIDES

Campylobacteriosis

Recently, interest has arisen concerning the possible role of domestic pets as reservoirs for campylobacteriosis. Little is known, however, con-

cerning the role of this organism as an etiologic agent of diarrhea in the cat. *Campylobacter fetus* subsp. *jejuni* is a curved, motile, gram-negative, microaerophilic rod with a wide range of avian and mammalian hosts. Isolation of the organism from stool specimens or rectal swabs requires selecting plating media such as Skirrow's, Butzler's or Campy-BAP.[4]

Considerable discrepancy exists between reported prevalence of Campylobacter infection in cats. Positive cultures were obtained from 45 per cent of clinically normal cats in an English humane society.[7] In a survey of animals less than three months old from kennels in the Denver area, 34.9 per cent of puppies and 6 per cent of kittens yielded positive stool cultures.[3] In contrast, fecal cultures from 92 diarrheic and 10 normal cats produced only two isolates.[21]

When pet cats have been incriminated with zoonotic spread of Campylobacter, family members have usually become ill several days after acquiring a kitten suffering from acute diarrhea.[51, 52] The risk of being infected with this organism by a normal cat or dog appears negligible, and the risk of spread can be minimized by frequent hand-washing and prevention of close contact between children and diarrheic kittens.[51] Humans appear to be more susceptible to a given dose of Campylobacter than are kittens or puppies.[50]

Clarification of the role of Campylobacter as a pathogen in the young cat will ultimately depend on the isolation (on selective media) of this bacteria from diarrheic feces in the absence of other bacterial, dietary, toxic, or viral causes of diarrhea, and observation of a specific response to antimicrobial therapy.

Tuberculosis

Although feline tuberculosis is an extremely uncommon gastrointestinal disorder, the infection may closely resemble other more common enteric diseases. The clinical signs of depression, weight loss, intermittent anorexia, and diarrhea, associated with splenomegaly, peripheral lymphadenopathy, and palpable abdominal masses, prompted a preliminary diagnosis of alimentary lymphosarcoma in one recently reported case.[44] Malabsorptive disease was eventually documented using D-xylose absorption—a finding unusual in alimentary lymphosarcoma, which more commonly causes obstructive disease. As with many other gastrointestinal disorders, Siamese cats appear to be more susceptible to tuberculosis.

VIRAL ENTERITIDES

Enteric Coronavirus

Enteric coronavirus may be the cause of a nonfatal enteritis in kittens from 4 to 12 weeks of age.[49] The virus is widespread in the cat population and almost all multiple-cat households and catteries are infected. The virus persists in recovered animals, many of whom are continuous shedders.

The virus is highly infectious by the oral route and has an affinity for the apical columnar epithelium of the intestinal villi from the mid-duo-

denum to the cecum. Although most adult cats develop an asymptomatic infection upon exposure, enteric coronavirus may cause a clinical enteritis of varying severity in recently weaned kittens. Low-grade fever, vomiting and diarrhea have been noted. Fresh blood in the stools and dehydration are present in severe cases.

Enteric coronavirus is closely related antigenically to feline infectious peritonitis virus and cross-reacts with this virus in serologic tests presently used in the diagnosis of feline infectious peritonitis. Previous infection with enteric coronavirus appears to hypersensitize cats to later infection with feline infectious peritonitis virus. The discovery of enteric coronavirus may also have provided clues as to the means of persistence of feline infectious peritonitis virus in the feline population. Although the two viruses differ markedly in their infectivity and tissue affinity, it has been postulated that feline infectious peritonitis virus is an infrequent but continually occurring mutation of the ubiquitous enteric coronavirus.[49]

Feline Rotavirus

Rotaviral particles have been observed in the diarrheic feces of two cats.[11, 53] A specific feline rotavirus was also isolated from five of 185 feline fecal samples, four of the isolates coming from normal cats.[33] From serologic surveys for rotavirus in the cat population, it would appear that subclinical infections are common but the virus does have pathogenic potential in certain cats.

Titers against lamb rotavirus antigen of 1:40 or greater were found in 28 per cent of 94 cats from colonies or households.[53] Twenty of 61 hospitalized cats (32.8 per cent) had an antibody titer of 1:5 or greater.[33] In Belfast, Ireland, five of six cats were found to have titers between 1:40 and 1:640.[40]

Experimental infection of a susceptible kitten with feline rotavirus caused diarrhea. By immunofluorescent studies, virus was observed in the villous epithelial cells in the mid and distal small intestine. Some villi were swollen and infiltrated with macrophages and neutrophils.[53] As with the other enteric viruses discussed here, the importance of feline rotavirus as a pathogen capable of inducing clinical disease remains to be clarified.

Feline Astrovirus

Electron microscopic examination of diarrheic feces from a young kitten revealed astroviral particles.[34] The kitten made an uneventful recovery from an illness consisting of mild dehydration, gas-distended loops of small bowel, and green watery diarrhea. At the present time, this is the only report of astroviral infection in the cat.

NON-NEOPLASTIC INFILTRATIVE DISEASES

Although preliminary observations concerning malassimilation syndromes in the cat have been reported,[38, 62] there remains a paucity of data concerning specific infiltrative small bowel disease.

Eosinophilic enteritis has recently been included as a component of a complex of hypereosinophilic syndromes in the cat.[30] Affected cats are presented because of vomiting, diarrhea with occasional blood, and variable weight loss. On abdominal palpation, small bowel thickening is consistent and some cats may also exhibit hepatosplenomegaly and mesenteric lymphadenopathy. A moderate to marked eosinophilia accompanies the condition. At necropsy, examination of small intestine shows eosinophilic infiltration into the mucosa and submucosa, fibrosis of the lamina propria, and hypertrophy of muscle layers. Some cats have multivisceral infiltration involving liver, spleen, mesenteric lymph nodes, or bone marrow. The syndrome does not appear to be induced by feline leukemia virus and, unfortunately, responds unsatisfactorily to corticosteroids.[30]

Methods used in the diagnosis of malassimilation syndromes of the dog are described elsewhere in this symposium. Quantitative fecal fat analysis is one method for confirming the presence of maldigestive or malabsorptive disease. It has recently been determined that more than 98 per cent of normal cats excrete less than 3 gm of fecal fat per 24-hour period. Diarrheic cats excreting more than 3.5 gm of fecal fat per 24 hours should be further evaluated for malassimilative disease.[36]

INTESTINAL NEOPLASMS

Intestinal Adenocarcinoma

Several recent articles have provided excellent new knowledge concerning this tumor, which comprises the single largest group of primary gastrointestinal neoplasms of the cat.[28, 46, 58]

The tumor occurs more commonly in older cats. Mean age at the time of diagnosis is approximately 11 years. The Siamese breed and the male cat are predisposed to development of this tumor.[46] Clinical signs are nonspecific and include progressive weight loss, depression, intermittent bouts of vomiting and diarrhea, and anorexia lasting from a few days to several months. Occasionally abdominal swelling, from the accumulation of ascitic fluid, may be noticed.

Intestinal adenocarcinomas are most commonly found in the jejunum,[58] ileum,[46] and ileocecocolic region.[6] Less frequently they arise in the colon. At laparotomy, small intestinal adenocarcinomas usually appear as pale annular strictures affecting 1 to 6 cm of intestine. Prestenotic and poststenotic dilatation of surrounding small intestine may be observed, and the proximal small bowel may be turgid owing to hypertrophy of muscle layers.[28, 46] Greater than 50 per cent of cats in one series had an accompanying neoplastic effusion in the peritoneal cavity.[46] On cut surface the tumor is white and firm because an abundance of fibrous tissue. As the tumor ages, this fibrous tissue tends to contract, thus forming an annular stricture with subsequent intestinal obstruction. Histologically, the tumor may be classified as a tubular adenocarcinoma, undifferentiated carcinoma, or mucinous adenocarcinoma. The prognosis for the patient, however, is

independent of the location of the tumor in the intestine or its histologic pattern.[58]

The tumor metastasizes most frequently to the abdominal serosa and mesenteric lymph nodes. Occasional metastases are found in the liver and lungs.[58] The mean survival time for 19 cats undergoing tumor resection was 20 weeks.[58] Recently, however, a postsurgical survival time of 28 months has been reported.[45]

Alimentary Lymphosarcoma

An alimentary lymphosarcoma must fulfill several criteria in order for a diagnosis of primary intestinal lymphosarcoma to be justified.[28] The tumor must arise within the intestine (and not in a mesenteric lymph node), it must invade the muscular layers of the intestine, and it must not be associated with peripheral lymphadenopathy.

Alimentary lymphosarcoma accounts for 15.2[24] to 46.6[41] per cent of feline lymphosarcomas. The tumor more commonly affects middle-aged to older cats (mean age, 7 to 10 years) and male cats.[28] Only 4.9 per cent of cats with primary intestinal lymphosarcoma are infected with feline leukemia virus.[24] All segments of the intestine may be affected, but the tumor most commonly arises in the small intestine. In comparison with intestinal adenocarcinomas, no segment of the small intestine is particularly predisposed to tumor development.

Clinical signs associated with intestinal lymphosarcoma tend to be insidious, progressive, and dependent on the segment of intestine involved. Cats with tumors in the proximal small bowel are usually presented with a history of vomiting 3 to 6 hours after the last substantial meal, progressive weight loss, and lack of appetite. Cats with ileal tumors often are presented with a history of chronic diarrhea and depression with a late onset of vomiting occurring 16 to 24 hours after the last meal.[28]

The tumor often appears as a fusiform thickening of the intestinal wall. Because of a lack of supporting structure in the area of the tumor, the intestinal wall may eventually dilate and even rupture at the tumor site.[28] Microscopic infiltration of main mesenteric lymph nodes is invariable.[39] The selective early infiltration of the germinal centers of mesenteric lymph nodes[39] and the presence of surface immunoglobulins and complement receptors on the neoplastic lymphocytes[31] confirm that the tumor is of B-cell origin. Malignant cells are frequently found in the splenic white pulp and renal cortices and, to a lesser extent, in the liver and bone marrow.[39]

Intestinal Mast Cell Tumors

Twenty-four cats with intestinal neoplasms were found to have primary intestinal mast cell tumors.[1] In contrast to other intestinal tumors, no purebred cats were affected. Once again, middle-aged to older cats (average age, 12.3 years) were susceptible.

Cats with intestinal mastocytomas were presented with histories of weight loss, vomiting, and diarrhea. Twenty-one of the 24 tumors were in the small intestine, where no predilection site was noted. The tumors appeared as firm segmental thickenings of the intestinal wall and on cut-

surface were a creamy-tan color. Metastases were found most frequently in the mesenteric lymph nodes, liver, and spleen.

In contrast to lesions reported in systemic mastocytosis, no intestinal ulceration was observed. This may be due to a lack of production of vasoactive substances since this neoplasm has the typical ultrastructure of degranulated mast cells.[1]

MECHANICAL DISORDERS

Presence of a Pancreatico-mesojejunal Ligament

Signs of chronic diarrhea and abdominal distention in 32 young cats (aged between 5 and 10 months) disappeared within 3 weeks of removal of a pancreaticomesojejunal ligament.[42] The 1- to 2-mm diameter ligament originated from the cranial pancreaticoduodenal vein, passed under the ileum and colon, and inserted onto the left side of the mesojejunum near the jejunal vein or jejunal lymph nodes. Necropsy examination of 2900 random cats revealed the ligament to be present in 202 (7 per cent).[42]

Further studies are needed to confirm the validity of this anomaly as a cause of diarrhea in the young cat. It would seem wise, however, based on the report cited above, to include a search for this ligament during celiotomies on young diarrheic cats.

Ileal Polyps

Ileal mucosal polyps associated with adjacent non-neoplastic intestinal stricture have been observed to cause intestinal obstruction in related Siamese cats.[43] Compared with ileal strictures caused by adenocarcinomas, this polypoid change is probably an unusual cause of obstruction.

GASTROINTESTINAL MANIFESTATIONS OF SYSTEMIC DISORDERS

Feline Dirofilariasis

Many cats infected with *Dirofilaria immitis* are asymptomatic, whereas others may be presented with acute or chronic clinical signs. Several reports have included vomiting and/or diarrhea as a manifestation of feline dirofilariasis.[9, 16, 17, 54, 57] The most common historical complaint, however, is respiratory distress associated with coughing, dyspnea, and lethargy.[9] Cats with acute manifestations may be presented with signs of syncope, dyspnea, collapse, vomiting, or neurologic signs. Chronically affected cats frequently exhibit intermittent bouts of similar signs over a prolonged period of time.[17] Feline dirofilariasis is currently an important diagnostic consideration when evaluating the chronically vomiting cat.

Feline Hyperthyroidism

It is unlikely that owners of hyperthyroid cats would present their pets solely because of the observation of gastrointestinal signs, since other

polysystemic changes are usually more dramatic. However, historical complaints concerning affected cats frequently include changes in the pattern of defecation. Stools are often frequent, soft, and voluminous. The clinician's suspicion of hyperthyroidism is aroused by the more characteristic signs of progressive weight loss in spite of a good appetite, hyperactivity and restlessness, polydipsia, cardiovascular arrhythmias, and palpable thyroid enlargement.[32, 56]

Systemic Mastocytosis

Chronic vomiting and diarrhea, weight loss, anorexia, and anemia frequently accompany systemic mastocytosis in the cat.[15, 37, 61] This neoplastic proliferation of mast cells arises in tissues containing hematopoietic precursors, and massive splenomegaly resulting in mild abdominal distention is a characteristic finding.[19, 37] In contrast to the primary intestinal mast cell tumor described earlier, the mast cells in systemic mastocytosis are rich in metachromatic granules containing histamine. Excessive histamine release is thought to damage the vascular network of the intestinal submucosa, resulting in ischemic necrosis and ulcerative gastroduodenitis. The mucosal ulceration is exacerbated by increased secretion of hydrochloric acid by parietal cells stimulated by histamine.[35] The vomiting associated with mastocytosis is thought to be due to irritation from ulcerative gastroduodenitis, gastric hypermotility induced by elevated histamine levels, and splenomegaly causing pressure on adjacent viscera.

SUMMARY

Several areas of feline gastroenterology deserve critical attention in the near future. For example, as compared with the dog, little is known about the various causes of malabsorptive disease in the cat. So frequently, intestinal biopsy samples reveal nothing more than intestinal thickening with fibrosis and nonspecific mild cellular infiltration, and the inciting cause is never determined.

It is, perhaps, wrong to be critical about the use of the bark of *Berberis vulgaris* and the root of *Rheum* in modern feline gastroenterology, since most of us occasionally use unconventional therapies. It has been rumored that I have been known to advocate the daily addition of a tablespoon of pumpkin-pie filling to the food of cats suffering from recurrent constipation and acquired megacolon. The rumors are true, and colonic evacuation is sometimes promoted with the use of this unusual bulking agent after traditional drug therapy has failed. We all have our weaknesses!

ADDENDUM

Since early 1982, numerous cats have been presented to British veterinarians with an apparently new disease characterized by autonomic nervous system dysfunction. Known as the "Key-Gaskell Syndrome," the disease is currently recognized as an autonomic polyganglionopathy because

the major pathologic changes are in the neuronal perikaryon of autonomic ganglia. Signs include persistent pupillary dilatation, decreased lacrimal and nasal secretions, bradycardia, megaesophagus, and constipation. Efforts to identify causative viral or toxic agents have so far been unsuccessful, and to this date no cases have been reported in the United States.

REFERENCES

1. Alroy, J., Leav, I., DeLellis, R. A., et al.: Distinctive intestinal mast cell neoplasms in domestic cats. Lab. Invest., 33:159–167, 1975.
2. Bishop, L. M., Kelly, D. F., Gibbs, C., et al.: Megaloesophagus and associated gastric heterotopia in the cat. Vet. Pathol., 16:444–449, 1979.
3. Blaser, M. J., LaForce, F. M., Wilson, N. A., et al.: Reservoirs for human campylobacterosis. J. Infect. Dis., 141:665–669, 1980.
4. Blaser, M. J., and Reller, L. B.: Campylobacter enteritis. N. Engl. J. Med., 305:1444–1452, 1981.
5. Bremner, C. G., Shorter, R. G., and Ellis, F. H.: Anatomy of the feline esophagus with special reference to its muscular wall and phrenoesophageal membrane. J. Surg. Res., 10:327–331, 1970.
6. Brodey, R. S.: Alimentary tract neoplasms of the cat: A clinicopathologic survey of 46 cases. Am. J. Vet. Res., 27:74–80, 1966.
7. Bruce, D., Zochowski, W., and Fleming, G. A.: Campylobacter infections in cats and dogs. Vet. Rec., 107:200–201, 1980.
8. Burgess, J. N., Schlegel, J. F., and Ellis, F. H.: The effect of denervation on feline esophageal function and morphology. J. Surg. Res., 12:24–33, 1972.
9. Calvert, C. A., and Mandell, C. P.: Diagnosis and management of feline heartworm disease. J. Am. Vet. Med. Assoc., 180:550–552, 1982.
10. Cameron, T. W. M.: On the pathogenicity of the stomach and lungworms of the cat. J. Helminthol., 10:231–234, 1932.
11. Chrystie, I. L., Goldwater, P. N., and Banatvala, J. E.: Rotavirus infection in a domestic cat. Vet. Rec., 105:404–405, 1979.
12. Clifford, D. H.: Myenteric ganglial cells of the esophagus in cats with achalasia of the esophagus. Am. J. Vet. Res., 34:1333–1336, 1973.
13. Clifford, D. H., Barboza, P. F. T., and Pirsch, J. G.: The motor nuclei of the vagus nerve in cats with and without congenital achalasia of the esophagus. Br. Vet. J., 136:74–83, 1980.
14. Clifford, D. H., Soifer, F. K., Wilson, C. F., et al.: Congenital achalasia of the esophagus in 4 cats of common ancestry. J. Am. Vet. Med. Assoc., 158:1554–1560, 1971.
15. Confer, A. W., Langloss, J. M., and Cashell, I. G.: Longterm survival of 2 cats with mastocytosis. J. Am. Vet. Med. Assoc., 172:160–161, 1978.
16. Dillon, R., Sakas, P. S., Buxton, B. A., et al.: Indirect immuno-fluorescence testing for diagnosis of occult *Dirofilaria immitis* infection in 3 cats. J. Am. Vet. Med. Assoc., 180:80–82, 1982.
17. Donahoe, J. M. R.: Clinical aspects of feline dirofilariasis. *In* Morgan, H. C. (ed.): Proceedings of the Heartworm Symposium '74. Bonner Springs, Kansas, V. M. Publishing, 1975.
18. Eastwood, G. L., Castell, D. O., and Higgs, R. H.: Experimental esophagitis in cats impairs lower esophageal sphincter pressure. Gastroenterology, 69:146–153, 1975.
19. Garner, F. M., and Lingreman, C. H.: Mast cell neoplasms of the domestic cat. Pathol. Vet., 7:517–530, 1970.
20. Gruffyd-Jones, T. J.: Clinical and radiologic features of anterior mediastinal lymphosarcoma in the cat: A review of 30 cases. Vet. Rec., 104:304–307, 1979.
21. Gruffyd-Jones, T. J., Marston, M., White, E., et al.: Campylobacter infections in cats and dogs. Vet. Rec., 107:294, 1980.
22. Gruffyd-Jones, T. J., Orr, C. M., and Flecknell, P. A.: A new syndrome in cats (diarrhea and protrusion of the third eyelid). Vet. Rec., 101:413–414, 1977.

23. Hanichen, Von T., and Hasslinger, M.-A.: Chronic gastritis caused by *Ollulanus tricuspis* (Leuckart, 1865) in a cat. Berl. Munch. Tieraerztl. Wochenschr., 90:59–62, 1977.

24. Hardy, W. D., Jr.: Epidemiology of primary neoplasms of lymphoid tissues in animals. *In* Twomey, J. J., and Good, R. A. (eds.): The Immunopathology of Lymphoreticular Neoplasms. New York, Plenum Publishing Corp., 1978.

25. Hargis, A. M., Prieuer, D. J., Blanchard, J. L., et al.: Chronic fibrosing gastritis associated with *Ollulanus tricuspis* in a cat. Vet. Pathol., 19:320–323, 1982.

26. Hargis, A. M., Prieuer, D. J., and Westcott, R. B.: A gastric nematode *(Ollulanus tricuspis)* in cats in the Pacific Northwest. J. Am. Vet. Med. Assoc., 178:475–478, 1981.

27. Hasslinger, M.-A., and Trah, M.: Distribution and detection of *Ollulanus tricuspis*, the feline stomach worm. Berl. Munch. Tieraerztl. Wochenschr., 94:235–238, 1981.

28. Head, K. W., and Else, R. W.: Neoplasia and allied conditions of the canine and feline intestine. Vet. Annu., 21: 190–208, 1981.

29. Henderson, R. D., Mugashe, F., Jeejeebhoy, K. N., et al.: The role of bile and acid in the production of esophagitis and the motor defect of esophagitis. Ann. Thorac. Surg., 14:465–473, 1972.

30. Hendrick, M.: A spectrum of hypereosinophilic syndromes exemplified by 6 cats with eosinophilic enteritis. Vet. Pathol., 18:188–200, 1981.

31. Holmberg, C. A., Manning, J. S., and Osburn, B. I.: Feline malignant lymphomas: Comparison of morphologic and immunologic characteristics. Am. J. Vet. Res., 37:1455–1460, 1976.

32. Holzworth, J.: Hyperthyroidism in the cat: 10 cases. J. Am. Vet. Med. Assoc., 176:345–353, 1980.

33. Hoshino, Y., Baldwin, C. A., and Scott, F. W.: Isolation and characterization of feline rotavirus. J. Gen. Virol., 54:313–323, 1981.

34. Hoshino, Y., Zimmer, J. F., Moise, N. S., et al.: Detection of astroviruses in feces of a cat with diarrhea: Brief report. Arch. Virol., 70:373–376, 1981.

35. Howard, E. B., Sawa, T. R., Neilsen, S. W., et al.: Mastocytoma and gastro-duodenal ulceration-gastric and duodenal ulcers in dogs with mastocytoma. Pathol. Vet., 6:146–158, 1969.

36. Lewis, L. D., Boulay, J. P., and Chow, F. H. C.: Fat excretion and assimilation by the cat. Feline Pract., 9:46–49, 1979.

37. Liska, W. D., MacEwen, E. G., Zaki, F. A., et al.: Feline systemic mastocytosis: A review and results of splenectomy in 7 cases. J. Am. Anim. Hosp. Assoc., 15:589–597, 1979.

38. Lott-Stolz, G.: Maldigestion and malabsorption in the cat. Kleintierpraxis, 22:110–112, 1977.

39. Mackey, L. J., and Jarrett, W. F. H.: Pathogenesis of lymphoid neoplasia in cats and its relationship to immunologic cell pathways. I. Morphologic aspects. J. Natl. Cancer Inst., 49:853–865, 1972.

40. McNulty, M. S., Allan, G. M., Thompson, D. J., et al.: Antibody to rotavirus in dogs and cats. Vet. Rec., 102:532–534, 1978.

41. Meincke, J. E., Hobbie, W. V., and Hardy, W. D., Jr.: Lymphoreticular malignancies in the cat: Clinical findings. J. Am. Vet. Med. Assoc., 160:1093–1099, 1972.

42. Ockens, N.: Ileus in cats—an unknown cause.: Ligamentum pancreaticoduodenale. Proceedings of the Twentieth World Veterinary Congress, Thessalonica, Greece, 2:1822–1824, 1975.

43. Orr, C. M., Gruffyd-Jones, T. J., and Kelly, D. F.: Ileal polyps in Siamese cats. J. Small Anim. Pract., 21:669–674, 1980.

44. Orr, C. M., Kelly, D. F., and Lucke, V. M.: Tuberculosis in cats: A report of two cases. J. Small Anim. Pract., 21:247–253, 1980.

45. Patnaik, A. K., Johnson, G. F., Greene, R. W., et al.: Surgical resection of intestinal adenocarcinoma in a cat with survival of 28 months. J. Am. Vet. Med. Assoc., 178:479–481, 1981.

46. Patnaik, A. K., Liu, S.-K., and Johnson, G. F.: Feline intestinal adenocarcinoma: A clinico-pathologic study of 22 cases. Vet. Pathol., 13:1–10, 1976.

47. Pearson, H., Darke, P. G. G., Gibbs, C., et al.: Reflux oesophagitis and stricture formation after anesthesia: A review of seven cases in dogs and cats. J. Small Anim. Pract., 19:507–519, 1978.

48. Pearson, H., Gaskell, C. J., Gibbs, C., et al.: Pyloric and oesophageal dysfunction in the cat. J. Small Anim. Pract., 15:487–501, 1974.
49. Pedersen, N. C., Boyle, J. F., Floyd, K., et al.: An enteric coronavirus infection of cats and its relationship to feline infectious peritonitis. Am. J. Vet. Res., 42:368–377, 1981.
50. Prescott, J. F., and Karmali, M. A.: Attempts to transmit campylobacter enteritis to dogs and cats. Can. Med. Assoc. J., 119:1001–1002, 1978.
51. Skirrow, M. B.: Campylobacter enteritis in dogs and cats: A new zoonosis. Vet. Res. Commun., 5:13–19, 1981.
52. Skirrow, M. B., Turnbull, G. L., Walker, R. E., et al.: Campylobacter jejuni enteritis transmitted from cat to man. Lancet, 1:1188, 1980.
53. Snodgrass, D. R., Angus, K. W., and Gray, E. W.: A rotavirus from kittens. Vet. Rec., 104:222–223, 1979.
54. Stackhouse, L. L., and Clough, E.: Five cases of feline dirofilariasis. Vet. Med. Small Anim. Clin., 67:1309–1310, 1972.
55. Strombeck, D.: Pathophysiology of esophageal motility disorders in the dog and cat. VET. CLIN. NORTH AM., 8:229–244, 1978.
56. Theran, P., and Holzworth, J.: Feline hyperthyroidism. In Kirk, R. W. (ed.): Current Veterinary Therapy VII. Philadelphia, W. B. Saunders Co., 1980.
57. Tornes, W. A., and Sambol, R. M.: Heartworm infection in a cat. Allied Vet., 30:150–152, 1959.
58. Turk, M. A. M., Gallina, A. M., and Russell, T. S.: Non–hematopoietic gastrointestinal neoplasia in cats: A retrospective study of 44 cases. Vet. Pathol., 18:614–620, 1981.
59. Twaddle, A. A.: Pyloric stenosis in three cats and its correction by pyloroplasty. N. Z. Vet. J., 18:15–17, 1970.
60. Twaddle, A. A.: Congenital pyloric stenosis in two kittens corrected by pyloroplasty. N. Z. Vet. J., 19:26–27, 1971.
61. Weller, R. E.: Systemic mastocytosis and mastocytemia in a cat. Mod. Vet. Pract., 59:41–43, 1978.
62. Wilkinson, G. T.: Some preliminary clinical observations on the malabsorption syndrome in the cat. J. Small Anim. Pract., 10:87–94, 1969.

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