intake with 8-cc water ration reported by Collier & Knarr may be very little beyond what rats would eat when not given water; thus water at this low level seems to be of very low efficiency compared with food intake with higher water ration. This low intake level of 0.04% quinine solution may be sufficient to correct the dehydrated state produced by eating dry food but not enough to produce additional eating.

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NOTE

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Neural control of feeding behavior in the pigeon[1]

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Anatomical studies have identified a network of structures at several levels of the avian brain whose location and connections suggest that they are involved in the neural control of feeding in the pigeon. The network originates in a cranial nerve sensory nucleus innervating the oral region and includes striatal and limbic components. Bilateral lesions placed in several of the structures of this network produced disturbances of feeding and drinking, including aphagia, adipsia, and hypophagia. It is suggested that further neurobehavioral analysis of these structures may clarify the role of extrahypothalamic mechanisms in the neural control of feeding.

Since the classic studies of Flourens, it has been known that total removal of the cerebral hemispheres abolishes feeding behavior in the pigeon, but the precise location of the regions involved has remained uncertain. The present preliminary results provide a more discrete localization within the avian forebrain of the structures responsible for the aphagia of the decerebrate bird. Furthermore, our findings indicate that these forebrain regions are components of a network of structures at several levels of the brain which is involved in the neural control of feeding. Figure 1 illustrates diagrammatically the location and some connections of several of these structures.

The network originates in the main sensory trigeminal nucleus, a sensory relay nucleus innervated by afferents from the oral region. An ascending pathway, the quinto-frontal tract, arises from this nucleus and projects to the forebrain, terminating in the nucleus basalis. The nucleus basalis is located in the basal region of the avian striatum, adjacent to the olfactory bulb and its associated structures. Axons of nucleus basalis contribute to the tractus fronto-archistriaticus which terminates in the dorsolateral nucleus of the archistriatum—an avian homologue of the mamalian amygdala. From this region efferent axons project to the hypothalamus. The implication of amygdala and hypothalamus indicates that the network includes components of the limbic system (Wallenberg, 1903; Huber & Crosby, 1929).

The existence of a series of pathways linking avian striatal and limbic structures with a sensory relay nucleus innervating the oral region suggested that it might be profitable to examine the effects of lesions in some of these structures on feeding and drinking.

SUBJECTS

This report is based on the data of 26 pigeons *(Columba livia)* obtained from a commercial supplier. They were housed in individual cages with food and water available ad lib. Room temperature was kept between 70 deg and 80 deg and a 12-h light/dark cycle was in effect in the laboratory.

PROCEDURE

Following a period of adaptation to the laboratory, food and water intake and

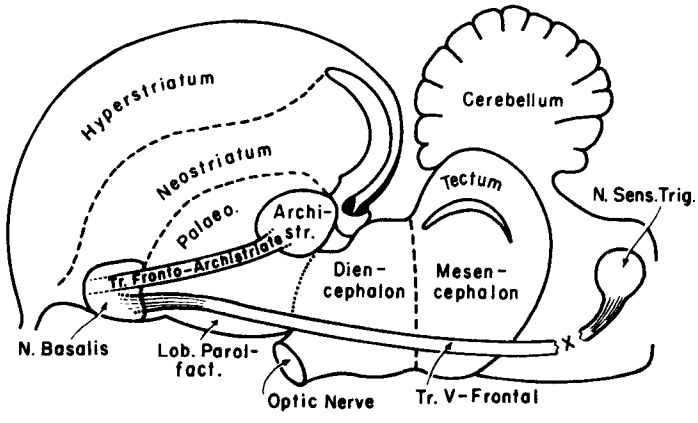

Fig. 1. Schematic diagram of the location and connections of some structures involved in the neural control of feeding in the pigeon.

Table 1
Effects of Brain Lesions upon Food and Water Intake in the Pigeon

Group	N	Days of aphagia	Days of adipsia
Control	6	0, 1, 1, 0, 1, 2.	0, 1, 0, 0, 1, 0.
Main Sensory Trigeminal Nucleus	4	4, 12, 18, 29*	0, 3, 0, 1*
Quinto-Frontal Tract	3	5, 7, 28*	2, 1, 28*
Nucleus Basalis	8	5, 7, 9, 13, 17, 22, 22*, 64*	2, 3, 1, 1, 2, 3, 22*, 64*
Archistriatum	5	3, 7, 7, 10, 16	0, 0, 2, 0, 0.

** Maintained on a liquid diet.*

weight data were recorded for several weeks prior to the placement of lesions. Postoperatively, this data was recorded for periods ranging from several weeks to several months. In birds (McFarland, 1964), as in many mammals, food and water intake are closely related. For this reason adipsic Ss were intubated with water daily. Some aphagic birds were maintained for varying periods by intubation of a liquid diet.

Bilateral electrolytic lesions were placed stereotaxically using a dc lesion-maker and No. 00 insect pins insulated to within .5 mm of the tip. The amperage and duration of the current and the number of lesions placed in each bird varied with the size and location of the structure being lesioned. Lesion coordinates were derived from a stereotaxic atlas of the pigeon brain (Karten & Hodos, 1967).

In the experimental group, lesions were directed at the main sensory trigeminal nucleus, the quinto-frontal tract, the nucleus basalis, and the dorsolateral nucleus of the archistriatum. Control birds were subjected to mock surgical procedures or to lesions directed at structures adjacent to those mentioned above.

Birds were sacrificed with an overdose of pento-barbitol and perfused with formol-saline. The brains were embedded in celloidin and cut at 40 μ, every other section being stained with thionin. The locus and extent of the lesions in each bird were then determined.

RESULTS

Table 1 summarizes the major findings with respect to lesion effects upon food and water intake. Surgical control procedures or control lesions produced little or no interruption of eating or drinking while striking deficits in these behaviors were found in the experimental group.

Effects Upon Food Intake

Lesions in the experimental Ss produced deficits in feeding behavior whose persistence and severity varied directly with the extent of the lesion. Thus, in a number of birds with massive lesions of the nucleus basalis or section of the quinto-frontal tract, aphagia persisted with no sign of recovery for as long as the birds were maintained on a liquid diet–17, 22, 28, and 64 days, respectively. In most cases, however, no attempt was made to maintain the birds artificially. Those Ss that did not recover were sacrificed after sustaining body weight losses of 25 to 30%, at which point their condition bordered on a state of inanition. In some birds with less extensive lesions, the aphagia was transient and was followed by recovery to preoperative intake levels. In others, several days of aphagia were followed by hypophagia persisting for periods of several weeks to several months.

Effects Upon Water Intake

Severe and persistent deficits in drinking behavior were found in three aphagic birds which were being maintained on a liquid diet. However, the persistent adipsia of these Ss may have been an indirect effect of the absence of dry food and the daily intubation of large amounts of liquid food. For the remainder of the experimental group such adipsia as did occur tended to be a transient phenomenon (1-3 days). The present data, therefore, suggests that the primary effect of the lesions was upon feeding rather than drinking behavior.

The rate of body weight loss in the experimental Ss did not differ from that of normal pigeons deprived of food for comparable periods. Furthermore, none of the lesioned birds showed any persistent impairment of posture or locomotion. Grooming and pecking behavior appeared normal and birds would swallow grain placed at the back of the mouth.

Such observations make it unlikely that the feeding deficits seen in these birds are attributable either to motor impairment or metabolic dysfunction. They suggest, rather, that the primary effect of the lesion is upon neural mechanisms related to food intake.

DISCUSSION

Previous investigations involving lesion and stimulation techniques have suggested that basal forebrain regions are involved in the neural control of feeding by the pigeon (Portmann & Stingelin, 1961; Phillips, 1964; Harwood & Vowles, 1966). The present study has localized the structures involved more precisely and shown that lesions in these structures produce disturbances in food and water intake.

Furthermore, our findings indicate that the neural mechanisms underlying feeding behavior in the pigeon are localized in a network of structures at several levels of the avian brain. The network originates in a sensory relay nucleus innervating the mouth and includes striatal and limbic components. Because some of the sensory inputs, central connections, and efferent projections of these structures are known, they lend themselves to analyses employing both neurobehavioral and electrophysiological techniques (Zeigler & Green, 1967; Zeigler & Witkovsky, in press). Such analyses may clarify the contribution to the neural control of feeding of structures at various levels of the brain.

With the exception of Dethier's studies of hunger in the fly (Dethier, 1967), the analysis of feeding behavior mechanisms has focused largely on one structure, the hypothalamus, and one vertebrate species, the rat. Data from a different vertebrate class and from a group of extrahypothalamic structures should, therefore, contribute to the comparative analysis of neural mechanisms underlying feeding behavior.

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NOTE

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