



CORNELL FELINE HEALTH CENTER NEWS

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New cardiovascular studies; Gastroenteritis outbreaks

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

During the past year several exciting developments have occurred at the Feline Center. One of these was the establishment of a new program to investigate cardiovascular diseases in the cat. Although the diagnosis of heart disease has increased dramatically in the last 5 years, very little is known about the etiology, prevention or therapy.

Dr. N. Syndey Moise is currently studying acute congestive cardiomyopathy as the first project in these cardiovascular studies. In addition to improving the diagnosis and therapy of clinical cases, she is investigating the possibility of a virus being the underlying etiology of cardiomyopathy. As announced on page 7, she would like you to refer any suspected cases of cardiomyopathy for evaluation.

We are all saddened by the untimely passing in early February of Dr. Gary Bolton. An Associate Professor of Small Animal Medicine specializing in cardiology, Dr. Bolton was a great inspiration to his colleagues, students, clients and friends. His empathy for and unswerving dedication to his patients were unmatched. The feline cardiovascular research program had long been a dream of his, and the continued contribution of this program to feline medicine will be a living memorial to him.

The Cornell Feline Health Center receives several consultation telephone calls daily from throughout the U.S. Aside from the usual calls on FIP and feline leukemia, an occasional call on kitten mortality complex, and various and sundry other problems, several outbreaks of severe gastroenteritis have been reported in catteries and veterinary clinics from several states. I think this is something clinicians should be aware of.

These outbreaks usually occur in adult cats with good vaccination histories, and the morbidity is high. There appear to be 2 general categories of these outbreaks. In the first, cats show anorexia, vomiting, diarrhea with mucus and blood, leukocytosis of 20,000 to 50,000 (but occasional severe leukopenia), and fever of 103°-105° (occasional fever spike to 106°). In hospital outbreaks, only stressed cats (i.e., cystitis, surgery) develop clinical signs. Viral studies (culture, fecal EM) have been negative, but Salmonella typhimurium has been isolated from all fecal cultures.

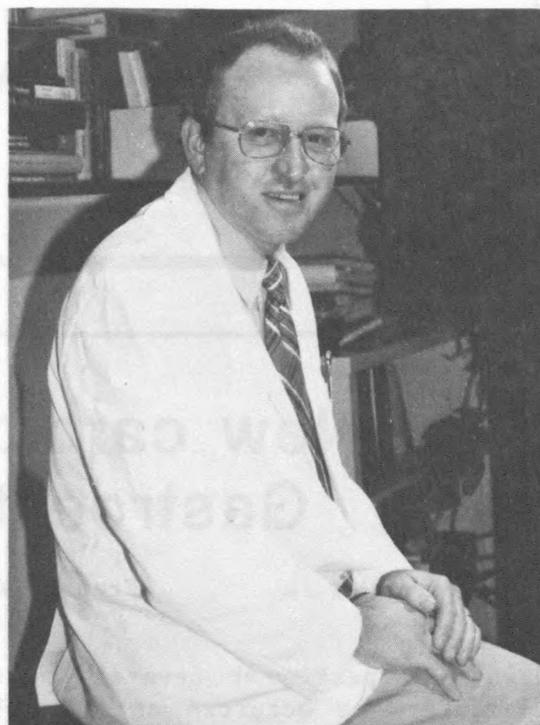
The second category of gastroenteritis is a panleukopenia-like syndrome occurring in adult cats. Cats have a fever and a mucoid enteritis, sometimes with blood,

(Cont. on page 2)

and they may vomit. Many affected cats have a relatively mild disease, but some have marked signs and severe leukopenia, and a few have died. Duration of illness has been 2 days to 2 weeks. Some outbreaks have followed a mild upper respiratory infection outbreak. To date, viral studies have been negative by culture and EM. Specialized bacterial studies are still in progress.

These descriptions are sketchy, but my main intent is to alert clinicians of the gastroenteritis outbreaks that have been reported. We urge you to contact the Feline Center with information about similar outbreaks.

Beginning with this issue of the Cornell Feline Health Center News, we will include condensed versions of interesting Senior Seminar reports. Our senior students expend considerable time and effort to provide an up-to-date review of specific clinical problems. Some are eventually published, but many are not. We believe inclusion of reports on feline topics will be helpful to clinicians in feline practice.



Dr. Fredric W. Scott, Director of the Cornell Feline Health Center since its inception in 1974, is also a Professor of Virology at the N.Y.S. College of Veterinary Medicine. The Cornell Feline Health Center serves the veterinary profession and the public as a reference laboratory for feline diseases.

CORNELL FELINE HEALTH CENTER NEWS

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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.

Feline Health Seminar for cat fanciers and breeders

Your clients may want to attend the second annual Feline Health Seminar taught by Cornell Feline Health Center personnel in Ithaca, N.Y., July 11-17, 1982. Topics of the week-long seminar will include Genetics of Coat Color and Disease Resistance, Feline Behavior, Nutrition, Medical and Surgical Problems, Intestinal and Viral Diseases, and General Health Care. Diagnostic tests will be demonstrated, and students will gain hands-on experience in the laboratory. Housing and meals will be provided for registrants. Please advise any clients who may be interested in this educational vacation to contact Mary K. Gloster, Cornell's Adult University, 626B Thurston Ave., Ithaca, NY 14853. Phone (607) 256-6260.

Feline hyperthyroidism

Christopher Olsen, D.V.M.

Feline hyperthyroidism is a disease, generally of older cats, which has only recently emerged as a syndrome of clinical importance to the veterinary clinician. Whether this disease simply did not occur with any regularity in the past or has been misdiagnosed is not known. It is a disease, however, which often presents with such striking clinical signs as to be readily recognized.

The disease is diagnosed most frequently in middle-aged to geriatric patients. These cats present with an insidious onset and progressive course of hyperactivity, weight loss despite polyphagia, polyuria and polydipsia, a matted, unkempt appearance to the haircoat, and occasionally soft stools or vomiting. The cats may be moderately pyretic, the mucous membranes and ear pinnae may appear pinker than normal, there may be a tachycardia, an abnormally strong precordial or femoral pulse, and a variety of cardiac arrhythmias and/or murmurs. The most informative physical exam finding is the presence of a uni- or bilateral cervical swelling anywhere from just caudal to the larynx to the thoracic inlet.

Based upon such a clinical presentation in an aged cat, diabetes mellitus should certainly be the foremost differential diagnosis to consider in light of the polyuria, polydipsia, polyphagia and weight loss. However, as clinicians become more cognizant of feline hyperthyroidism, these cats may be recognized before demonstrating the entire array of classical signs. In these cases, it will be important to expand the differential diagnosis list to encompass such diseases as chronic renal disease, cardiomyopathy, a primary dermatosis, primary gastrointestinal diseases, and other diseases as they may relate to the specific set of signs demonstrated by individual patients.

The hyperthyroidism syndrome in cats is

a clinical disease manifestation of a functional thyroid tumor which elaborates excessively high levels of the thyroid hormones, T_4 , and to a lesser degree in most cases, T_3 . (It is currently accepted by most authorities that T_3 is the physiologically active compound, formed in most part by peripheral tissue conversion from T_4 . However, direct glandular secretion of T_3 , in man at least, can be increased during hyperthyroidism.) This functional or "toxic" thyroid tumor is most often benign, categorized as multinodular adenomatous hyperplasia or as discrete adenomas. Adenocarcinomas can occur, but are less frequent and, even when present, rarely metastasize, as opposed to the situation in dogs in which thyroid tumors are often aggressively malignant.

In addition to the thyroid glandular tissue in the neck, there may be accessory thyroid tissue present in the anterior mediastinum. This is due to a developmental descent from the thyroid's origin from endodermal epithelial cells on the foregut floor between the first 2 pharyngeal pouches. This accessory thyroid tissue may be involved in the neoplastic process, an important diagnostic and therapeutic concern.

Over the years, a variety of techniques have been designed and utilized to evaluate T_3/T_4 levels, mostly in man and dogs. A great deal of controversy has surrounded the relative value of each technique. Today, most labs utilize a competitive protein binding or radioimmunoassay technique. In general, several factors are important to consider: values from one lab to another cannot be compared without reference to each lab's normal values; normal feline values are still being defined; lab results must be correlated to clinical signs; and thyroid hormone levels can vary with time of day, age, breed, and pregnancy status, as well as the effects of many drugs including estrogens, andro-

Hyperthyroidism (Cont.)

gens, glucocorticoids, salicylates, phenobarbital, phenylbutazone and diphenylhydantoin. However, in most cases of feline hyperthyroidism, hormone levels will be markedly elevated, often 2 to 4 times normal.

In addition to evaluation of hormone levels, another diagnostic tool available at many large teaching institutions is nuclear medical imaging. Because radioactive isotopes are handled by body systems in the identical manner to endogenous stable forms of the same element, radioimaging allows one to trace a body system's anatomical as well as functional structure through the use of a scintillation or Anger camera from outside the body. In the case of the thyroid gland, ^{131}I , ^{125}I , or $^{99\text{m}}\text{TcO}_4$ can be used. ($^{99\text{m}}\text{TcO}_4$ is technetium, a radioisotope derived from molybdenum, which can be used because the thyroid traps it as it would I. It is in fact preferred over ^{131}I or ^{125}I because of its lower energy level, lack of particulate radiation, short $T_{1/2}$ and decreased employee and patient exposure.) Imaging can also be used to evaluate involvement of accessory thyroid tissue and functional metastatic masses.

In addition to thyroid testing, several other diagnostic tests may lend support to the diagnosis of feline hyperthyroidism. Many of these cats will have elevated levels of serum AST (SGPT), ALT (SGOT), and alkaline phosphatase (AP). The elevation in AP is attributable to the bone isozyme and increased bone turnover secondary to the hypermetabolic state established in hyperthyroidism. The elevations of AST and ALT relate to the presence of a poorly defined hepatopathy with minimal alteration of hepatic structure. One suggestion has been increased hepatocyte permeability secondary to glycogen depletion. Similar findings occur in human thyrotoxic patients, and as in man, these values generally return to normal subsequent to reestablishment of euthyroidism in these cats.

The cardiac manifestations of hyperthyroidism as described above can be striking

and clinically very significant. In man, these abnormalities may largely determine the course, progress and prognosis of thyrotoxicosis.

Electrocardiography may reveal any of a number of disorders in feline hyperthyroids, including: sinus tachycardia (rate > 240 beats/minute), tall R waves suggesting left ventricular enlargement, premature atrial contractions, a shortened Q-T interval (probably related to the rapid rate), prolongation of the QRS complex, and paroxysmal arrhythmias consistent with a re-entry mechanism (including atrial bigeminy, ventricular trigeminy and runs of ventricular premature beats and gallop rhythms). Additionally, a pre-excitation syndrome has also been documented. The value of ECG in diagnosis and management of these cats should be obvious. As with the serum enzyme abnormalities, most of the cardiac manifestations will usually also resolve after correction of the thyrotoxic state.

Understanding the pathology behind the feline hyperthyroidism syndrome, it should not be surprising that surgery is the treatment of choice. However, prior to surgery, two concerns must be addressed. First of all, the presence of metastatic disease should be ruled out prior to surgery. This may be investigated via thoracic radiographs and/or nuclear imaging, looking for abnormal areas of increased uptake.

The second concern should be the general preoperative condition of the patient. If a cat exhibits the cardiac abnormalities that can occur with this disease, it is recommended that the cat be stabilized medically prior to surgery. Two drugs have been used in this regard. The first is propranolol, a beta adrenergic blocking agent, useful because of the increased adrenergic stimulus in hyperthyroidism. Propranolol has been beneficial at a dose of 2.5 mg BID to TID for as little as 2 days prior to surgery.

Because it is now clear that the cardiac side effects in hyperthyroidism are

also due to direct effects of the increased levels of thyroid hormone, the antithyroid drug propylthiouracil (PTU) is useful at a dose of 50 mg BID-TID. PTU has been found to exert its antithyroid function by blocking the binding of iodine to the tyrosyl residues of thyroglobulin and the coupling of MIT and DIT to produce T_3 and T_4 , via peroxidase inhibition. The duration of treatment needed to return thyroid hormone levels to normal has varied from 3 days to 3 weeks.

The surgical approach for removal of both thyroid lobes begins with a mid-ventral cervical incision from the hyoid region almost to the thoracic inlet. The sternocephalicus, and then sternohyoideus muscles should be defined, separated left from right, and laterally retracted to expose the thyroid lobes adjacent to the trachea. Excision of the lobe(s) involves ligation of only 2 main vessels, the cranial and caudal thyroid, with excision considered relatively simple.

There is some debate as to whether both thyroid lobes should be routinely excised in all cases. Usually only one lobe is significantly enlarged. According to one theory, the second thyroid lobe should be excised if it is found at surgery to be abnormal in appearance, color, or consistency, as is the usual case, despite a lack of enlargement. In a minority of cases, only one thyroid lobe will appear abnormal at surgery, with the opposite lobe atrophied or even absent because of TSH suppression induced by the elevated hormone levels from the abnormal lobe. In these cases, it is recommended that only the abnormal lobe be excised because postoperatively the atrophied lobe resumes function. The obvious counter argument in these cases is that in the event of a thyroid adenocarcinoma, one would want to remove all thyroid tissue at the time of surgery to prevent recurrence of the carcinoma; thus some would recommend total thyroidectomy in all cases.

There are 4 complications to consider following thyroidectomy, namely, hypothy-

roidism, hypoparathyroidism, interruption of recurrent laryngeal nerve function, and recurrence of the hyperthyroid syndrome, especially if a unilateral thyroidectomy is performed.

To abate hypothyroidism after total thyroidectomy, replacement therapy L-thyroxine should be instituted within 24 hours of surgery, usually at a dose of 0.05 to 0.1 mg SID. This dose should be individually adjusted, with the addition of sodium liothyronine as necessary based on sequential postoperative evaluation of T_3 and T_4 levels. Lifetime replacement therapy will usually be necessary at some level in cases of bilateral thyroidectomy. In the case of unilateral thyroidectomy, however, the supplemental thyroxine required will be minimal, if any.

Postoperative hypoparathyroidism, with consequent hypocalcemia, can be a problem because of the intimate association of the parathyroid glands to the thyroid gland. Unfortunately, the parathyroid glands can be difficult to identify at surgery. One parathyroid gland should be spared, along with its blood supply, if possible, but ectopic parathyroid tissue is usually present in sufficient amounts to maintain function or to restore it within a few days. To avoid problems, signs of hypocalcemia (weakness, tetany, convulsions) should be monitored and therapy initiated as necessary. One protocol for treatment is as follows: initially 1-2 ml 10% calcium borogluconate slowly IV while auscultating the heart (or preferably monitoring an ECG) followed by 20 ml/24 hours calcium borogluconate in an IV drip, 2.5 grains calcium lactate PO BID and PO Vitamin D_2 BID.

Damage to the recurrent laryngeal nerves is also a problem because of their close anatomic relationship to the thyroid lobes (lying dorsomedial to the lobes, loosely adherent to the trachea), and their small size (less than 1 mm in diameter). Damage to the nerves has produced signs such as altered voice, cough, and harsh respiratory sounds.

(Cont. on page 8)

In Memoriam



Gary R. Bolton, D.V.M.

September 21, 1942 - February 10, 1982

During his short life of 39 years, Gary Robert Bolton established an outstanding reputation as a veterinary cardiologist, a dedicated teacher, a clinician par excellence, and a devoted family man.

A native of Racine, Wisconsin, Dr. Bolton attended the University of Wisconsin and Iowa State University, where he received his D.V.M. degree in 1967. He then took an internship, a medical residency, and a residency in cardiology at The Animal Medical Center in New York City.

In 1970, Dr. Bolton joined the faculty of the N.Y.S. College of Veterinary Medicine as an Assistant Professor of Small Animal Medicine - Cardiology. He was appointed Associate Professor in 1974. He was a gifted teacher with the unique ability to pinpoint essential clinical aspects while radiating interest and enthusiasm for his subject. He was the recipient of the Norden Distinguished Teacher Award in 1977, and the Class of 1982 dedicated their yearbook to him.

A board certified veterinary cardiologist, Dr. Bolton authored the Handbook of Canine Electrocardiography, one of the leading reference texts on veterinary

cardiology, which was also published in Japanese. He was working on the second edition of this text at the time of his fatal illness. He also was one of the first veterinary cardiologists to develop the technique of ECHO cardiography, and was instrumental in developing the feline cardiovascular research section of the Cornell Feline Health Center. His contribution to the veterinary literature included over 30 publications in texts or periodicals.

Dr. Bolton was a very popular lecturer at local, state, and national meetings throughout the country. His dry sense of humor and practical approach to clinical problems were constantly in demand. Only he could have enlisted the entire audience of several hundred Small Animal clinicians at an AAHA meeting in Boston to participate in 2-minute exercise drills during the seminar breaks. He was invited to Japan to give an intensive series of lectures during a 3-week tour of that country.

Without question, his empathy for his patients and his compassion and understanding for their owners went far beyond the call of duty. He would spend hours at night by the cage of a critically ill dog encouraging and nursing this dog back to health. Hundreds of letters have been sent by clients attesting to his unusual compassion and empathy.

Dr. Bolton generated such a lasting impression that an endowed award, "The Gary Bolton Memorial Cardiology Award," arose from donations in memory of his outstanding contributions to the field of small animal cardiology. The first award was presented to a senior veterinary student this year.

There is no question that Dr. Bolton is missed by his wife Jean, son Kerry (10), daughter Mickey (8), the rest of his family, and by all of the numerous colleagues, students and friends whose lives were touched by him. The veterinary profession has been privileged to have Gary Bolton as a dedicated member.

Referrals requested

Feline Cardiomyopathy - At the Cornell Feline Health Center, we are investigating the possible cause or causes of feline cardiomyopathy. Electrocardiography, phonocardiography, echocardiography, and angiography are some of the clinical studies which are being used to establish the diagnosis of congestive and hypertrophic cardiomyopathy. Additionally, viral isolation attempts and transmission studies are planned. Specifically, we are interested in evaluating the possibility of a viral etiology in feline congestive cardiomyopathy. In addition to the diagnostics and investigation of etiology, therapeutic regimens will be individualized in attempts to manage these cats.

We are calling on you for assistance. If you encounter cases of cardiomyopathy, we would appreciate hearing from you, as well as having these cases referred for evaluation. If you are interested in our study or have a case to refer, please contact Dr. N. Sydney Moise through the Cornell Feline Health Center, (607) 256-7663.

Craniofacial Malformation in Burmese Kittens - Over the past few years we have accumulated information and specimens on a malformation in Burmese kittens that may be an inherited defect. This malformation appears to be limited to the head, affect-

ing the brain, cranial cavity and face. Most have an enlarged cranial cavity with a bulging of the area occupied by the brain. The top of the brain is only covered by skin. This consists of a duplication of the cerebral hemispheres which sometimes are distended with cerebrospinal fluid. The eyes may be missing or very small. There may be no nostrils or nasal cavities but the upper jaw and palate may be partially duplicated. The "whisker pads" may also be double.

This has been recognized by breeders of Burmese cats in states on both the East and West coasts of the United States. It usually affects only one or a few in any one litter and it is hypothesized that this is inherited as an autosomal recessive gene. A group of breeders has organized in California to prove this hypothesis by test breedings and to utilize these breedings to identify carrier and non-carrier cats for future breeding of cats free from this disease.

If you have observed one of these malformations, please contact Dr. Alexander de Lahunta, Department of Anatomy, New York State College of Veterinary Medicine, Ithaca, NY 14853, or Ms. Margherita G. Harris, who represents the Burmese Cooperative Research Group that is investigating the genetics of this disease, 27470 Loma Del Rey, Carmel, CA 93923.

- Please send donation cards for the euthanasia memorial program.
- I would like information on bequests and deferred giving opportunities to benefit the Cornell Feline Health Center.
- I would like to read an article on _____.

Name _____

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Hyperthyroidism (Cont. from page 5)

Recurrence of the hyperthyroid syndrome can be due to local regrowth of a tumor, pathologic involvement of accessory thyroid tissue not diagnosed preoperatively, or development of functional metastases.

Despite these possible complications, it is important to realize that in the majority of cases, surgery and the postoperative course are uneventful, with resolution of almost all clinical signs and decrease of thyroid hormone levels to the normal range by 3-4 weeks postoperatively.

Propylthiouracil can be used to medically control chronic hyperthyroidism. One protocol recommends a loading dose of 50 mg TID until thyroid hormone levels have normalized, followed by a maintenance dosage of 50-150 mg per day individualized to each patient. Physical examinations and hormone level assays should then be repeated at 2-3 month intervals. Several complications can be encountered using PTU chronically. Hormone levels may actually fall below normal requiring reduction in PTU dosage or concomitant administration of supplemental L-thyroxine. Other side effects of prolonged PTU therapy may include vomiting and anorexia, allergic reactions/ facial swelling and pruritis,

hepatopathy, Coomb's positive/ANA negative anemia, and thrombocytopenia, although these side effects are not common.

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Christopher Olsen is a May 1982 graduate of the N.Y.S. College of Veterinary Medicine and a member of the Phi Zeta Veterinary Medicine Honor Society. He is now associated with the East Hampton Veterinary Group, East Hampton, N.Y.

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