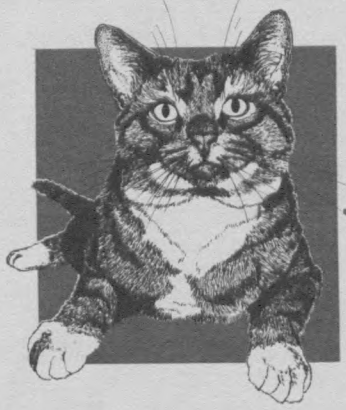


Cornell Feline Health Center
Veterinary News
Summer 1983

Catpox Virus Infection

Fredric W. Scott, D.V.M., Ph.D.

Recent reports in the veterinary literature clearly show that domestic and exotic cats are susceptible to a virus similar to "cowpox" virus. This brief review is to alert small animal clinicians to this disease of cats. Catpox virus infection generally presents as a dermatological disease with characteristic "pock-like" lesion of the skin, but some cats may have lung involvement.

The first reported cases of pox virus invections in felidae occurred in an outbreak in exotic cats in the Moscow zoo in 1972-73, but this report remained relatively unknown. In the late 1970s in the United Kingdom, cases of suspected "cowpox-like" virus infections in domestic cats were reported. In the February 19, 1983 issue of Veterinary Record, 2 reports of pox virus infections of domestic cats were reported. In one of these reports, Dr. Rosalind Gaskell and co-workers isolated and characterized a virus with properties identical to cowpox virus. They inoculated 4 cats with this catpox virus isolate and reproduced clinical disease in all inoculated cats.

Clinical signs

The incubation period is approximately 6 days. Localized crusty and slightly proliferative skin lesions 2 to 3 mm in diameter appear initially at the site of inoculation or scarification. Lesions may appear first on the paws, head, or lips, or around the edges of the conjunctiva. Lesions often spread within a few days to involve the skin over much of the body. Some cats have a mild conjunctivitis with slight purulent ocular discharge, and some cats may show signs of lung involvement. Temperature usually remains normal, and appetite may be normal to severely depressed. Cats may show severe edema of the distal limbs, head, and neck.

The course of the disease is usually several weeks, with most cats making a complete recovery. However, complications or severe pneumonia may alter the outcome. In the Moscow zoo outbreak, all cats that developed pneumonia died within 3 to 8 days, whereas only one of 7 cats with dermal infection without lung involvement died. Other signs or concurrent problems noted in the reported cases included the following: abscess, sloughing of metacarpal pad, stertorous breathing, ulcerated lick granuloma of lip, blepharitis, transient neurological signs. Pruritis was reported to be slight in some cases, but other cats exhibited severe pruritus with self-inflicted exacerbation of lesions.

The skin lesions were reported by Gaskell and co-workers to be circular, 3 to 6 mm in diameter, and to be one of 3 types. The first type appeared as "flat, glistening, red, hairless areas." Type 2 lesions were thick scabs which covered "shallow, crater-like ulcers filled with white pus," while type 3 lesions consisted of thick scabs over "smooth to granular red areas."

Diagnosis

Tentative diagnosis of pox virus infection can be made on the basis of clinical

signs. Confirmation of this diagnosis requires histopathogical examination of skin lesion biopsies or necropsy tissues, isolation of virus, or serological identification of specific pox virus antibodies. Catpox virus produces characteristic intracytoplasmic inclusion bodies, and these can be detected in skin or lung lesions. Virus has been isolated from scabs, skin lesions, lung, tonsil, lymph nodes, thoracic fluid, thymus, spleen, liver, and throat swabs. For serological studies, acute and convalescent serum samples are required. If pox virus infection is suspected, the clinician should contact the local diagnostic laboratory or the Cornell Feline Health Center before submitting samples.

Treatment

Treatment of catpox virus infection is symptomatic and supportive. Most cases have been treated with broad-spectrum antibiotics. One significant word of caution--it appears that treatment with corticosteroids or oral progestogen may transform a localized, relatively mild disease into a generalized, more severe disease which may even be fatal.

Source of infection

The source of infection in the reported cases has not been identified. The isolated virus has been identified as an orthopox virus similar to cowpox. There was no known clinical disease of cattle suggestive of cowpox in the areas from which the catpox virus infections occurred. One theory suggests that the source of infection is an as yet unidentified small wild animal.

While there has not been any indication of human infection with the catpox virus, one should use caution with these cases since cowpox and several other pox viruses of animals can result in human infection.

References

Baxby D et al.: Cow pox virus infection in unusual hosts. Vet Rec 104:175, 1979.

Gaskell RM et al.: Natural and experimental pox virus infection in the domestic cat. Vet Rec 112:164, 1983.

Marrenikova SS et al.: Outbreak of pox disease among Carnivora (Felidae) and Edentata. J Inf Dis 135:358, 1977.

Martland MF: Pox virus infection of a domestic cat. Vet Rec 112:171, 1983.

Fredric W. Scott (Cornell '62; Ph.D., Cornell '68), is a Professor of Veterinary Virology. He has been the Director of the Cornell Feline Health Center since its founding in 1974.

Correction

The word "ankylosis" was incorrectly printed as "amylosis" in the article on "Feline Chronic Progressive Polyarthritis" in our Spring 1983 issue. The correct sentence is: "This may progress, producing moderate periarticular bone erosion, pannus formation, collapse of the joint space and, finally, fibrous ankylosis of the joint."

Cornell Feline Health Center
Veterinary News

A publication for veterinary professionals

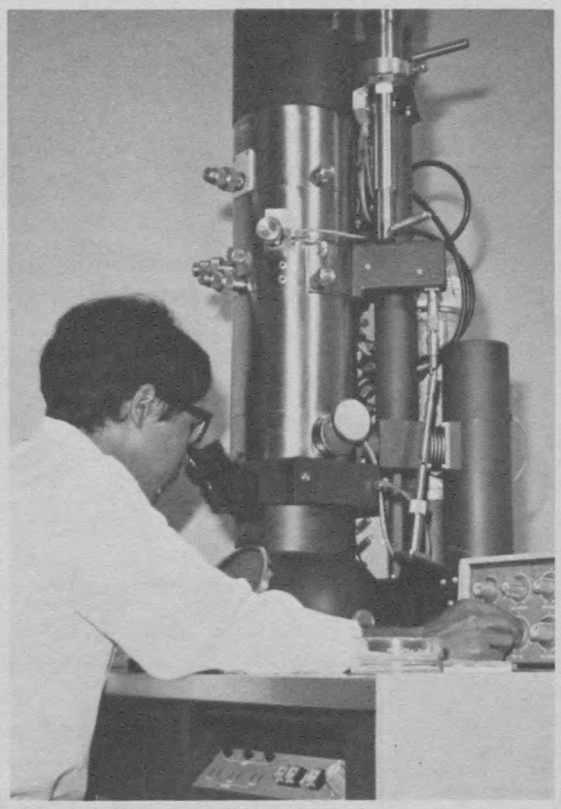
The ultimate purpose of the Cornell Feline Health Center is to improve the health of cats everywhere, by developing methods to prevent or cure feline diseases, and by providing continuing education to veterinarians and cat owners. All contributions are tax-deductible.

Director: **Fredric W. Scott, D.V.M., Ph.D.**
Asst. Director-Public Affairs: **Edna H. Federer**
Editor: **Joyce Tumbelston**
Secretary: **Sheryl A. Bronger**
Special Consultant: **Leo A. Wuori, D.V.M.**

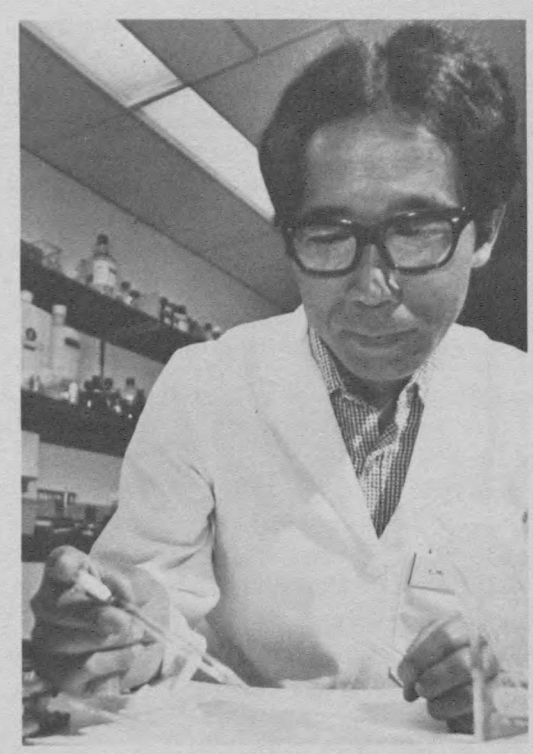
Dr. Taka Hoshino Wins Small Animal Research Award

Dr. Yasutaka (Taka) Hoshino, a former member of the Cornell Feline Health Center, has been named to receive one of the prestigious 1983 Ralston Purina Small Animal Research Awards for his outstanding work in virology conducted while at Cornell. He was chosen for this honor by the AVMA Council on Research, in recognition of the originality and significance of his research and the quality of his scientific publications.

Dr. Hoshino was the first to propagate feline infectious peritonitis virus in the laboratory, thus opening the door to other in vitro propagations which are slowly unlocking the secrets of this devastating disease. In addition, by electron microscopy of fecal samples, he isolated and described four new enteric viruses: feline rotavirus, canine rotavirus, feline astrovirus, and feline enteric coronavirus-like particles. His discoveries will pave the way for further understanding of the whole spectrum of enteritis in cats and dogs. In recognition of these landmark contributions to small animal medicine, he was presented this June with $1,000 and an engraved wall plaque by the Ralston Purina Company.



Dr. Hoshino focuses in on enteric virus particles highly magnified by the electron microscope.



Dr. Hoshino performs the immunofluorescence assay for the detection of specific antibodies against his newly discovered viruses.

Dr. Hoshino received his D.V.M. from Nihon University, Japan, in 1965, and completed his master's degree in Veterinary Virology at Cornell in 1980. Now a visiting scientist at the National Institutes of Health, he is continuing his work with animal and human rotaviruses in attempts to develop a vaccine for humans. We are honored to count Dr. Hoshino among the distinguished alumni of the Cornell Feline Health Center.

Feline Haemobartonellosis

Corisse N. Vaughan, D.V.M.

Feline haemobartonellosis is caused by the rickettsial organism Haemobartonella felis, an obligate parasite of erythrocytes of the cat. H. felis causes immune-mediated damage leading to increased red cell fragility and destruction. The disease is characterized by anemia, icterus, fever, anorexia, and weakness.

Prevalence and transmission

The exact percentage of the total feline population affected with H. felis cannot be accurately determined because most cases are latent and therefore go undiagnosed. But the prevalence is higher than expected, based on experimental work in which blood from "normal," randomly chosen cats, when pooled and injected into a susceptible cat, brought about clinical signs of the disease. Sometimes its presence is only discovered when a cat is used as a blood donor. Other times its presence is discovered coincidentally during blood testing for some other disease.

The disease is seen most often in young male cats 1 to 3 years of age, although it is seen in both males and females of all ages. Risk for males is 2 1/2 times that for females.[2] Seasonally, feline haemobartonellosis occurs slightly more frequently in the late spring.[2]

The precise mode of its transmission has not been proven, but it is known that blood from an already infected cat must somehow be transferred into the bloodstream of a healthy cat to produce the disease.[3] Biting insects, particularly fleas, are likely carriers of the parasite but definitive experimental evidence to that effect is lacking.[4] Bite wounds may be a possible mode of transmission. Fight wounds leading to abscesses may be one factor that lowers the animal's resistance, allowing the parasite to multiply and produce clinical signs of the disease.

Intrauterine transmission is well established. The organism has been found in still-born kittens and in kittens within 3 hours of birth. The feline placenta is of the chorio-endothelial type, in which the maternal blood vessels are in contact with the fetal chorion, the placental barrier being only about 4 cells thick. As endometrial cellular debris is absorbed by the outer fetal layer, transmission of infection by this route would seem to be feasible.[5]

In addition, infections can be transmitted iatrogenically during blood transfusions. Blood from adult cats which appeared normal clinically and hematologically produced the disease when injected into susceptible cats, indicating the donors were carriers. To ensure that a prospective blood donor cat is not chronically infected, it is recommended that the cat be splenectomized and blood films be examined for organisms on alternate days for 2 weeks after splenectomy.[6]

Experimentally, the infection can be transmitted by injecting blood via the intraperitoneal, intravenous, or oral routes.[4] Direct contact does not spread the disease, nor does contact with body excretions of infected cats. H. felis does not affect species other than the cat.

Clinical signs

The most common clinical signs are depression, weakness, anorexia, and pallor. Many times splenomegaly, icterus, weight loss, vomition, dehydration, and pyrexia are noted.

Clinical signs are somewhat dependent on the rapidity with which the anemia develops. If anemia develops gradually, the cat may lose a considerable amount of weight but be bright and alert.[6] If anemia develops quickly, the cat will have lost little weight, but be markedly

depressed and icteric. If the blood destruction is severe enough, an anemic bruit will be heard. Also, with loss of erythrocytes, there is a loss of oxygen-carrying capacity and thus dyspnea may be present. Early during the disease process, the rectal temperature is high (104°-105° F) but it will drop to sub-normal in the moribund cat.

Diagnostic tests

A blood smear stained with Wright's, Giemsa, Diff-Quik®, or acridine orange, revealing the organism attached to the erythrocyte, is the only positive diagnostic test for feline haemobartonellosis. Unfortunately, the organism is not always demonstrable in the peripheral blood, as parasitemia only lasts 1 to 2 days,[7] and parasitemic peaks occur every 3 to 11 days. Consequently, cats with haemobartonellosis may need to be examined daily for a week or more before parasites are observed. Where the organism goes when not in the peripheral circulation has been studied very extensively. It has been shown that the organism is sequestered by the reticuloendothelial system, particularly the spleen.[8]

Preferred stains for light microscopy are those of the Romanowsky type. Various investigators reported that Giemsa stain produced an intense red coloration, a bluish tinge with distinct pink shades, or blue shades with purple granulation. Wright's stain imparts a bluish color with reddish granules at the periphery of the organism. With Schilling's methylene blue-eosin stain, the organisms stain a bright red, and the erythrocytes stain blue.[4] New methylene blue is not recommended for detection of H. felis because it is difficult to distinguish this small organism from specks of reticulum of the more mature reticulocytes.[9]

The parasite appears mainly in 3 forms on the outside of the red blood cell. Most common are the small coccoid and the short rod forms adhering to the erythrocyte. During active multiplication, the parasites frequently appear as delicate rings, occurring singly or in clusters on the surface of the erythrocytes.

Characteristically, a stained blood smear will show evidence of a hyperactive bone marrow as demonstrated by the presence of immature erythrocytes. This is evidenced by polychromasia, anisocytosis (macrocytosis) and nucleated red blood cells. Occurrence of nucleated erythrocytes in the peripheral blood alone is not a reliable measure of effective erythrogenesis. More reliable indicators of anemia in remission are a marked anisocytosis and polychromasia[9] and a rising hematocrit. A round, deeply stained nuclear remnant near one edge of the erythrocyte is called a Howell-Jolly body. In anemia the number may increase.

On a routine hemogram the hematocrit is low (less than 24%), hemoglobin is low (less than 8 g/dl), and total red blood cell count is low (less than 5 million/ul). These values are associated with a macrocytic (MCV greater than 55 fl), normochromic (MCHC equal to 30-36%) anemia. The bone marrow is responding intensely to the hemolytic crisis, pouring many red blood cells into the blood stream.

The cat may have a leukopenia, a normal total white blood cell count, or leukocytosis. Because nucleated red blood cells may be present, a corrected total white blood cell count must be calculated using the following formula:

$$\text{Corrected total WBC count} = \frac{100 \text{ WBC}}{100 \text{ WBC} + \# \text{ nRBC}} \times \text{uncorrected total WBC count.}$$[9]

Absolute monocyte counts are, however, frequently increased and monocytes are often bizarre during the acute phase of the disease. Erythrophagocytosis by monocytes or macrophages may be seen if blood films are scanned at low magnification.

Treatment

Attention should be first directed to life-supportive treatment. If the cat is severely dyspneic, it should be given oxygen by mask or endotracheal tube or placed in an oxygen cage. If the cat is

severely anemic (hematocrit less than 15%), fresh, whole cat blood should be given intravenously. Forty to 50 cc of blood is sufficient for an average size cat. If the cat is too young and the veins too small to give an intravenous injection, intraperitoneal or intramedullary injection of blood is satisfactory. The number of transfusions depends on the animal's response and condition.

Antibiotics or arsenical drugs and steroids are used to treat H. felis. Broad spectrum antibiotics such as tetracycline, oxytetracycline, and chloramphenicol have been used with variable results. Oxytetracycline has been the most widely used and seems to be the most effective. Cats should be treated orally for 3 weeks at a dosage of 20 mg/kg 3 times a day.[6] Chloramphenicol is effective in approximately 50 percent of the cases. The dosage is 5-7 mg/lb of body weight 2 times a day for 10 to 14 days.[4] Because chloramphenicol produces significant (though reversible) bone marrow injury at therapeutic dosages recommended for cats, it may not be rational to use when a regenerative erythroid response is desired.[6] Thiacetarsamide sodium (Caparsolate: Abbott Laboratories) given at 1 mg/kg intravenously, twice 48 hours apart, may also be used. Fishler[13] has treated 40 cats with this drug and reports good results. Previously, Fishler had used tetracycline and chloramphenicol with poor results. Fishler reports that the only adverse reaction observed was excessive salivation in a few cats. If accidentally overdosed, the cat can be treated with dimercaptol (BAL: Hynson, Westcott and Dunning, Inc.) at a dosage rate of 2 mg/lb 3 times a day until recovery.[13] Others report the drug to be less effective. Thiacetarsamide sodium is not approved for cats.

In addition, treatment with a glucocorticoid such as prednisolone (1-2 mg/kg body weight twice a day) is indicated based on evidence of immune-mediated injury to erythrocytes. The glucocorticoid dosage should be decreased gradually as desired increases in hematrocrit are measured.[6]

No drug appears to totally eliminate organisms from infected cats, and consequently "recovered" animals remain chronically infected. With proper therapy, most cats recover from the acute phase but remain inapparent carriers. No immunity is conferred.

Post-mortem findings

On gross examination, the animal is noted to be anemic with varying degrees of icterus.[10] The animal is usually emaciated. The spleen is large with rounded edges and darker than normal. The liver is large and may have small, 2- to 3 mm, raised, gray-white nodules on its surface. Cardiomegaly may be present. The lymph nodes may also be larger than normal and hemorrhagic or edematous.

The cause of death is attributed to the severity of the anemia. Parasitized erythrocytes are sequestered by macrophages, particularly of the spleen. Some of these erythrocytes do return to the circulating blood, but they are damaged so that osmotic fragility increases and erythrocytes suffer a premature death.[11]

Geographic incidence

The genus Haemobartonella is widespread in animals throughout the world and has been reported in many species of rodents, cattle, dogs, and cats. H. felis may also have a worldwide distribution[5] but thus far has been reported in Australia, New Zealand, Finland, Great Britain, France, Hungary, Chad, Japan, Bulawayo, Africa, Mexico, the United States, and Canada.

References

[1]Hayes HM, Priester WA: Feline infectious anemia. Risk by age, sex, and breed: prior disease: seasonal occurrence: mortality. J of Sm An Pract 14 (12):797-804, 1973.

[2]Gilbride AP: Feline Infectious Anemia. Friskies Research Digest 6 (3):6-12, 1970.

[3]Small E, Ristic M: Haemobartonellosis. Vet Clin of N Am 1:225-230, May 1971.

[4]Wilkinson GT: Feline Infectious Anemia. Vet Rec 331-333, Mar 29, 1969.

[5]Harvey JW: Feline Haemobartonellosis. Current Veterinary Therapy VII, Small Animal Practice. Philadelphia, WB Saunders Co., 1980.

[6]Harvey JW, Gaskin JM: Experimental Feline Haemobartonellosis. J Am An Hosp Assoc 13:28-38, Jan-Feb 1977.

[7]Maede Y, Murata H: Ultrastructural Observation on the Removal of Haemobartonella felis from Erythrocytes in the Spleen of a Cat. Jap J Vet Sci 40:203-205, 1978.

Cats and Tuberculosis

Jeffrey E. Barlough, D.V.M.

Tuberculosis is an important worldwide disease of man and animals that has been recognized since ancient times. The term "tuberculosis" derives from the multiple small nodules of inflammatory tissue, or tubercles, that are characteristic of the disease in some species. Tuberculosis is caused by several members of the acid-fast genus Mycobacterium: M. tuberculosis, M. bovis, and M. avium ("tubercle bacilli").

Human beings are susceptible to infection with all three types of tubercle bacilli. Dogs are equally susceptible to both M. tuberculosis and M. bovis, but seem to acquire the infection more often through contact with tuberculous humans actively shedding organisms in respiratory secretions (so-called "open" cases of tuberculosis). These dogs most often show clinical signs referable to the lower respiratory tract, such as coughing, retching, and dyspnea.

Cats, on the other hand, are highly resistant to M. tuberculosis but are highly susceptible to M. bovis and most commonly acquire the infection by ingestion of contaminated cows' milk. Thus they usually show clinical signs of abdominal disease, such as diarrhea, mesenteric lymphadenopathy, and ascites. However, pulmonary, renal, and genital tuberculosis have also been reported in cats, as have ocular and skeletal manifestations of the disease. Rapidly progressive, disseminated infections may also occur, with widespread invasion of the thorax and abdomen. M. avium, deposited in soil by infected birds, may also on rare occasions infect cats and produce fatal disease. Tuberculosis is infrequently diagnosed in cats in the United States but is more common in other areas of the world where bovine tuberculosis is prevalent.

Antemortem diagnosis of tuberculosis in the cat can be difficult. History and clinical signs may be suggestive of tuberculosis, especially if a recognized source of tubercle bacilli can be identified in the animal's environment, and thoracic radiography may be helpful in cases with pulmonary involvement. A more definitive diagnosis, however, can be elusive. Although organisms are frequently shed in body secretions and excretions, their demonstration by Ziehl-Neelsen acid-fast staining may be difficult because of their low numbers. Tuberculin reactions in cats are inconsistent and therefore unreliable, and serology is of little value. Culture and identification of the causative bacterium are definitive, but require weeks to months of incubation in the laboratory.

Biopsy of enlarged lymph nodes is probably the most rapid method of obtaining a reliable diagnosis in the living animal. Histopathologic examination of biopsy tissue combined with Ziehl-Neelsen staining of lesions will provide a definitive answer in most cases. Biopsy of the cecal lymph node at laparotomy is especially helpful when mesenteric lymphadenopathy is present because the disease may localize in lymph nodes without gross involvement of other organs. In addition, cecal lymph node biopsy will differentiate between

Haemobartonellosis

[8]Schalm OW, Switzer JA: Feline Infectious Anemia. Fel Pract 2:26-30, Sept-Oct 1972.

[9]Hatakka M: Haemobartonellosis in the Domestic Cat. Acta Vet Scand 13 (3): 323-331, 1972.

[10]Maede Y: Studies on Feline Haemobartonellosis II. The Mechanism of Anemia Produced by Infection with Haemobartonella felis. Jap J Vet Sci 37:49-51, 1975.

[11]Fishler JJ, Birzele FD: A Practical Approach to Diagnosis and Control of Feline Infectious Anemia. Fel Pract 2:36-37, Sept-Oct 1972.

Corisse N. Vaughan is a 1982 Cornell graduate currently in small animal practice in Springfield, Va. This article is derived from her Senior Seminar presentation.

tuberculosis and alimentary lymphosarcoma, the most common disease of cats with which tuberculosis can be confused. When ascites is present, tuberculosis can mimic feline infectious peritonitis. In all cases biopsy should be accompanied by bacteriological culture so that the infecting strain can be identified. This will facilitate identification of the source of the infection.

Treatment of feline tuberculosis with several drugs, including streptomycin and isoniazid, has been reported. However, the required therapy is prolonged and cats undergoing treatment are a potential threat to humans (and to other animals) with which they come into contact. For this reason treatment of tuberculous cats is strongly discouraged, and the owner should be informed of the zoonotic potential of the infection. A search for the source of the infection, if unidentified, should be instituted as soon as a definitive diagnosis has been made.

Jeffrey E. Barlough (Davis '79) has been with the Cornell Feline Health Center since 1980, studying the feline immune response to feline infectious peritonitis (FIP).

Newly Revised Book Available

A vastly expanded, updated version of our feline health manual is now available. Entitled "Felis domesticus: A Manual of Feline Health 1982-1983," the book combines the proceedings of the Second and Third Annual Feline Health Seminars into a valuable compendium of 201 pages. New topics have been added to last year's manual, meshing two books into one. Topics include:

Anatomy; Reproduction; Genetics; Fetal Development and Congenital Birth Defects; Raising Kittens; First Aid; Operating a Successful Animal Shelter; Design and Management of Shelters and Catteries for Prevention of Infectious Diseases; Zoonoses; Computerized Diagnostics; Infectious Diarrheas; Feline Behavior; Nutrition; Common Dermatological Disorders; Internal Medicine; Respiratory Diseases; Feline Panleukopenia; Immunization; Coronavirus Infections; Kitten Mortality Complex; Feline Leukemia Virus; Surgery; Suggested Readings; Glossary.

At a cost of only $15.00 (plus $1.50 for postage and handling), this book offers an excellent survey course in modern feline medicine that is of benefit to veterinarians and lay people alike. Order your copy today by sending your check for $16.50 (U.S. funds) made out to: Cornell Feline Health Center.

Cornell Feline Health Center
Cornell University
College of Veterinary Medicine
Ithaca, New York 14853