2. Brodey, R. S., and Prier, J. E.: Clinico-Pathologic Conference. JAVMA, 140, (Jan 15, 1962): 173–176.
3. Dingwall, J. S., Eger, C. E., and Owen, R. R.: Clinical Experiences with the Combined Technique of Ureterovesicular Anastomosis for Treatment of Ectopic Ureters. J Am Anim Hosp Assoc, 12, (1976): 406–410.
4. Finco, D. R., Kneller, S. K., and Crowell, W. A.: Diseases of the Urinary System. In *Feline Medicine and Surgery*. 2nd ed. American Veterinary Publications, Inc, Santa Barbara, Ca (1975): 285.
5. Johnston, D. E., and Archibald, J.: Male Genital Systems. In *Canine Surgery*. 2nd Archibald ed. American Veterinary Publications, Inc, Santa Barbara, Ca (1974): 713.
6. Johnston, G. R., Osborne, C. A., Wilson, J. W., and Yano, B. L.: Familial Ureteral Ectopia in the Dog. J Am Anim Hosp Assoc, 13, (1977): 168–170.
7. Lane, J. G.: Canine Ectopic Ureter—Two Further Case Reports. J Small Anim Pract, 14, (1973); 555–560.
8. Osborne, C. A., and Hanlon, G. F.: Canine Congenital Ureteral Ectopia: Case Report and Review of Literature. Anim Hosp, 3, (1967): 111–122.
9. Osborne, C. A., and Perman, V.: Ectopic Ureter in a Male Dog. JAVMA, 154, (Feb 1, 1969): 273–278.
10. Osborne, C. A., Dieterich, H. F., Hanlon, G. F., and Anderson, L. D.: Urinary Incontinence Due to Ectopic Ureter in a Male Dog. JAVMA, 166, (May 1, 1975): 911–914.
11. Owen, R. R.: Canine Ureteral Ectopia—A Review. 1. Embryology and Aetiology. J Small Anim Pract, 14, (1973): 407–417.
12. Owen, R. R.: Canine Ureteral Ectopia—A Review. 2. Incidence, Diagnosis, and Treatment. J Small Anim Pract, 14, (1973): 419–427.
13. Patten, B. M.: Foundations of Embryology. 2nd ed. McGraw-Hill Book Company, New York, NY (1964): 480–512.
14. Reis, R. H.: Renal Aplasia, Ectopic Ureter, and Vascular Anomalies in a Domestic Cat *(Felis domesticus)*. Anat Rec, 135, (1959): 105–107.
15. Seidenberg, L., and Knecht, C. D.: Ectopic Ureter in the Dog. JAVMA, 159, (Oct 1, 1971): 876–877.

CLINICAL REPORT

Feline Infectious Peritonitis

THE OWNER of a small cattery breeding Persian cats sent 3 dead kittens (one 4 months old, and two 4½ months old) to the necropsy service of the Faculty of Veterinary Medicine, University of Liège. These kittens constituted the 3rd litter of a 3-year-old female. Feline infectious peritonitis (FIP) was diagnosed in all 3 kittens on the basis of characteristic macroscopic and microscopic lesions.[3]

Five months later, the same female gave birth to 3 kittens. One of them died of FIP at the age of 2½ months; the diagnosis was based on macroscopic findings. We then proceeded with clinical examinations of the mother and the 2 other kittens. The mother had lost weight and was weak. The 2 kittens were clinically normal.

Two months later, the kittens had the same pattern of weight loss and weakness, accompanied by retarded growth. Five months later, the mother and the 2 kittens appeared clinically normal. During the whole observation period, no other cat in the cattery had clinical signs of FIP. Specific precautions were not taken to avoid contagion in the cattery.

The etiology of FIP is slowly becoming clear, based on the experimental transmission of the disease agent in filtrates of organ extracts and the electronmicroscopic observation of viral particles in diseased tissues. Some specific characteristics of the etiologic agent have been reported.[2,6]

Up to now, all theories concerning spread of FIP have been based on clinical observations, serologic tests being only recently available.[5] Neonatal FIP has occurred among kittens produced by certain queens.[4] In a recent review, the different forms of the disease as well as the possible modes of transmission were described.[4] Transplacental transmission of the disease agent seems applicable to the kittens of this report.

Unfortunately, the feline leukemia status of the affected kittens was unknown. Concurrent diseases, especially feline leukemia, apparently can

suppress a cat's resistance enough to allow the FIP virus to cause disease when it might otherwise pass unnoticed.[1]

One of the affected kittens died during a period in which it should have been protected by maternal immunity. Regardless of this fact, it cannot be excluded that the kittens obtained the infection by direct contact at around 6 to 9 weeks of age, either from the mother or from clinically normal carriers in the immediate environment.—*P.-P. Pastoret, M. Gouffaux, M. Henroteaux, F. Schoenaers, Faculté de Médecine vétérinaire ULg, 45, rue des Vétérinaires, 1070 Brussels, Belgium, and J. Tepper, 2 Penn Ct, Dix Hills, NY 11746.*

1. Cotter, Susan M., Hardy, William D., Jr., and Essex, Myron: Association of Feline Leukemia Virus with Lymphosarcoma and Other Disorders in the Cat. JAVMA, 166, (March 1, 1975): 449–454.
2. Osterhaus, A. D. M. E., Horzinek, M. C., and Ellens, D. J.: Untersuchungen zur ätiologie der felinen infektiösen peritonitis. Berl Munch Tieraerztl Wschenschr, 89, (1976): 135–137.
3. Pastoret, P.-P., Gouffaux, M., and Henroteaux, M.: Description et étude expérimentale de la péritonite infectieuse féline. Ann Med Vet, 118, (1974): 479–492.
4. Pedersen, N. C.: Feline Infectious Peritonitis: Something Old, Something New. Feline Pract, 6, (1976): 42–51.
5. Pedersen, Niels C.: Serologic Studies of Naturally Occurring Feline Infectious Peritonitis. Am J Vet Res, 37, (Sept, 1976): 1449–1453.
6. Starks, Barbara W., Corstvet, Richard E., and Buckner, Ralph G.: Certain characteristics of the Infective Agent of Feline Infectious Peritonitis. Am J Vet Res, 37, (March, 1976): 335–338.

Equine Intestinal Clostridiosis

Clostridium perfringens (type A) infection in the horse was studied because this organism had been isolated in large numbers from the intestinal content and feces of horses with acute and often fatal disease.

Affected horses typically had an acute onset, with apathy, foul-smelling diarrhea, discolored mucous membranes, and elevated pulse rate and temperature. Not infrequently, affected horses died shortly after onset of clinical signs. Case histories revealed that affected horses had been subjected to stress prior to onset of the disease. There were widespread angiopathies with hyperemia, edema, hemorrhages, and poorly coagulated blood. The changes were most marked in the cecum and large colon and were diagnosed as acute typhlitis and colitis. Some horses had lesions in the myocardium, liver, and kidneys.

In horses that survived, the disease regressed, and parallel to this the *C perfringens* counts decreased to normal levels. A positive correlation between the numbers of *C perfringens* in feces and the severity of illness was established; *C perfringens* was detected only in exceptional cases in the intestinal contents and feces of healthy horses and then only in very low numbers.

Immunologic investigations indicated that sick horses and horses exposed via the intestinal tract to *C perfringens* were immunologically stimulated by its extracellular antigens.

The cause of the rise in the *C perfringens* counts in the sick horses could not be established with certainty. Treatment with antibacterial drugs could not be excluded as a contributing factor in a few of the cases. It was proposed that fodder rich in protein with a relatively low content of cellulose and in combination with stress might be of etiologic importance.

The disease resembles colitis X and similar disorders. However, the prevalence of *C perfringens* above a certain level combined with the described clinical and pathoanatomic findings was regarded as the best diagnostic criterion for the disease. Because *C perfringens* seemed to be of etiologic importance, the disease was named equine intestinal clostridiosis. —*Martin Wierup in Acta Vet Scand, Suppl 62, Uppsala, Sweden, 1977.*