

This pattern of feeding being restricted to two nestlings may have resulted from the need for restricting demands on available food supplies and thus competition for food among nestlings. This is suggested by the fact that selective feeding favored the larger birds. However, competition for food among the nestlings is not now the effective operating principle restricting the brood-size of Black Vultures to two birds. I believe the two birds could not have demanded the full time of the adults for their feeding, unless the third bird was excluded from feeding by the selective feeding of the adults and thus it should have received some food and not died so soon. Also, I earlier found (Stewart 1974) that parent Black Vultures normally initiate feeding and that individual young birds await an opportunity to be fed. The selective feeding practiced thus appears to be a behavioral adjustment associated with the number of young birds to be fed and not the excessive demand on the food supply. Acceptance of the introduced bird and neglect of the smaller of their own nestlings by the pair suggests that larger size instead of kinship elicited preferential treatment from the parent birds.—PAUL A. STEWART, 203 Mooreland Dr., Oxford, North Carolina 27565. Accepted 1 Aug, 1982.

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**Malformation of the oviduct in a Canada Goose.**—After pair formation and nest construction, failure to lay followed by incubation behavior on an empty nest is a rare phenomenon in wild birds. Heusmann and Pekkala (*Wilson Bull.* 88:148–149, 1976) recorded such an example in a Wood Duck (*Aix sponsa*) and Fjetlund (*Wilson Bull.* 90:456–457, 1978) and Lumsden (*Wilson Bull.* 92:415, 1980) each reported a case in a Canada Goose (*Branta canadensis*). Male (Br. Birds 70:394, 1977) recorded a Great Tit (*Parus major*) incubating in an empty nest, and Kettle (Br. Birds 71:12, 1978) reported a similar incident involving a Blue Tit (*Parus caeruleus*). Dhondt and Eyckerman (Br. Birds 71:600, 1978) found 7 of about 1000 pairs of Blue Tits and 2 of about 1500 pairs of Great Tits incubating empty nests on their study areas around Ghent, Belgium. In these examples the cause of failure to lay was not determined. We here discuss the causes of failure to lay in a Giant Canada Goose (*B. c. maxima*).

The nesting behavior of a wild Giant Canada Goose was recorded from 1976–1978 when she annually built nests but did not lay (Lumsden 1980). This female was banded F13 as an after-hatching-year bird, and returned with the same mate to nest at the same location for 3 consecutive years. When given fertile eggs she incubated, hatched, and raised goslings.

In 1979 this female built a nest by 25 April, but still had not laid eggs by 22 May. When given dummy eggs, she incubated steadily until 21 June, but later deserted.

In 1980 her nest was built by 9 April and she was sitting steadily by 26 April. By 9 May she had not laid and was given dummy eggs which she incubated for 61 days until 26 June, when she deserted.

On 14 April 1981, F13 and her mate returned to the nesting island and began to prepare a nest-site. By 24 April she had completed a new nest lined with a small quantity of down and was given dummy eggs. On 26 April she started to sit continuously and on 27 April her nest contained an abundance of down. In Canada geese some down is usually present in the nest when the penultimate egg is laid and when the last egg in the clutch is laid an abundance of down is shed into the nest. It was decided to collect the female and ascertain what was responsible for the inability to lay eggs, yet manifest normal reproductive behavior. The collection of this 7-year-old bird was timed to coincide as closely as possible with the time when the last egg could be expected to be laid. The carcasses of F13 and her mate were frozen immediately and stored for study.

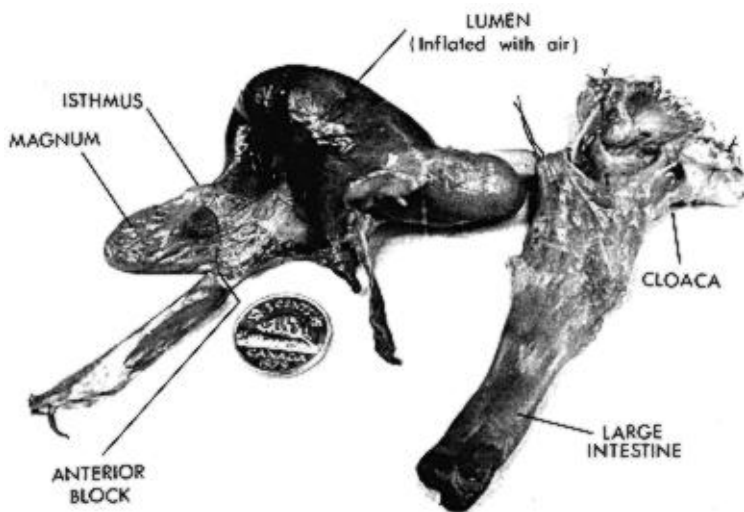


FIG. 1. Deformed oviduct of a Giant Canada Goose lacking an infundibulum.

*Body condition of F13.*—In 1981, as in previous years, this goose fattened normally (Hanson, Arctic Inst. Tech. Paper No. 12, 1962), and by late April, had reached the “rounded” abdominal profile class 3 (Owen, J. Wildl. Manage. 45:227–230, 1981). Giant Canada Geese of this southern Ontario population (Lumsden, Ont. Field Biol. 35:49–56, 1981) do not acquire the massive fat loads of more northern breeding stocks (Mainguy et al., unpubl.) and probably seldom achieve the sagging class 4 abdominal profile. The live weight, mass of the pectoralis major and supracoracoideus muscles, weight of the empty gizzard and heart, and weight of the abdominal fat depot conformed to the values encountered in wild Giant Canada Geese breeding along the Toronto waterfront 55 km to the south. The liver weight fell within the normal range for breeding geese and showed signs of lipid accumulation (Mainguy, M.Sc. thesis, Univ. Guelph, Guelph, Ontario, 1982).

*The ovary.*—The ovary was without sign of disease or deformity and appeared normal for a goose killed at that stage of the breeding cycle. There was one yellow follicle, smooth, firm, and round, apparently still under rapid yolk development. This weighed 4.4 g or about 7% of the weight of an average yolk from an egg of this goose population. This follicle was probably destined to be resorbed, because to attain maturity, it would have had to undergo about another 6 days of rapid yolk synthesis (Lumsden, unpubl.). The abundance of down in the nest suggested that her laying cycle was completed 1 or 2 days before collection. There were six well-defined atretic follicles heavily vascularized with wrinkled, flaccid pinkish-grey walls. These weighed sequentially, 3.2, 1.3, 0.8, 0.2, 0.1, and 0.07 g. Also present were numerous small follicles of the normal size for ova in the resting stage. Ovulated follicles were not present.

*The oviduct.*—The oviduct and the entire cloacal region when dissected from the carcass, revealed that the oviduct structure was grossly deformed (Fig. 1). The oviduct weight was 21.7 g compared to  $83.7 \pm 3.27$  g ( $N = 18$ ) in a normal goose during the laying period. The

exit from the oviduct into the cloaca was open. Since this bird could never have laid an egg, the oviduct likely opened by hormonal means.

The lumen appeared normal, and no tumor or polyp was present. The exit from the lumen was tied off with thread at its junction with the cloaca and inflated with air by means of a pipette. It was apparent that the isthmus was blocked, because no air passed into the magnum. The magnum was grossly foreshortened and a further constriction was present at its anterior end. This constriction was also blocked and prevented the passage of air. When the magnum was punctured for the injection of air, a quantity of pink viscous (albumen?) material flowed out. Anterior to the upper blockage the oviduct was narrow, 27.7 mm long, enshrouded in mesenteric tissue and terminated in a point. The funnel-shaped infundibulum was missing. The failure to lay in this goose was clearly a result of a deformed and blocked oviduct.

It is of interest that the ovary which appeared to be normal contained six atretic follicles, but no indication of ovulated follicles. These atretic follicles were graded in size indicating that at maturity each in turn began to resorb. If the endocrine system as a whole had been functioning properly we would expect that ovulation would have taken place, but into the body cavity, since the infundibulum was missing.

Cole and Hutt (Poult. Sci. 32:481-492, 1953) described a condition in 26 of 324 fowl in which the cause of non-laying was an incomplete oviduct. They stated that in most of these the infundibular portion of the oviduct persisted, while a section of the albumen secreting region degenerated. They did not specifically discuss the state of the ovaries in these birds and nowhere described the presence of atretic follicles. In their discussion of non-layers with normal oviducts they noted there was often liquid yolk in the body cavity indicating that the bird had ovulated but the yolk had not been picked up by the infundibulum.

Failure to ovulate suggests a deficiency of LH, and atresia is controlled by the secretion of progesterone (Van Tienhoven, *Endocrinology of Reproduction in Birds*, pp. 1088-1169, in *Sex and Internal Secretions*, W. C. Young, ed., Williams and Wilkins, Baltimore, Maryland, 1961). It seems likely that the massive deformity of the oviduct of the Giant Canada Goose F13 probably occurred during early ontogeny. We speculate that the deformity and the absence of the infundibulum may have prevented adequate secretion of LH, hence the absence of ovulated follicles. However, adequate secretion of sex hormones occurred to allow copulation, pair-bonding, nest-building, incubation, brooding behavior, and presumably five seasonal proliferations and regressions of the oviduct. The development of the magnum and the presence of gelatinous material suggests that this zone of the oviduct, although reduced in size, was still able to respond to the synergistic effects of estrogen and progesterone. This is Ontario Ministry of Natural Resources, Wildlife Research Contribution No. 82-05.—HARRY G. LUMSDEN, *Ontario Ministry of Natural Resources, Wildlife Research Section, P.O. Box 50, Maple, Ontario L0J 1E0, Canada*; AND VERNON G. THOMAS, *Dept. Zoology, Coll. Biological Science, Univ. Guelph, Guelph, Ontario N1G 2W1, Canada. Accepted 15 Sept. 1982.*

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**Black-capped Chickadee performs "hiss-display" while in wire-mesh trap.**—On 15 July 1981, I re-trapped a color-banded female Black-capped Chickadee (*Parus atricapillus*) that had visited feeders in my yard in Logan, Cache Co., Utah, intermittently between her initial capture on 13 December 1980 and 21 April 1981. The bird was not re-trapped during that interval. I carried her into the house (a distance of 11 m) in the wire mesh trap 17 × 17 × 16 cm. As I placed the trap on the table the chickadee, which crouched on the