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PEROSIS IN CANADA GEESE (Branta canadensis)

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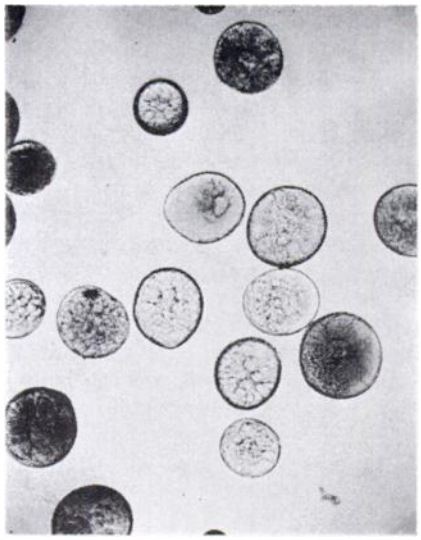


FIGURE 1. *Suspension of* Leucocytozoon simondi *megaloschizonts free of host cells.*

transfused stages since parasite development proceeded simultaneously in both groups. This is in agreement with Dresser (1967, J. Protozool., 14: 244-254), who transfused blood from a newly infected duck to an already patent duck and got an increase in round gametocytes followed by elongate gametocytes and with Fallis et al. (1956, Canad. J. Zool., 34: 389-404) who did the same with uninfected recipients. Having killed our ducks prior to the 11th day, the expected day of elongate gametocyte appearance (Kocan & Clark, 1966, J. Parasitol., 52: 962-966), we have no way of knowing if they would have occurred at all. Since megaloschizonts were present, it is possible that they would have produced elongate gametocytes if the birds had not been killed.

The experiment to determine what developed from megaloschizonts was negative. Neither the duckling receiving whole megaloschizonts nor the one receiving disrupted megaloschizonts showed any circulating parasites for 7 days following inoculation.

Different results might have been obtained if a more precise timing of transfer were employed, or the manipulation of the megaloschizonts might have altered them sufficiently to render them noninfectious. It is also possible that megaloschizonts are not the source of elongate gametocytes and that their disappearance in the 11th day post infection, and the simultaneous appearance of elongate gametocytes, is only a coincidence. As Wehr (1962, Avian Diseases, 6: 195-210) pointed out, elongate gametocytes are present in turkeys infected with *L. smithi* but no megaloschizonts have been found.

Whatever the reason for the negative results, it is hard to imagine that the megaloschizont does not play some major role in the development of *L. simondi*. Further research is needed to clarify the role of the megaloschizont in the life cycle of *Leucocytozoon*.

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PEROSIS IN CANADA GEESE

(Branta canadensis)

Perosis (slipped tendon), a condition characterized primarily by deformities of the leg bones, is commonly encountered in young chickens and turkeys maintained on rations either deficient in one or more essential nutrients or containing them in improper balance. A lack of manganese is a common cause in poultry (Norris and Scott, 1965. *In* Biester and Schwarte (ed.) Diseases of Poultry:144-180. Fifth Ed. Iowa State Univ. Press, Ames; Siegmund (ed.), 1967. *In*

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The Merck Veterinary Manual: 1425-1427. Third ed. Merck and Co., Rahway, N.J.), and the availability of this element is decreased by a large excess of calcium or phosphorus in the diet (Siegmund, *op. cit.*). Choline, biotin, folic acid, niacin, vitamin E, and pantothenic acid deficiencies, particularly the first four, are sometimes implicated in perosis and perosis-like disorders (Norris and Scott, *op. cit.*:181-219). Other pathologic changes, sometimes causing death, occur in young birds when their diet or that of the breeding stock contains inadequate levels of these vitamins.

Norris and Scott (*op. cit.*:165) state that perosis affects young pheasants, grouse, and quail. Similar bone defects were induced in Pekin ducks (Scott and Heuser, 1952, Poultry Sci. 31:752-754; Heuser and Scott, 1953, Poultry Sci. 32: 137-142) and in Emden and White Chinese geese (M. L. Scott, pers. comm.) on niacin-deficient diets. Prior to the observations recorded here, however, the disease has not been reported in geese inhabiting a natural marsh.

History and Procedures

A severe loss of captive goslings occurred on the Fish Springs National Wildlife Refuge (Dugway, Utah) in the spring of 1964. According to the Refuge Manager, 30 of 41 birds of the year presumably died within 2 days to 6 weeks after hatching, although the carcasses of 11 were not found.

The parent stock, 20 adult birds captured and pinioned in other areas a few years earlier, were held with 15 juveniles in a 550 x 1,000 ft. pen enclosing approximately 12½ acres of a spring-fed salt marsh described by Bolen (1964, Ecol. Monog. 34:143-166). Free-flying wild geese at times increased the population to as many as 80. The first of nine broods hatched on May 11, the last on June 18.

Burning of the previous year's vegetation in early spring had resulted in heavier-than-usual stands of salt grass (*Distichlis stricta*) and rushes (*Juncus* sp.). Although a mixture of grains (barley, wheat, and corn), poultry pellets (composition unknown), and alfalfa hay was provided, the young birds did not accept it readily, and their diet consisted largely of new shoots of the two native plants.

Two deaths occurred on May 15, and further losses were recorded every 1 to 4 days from May 23 until the last week of June, when eight apparently healthy goslings remained. Only eight obviously crippled birds were seen during that period, but others were not examined closely for minor abnormalities. No adult geese were grossly affected.

Five goslings were submitted to this laboratory for diagnosis: two that died at 4 days of age, an approximately 1-month-old crippled bird killed for examination, and two severely affected living ones that had survived for 2 months. The latter two were killed for detailed study after 5 days of observation. As a part of each necropsy, liver, spleen, lung, and heart blood were streaked on plates of Brain Heart Infusion Agar and Cystine Heart Agar containing 1.0% hemoglobin (all Difco* products), which were incubated at 37 C.

RESULTS

The carcasses of the two 4-day-old goslings appeared to be normal, both internally and externally. Since dietary deficiencies were not suspected at that time, neither food nor water was requested for chemical analysis. The methods used detected no pathogenic organisms in the tissues of these or the three birds submitted later.

The 1-month-old crippled bird examined in June provided the first clear-cut evidence of the nature of the condition. Its right tibial-metatarsal joint was enlarged, and the gastrocnemius tendon had slipped laterally from its condyle

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(Fig. 1). No other abnormalities were noted.

Except for their markedly deformed legs, the two 2-month-old birds were in good condition. The left tibial-metatarsal joint of one was enlarged, and the articulating ends of the bones were twisted so that the metatarsus extended outward at almost a right angle to the body axis (Fig. 2). Again the gastrocnemius tendon had slipped to the lateral side of the joint (Fig. 3).

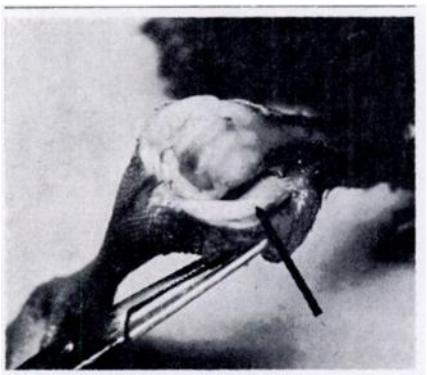


FIGURE 1. *Enlarged right tibial-mentatarsal joint of 1-month-old gosling. Arrow points to gastrocnemius tendonly lying on lateral side.*

Both legs of the second 2-month-old bird were affected so severely that it was unable to stand (Fig. 4). The right leg was extended posteriorly. The tibia was flattened, twisted, and bent at about a 45° angle; the tibial-metatarsal joint was enlarged and stiff (Fig. 5). Hemorrhage at the coxofemoral joint may have been caused by the bird's repeated attempts to stand, which forced the distal end of the femur upward and outward. Except for the enlarged, stiffened hock joint, the left leg was normal.

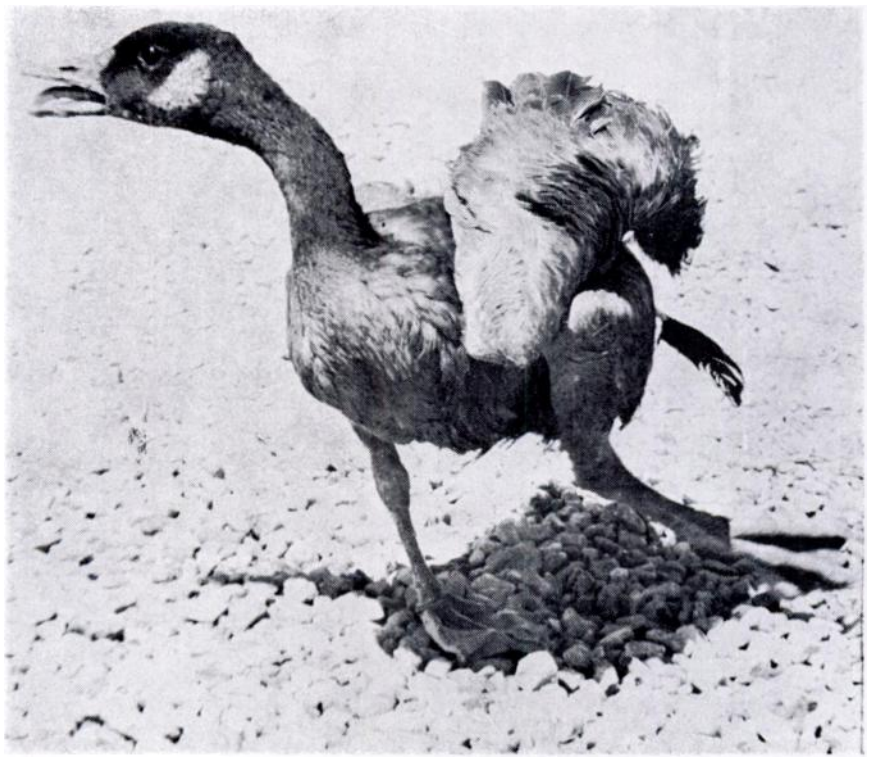


FIGURE 2. *Effects of perosis on left leg of a 2-month-old gosling.*

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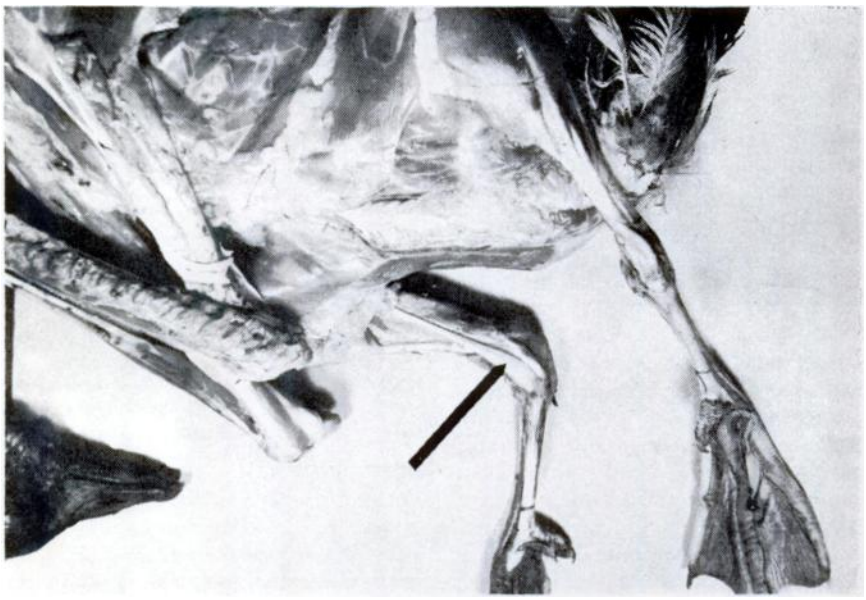


FIGURE 3. *Slipped gastrocnemius tendon of bird shown in Fig. 2.*

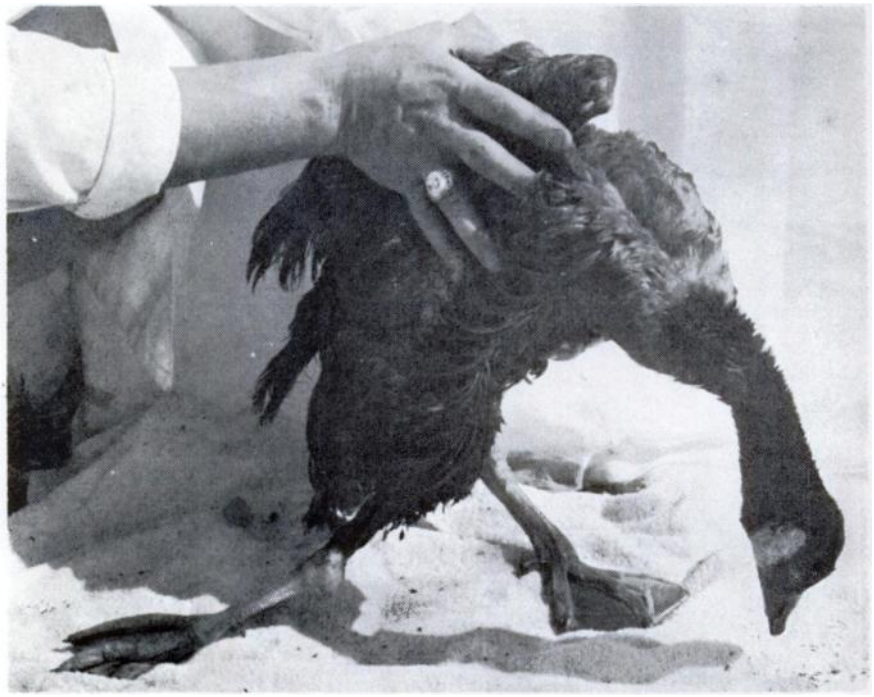


FIGURE 4. *Two-month-old gosling with severe perosis. Right tibial-metatarsal joint is enlarged and leg bones are twisted.*

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Neither gastrocnemius tendon had slipped from its groove. Grossly, the internal organs were normal.

In both 2-month-old birds, the wing on the side of the most severely affected leg was almost constantly held over the back so that it rested on the opposite side (Fig. 2). The wings offered little resistance to manual replacement, but they immediately returned to the abnormal position when the hand was removed. The left wing of one bird drooped, largely, it appeared, because it served as support for the opposite wing (Fig. 2). No defects of the wing bones were recognized. It is possible that the tendency developed over a period of weeks as the birds attempted to maintain balance on their deformed legs.

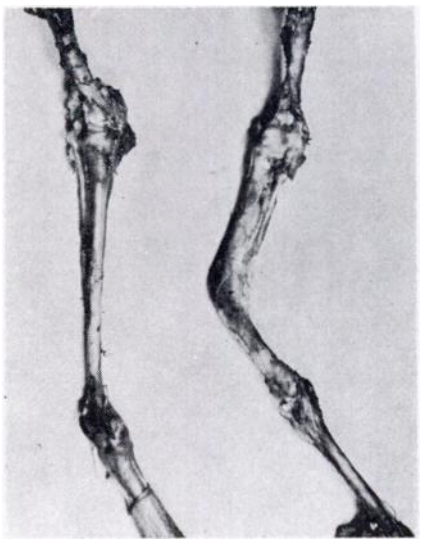


FIGURE 5. *Left and right leg bones of gosling shown in Fig. 4.*

DISCUSSION

The one or more dietary deficiencies responsible for perosis in the Fish Springs flock were not identified. By the time the first laboratory diagnosis was made in late June, the vegetation had matured and probably was not comparable in nutritive value to the new shoots consumed earlier by the young birds. Moreover, because continuous observation of feeding activity was impractical, it was difficult to estimate what proportion of the goslings' diet was made up of the supplementary feeds. Heuser and Scott's studies (1953, Poultry Sci. 32:137-142) showed that Pekin ducklings require more niacin for the prevention of bowed legs than is present in the usual practical chicken ration. Emden and White Chinese geese also have a high requirement for niacin, not only to prevent bowed legs, but also to promote normal growth and prevent mortality (M. L. Scott, pers. comm.). It is possible, then, that the goslings' diet would have been inadequate even if it had been made up entirely of the supplements.

Because only five goslings were examined in detail and only three cases of perosis were diagnosed by laboratory examination, the cause of the major part of the mortality is in doubt. The observation of eight cripples, however, suggests the possibility that about 20 per cent of the young birds had clinical perosis. Others may have died of dietary deficiencies of another kind or before the characteristic signs of perosis developed.

According to more recent records of the Fish Springs Refuge, the mortality rates in the flocks of young geese were 52.4 per cent in 1965 and 70.0 per cent in 1966, but the causes were not determined. The adult flock was transferred from the Refuge to another state, and no information on survival of goslings in the new area was received at this laboratory after that time.

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