An ulcerative enteritis of racing pigeons

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Ulcerative enteritis was first reported in quail in 1907, and, as a result, earned the name “quail disease” (1). During the next several decades, it became apparent that the condition could also affect a variety of other young gallinaceous birds, including various species of quail, grouse, pheasants, wild and domestic turkeys, and chickens (1). However, there are few reports of ulcerative enteritis in pigeons. Levi (2) reported the occurrence of a disease resembling ulcerative enteritis in pigeons and doves in the state of New York in 1940. In 1951, Glover (3) recorded ulcerative enteritis in young pigeons in Ontario. We report herein the existence of a similar disease of young racing pigeons in southern Alberta.

The disease was characterized by sudden onset of profuse, odorous diarrhea that was accompanied by relatively rapid emaciation

Reports of illness and death in nestlings and recently weaned racing pigeons in local lofts resulted in the submission of representative birds from each loft to the Regional Veterinary Laboratory, Lethbridge. Affected birds ranged in age from 12–35 days, but most were in the narrower range of 12–21 days. Clinical signs of illness were not evident in older juvenile birds or adults. Occurrences of the disease in any loft were sporadic, and were noted in nestlings reared in some nest boxes, but not those in adjoining boxes. Often, several successive nests of youngsters reared in certain identified boxes became ill. The disease was characterized by sudden onset of profuse, odorous diarrhea that was accompanied by relatively rapid emaciation. A characteristic feature of the disease was that, in spite of diarrhea and emaciation, the general demeanor and appetite of birds observed early in the course of the disease were not markedly different from those of healthy birds of similar age. However, as the disease progressed, these nestlings were often depressed and assumed a huddled posture with ruffled feathers. Physical examination of such birds usually confirmed the fact that they were very thin and underweight. On average, 25% of nestlings in each loft became ill.

Birds submitted to the laboratory were killed by cervical dislocation and a routine postmortem examination was conducted. Tissues, including intestine and liver, were inoculated on the following media: bovine blood, MacConkey’s, chocolate, brilliant green, Hektoen’s, and bismuth sulfide agar. Standard methods for the isolation of *Salmonella* spp. were used. Inoculated plates of blood agar were incubated aerobically, anaerobically, and in an atmosphere of 10% CO₂ at 37°C for 48 hours. As well, prior to inoculation of media for anaerobic incubation, minced tissues were heated in a water bath at 70°C, 80°C and 100°C for three hours, one hour, and three minutes, respectively (1). Selected tissues were fixed in 10% neutral buffered formalin, trimmed, processed routinely, cut at 4 μm, and stained with hematoxylin and eosin (H & E) and by the Gram method.

At necropsy, it was evident that affected birds were emaciated, with decreased volume of pectoral muscles. The crop of most birds was distended with grain. Significant lesions were confined mainly to the intestines where they were most prominent in the mid to lower jejunum; in all birds examined, the duodenum, ceca, and colon were devoid of ulcerative lesions. Tan, multifocal ulcers with hyperemic borders were prominent and could be seen readily through the serosa (Figure 1). As seen from the mucosal surface, intestinal lesions were discrete or confluent, round, oval,

Figure 1. Loop of intestine from a young pigeon affected with ulcerative enteritis. Note the prominent focal ulcers visible through the serosal surface. Bar = 1 cm.

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or irregularly shaped ulcers filled with necrotic debris, and measured 2–5 mm in diameter. In some birds, there were serosal perforations of ulcerated foci and resulting air sacculitis/peritonitis and serosal adhesions. In others, segments of intestine were distended and often obstructed by inspissated cores or large flakes of yellow, necrotic debris associated with areas of ulceration. Occasionally, there were tiny, pale foci in the liver. Examination of gram-stained smears prepared from areas of intestinal ulceration revealed a mixed population of bacteria, of which a predominant number were slender, gram-positive rods, some bearing oval sub-terminal spores and resembling *Clostridium* spp. Aerobic cultures of liver and intestine from affected birds yielded only coliform organisms; anaerobic cultures of heated and unheated tissues did not yield bacterial organisms.

Histologically, there was severe ulceration, often associated with extension of the inflammatory reaction through the layers of the longitudinal and circular smooth muscle to the serosa. Heterophils, which were most numerous in the more superficially ulcerated areas, also infiltrated from these sites to the serosa. In addition, many mononuclear cells, mainly lymphocytes, infiltrated all layers and formed nodules in the tunica muscularis and periserosal fat. Cellular detritus, many colonies of gram-positive rods, and rapidly proliferating intestinal epithelial cells were associated with the ulcerative process. Examination of grossly visible hepatic lesions seen in one affected bird revealed a massive infiltrate of heterophils, particularly in the area of portal triads. Thick collars of such inflammatory cells were evident throughout and were prominent on low-power view. Occasional foci of hepatic necrosis were evident.

Treatment with oxytetracycline (Terramycin Soluble Concentrate, Pfizer Canada Inc., Montreal, Quebec) at the rate of 0.5 g/L in drinking water for five days resulted in improvement in the health of affected birds in all flocks, often within 24 hours of the onset of treatment. In general, birds in affected flocks responded well, in that the diarrhea ceased and clinically affected youngsters began to gain weight. Despite the early success with the use of oxytetracycline, however, it became apparent that, in several instances, treated birds relapsed within several days after withdrawal of medication, or after a period of additional stress such as that of transportation. The use of chloramphenicol (Quanto-chlor, Quantum Pharmaceuticals, Toronto, Ontario) in drinking water at a rate of 0.22 g/L for three to five days in initial or recurring infections provided more satisfactory treatment and resulted in fewer recurrences of the disease. Untreated birds usually died. The recurrence of the condition supported the published view that the causative organism is shed in the feces and remains viable indefinitely in litter or in remaining fecal material on perches and floors (1). As well, it is possible that the infection was not eliminated completely by the initial treatment. As a result, the disease may have recurred in some birds that then became another source of organisms for young birds in the environment. The disease occurred in nestlings in one loft each year for several years. The use of chloramphenicol in drinking water for five days when nestlings were approximately 12 days of age usually prevented, or reduced the severity of, the disease in this loft.

## The use of chloramphenicol in drinking water for three to five days in initial or recurring infections provided satisfactory treatment

Ulcerative enteritis in gallinaceous birds is caused by *Clostridium colinum*, a slender, motile gram-positive rod with oval, sub-terminal spores (1,5). *In vitro* growth requirements of this organism are difficult to achieve because of its fastidious nature and requirement for enriched media and anaerobic conditions. The organism is extremely resistant to common disinfecting agents as well as to physical changes in the environment. For example, it survives heating at 70°C for three hours, 80°C for one hour, and 100°C for three minutes (1). It is sensitive to a number of antibiotics including streptomycin, tetracyclines, bacitracin, chloramphenicol, and penicillin, and to furazolidone (1,4,5). In chickens, ulcerative enteritis tends to occur after a bout of other infectious disease such as coccidiosis; procedures to control other diseases are useful in reducing losses from ulcerative enteritis (1). However, in the pigeons examined here, there was no evidence of pre-existing or intercurrent disease.

The lesions of ulcerative enteritis recorded here were similar to those reported previously in gallinaceous birds (1) and pigeons (2,3). The cause of the disease reported here was considered to be *C. colinum*, or a similar organism, despite our inability to culture the causative organism from tissues of affected birds. The history, gross and histological lesions, Gram stains demonstrating typical organisms in tissues, and the response of affected birds to treatment, supported this view. This report should serve to familiarize veterinary practitioners with this infrequently reported disease of young pigeons, and to provide information on treatment.

### References