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volving approximately 65–75% of the cerebral hemispheres of the brain and approximately 30 bots in the retropharyngeal pouches (Fig. 1). No other gross lesions were present.

Bacteriologic examination, utilizing 5% sheep blood agar, revealed *Klebsiella pneumoniae* from the kidney, liver, spleen, and cerebral spinal fluids. Cultures of the brain, utilizing 5% sheep blood agar and chocolate agar, revealed alpha-*Streptococcus* and *Corynebacterium pyogenes*. The bots were identified as *Cephenemyia phobifer*. Representative specimens have been deposited in the U.S. National Parasite Collection in Beltsville, Maryland (Accession No. 77316). No virus was isolated via inoculation of tissue cultures or embryonated chicken eggs.

Utilizing routine histopathologic methods (Luna, 1968, *In* Manual of Histologic Staining Methods of the Armed Forces Institute of Pathology, McGraw-Hill, Inc., New York, pp. 32–39), examination of the brain revealed areas of necrotic debris surrounded by degenerative neutrophils encapsulated by inflammatory cells and connective tissue.

Five species of *Cephenemyia* have been reported in North America: *C. apicata*, *C. jellisoni*, *C. phobifer*, *C. pratti*, and *C. trompe*. *Cephenemyia pratti* is most commonly found in mule deer (Harwood and James, 1979, *In* Entomology in Human and Animal Health, Macmillan Publishing Co., New York, pp. 311–312). *Cephenemyia phobifer* is usually present in eastern United States and generally is found in white-tailed deer (*Odocoileus virginianus virginianus*) (Davis and Anderson, 1971, *In* Parasitic Diseases of Wild Mammals, Iowa State University Press, Ames, Iowa, p. 283).

Cerebral abscesses may arise from septic thromboemboli or bacterial emboli, or by direct invasion of the brain from an adjacent structure. It may be possible that migration of the larvae of *C. phobifer* may play a role in the development of the cerebral abscess. Myiasis producing frontal abscesses with *Corynebacterium pyogenes* has been reported in sheep and cattle (Jubb and Kennedy, 1970, *In* Pathology of Domestic Animals, Academic Press, New York and London, p. 402).

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Lymphoproliferative Disease in the American Goldfinch, *Carduelis tristis*

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During a long-term study of the American Goldfinch near Guelph, Ontario (Middleton, 1978, Condor 80: 401–406; Middleton, 1979, Ecology 60: 418–432), various attempts have been made to establish a research population from wild-trapped stock. These efforts have not met their objectives as the majority of the captives have either died before the experiments were started or during their course. The type of experiments have varied from placing individually caged birds in an environmental chamber to test the effect of photoperiod in gonadal cycles and molt, to placing free-flying birds in outdoor flight pens with natural vegetation in efforts to induce reproduction. With few exceptions birds have not survived in captivity for longer than a year, and death has consistently occurred at times of apparent stress, such as changing holding conditions during experimentation, or during molt. Routinely, carcasses in suitable condition were sent for necropsy to the Department of Pathology, Ontario Veterinary College, Guelph. When cause of death could be established the reports most frequently suggested that it was due to enteric

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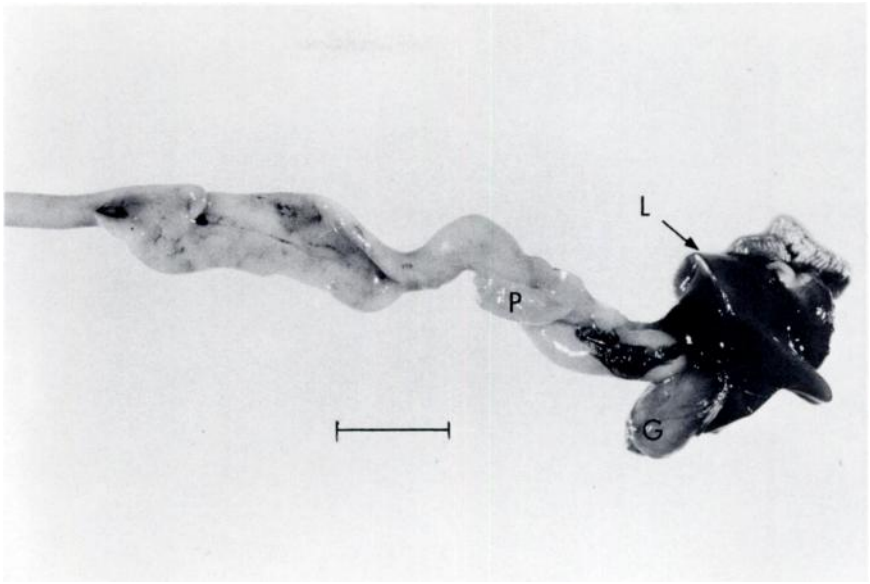


FIGURE 1. Greatly thickened duodenum of goldfinch with lymphoproliferative disease. P, pancreas. G, gizzard. L, liver. Bar = 1 cm.

Isospora coccidia infection (species not identified).

In September 1980, three male and three female goldfinches were trapped from the wild and placed in an outdoor aviary (3 × 6 × 8.5 m) covered with 1.25 cm mesh chicken wire on wooden framing, and placed on a concrete floor, at the Zoology aviary, University of Guelph. Two large planters (1.2 × 1.2 × 1 m) containing small deciduous shrubs (*Acer* sp.) were located at each end of the flight enclosure. The west end had a roofed enclosure but with access to the flight. Food (sunflower chips, niger, millet and canary seed) was provided in hanging feeders suspended from the roof of the enclosure and water was provided in a creep-flow container placed on its floor. Water was replaced twice every week and food as necessary. The entire complex was cleaned once a week, except during winter when snow accumulation prevented clearing of the uncovered flight. During the breeding season various natural foods, including grasses (Fam. Gramineae) and composites (Fam. Compositae), were provided for the birds.

The birds survived the winter and summer without any apparent signs of illness, although no attempts were made to nest. In late August 1981, with the onset of the post-breeding molt (Middleton, 1978, Condor 80: 401–406), the first signs of disease appeared. The birds showed gradual loss of weight, accompanied by poor flight capacity and ultimately extreme lethargy with feathers fluffed. Three birds died in late August but their carcasses were not recovered until autolysis was too advanced to make necropsy worthwhile. However on 2 and 4 September single carcasses were submitted for necropsy. The last surviving bird was removed from the pen on 25 September and was close to death at that time. The subsequent analyses are based on these three specimens.

The first bird submitted had a hyperemic swollen protruding cloaca and wet feces on its tail feathers. The abdomen of all three birds was distended and the birds were dehydrated and lacking in body fat. There was moderate muscle atrophy. Except for autolytic changes in the two dead birds, all three showed similar internal lesions. The duodenum and proximal jejunum were thickened (Fig. 1). The wall of the duodenum was 2 mm thick in some areas to give a total diameter of over 4 mm. The wall of the small intestine became less affected posteriorly and the ileum and large bowel were more normal. The upper digestive tract was

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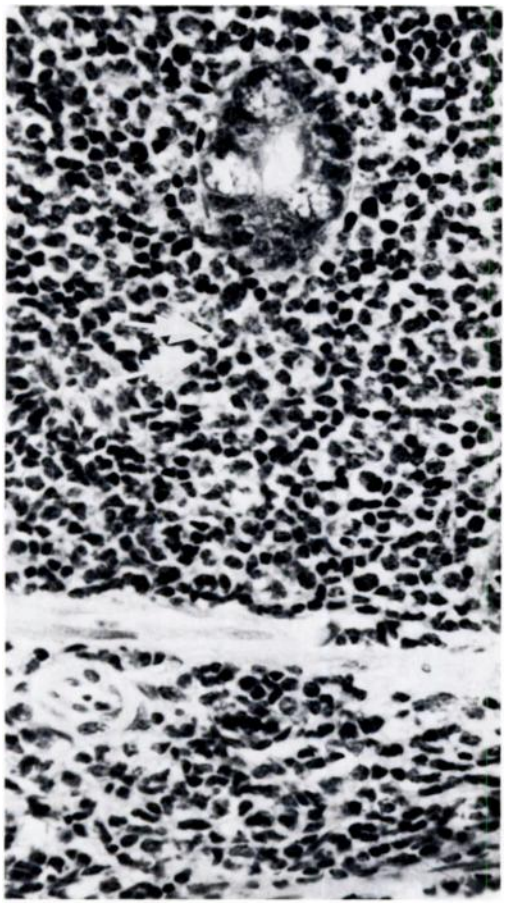


FIGURE 2. Neoplastic lymphocytes infiltrating through lamina propria, submucosa and muscularis of the duodenum. The serosa is at the bottom and part of two crypts is present. There are merozoites in the cytoplasm of many neoplastic lymphocytes (two are identified by white arrows) frequently causing indentation of the nucleus. ×500.

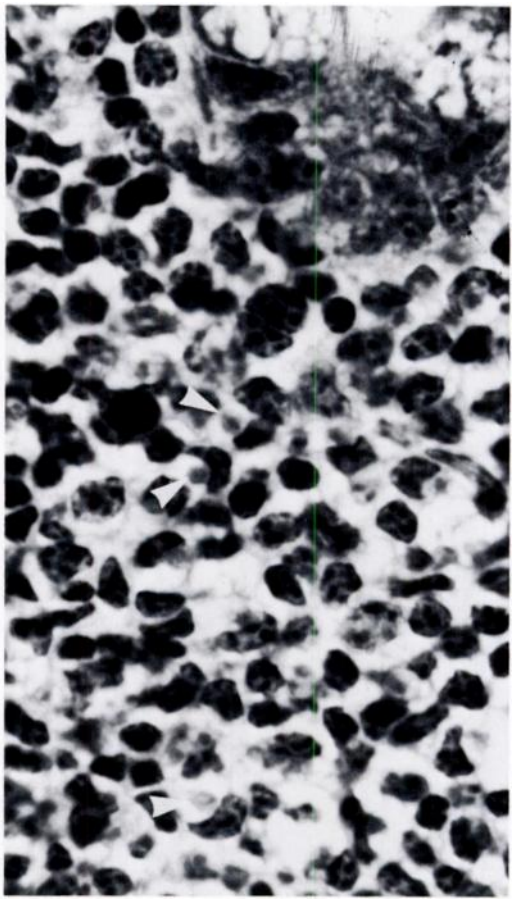


FIGURE 3. Higher power of the central part of Figure 2. ×1,250.

empty. The intestinal content varied from slightly fluid in the affected portion to fluid, gas and more normal content in the ileum and large bowel. Other organs were of normal color and of normal to small size.

A blood smear from the bird submitted alive did not reveal abnormalities. There was no evidence of anemia, blood parasites or neoplastic lymphocytes.

Cultures from intestines and organs from all three birds were negative for pathogenic bacteria. Large numbers of coccidia, mainly sexual stages, were present in smears from the affected portions of the intestines.

Histologic examination of intestine revealed that the lamina propria, submucosa and muscularis were infiltrated with sheets of monomorphic, medium sized, neoplastic lymphocytes (Figs. 2, 3). Nuclei were a moderate size, with a diameter about equal to the length of a red blood cell nucleus. They were non-cleaved, quite vesicular, contained prominent chromatin clumps and one or more hypertrophic nucleoli. Cytoplasm was abundant but indistinct. Few mitotic figures were present. The neoplastic cells were most prominent in the lamina propria and villi were distended with sheets of uniform cells. Crypts were absent or widely separated. Many sexual stages of coccidia were present in epithelial cells but there was no reaction or hemorrhage and the epithelium was intact in the bird submitted alive. Merozoites

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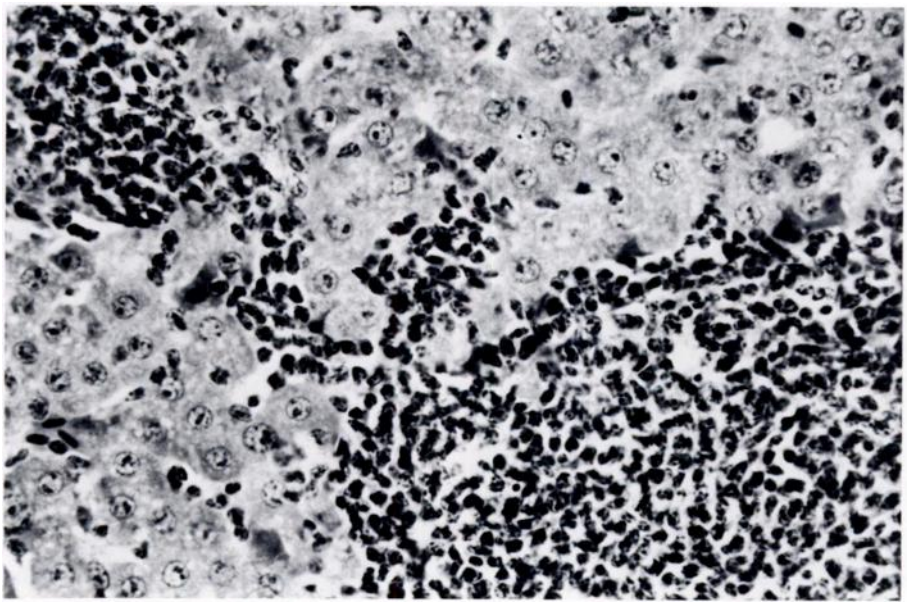


FIGURE 4. Neoplastic lymphocytes in the liver and infiltrating between cords of liver cells. ×500.

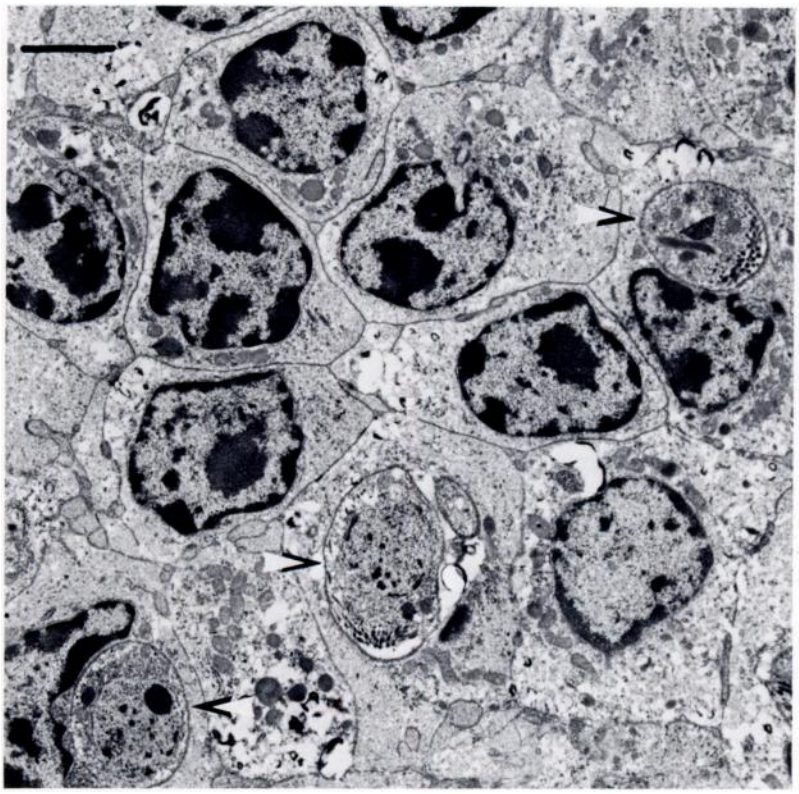


FIGURE 5. Neoplastic lymphocytes in lamina propria of duodenum of goldfinch. Zoites (arrows) are present in the cytoplasm of some of these lymphocytes. Bar = 200 nm. ×7,500.

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were present in the cytoplasm of some of the neoplastic lymphocytes in all three birds (Figs. 2, 3). Large numbers of neoplastic lymphocytes were present in the submucosa and smaller accumulations were infiltrating through the muscularis, and were scattered on the surface of the serosa and in the subserosal tissue (Fig. 2). Similar neoplastic cells were present in foci around portal triads and scattered diffusely between cords of liver cells of all three birds (Fig. 4). They were also present in the kidney, spleen and lung of one or two of the birds. No neoplastic lymphocytes were found in bursa, muscle, gonad or central or peripheral nervous system.

Electron microscopy on tissue from the bird submitted alive revealed occasional plasma cells scattered among the neoplastic lymphocytes and under light microscopy cells with the nuclear morphology of plasma cells were seen in the lamina propria close to crypt and villous epithelium. Parasitic forms (zoites) were present in the cytoplasm of approximately 10% of the lymphocytes in the intestine (Fig. 5).

A review of seven previous cases from the same source necropsied over the past 10 yr revealed that all had been diagnosed as dying from coccidiosis. Histologic material was available from four of these birds and examination of slides from these cases suggested that two cases, one submitted in June 1979 and one in August 1971, had lesions in the intestine similar to those reported here. Two cases submitted in April and July 1971 had a predominantly inflammatory infiltrate in the lamina propria of the intestine, although in the case submitted in July 1971 accumulations of neoplastic-like lymphocytes were present on the serosal surface of the duodenum. In all four cases zoites were present in cells in the lamina propria. These cells were identified as lymphocytes by light microscopy.

The results of previous necropsies had led to the suggestion that most mortality in captive goldfinches resulted from coccidiosis. In turn it was felt that this disease might be a significant mortality factor in wild populations, particularly during the two periods of extensive molt (Middleton, 1977, Condor 79: 440–444; Middleton, 1978, Condor 80: 401–406). This suggestion was supported by the isolation of coccidial oocysts in the droppings of recently captured goldfinches (Middleton, unpubl. data). However, the results reported here indicate that although coccidia may have been implicated in the death of some of the birds, lymphoproliferative disease affecting the intestine and other organs was the primary problem.

The presence of zoites in intraepithelial lymphocytes is an interesting feature of this condition but may only indicate the usual transport mechanism of coccidia merozoites from the tips of villi where they penetrate the epithelium to the crypt where they re-enter epithelial cells to continue their cycle as suggested for *Eimeria* sp. by Fernando (pers. comm.). If the merozoites entered neoplastic intraepithelial lymphocytes they would not be carried to the crypt and would remain in the neoplastic cells unable to continue their cycle. Schizonts were found only in intestinal epithelium and there was no other evidence of systemic isosporan infection.

Unfortunately little is known about disease in the American Goldfinch or other wild passerine birds. Lymphoproliferative disease in the form of malignant lymphoma has been seen as an outbreak of disease in caged canaries in Ontario (Julian, unpubl. data) and has been reported elsewhere (Purchase and Burmester, 1978, *In* Diseases of Poultry (7th Ed.), Hofstad (ed.), Iowa State Univ. Press, Ames, Iowa, pp. 418–468; Jackson and Cooper, 1981, Refresher Course for Veterinarians on Aviary and Cage Birds, Univ. of Sydney, Post-Graduate Comm. in Vet. Sci. Proc. #55, p. 624; Wadsworth et al., 1981, Avian Pathol. 10: 499–504; Cavil, 1982, *In* Diseases of Cage and Aviary Birds (2nd Ed.), Petrak (ed.), Lea and Febiger, Philadelphia, Pennsylvania, pp. 523–524).

Coccidia infection of passerines is apparently usually due to *Isospora* sp. and has been reported as causing disease in canaries and finches including goldfinches (Harrigan, 1981, Refresher Course for Veterinarians on Aviary and Cage Birds, Univ. of Sydney, Post-Graduate Comm. in Vet. Sci. Proc. #55, pp. 347–349; Keymer, 1982, *In* Diseases of Cage and Aviary Birds (2nd Ed.), Petrak (ed.), Lea and Febiger, Philadelphia, Pennsylvania, pp. 541–543). Lesions were not described in these outbreaks. However, Box (1975, J. Protozool. 22: 165–169) described two different species of *Isospora* in the canary, *I. serini*, causing a disseminated infection of the mononuclear phagocytes and *I. canaria* producing a typical intestinal infection.

It seems unlikely that coccidiosis played any

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role in the etiology of lymphoproliferative disease in the goldfinches studied here. It is more likely that immunosuppression resulting from the neoplasia permitted the development of coccidiosis.

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Intracytoplasmic Neuronal Inclusions in the Hippocampus of Non-rabid Moose, *Alces alces* (L.)

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Intracytoplasmic acidophilic neuronal inclusions in the brain are characteristic of rabies virus infection, but they are also described in non-rabid animals of several species including domestic cat (Szlachta and Habel, 1953, Cornell Vet. 43: 207–212), dog (Cameron and Conroy, 1974, Vet. Pathol. 11: 29–37), cattle and sheep (Stovall and Pessin, 1942, Am. J. Public Health 32: 171–175), skunk (Jubb and Kennedy, 1970, Pathology of Domestic Animals, Vol. 2, Academic Press, New York, pp. 414–416), fox, and laboratory mouse (Smith et al., 1972, Veterinary Pathology (4th Ed.), Lea and Febiger, Philadelphia, pp. 351–356). Such descriptions have provided useful baseline data for histological interpretation of diseased brains. This paper provides the first description of intracytoplasmic neuronal inclusions in the hippocampus of non-rabid adult moose.

In August 1979, a cow moose (Case 1) showing abnormal behavior was reported by tourists to officials of Prince Albert National Park, Saskatchewan, Canada. The animal was killed and the head was removed, frozen, and submitted for post mortem examination 1 mo later. In October 1980 a 2½ yr old cow moose (Case 2) raised at the Wyoming Game and Fish Department's Sybille Wildlife Research Unit became acutely ill. Clinical signs included weakness, anorexia, and bilateral limbal corneal opacity. The moose would drink water and eat willow branches when these were held for her. After 2 days the moose was killed due to her deteriorating condition and a post mortem examination was conducted. Intracytoplasmic neuronal inclusions were observed in the brains of these two moose, and additional moose brains were obtained for comparison. These included a cow moose (Case 3) which died of chronic enteritis at Sybille, and two free-ranging cow moose from Wyoming, one with no history of clinical disease (Case 4) and one with severe keratoconjunctivitis (Case 5).

At necropsy, brain tissue was fixed in 10% neutral buffered formalin. Half of each brain was frozen unfixed and submitted either to the Western Animal Disease Research Institute (Agriculture, Canada) or to the Wyoming State Veterinary Diagnostic Laboratory for possible detection of rabies virus antigen by fluorescent antibody technique (FAT). Mouse inoculation studies were conducted in Cases 2–5. Samples of fixed brain, which included frontal and occipital cortex, basal ganglia, hippocampus, thalamus, mesencephalon, cerebellum, and medulla oblongata in most cases, were embedded in

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