REVIEW ARTICLE

THE NEURAL BASIS OF FOOD AND WATER INTAKE IN BIRDS*

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INTRODUCTION

The purpose of this review is to outline research on the avian brain with respect to the regulation of food and water intake. Comparisons with the more widely known mammalian literature will be made wherever possible, and, in particular, emphasis placed on more behavioural approaches. Richter (84) first applied the concept of homeostasis to behaviour when he showed in his 'cafeteria' experiments that rats develop motivated behaviour appropriate to the correction of their own physiological needs, and to the maintenance of their internal environment. Since this time it has been recognized that behaviour and its underlying physiology are needed to fully understand motivational problems, and there are now many excellent and comprehensive reviews which emphasize the role of the central nervous system in the regulation of food intake (4, 13, 30).

Experiments with rats and other small mammals have established a key role for the hypothalamus in the control of both food and water intake. As the hypothalamus has a relatively constant anatomical organisation in all mammals (57), it is not very surprising that little sign of variation in its functional organisation with respect to feeding and drinking has so far been detected. Mammals and birds are both descended from reptiles, but have diverged and taken separate evolutionary paths, such that the diencephalon of birds is much closer to the reptile than to the mammalian brain, and, in contrast to mammals, birds show very little neocortical development.

If we are to attempt to build general models of motivation (91), then comparative studies are required to clarify the neural organisation of behaviour.

Species variation in the role of oral, alimentary and systemic factors: Feeding and drinking are thought to be initiated by changes in the level of blood glucose (7), osmotic pressure (27), and other systemic factors. Food and water intake are monitored at 3 levels : oral, alimentary and systemic and all three may contribute to ultimate satiety. In mammals, it is

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likely that a ventromedial hypothalamic 'satiety centre' receives information relating to gastric distension (89), blood glucose levels (7), and possibly fat metabolism (47). In the case of drinking, no corresponding centre has been found.

The alimentary and digestive system of birds differ in a number of aspects from mammals - most noticeable is the absence of teeth and the presence of three storage components for food - the crop, proventriculus, and stomach. The size and shape of the crop varies widely between species, e.g. in the pigeon it serves as a storage organ (25). Removal of the crop (81) does not appear to impair food intake and weight regulation. There are major differences in metabolism (94), e. g. insulin injections do not lead to increases in food intake (23) and experiments with goldthioglucose have not revealed the existence of 'glucosensitive' regions in the hypothalamus (14, 29, 100) as suggested by Mayer *et al.* (62) in mammals.

Sensory factors, particularly those of taste, are of considerable importance in the regulation of appetite (73, 74). Birds have a small number of taste buds(45) and it is unlikely that taste is as important a factor in the selection of food as it is with mammals. With respect to sweet tasting substances, quail, jungle-fowl, herring-gulls, starlings, finches, pigeons and geese, are all indifferent to sucrose (46). Visual properties and the surface texture of food are reported as taking preference over all other qualities in the bird's selection of food (69).

Satiation is a multifactor process, but there do appear to be considerable species differences in the weighting that is given to the various factors (1). Thus sham feeding and drinking postpones satiation in rats and dogs (10, 39, 48) and in man it appears that oral factors override gastric factors since subjects overeat when simultaneously fed a liquid diet both orally and intragastrically (40). However, similar experiments in pigeons (65) indicate that oral factors play no role in the satiation of drinking. Thus, while the delays involved in digestion and absorption entail some sort of short-term satiation mechanism, it appears that different species have solved the problem in different ways.

Anatomy of the Avian Brain

Forebrain: The anatomy of the avian brain has been extensively reviewed (36, 44, 79). Cerebral evolution in birds is dominated by the growth and development of the corpus striatum , neopallial structures are relatively small and show a striatal pattern due to their three dimensional development, which is in contrast to the surface growth of neocortex that characterises cerebralisation in mammals. In the avian forebrain there is no corpus callosum or tracts corresponding to the mammalian pyramidal tract, and the archipallium is represented by a highly rudimentary hippocampus in the medial walls of the hemispheres.

Homologies between the various parts of the mammalian and avian forebrain have been investigated in the ontogenetic studies of Kappers (1923), Kuhlenbeck (1938), Kallen (1953), and Haefelfinger (1958). With the exception of Kallen, who regarded most of the

avian striatal structures as being homologous to the mammalian neopallium, the above authors are in substantial agreement on the main mammalian homologues of the avian forebrain structures, especially of its basal parts. According to cytoarchitectural studies (92), the paleostriatum and archistriatum present rather general features in various avian species systematic groups, whereas qualitative evolution in birds is reflected in differences of relative growth and cytoarchitecture in the dorsal parts of the forebrain.

Diencephalon: The diencephalon in birds is divisible into the epithalamus and the dorsal and ventral thalamus, as in other vertebrates. The thalamus is composed of a number of well developed nuclei and has abundant connections with the telencephalon and the mesencephalic tectum. The laterally displaced avian tecti form large and complicated optic lobes that play an important role in sensory-motor integration. Attempts to homologise the nuclei of the avian thalamus with the thalamic nuclei of mammals on the basis of their morphologic or ontogenetic relations or characteristics have led to controversial interpretations (38, 52). Powell and Cowan (80) investigated the projections of the thalamic nuclei upon the telencephalon by the technique of retrograde cell degeneration, considered that it was not possible to homologise solely on the basis of telencephalic projections, the individual nuclei or nuclear groups of the avian and mammalian thalamus. The functional significance of the avian dorsal thalamus was questioned by Erulkar (1955), who could not record evoked potentials to tactile or auditory stimulation from any of the thalamic nuclei in pigeons even when such stimulation evoked potentials in caudal parts of the neostriatum. In the light of recent data (34, 76) the nucleus rotundus, the most conspicuous nuclear mass in the avian thalamus, appears to be a relay of visual information mainly to the paleostriatum and neostriatum caudale. However, the functional organisation of the major part of the avian thalamic nuclei still remains unsettled.

We can consider the preoptic region with the hppothalamus proper, although it is more correctly a part of the telencephalon. The preoptic region is grouped around the third ventricle in the region anterior to the entrance of optic fibres into the brain. A preoptic nucleus can be distinguished which is probably the homologue of the mammalian nucleus of that name (31), and in the dove consists of fine densely packed cells on either side of the ventricular wall.

A series of discrete nuclei with similar organisation to mammals can be distinguished in the anterior hypothalamic region (19). However, in the present context, an important difference between birds and mammals is the complete absence of a well-defined ventromedial nucleus, and absence of the medial forebrain bundle as a component of the lateral hypothalamus.

Connections between the forebrain and hypothalamus: Considerable confusion can arise when comparing the major forebrain bundles in the mammalian and avian brains, as the medial forebrain bundle or *tractus septo-mesencephalicus* is not directly homologous with the mammalian medial forebrain bundle. In mammals the latter has fibres of striatal origin (31) and the hippo-

campal projection system, the fornix, is distinct from the bundle—neither of these facts is the case with birds. In the avian brain the tractus septo-mesencephalicus originates from the front three quarters of the septum (19) and the fibres converge ventrally into a large bundle which passes ventrally the anterior commissure and dorsally the ventral peduncle. It then bends latero-dorsal and splits into several branches. However, according to Gurdjian, by far the largest component of the rat medial forebrain bundle is the septo-hypothalamic component, and in view of this similarity to birds, it is interesting that MacPhail (58) found stimulation of the avian forebrain bundle to be aversive in contrast to the rat (72). This in itself suggests a significant difference between the organisation of the motivational systems of the two classes of animal.

The lateral hypothalamic feeding centre of mammals (5,6) is inextricably associated with longitudinal fibres of passage comprising the median forebrian bundle. Nauta (70) has defined the medial forebrain bundle as the cardinal connection of the septum and preoptic regions with the midbrain, and considers it to be a common trajectory for mingled ascending and descending projections of various origins.

In transverse silver sections of the dove brain (Fig. 1), the most distinctive feature at the levels of the forebrain and diencephalon is the *lateral forebrain bundle*. This bundle shows a great increase in the number of fibres and in the richness of their distribution over its homologue in the reptiles, but is built on the same general plan. Various components of the system have been described (15, 20, 37), but the analysis of the components in 1903 by Edinger and colleagues still remains the classic description on which all later work is based.

Kappers, Huber, and Crosby (44) point out that, using silver stains, the lateral forebrain bundle is seen, not as made up of distinct and separate components, but a broad fan-shaped mass of fibres connecting the major portion of the lateral wall with most of the dorsal thalamus, certain pretectal areas, and portions of the central thalamus, hypothalamus, and tegmentum. Important components of this bundle are the frontothalamic tract, tractus thalamo-frontalis intermedialis, tractus striocereberallis, tractus thalamo frontalis-medialis, and the tractus striohypothalamius medialis. An avian peculiarity, the quinto-frontal tract, is also incorporated within the LFB (71).

Edinger *et al.* (1903) include the tractus *occipito-mesencephalicus* as a component of the lateral forebrain bundle, but in doves it is easily distinguished from the latter. At the level of the anterior commissure, it bends to the median and in the brain stem, again to the caudal. Phillips (76) found that electrical stimulation of the archistriatum produced evoked potentials in the nucleus ruber, posterior commissure, septum, pre-optic area, anterior hypothalamus and dorsal supra-optic decussation. With the possible exception of the septum, all these regions receive fibres from the tractus occipito-mesencephalicus.

In summary, in addition to inputs from all sensory modalities, the avian hypothalamus receives fibres from the archistriatum (homologue of the mammalian amygdala) and from the

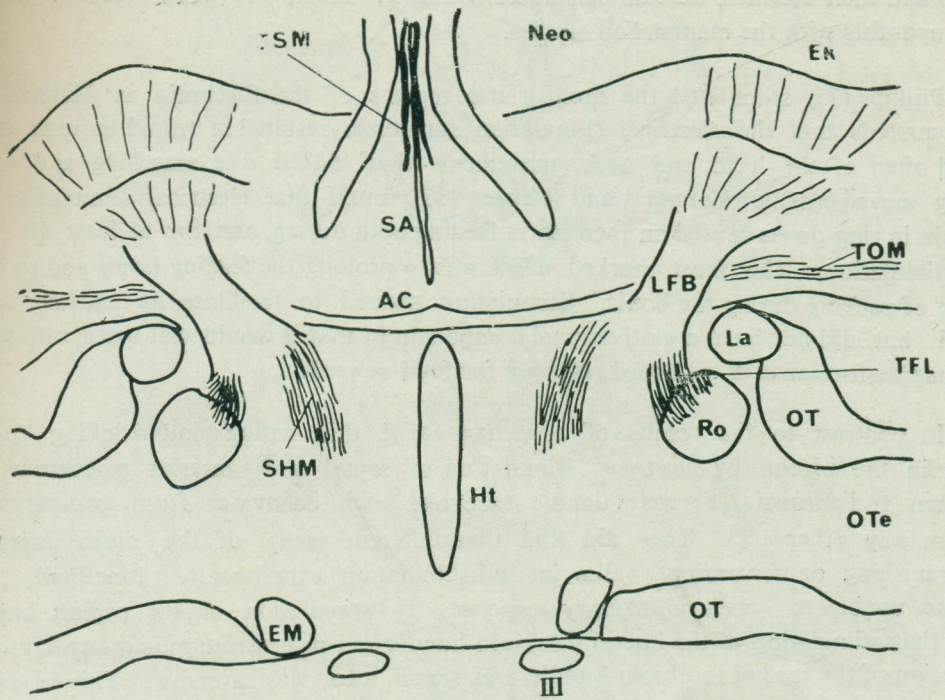

Fig. 1: Frontal section through the brain of the Barbary dove at the level of the anterior commissure.

Key to abbreviations.

AC=anterior commissure, EK=ektostriatum, EM=nucleus ectomamillaris, Ht=hypothalamus, La=nucleus lateralis anterior thalami, LFB=lateral forebrain bundle, Neo=neostriatum, OT=optic tract, OTe=optic tectum, Ro=nucleus rotundus, SA=septal area, SHM=tractus strio-hypothalamicus medialis, TFL=tractus thalamo-frontalis lateralis, TOM=tractus occipitomesencephalicus, TSM=tractus septo-mesencephalicus

paleostriatum primitivum (homologue of the mammalian globus pallidus) via the tractus occipito-mesencephalicus and the lateral forebrain bundle, and from the septal region via the tractus septo-mesencephalicus or medial forebrain bundle.

Brain Stimulation and Feeding Behaviour: Akerman *et al.* (2) elicited hyperphagia, polypneic panting and polydipsia, by electrical stimulation from implanted electrodes in the hypothalamus of pigeons (*Columba livia*) and concluded that the central regulation of body temperature, and of food and water intake is principally the same in birds and mammals. From the diagrams of Akerman it appears that stimulation of the area ventralis anterior, the stratum cellulare externum, and the septum, caused eating and prolonged stimulation, over-eating. However, there are no quantitative observations in this paper, and no indication of the repeatability of the results. Indeed, since implantation of the electrodes, testing of the

animals and their sacrifice, were all performed within 24 hours, the work was semi-acute, and not comparable with the mammalian studies.

Phillips (75) stimulated the archistriatal regions of the forebrain in Mallard ducks, and comments that the electrical stimulation sometimes resulted in "rapid movements of the bill and often of the head and neck, movements that looked like searching and gabbling (feeding movements)". Harwood and Vowles (33) found that electrical stimulation of the forebrain in ring doves caused an increase in feeding both during, and for at least 10 minutes after stimulation. The most marked effect was to prolong the feeding bouts and to increase the rate of pecking during the bouts. Stimulation seemed to facilitate an existing tendency to feed, but did not have a motivational component in that it would not cause satiated birds to resume performance of a learned response for food reward.

In contrast to the results of Akerman *et al.* that eating and drinking could be evoked in the pigeon by electrical stimulation of septal and anterior paleostriatal sites, Goodman and Brown (28) were unable to evoke such behaviour from comparable sites or from any other loci. They did find that different areas of the brain appeared to have punishing or rewarding value in self-stimulation experiments. MacPhail (58, 59) was also unable to elicit alimentary responses by stimulation of the pigeon brain, but found that stimulation of the lateral forebrain bundle and paleostriatum is rewarding, whereas stimulation of the medial forebrain bundle and septal area was aversive. This is in sharp contrast to mammals where the septal area and medial forebrain bundle are generally accepted as part of the reward system (72); support for this observation comes from Phillips (75), who found that electrical stimulation of the septal area in six Mallard ducks yielded "escape" responses. There is some evidence, then, for a contrast between avian and mammalian organisation with respect to self-stimulation. More work is needed, as Andrew (9) working with young chicks describes an anatomical organisation similar to the mammalian pattern.

Wright (100) stimulated over 100 sites in 37 Barbary doves in an attempt to find areas which would elicit feeding behaviour. Some 14 electrodes appeared to be associated with feeding behaviour (Fig. 2), but this was typically highly variable. "On one day the feeding might be quite vigorous and prolonged, and another day the same site would yield either no feeding at all, or very weak pecking at food. The latter was more commonly the case, the pecking was slow and cautious, often broken by periods of walking around the cage and pecking at inanimate objects. A good deal of attentive postures were seen in association with feeding, and in some animals signs of fearful behaviour, which at higher intensities resulted in escape behaviour. In twelve of these sites, the feeding usually occurred *after* stimulation had ceased, sometimes with a latency of two or three minutes. In any one session it was quite impossible to predict whether feeding would occur or not".

Phillips and Youngren (78) in an extremely comprehensive study in which over 1500 brain loci in 87 birds were tested found no ESB-evoked feeding in chickens or ducks.

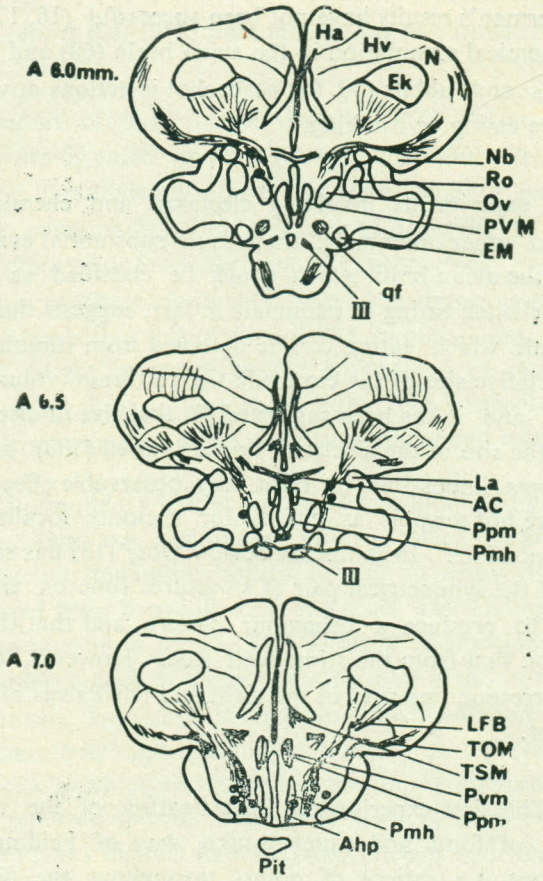

Fig. 2: Sites associated with feeding behaviour on more than 50% of the experimental sessions.

Key to abbreviations

AC=anterior commissure, Ahp=nucleus hypothalami anterior, Ek=ektostriatum, EM=nucleus ectomamillaris, Ha=hyperstriatum accessorium, Hv=hyperstriatum ventrale, La=nucleus lateralis anterior thalami, LFB=lateral forebrain bundle, N=neostriatum, Nb=nucleus basalis, Ov=nucleus ovoidalis, Pit=pituitary, Pmh=nucleus medialis hypothalami posterioris, Ppm=nucleus principalis precommissuralis, Pvm=nucleus periventricullaris magnocellularis, qf=tractus quinto-frontalis, Ro=nucleus rotundus, TOM=tractus occipito-mesencephalicus, TSM=tractus septo-mesencephalicus, III=third cranial nerve.

Putkonnen (31) similarly did not report feeding with chicknes, and Delius (16, 17) could not obtain reliable eating from stimulation of the diencephalon in Herring and Lesser Black-backed gulls (*Larus aregntatus* and *Larus fuscus*).

In summary, only von Holst and St. Paul (35) and Akerman *et al.* have reported compulsive eating or drinking in birds, the former give no histological details at all, and

attempts to replicate Akerman's results have not been successful (16, 17, 33, 58, 59, 75, 78, 81). There is one report of chemical stimulation in the avian brain (60) and this study also showed that in contrast to rats and rabbits (30, 68) carbachol injections at various diencephalic sites in the dove do not induce eating or drinking.

On the basis of experiments involving electrical and chemical stimulation, and in contrast to the rat and other mammals, there is no substantial evidence for a region in the lateral hypothalamus of the avian brain which could be classified as a feeding centre. The literature on electrically elicited eating in mammals in fact, suggests that the rat is rather unique in the relative ease with which eating can be obtained from stimulation of the lateral hypothalamus. In rats the relative size of the electrode tip to brain volume is much larger than in other larger species, and it has been suggested (8) that size of electrode tip is important in this context. In doves, the size of the electrode tip was varied (100) but with a tip exposure of under 0.2 mm. it was impossible to elicit any observable effects, and tip sizes of above 0.5 mm. overall length are not sensible as claims for accurate localisation within the hypothalamus would be impossible. In a recent review Doty (18) has suggested that stimulation may activate only one of the symmetrical pair of structures (one on either side of the brain) that normally interact to produce a behaviour pattern, and that the output from the unstimulated side might inhibit that from the stimulated side. However, the difficulty of getting two electrodes into corresponding sites in the right and left halves of the brain will make this suggestion difficult to test.

Lesion studies: The first experimental investigation of the role of the avian hypothalamus in regulation of food and water intake, was of Feldman *et al.* (26) who placed bilateral electrolytic lesions at a variety of points throughout the diencephalon of some 350 domestic chickens. They reported 12 animals which lost the ability to eat, some of which had accompanying disturbances in temperature regulation from which they subsequently died, and all birds showed almost no voluntary activity. They describe the aphagia as resulting from widely scattered lesions in the medial and lateral, anterior and posterior regions of the hypothalamus. Inspection of their photographs indicates that the lesions were in the stratum cellulare externum and in the medial posterior hypothalamic nucleus. Smith (90) reports 6 hens becoming aphagic follwing lesions of the lateral hypothalamic nucleus, but gives no description of the postoperative behaviour, or whether the birds eventually recovered.

Leprovsky and Yasuda (55) report hyperphagia in chickens from lesions "in what may be assumed to be the ventromedial area of the hypothalamus." However, none of their data indicates a significant increase in the food intake of lesioned animals, and the authors appear to use the term 'hyperphagia' incorrectly. The graphs shown in this report, and a later review (56) clearly show that the lesions in both sexes produce gains in weight, without accompanying increases in food intake, and the graph for female hens actually shows a decrease in food

intake. The authors again report that there were indications of a functional castration in male and female animals.

A more convincing report of hyperphagia describes (50) eleven cases following hypothalamic lesions in the white-throated sparrow (*Zonotrichia albicollis*). Lesion damage was restricted to the ventromedial hypothalamus above the optic chiasma and supra-optic decussation, below the anterior commissure, and adjacent to or across the ventral portion of the third ventricle at an anterior-posterior level between the supra-optic nuclei and the median eminence. The weight gain of the experimental birds was almost identical to that of photostimulated controls which suggests that this region of the hypothalamus may be involved in the normal photo-induced weight increases of the white-throated sparrow before its Spring migration. In a later report (51) these authors indicate that the hyperphagia is often accompanied by a polydipsia, and associated with gonadal disturbances and moult.

Wright (99) using radiofrequency lesions described two kinds of aphagia in the Barbary dove (Streptopelia risoria). One was a short-term effect in which the animals appeared highly motivated to eat but were unable to mandibulate *i.e.* to pick up seed with the beak and carry it to the back of the throat prior to swallowing; the other was a more permanent aphagia in which the birds displayed no interest in food. This short-term aphagia was initially accompanied by adipsia. The time course of a representative bird is shown in Fig. 3. Special care was taken in this study to ensure that the birds were not showing altered temperature regulation which might secondarily result in aphagia. Kanematsu (41) has reported that lesions of the lateral hypothalamic nucleus in chickens resulted in prolonged hypothermia, the same region from which Smith (90) has reported aphagia. In a continuing series of experiment Wright (100) was unable to clearly associate any distinct hypothalamic nucleus with feeding deficits. Analysis of the anatomical damage underlying aphagia in the dove suggests that damage to lateral aspects of the lateral forebrain bundle, and in particular to the tractus thalamo-frontalis and tractus strio cereberallis would account for the mandibulation deficits (see Fig. 1). These two tracts include sensory fibres innervating the mandibles (tractus quinto-frontalis) and glossopharyngeal fibres innervating the salivary glands and muscles of the tongue and pharynx.

Substantial agreement with the results in the dove has come from two recent reports; Zeigler (103) found that pigeons with lesions of the main sensory trigeminal nucleus and the nucleus basalis were able to peck normally at food and to swallow seed placed at the back of the mouth, but were unable to mandibulate their food; in a single-unit analysis of the trigeminal nucleus, Zeigler and Witkovsky (102) report that 72% of the cells were activated by mechanical stimulation of the beak, ranging from very light touch to pressure. Lesions of the quinto-frontal tract (103) produced both aphagia and adipsia in pigeons.

Cerebral Influences: Early reports (22, 60, 88) indicate that feeding behaviour persists after incomplete isolation of the cerebral hemispheres, whereas Rogers (85) and Ten Cate (96)

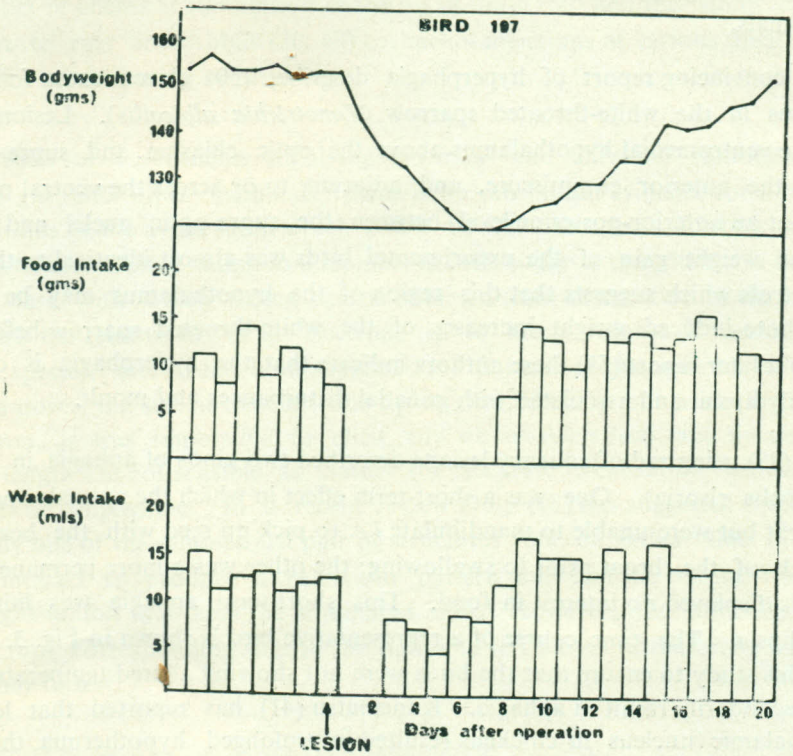

Fig. 3: Recovery from Aphagia and Adipsia in the Barbary dove. This bird had bilateral lesions in the lateral hypothalamus impingeing on lateral aspects of the lateral forebrain bundle.

concluded that without the hemispheres, the ability to eat and drink is lost. However, Thauer and Peters (97) kept a pigeon alive for two years without its hemispheres and the pigeon was neither blind, nor had it lost the ability to take up food. These contradictions are in part resolved by two later studies (3, 24) in which pigeons with all connections between the telencephalon and the rest of the brain cut, showed neither eating or drinking.

The tentative conclusion which can be drawn from these and more recent work (99, 103) is that for normal feeding behaviour the integrity of connections between the forebrain and the diencephalon is essential; and that the development of the anterior neostriatum and the immediately overlying hyperstriatum ventrale of various avian species, is postively correlated with the complexity of their feeding habits (17, 67, 93).

Association between feeding and drinking: A hungry animal is also slightly thirsty, and vice versa, this reciprocity between hunger and thirst has been described in a wide number of mammalian species (12, 54) and extensively investigated in the Barbary dove

(63, 64). Characteristically the lateral hypothalamic syndrome in mammals results in both aphagia and adipsia, and invariably the aphagia recovers before the adipsia (95). The early reports of aphagia in birds do not give details of their durations, and it is difficult to say how far there is any association between adipsia and aphagia.

Recent work on doves and pigeons indicates that, in marked contrast to the rat, adipsia always recovers before aphagia; Figure 4 summarises the length of aphagia and adipsia in doves taken from Wright (1969). This contrast with mammals may in part be due to the eating and drinking response patterns being behaviourly distinct from each other in pigeons which as a group are characterised by their drinking response of siphoning water into the oesophgus with the beak held under the water (98).

Recovery occurs abruptly in the dove (see Fig. 3) and is unaccompanied by any of the signs of aversion to food which are so characteristic of the rat. In chickens two cases have

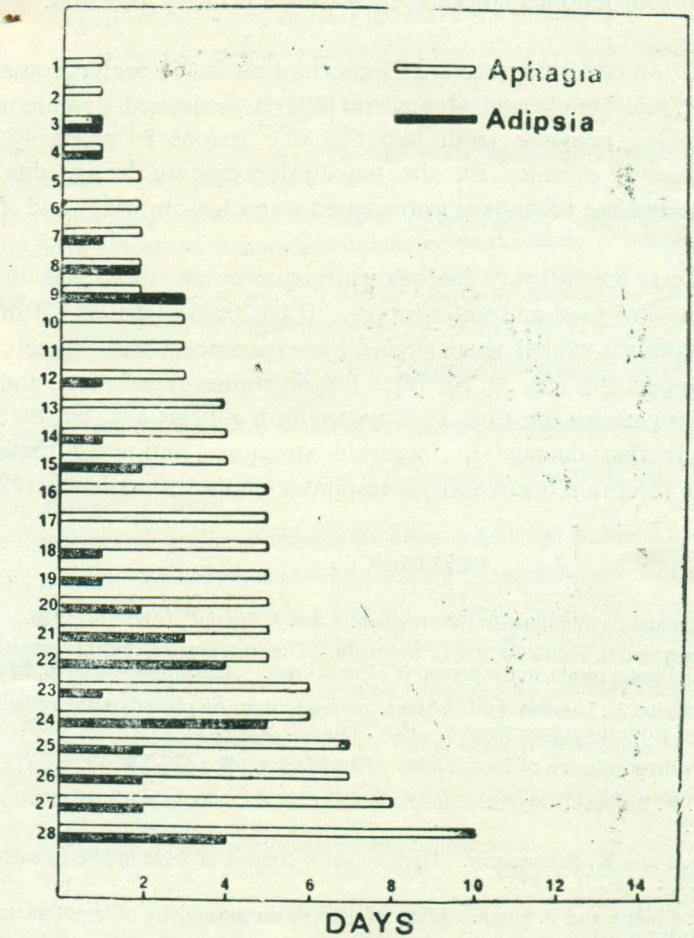

Fig. 4 : Relative durations of Aphagia and Adipsia in the Barbary dove.

been described in which adipsia occurred independently of aphagia (56), but no histological details are given.

Increases in Food and Water Intake: The hyperphagic response in birds has already been discussed (50, 51, 55, 56) and it is clear that there are no instances of increased food intake following ventromedial lessions which can be compared to the mammalian literature. The typical polyuric response of marked diuresis and excessive loss of bodyweight during water deprivation was described by Rogers (85) following complete destruction of the thalamus in pigeons. Ralph (81) lesioned the supraoptic region in hens and produced 2-3 fold increases in water intake, but the exact course of water balance was impossible to assess as the rate of urine output and water content of faeces was not examined. Attempts to distinguish between primary polydipsia and primary diabetes insipidus in chickens by measuring blood sodium levels have been without success (49), but low levels of arginine vasotocin were found in the posterior pituitaries of four lesioned chickens, indicating a primary polyuria.

In doves, inhibition of food intake is an important means of water conservation during water deprivation (66), and Wright and McFarland (101) have devised a technique to determine whether the overdrinking response from hypothalamic lesions is primarily a polyuria or a polydipsia. They found evidence for the polydipsic response being due to increased evaporative water loss and not secondary to increased water loss in urine and faeces.

Conclusion: There are still very few quantitive studies on the effects of brain damage in birds on the control of food and water intake. It has been suggested (2) that organisation of the avian hypothalamus is similar to mammals, but experiments with direct stimulation of the brain do not support this (16, 33, 58, 76). Lesion studies remain few, and the number of animals small, and it is perhaps too early to draw any firm conclusions, but there is suggestive evidence with pigeons that damage to forebrain structures, and nerve tracts connecting the diencephalon with the forebrain, yield aphagic responses similar to mammals (99, 100, 102).

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