

A Persistence of Responding in Hyperstriatal Chicks

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Two-week-old Gallus chicks after lesion of the dorsal midline hyperstriatum accessorium are less easily distracted by novelty from the performance of a trained runway response than those with more lateral or posterior hyperstriatal damage or sham-operated controls. With dorsal midline hyperstriatal lesions, chicks also show delayed acquisition of a passive-avoidance task and an impaired response pattern on a delayed-response task compared to controls. The apparent continuation of the trained responding characteristic of chicks with these lesions when experimental contingencies change is tentatively compared with the behavior of mammals with limbic lesions.

INTRODUCTION

The purpose of this paper is to show that there is a tendency for Gallus chicks with lesions in the dorsal midline hyperstriatum accessorium (DMHA) to persist with acquired behaviors when the experimental conditions change. The behavior of chicks with more lateral or posterior damage changes in a manner similar to sham-operated controls (Cs).

Oades (1976) has reported that changes in the pattern of choice of differently colored food grains were more difficult for chicks with DMHA lesions than for sham or lesioned controls. It was argued that a chick with DMHA lesions is less susceptible to influence by changes in its environment, for by not responding so readily to the less-preferred food color it was taking longer to learn that these grains were an equally acceptable food. In contrast, chicks with ventral midline damage would change their responding to an even greater extent. Supporting preliminary evidence that DMHA-lesioned chicks appeared less likely to pause to examine and react to novelty or to features

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which were not relevant to the learned response or goal of appetitive search was obtained by finding that they responded less than Cs toward pebbles which had been introduced into the food choice.

A hungry chick moving down a straight runway for a reward of food provides a situation into which distracting stimuli may be easily introduced. The function of a novel feature in the following experiments was to distract a chick from the ongoing approach behavior. Four different areas of the avian forebrain were lesioned to investigate whether continued responding was a result of a lesion damage restricted to the DMHA. In addition to the dorsal midline hyperstriatum accessorium, the areas immediately posterior (posterior HA), lateral (DLH), and ventral in the septum were also lesioned.

These results are described for experimental situations in which mammals with damage to the limbic forebrain have been tested. In the extensive literature on hippocampal function (see Kimble, 1968; Jarrard, 1973; Isaacson, 1974 for reviews), many behavioral deficits following lesion damage have been described and interpreted. From these it is tentatively suggested that the behavior resulting from lesion of the DMHA of birds and parts of the limbic forebrain of mammals brings about a tendency for a continued direction for the trained/learned behavior which is not attributable to primary sensory or motor deficits despite changes of the contingencies in the environment. Thus, these data may provide preliminary evidence for an analogy between these structures.

If a lesioned chick shows a shorter runway latency than a control, then this result would be similar to reports that hippocampally lesioned rats (Wickelgren and Isaacson, 1963; Cohen, 1970) are less distracted by novel alleys and novel characteristics (e.g., sandpaper on the floor) added to a runway task. These rats exhibited low running latencies (Wickelgren and Isaacson, 1963) compared to sham-operated controls, and neocortically lesioned animals (Cohen, 1970).

There are several ways in which the results could be influenced by the way in which the experiments were conducted. The age at which chicks are trained and the amount of training that each receives could affect the speed of running and the nature of the response to the novel stimulus. The novel stimulus should not be too frightening or too difficult to observe. The order in which a series of novel stimuli is presented may also affect responsiveness to those stimuli that are presented later. These effects were investigated and are briefly described in the first section. The second section describes the continued approach of DMHA-lesioned birds in the presence of novel stimulation in a runway in contrast to the increased latency for birds with lesions elsewhere in the hyperstriatum. The third and fourth sections describe two other examples of situations where the results may show the persistence of DMHA-lesioned chicks, namely, a passive-avoidance task and on a schedule of differential reinforcement at low rates of responding (DRL-10).

METHODS

Male domestic fowl chicks (Warren sex-link, Southdown, Uckfield) were individually housed in cages 30 x 23 x 45 cm long. On one of the long sides was a pane of frosted glass through which the cage was continuously illuminated by a 40-W bulb. The temperature was maintained at $27 \pm 2^\circ\text{C}$. Food and water were freely available in small Perspex dishes on the floor of the cage, which was covered with a white absorbent paper towel.

Operation

The coordinates for the operation were taken from an atlas prepared for this strain of chick (Andrew, 1973; Andrew and Oades, 1973). The coordinates given are for 2 to 3-day-old chicks weighing about 40 g, but other work at the University of Sussex (unpublished) has shown that they are reasonably accurate in the second week of life. A modified mouse stereotaxic instrument was used (Andrew and Oades, 1973). Bilateral electrolytic brain lesions were made by a constant-current generator delivering 0.1 mA DC for 20 sec with bipolar electrodes. The electrodes were 00 insect pins insulated with Formvar varnish with a diameter of 0.33 mm and an abraded tip of 0.25-mm diameter. The insulated pins with small amphenol connectors were mounted approximately parallel in dental cement with their tips 2 mm apart. These electrodes were implanted with an antero-posterior orientation. All control birds were sham-operated. A sham operation consisted of the delivery of an anesthetic (0.04 ml, 60 mg/ml of Nembutal for an 80-100-g bird) and an incision to the skin, skull, and dura.

DMHA: 14.5-16.5 mm antero-posterior, 1 mm lateral to midline, 4 mm deep.

Posterior HA: 11.5-13.5 mm antero-posterior, 1 mm lateral, 4 mm deep.

DLH: 14.5-16.5 mm antero-posterior, 4 mm lateral, 4 mm deep.

Septal: 14.5-16.5 mm antero-posterior, 1 mm lateral, 5.3 mm deep.

Day of operation: for Section 2, Day 7 of life; for Section 3, Day 5 of life; for Section 4, Day 6 of life.

Histology

After the completion of testing the operated birds were given an overdose of 0.1 ml of Nembutal anesthetic. The blood stream was then perfused with 10 ml of avian Ringer's solution followed by 10 ml of Heidenhain's Susa fixative. After the brain had remained in this reagent in the opened skull for up to 48 hr, after the method of Andrew (1973) and Andrew and Oades (1973), a vertical reference plane for sectioning was cut with the head in the normal operating position in the stereotaxic apparatus. The brain was then removed to alcohol with 5% iodine, dehydrated, and

embedded in wax, and 12- μ m serial sections were cut, stained in cresyl violet, and all were mounted for examination.

Drawings are presented (Fig. 1) of the tissue damage for examples of each type of lesion, approximately every 400 μ m or when a significant change in the extent of the tissue damage occurred in the series of sections which was cut in the planes coincident with those represented in Andrew's stereotaxic atlas of the chick. Further material has been presented elsewhere (Oades, 1976.) The bilateral extent of the lesion is shown by the shaded area; the maximum unilateral extent is shown by the hatched area. The occipito-mesencephalic tract never showed any bilateral degeneration. The extent of the degeneration of the septo-mesencephalic tract is indicated in the figure legend. Degeneration may be seen in these preparations by gliosis where the tract passes through the septum and anterior dorso-lateral midbrain. Further analysis of the extent of degeneration of the septomesencephalic tract following DMHA lesion and discussion of the lack of discernible effect may be found in the report by Oades (1976).

Where coordinates are given, lateral measurement refers to a zero on the valleculla, vertical measurements refer to a zero on the valleculla at a point on the midline between the ear-ear bars, and antero-posterior measurements give 15 mm as the point where the valleculla and the ear-ear midline cross.

Each bird was numbered (and received a letter prefix—e.g., D70). In the diagrams, arranged vertically for each bird, there is a second figure which refers to the slide from which the diagram was taken. The first and last diagrams have an extra figure which represents the first (anterior) and last (posterior) slide in the series where the histological damage was recorded. This allows the length of the lesion to be calculated in the antero-posterior axis (four sections per slide).

Sections 1 and 2

Experimental apparatus. At one end of the runway, 21 x 35 x 130 cm long, was a start box, 21 x 21 x 35 cm high; at the other end was a clear Perspex food dish, 1.4 cm high and 5 cm in diameter. The following were the distracting stimuli used.

Black-and-white food dish (BWFD): The sides were painted black and white for succeeding quarters of the circumference. The size was the same as the food dish used in training.

Black-and-white sides (BWS): Two panels could be added, one to each side of the runway, consisting of three vertical stripes 6 cm wide (black-white-black). These were 35 cm high and placed 35 cm in front of the food dish.

Bell: A loud doorbell situated halfway along but outside the runway was rung for 5 sec after any movement of the chick during the test trial.

Grid: A white grid (mesh, 1 x 1 cm) 10 cm wide was placed halfway down the runway across the floor of white absorbent paper.

Alleys: Halfway down the runway on both sides, alleys 7 cm deep and 18 cm long were exposed. A food dish was located in the corner of both alleys.

Empty dish: An empty Perspex food dish of the same type used in the training trials was located at the end of the runway where food was usually placed.

Grid, hopper, and alley (GHA): With the grid *in situ* and the alleys exposed, a metal food hopper was placed across the farther corner facing the start box, extending into the runway by 1 cm.

Grid, hopper, alley, and BWS (GHAB): In addition to the exposed alleys and the grid, a food hopper was placed immediately beyond the alleys from the start box extending into the runway by 4 cm on either side. On the start box side of the alleys the BWS panels were exposed.

Flag: From the one open alley a cardboard "Y," black on one side and

Fig. 1. Representative diagrams are taken from serial sections of the histological damage (blackened, bilateral; diagonal lines, unilateral) sustained by bilateral electrolysis of (from left to right) the DMHA, posterior HA, DLH, and septum of Gallus chicks. DMHA: The lesions caused some bilateral damage for 1.9-2.4 mm on the A/P axis, with maximum bilateral involvement in the plane of the AC. Damage exceptionally occurred 0.5 mm anterior to the plane of the AC and usually was contained between the planes of the AC and P1C. Bilateral damage affected the hippocampus and the medial half of the HA toward the dorsal surface. The HV received slight bilateral damage close to the ventricles. The TSM shows some bilateral degeneration in its posterior extent. Posterior HA: These superficial lesions lie on the dorso-medial and posterior dorsal surface of the hippocampal/HA area which starts just posterior to the plane of the PC. Greatest bilateral damage occurs about 0.5 mm posterior to the plane of the PC. Damage extends for 2.5-3.5 mm in the A/P axis. Some of the medial HV was also removed. There is no TSM degeneration. DLH: The lesions were placed laterally, well clear of the midline ventricles. There is some medial neostriatal and HV damage anterior to the plane of the PC extending posteriorly and dorsally to the dorso-lateral HA surface. The A/P extent of the lesion varied from 1.6 to 3.2 mm. There is no TSM degeneration. Septum: k; the damage is slight in extent but medial and bilateral in position (0.32-0.62 mm). The TSM degeneration was assessed as ranging from partial to extensive. l: Unilateral damage extended 1.0-1.7 mm equally on either side of the AC, but bilateral medial damage was slight. In all birds the TSM was degenerating. m: Bilateral damage varied in extent from 0.7 to 1.8 mm. The TSM tended to show considerable degeneration.

Abbreviations: A, archistriatum; Aa, archistriatum anterior; Ai, archistriatum intermedium; Ap, archistriatum posterior; Av, archistriatum ventrale; AC anterior commissure; A/P, antero-posterior; CIO, capsula interna occipitalis; E, ectostriatum; HA, hyperstriatum accessorium; HIS, hyperstriatum intercalatus superior; HV, hyperstriatum ventrale; LFB, lateral forebrain bundle; N, neostriatum; OM, occipitomesencephalic tract; PA, paleostriatum augmentatum; PC, posterior commissure; P1C, pallial commissure; PP, paleostriatum primitivum; SL, lateral septum; SM, medial septum; TSM, tractus septo-mesencephalicus.

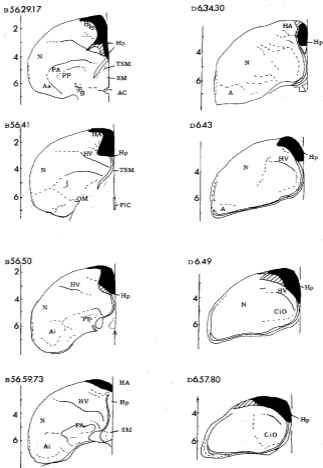
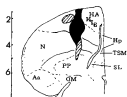
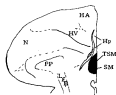


Figure 1

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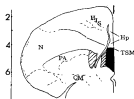
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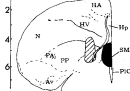
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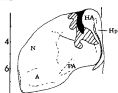
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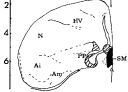
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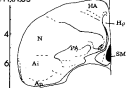


Fig. 1 continued

white on the other and 9 cm long, extended into the runway. It was rotated once every 5 sec by a silent electric motor.

Experimental procedure. The chicks were trained to run from the start box to the food dish at the farther end of the runway. Ten training trials from half the runway length were conducted on Day 5 and ten trials at full runway length were conducted on Day 6, without any distracting stimuli being present. Each chick was placed in the start box for 15 sec before the commencement of a trial. After the door of the start box was raised, food was available for 1 min. Once started, feeding was allowed to continue for 10 sec. Only those that had learned to run consistently in under 5 sec over the last three training trials were used. Four chicks would be run in sequence which gave an intertrial interval for trained birds of about 2-3 min.

The first three experiments investigated the effects of different training regimes on the performance of intact chicks. Experiment a investigated the effect of the number of training trials received in the first week (16 birds) and b investigated the effect of the number of training trials received in the second week of life (14 birds). The training sessions were conducted over 3 days for experiments a and b and over 2 days for c. In experiment c, eight birds had passive experience of the runway before training and eight birds had not. Experiments a and b employed different combinations of distracting stimuli.

The effects of the presentation of the novel stimuli on the latency of chicks to run to feed were examined in experiments d to m. The following birds were tested: d, e, f: 21 DMHA-lesioned and 21 C chicks; g, h: 10 posterior HA-lesioned and 8 C chicks; j: 6 DLH-lesioned and 9 C chicks; k, l, m: 20 septal-lesioned and 22 C chicks.

Tests were performed between Day 9 and Day 13 of life. The order of presentation of the stimuli may be seen in Table 2. Three postoperative control trials established speeds of running comparable to preoperative training. Different postoperative novelty trials were presented in alternation with post-test control trials which reestablished speeds of running for those birds whose behavior had been disrupted by the presentation of a distracting stimulus. Thus, any increases in latency which were observed were not due to overt additive effects of fearful behavior.

The duration spent in the start box after the door was raised and the latency to feed once the chick had started to move were measured. Although the speeds of running after training were stable for any particular chick, between chicks, the speeds varied. Therefore, the scores are rated as +1 if the chick ran to eat within 5 sec of the latency it recorded on a control run with no distracting stimuli, +½ if the chick ate at all during the trial, and 0 if no feeding was recorded.

Section 3

Experimental apparatus. A pale-blue runway 21 x 35 x 130 cm long was used with a start box 21 x 21 x 35 cm high. At the opposite end of the runway was a Perspex dish containing water and resting on a small metal plate which extended 5 cm in front of the dish of water. This plate was connected to a Grason Stadler constant-current electric shock generator E1064GS at 117 V ac 60 Hz by way of two wires, of which one was submerged into the drinking dish and the other was connected to the metal plate. Thus, the chick would complete the circuit by standing on the metal plate and dipping its bill into the water.

Experimental procedure. During the morning of Day 3 of life each chick was placed in the runway for 5 min of familiarization with the novel environment. In the afternoon, after 4.5 hr of water deprivation, they received five training trials in half the runway. On Days 4 and 5, after a 6-hr deprivation of water, they received five training trials without shock on each day in the runway.

At the commencement of a trial the bird was placed in the start box. After 15 sec the door was carefully raised and the bird was allowed up to 60 sec to run to the dish of water to drink. For the first four birds, 10 sec of drinking was allowed. For succeeding birds, a criterion of 4 "scoops" in the water was adopted to prevent the risk of satiation before the 10 trials were completed. The bird had to stand on the metal plate to drink. If the bird retreated off the plate, this prematurely ended the trial.

On Day 6, the first day of testing after the operation, three further nonshocked control trials were performed to ensure maximum running speed before the test (shocked) situation was applied. These control trials were not run on the second day of testing.

Eight birds with DMHA lesions and eight Cs were deprived of water for 6 hr before being tested for learning of the passive-avoidance task. Ten trials were conducted on Day 6 and a further 10 trials were conducted 72 hr later on Day 8. The chicks were run for up to 20 test (shocked) trials in total. If they did not drink for three trials in succession then the session was terminated.

It was decided to use enough current to cause the chick to visibly react with bill withdrawal and a mild jump (padding of the feet). A chick making the electrical connection has at one end well-keratinized feet and at the other a sensitive tongue. It was believed that individual variability of response warranted the use of a current between 0.25 and 0.4 mA, depending on this response. Thus, the current delivered on the first experimental trial may have altered by up to 0.1 mA on the succeeding trial, but thereafter was held constant. The usual value was 0.3 mA. The shock delivered was relatively mild as it was the intention to make the act of drinking more a distasteful and non-rewarding than a highly distressful experience for the chick.

Three measures of the chick's ability to withhold the trained response were taken—the number of experimental trials each bird was given, the latency of a bird running from the start box to the dish to drink, and the number of scoops which were shocked before the bird backed off the metal floor.

Section 4

Experimental apparatus. The pale-brown wooden Skinner box (35 x 35 x 50 cm high) was illuminated by a 40-W lamp resting just above the box, facing down and away from the key lights. The keys were 6 cm from the floor of the box and consisted of two translucent Perspex panels side by side, each exposing an area of 2.5 x 2.5 cm to the inside of the box. In the center of each key was a black dot to encourage the chick to peck the key. A peck on the keys resulted in a simple mechanical contact being made, completing an electrical circuit which raised the keys to make the hopper of chick starter grain available. The circuit allowed 4 sec of access to food before the keys descended. The keys were connected to a time clock so that for 5 sec after the original peck, on a continuous reinforcement schedule (CRF), no further key operation would result in reward. A Wratten deep-red filter (passing a dominant wavelength of 700 nm) was projected onto the keys, which operated as one. A mechanically operated flag cut off the keys' illumination from a Kodak Carousel S slide projector to avoid the glare being directed into the face of the chick.

On the DRL-10 schedule, pecking would not result in the presentation of the food hopper for 10 sec after the termination of a reward (15 sec after the rewarded peck). After each peck within this 10-sec "time-out" a clock recycled so that a further 10 sec would have to elapse with no pecking before a peck would be rewarded. Thus, with the key lights illuminated the task presented was a noncued DRL-10 schedule.

Experimental procedure. Six DMHA-lesioned and six sham-lesioned chicks were deprived of food for 4 hr before each conditioning session. After six 10-min CRF training sessions on Days 3, 4, and 5, the final (seventh) preoperative session was recorded on a multichannel Esterine Angus pen recorder. After surgery on Day 6, a 24-hr period of recovery was allowed before a further 10-min CRF session was recorded to ascertain that the acquisition of the conditioned response and general behavior were not impaired. In all cases no such impairment was observed.

On Day 8, 10-min training sessions on a DRL-10 schedule were commenced. Two sessions were delivered each day, the first and final (tenth) sessions were recorded for detailed analysis. To allow adequate assessment of the histological damage according to the prepared stereotaxic atlas for young chicks, in view of the rapid growth of the chick brain at this age, DRL training was restricted to the second week of life. One hundred minutes of

DRL training is a short period for chicks to learn a complex operant schedule; therefore, the results are analyzed in terms of the frequency of response for the introduction of increased inter-response times (IRTs) after the method of Messent (1973) and Duncan *et al.* (1970), in addition to the more usual measures of the number of responses and rewards as criteria of acquisition of appropriate responding. Both mean and median values for scores are quoted in Table 6 to show the extent and direction of skew in the population. (These simple alternative treatments are designed to show whether all the experimental animals, which have not acquired the criterion for a new DRL schedule, may yet have learned to introduce appropriately longer intervals between responses in comparison with a CRF schedule.

RESULTS

Section 1

Variations of the runway training regime. All the chicks were intact and unoperated birds. There is a slight and nonsignificant trend for a test-trial latency to increase when fewer training trials were received (Table 1). This occurs in the first week of life (BWS, first week, a, $P < 0.14$, two-tailed Mann-Whitney U test), but not in the second week when the latency scores are equivalent (b, BWS). But the latency for birds receiving 10 trials (C10s) may become longer if they receive their training in the second week rather than in the first (BWFD, $P < 0.04$, two-tailed Mann-Whitney U test). The latency for birds receiving 20 trials (C20s) is also slightly longer in the second week than in the first (BWS, $P < 0.1$, Mann-Whitney U test).

The birds of Experiment c, with more experience of the runway, ran faster and were not as distracted by the novel stimuli as those with less experience (BWS, $P < 0.02$, two-tailed Mann-Whitney U test). Where there was an obstacle (e.g., GHAB), the less experienced birds were distracted for a longer period of time ($P < 0.05$, Fischer test).

In summary, the latency increases if training does not occur until the end of the second week, but this effect is less marked if 20 trials are delivered. Less than 20 training trials increases the latency of running for food for birds faced with novel stimuli. Pretraining experience of the runway can bring about a later decrease of latency on novelty trials. Such birds are not so wary of the novel obstacles and will approach these and the food dish quicker.

The order of the presentation of the novel stimuli used here did not influence the latency. However, the nature of the distracting stimuli affects the latency to a varying degree. Thus, with objects (e.g., GHA, GHAB) or open alleys, chicks appear to either run or not run. There is rarely a gradual approach to the food dish (a majority of chicks does not run). These were

TABLE 1
 Means (and Medians for Each Group Over All Tests) of the Rated Latencies to Feed in Each of the Test Runways
 Containing the Novel Stimuli Shown for Birds Which Had Received 10 or 20 Training Trials in Their First
 Week of Life (a) or Second Week of Life (b) and for Birds with and without
 Experience of a Runway before Training (c)^d

	N	BWFD	BWFD	BWS	BWS	GHA	GHAB	Alley	Overall medians
Experiment a	8								
Half-trained in first week (C10s)		0.63*	0.88**	0.75	0.81	0.56	0.50	0	0.63
Wholly trained in first week (C20s)	6	0.67	0.58	1.00	1.00	0.58	0.42	0.17	0.58
Experiment b	7								
Half-trained in second week (C10s)		0.14*	0.14**	0.57	0.64	0.50	0.36	-	0.43
Wholly trained in second week (C20s)	7	0.43	0.43	0.86	0.57	0.29	0.07	-	0.43
Experiment c	8								
With runway experience		(BWFD) 0.56	(BWS) 0.75	(BWS) 1.00***	(GHAB) 0.25	(Empty) 0.75	(Alley) 0.38	-	0.66
Without runway experience	8	0.50	0.69	0.56***	0.25	0.50	0.50	-	0.50

^dLatencies were rated as +1 if chicks fed within 5 sec of the final control trial latency, +½ if chicks fed between 5 and 60 sec, and 0 if chicks did not feed. High scores indicate consistent short latencies to feed. Similar numbers of asterisks show the two groups that are compared.

* $P < 0.04$, two tailed.

** $P < 0.04$, two tailed.

*** $P < 0.02$, two tailed.

TABLE 2
Distractibility Ratings for Experimental and Control Groups on
Separate Novelty Trials in a Runway for Food Rewards^a

Birds	N	BWFD	BWS ₁	BWS ₂	GHAB	Flag	Alley	Empty	Median
d									
DMHA	6	0.33*	0.83	0.83	0.17	0.17	0.67	0.25	0.33 [†]
C	8	0.06	0.56	0.88	0	0	0.44	0.19	0.19
e									
DMHA	7	0.43	0.86	0.86	0.14	0.36	0.64**	0.71	0.64 ^{††}
C	7	0.21	0.86	0.64	0.07	0.14	0.29	0.57	0.29
f									
DMHA	8	0.88	0.88	0.88	0.31	0.38	0.31	0.86	0.86 ^{†††}
C	6	0.33	0.92	0.92	0.17	0.17	0.53	0.50	0.33
g/h									
DLH	6	0.66	0.75	0.83	0.50*	1.00*	0.33	0.42	0.66
C	9	0.44	1.00	0.72	0	0.44	0.39	0.55	0.44
					(Grid)	(Bell)			
i									
Posterior HA	10	0.33	0.42		0.27	0.42	0.13		0.33
C	8	0.51	0.33		0.36	0.51	0.26		0.36
k									
Septal	7	0.88	0.75 [†]				0.38		0.75
C	7	1.00	0				0.75		0.75
l									
Septal	6	0.85	1.00		0.40	0.20	0.30		0.40
C	7	0.80	0.70		0	0.80	0.50		0.70
m									
Septal	6	0.95	1.00 ^{††}		1.00	0.95	0.60		0.95
C	8	0.60	0.40		0.35	0.65	0.65		0.60

^aThe higher the rating, the closer the performance to the trained, short latency. All *P* values given are two tailed.

*,***P* = 0.05.

[†]*P* = 0.002.

^{††}*P* = 0.03.

^{†††}*P* = 0.05 (concordance, *P* < 0.005).

^{††††}*P* = 0.049 (concordance, *P* < 0.001).

^{†††††}*P* = 0.047 (concordance, *P* < 0.01).

always presented last to the chicks. The BWS and BWFD will cause a latency to increase, but few chicks will not pass them. Chicks tend to show a longer latency on the BWFD trial, where the novelty is at the food source, than on the BWS trial. The BWFD may be more difficult to approach than the BWS, which can be passed on the way down the runway to the food goal.

The medians show a negligible difference between C10s and C20s, but as runway experience does increase the chance of running (Experiment c, 0.66 against 0.50), 20 preoperative training trials were conducted by the early part of the second week of life.

Section 2

Runway performance—DMHA-lesioned chicks. With each of the three batches of chicks tested in this section (Table 2) there is at least one test on which Cs showed a significantly longer latency than DMHA-lesioned chicks on a novelty trial.

TABLE 3
Delays in the Start Box of the Novelty Runway^a

Experiment	N	BWFD	BWS ₁	BWS ₂	GHAB	Flag	Alley	Empty	Median for all tests
d	6 Es	1.7	3.8	1.5	21.0	23.7	0.3	7.6	3.8*
	8 Cs	8.2	12.5	1.3	34.8	41.6	4.9	12.5	12.5*
e	7 Es	9.1	3.0	1.6	29.1	2.8	—	5.7	4.4
	7 Cs	6.9	1.0	6.0	29.8	1.0	—	6.6	6.3
f	8 Es	1.0	0.5	1.0	12.0	11.0	0	0.8	1.0**
	6 Cs	8.3	0.5	3.1	26.5	23.0	3.1	11.0	8.3**

^aThis table shows, for experiments d, e, and f, the mean period (seconds) which DMHA and C chicks spent in the start box after it was opened at the beginning of each of seven test trials on the distractibility in runway task. DMHAs tend to spend less time in the start box before running past a novel feature to feed. Similar numbers of asterisks show the two groups that are compared.

* $P < 0.03$.

** $P < 0.041$.

In Experiment d, lesioned chicks ate from the BWFD quicker than did the Cs ($P < 0.05$, two-tailed Mann-Whitney U test). In Experiment e, lesioned birds ran down to feed earlier than did Cs in the presence of open alleys to the side ($P < 0.05$, two-tailed Mann-Whitney U test). In Experiment f the result is not significant, but DMHA-lesioned chicks do eat from the BWFD sooner ($P < 0.1$, two-tailed Mann-Whitney U test). On the BWS trial, Cs (unlike the DMHA-lesioned chicks) took longer to pass the panels than on control trials ($P < 0.016$, two-tailed Wilcoxon matched-pairs test).

In all three experiments there is a similar tendency for DMHA-lesioned chicks to be less distractible than Cs on each test session. A measure of this is the Kendall coefficient of concordance (Siegel, 1956); d, $P < 0.005$; e, $P < 0.001$; f, $P < 0.01$.

On a test trial a chick which runs straight to the end of the runway when the door of the start box is lifted is likely to be a DMHA-lesioned chick, while one which leaves the start box and pauses before passing or approaching the novel stimulus is likely to be a C. Pauses did not always occur by the novel stimulus, for many chicks showed a delay to leave the start box when it was opened. These mean delay periods are shorter for DMHA-lesioned chicks than for Cs (Table 3—d, $P < 0.03$; e, $P < 0.09$; f, $P < 0.041$). This supports the results above, which showed that there is a greater probability that DMHA-lesioned chicks will run fast down the runway to the food dish.

Posterior-HA-lesioned chicks. The latencies shown by chicks with lesions in a more posterior region of the hyperstriatum in the midline are broadly similar to those shown by sham-operated birds (Table 2). The removal of some tissue in approximately equal amounts from different sites in the brain does not produce a lack of distractibility like that seen with DMHA-lesioned chicks.

DLH-lesioned chicks. In the presence of the flag and GHAB, DLH-lesioned chicks continue to run to feed significantly more often than sham-operated Cs (Table 5, $P < 0.05$, Fischer test). However, there is no tendency across all the distractibility trials for DLH-lesioned chicks to run to feed more rapidly than Cs ($P < 0.05$ – 0.1 , Kendall coefficient of concordance; the Mann-Whitney U test is not significant).

Septal-lesioned chicks. In the first (k) of the three batches of chicks tested, septal-lesioned chicks clearly show a shorter latency than Cs (Table 2, $P < 0.002$, two-tailed Mann-Whitney U test). It was shown in Section 1 that BWS were very effective in decreasing the speed with which chicks ran to the food rather than causing many not to approach the dish. This effect carries over to the control trials conducted after the BWS were removed ($P < 0.003$, two-tailed). The results of Experiment 1 show that unilateral forebrain damage does not affect the latency to run, for there are no overall significant differences between septal-lesioned and control birds. However, two birds

TABLE 4
Approach to Colored Foods in a Runway^a

Birds	N	Trial 2 (G)		Trial 3 (R)		Trial 4 (G/Y/R)	
		Approach	Peck	Approach	Peck	Approach	Peck ^b
DMHAs	16	14	0	14	4	15	7
Cs	13	9	0	9	0	10	3
DLHs	6	4	0	5	0	4	3
Cs	9	3	0	7	0	4	3

^aThis table shows the number of DMHA-lesioned, DLH-lesioned, and control chicks which approached (within 5 cm) and pecked colored foods on three presentations at the end of a runway: green (G) alone, red (R) alone, and green, yellow, and red (G/Y/R) together.

^bY food.

from this poorly lesioned group showed large and bilateral medial septal damage and degeneration of the tractus septomesencephalicus. These two birds were not easily distracted by novel features (median latency was rated at 0.9 per trial for both birds).

In experiment m, septal-lesioned birds tended to show a shorter latency than did Cs on the test trials (e.g., BWS, $P < 0.03$, two-tailed; grid, $P < 0.08$, two-tailed Mann-Whitney U test). A significant coefficient of concordance ($P < 0.015$) might seem to suggest that septal-lesioned birds showed a shorter latency, but a Mann-Whitney U test proved nonsignificant ($P < 0.114$).

DMHA- and DLH-lesioned chicks and colored food. After the presentation of the novel stimuli described above, five trials were performed using combinations of colored foods at the end of the runway with 16 DMHA-lesioned, 6 DLH-lesioned, and 22 Cs. All birds had been trained to run to yellow (Y) food in under 5 sec. All birds showed this short latency on the first and fifth trials with Y food alone. On the second trial, green (G) food was presented, on the third, red (R) food was presented, and on the fourth, G, Y, and R foods were presented in a line across the runway. Table 4 shows that over the three tests, more DMHA-lesioned chicks approach (but do not eat) the foods than do Cs ($P < 0.05$, χ^2 test). No chicks fed immediately when the three foods were presented on Trial 4. Chicks only pecked Y food on Trial 4. Three foods were presented on Trial 4. Chicks only pecked Y food on Trial 4. Two chicks (from lesioned and C groups) pecked R food on Trial 3 and no chicks pecked G food. DLH-lesioned chicks showed no significant differences in comparison with the sham-operated Cs.

TABLE 5a

Passive-Avoidance Performance after DMHA and TSM Damage^a

Birds	Number of trials receiving shocks	Latency on trial (sec)		Total number of scoops	
		5	10	Days 1 and 2	Day 2
DMHAs	18.9(20)*	5(4.5)	9.6(5.0)**	54.7(55)	31.5(33) ⁺
Cs	11.6(12)*	12.2(4.5)	60(21.5)**	30.3(28)	11.5(12.5) ⁺

^aThis table shows the results for eight DMHA-lesioned chicks and eight Cs on a passive-avoidance task where the reward was water and the electric current delivered was 0.35 mA. The mean and the median (in parentheses) values are given for the three measures of continued approach to the water dish: number of test trials where electric shock was received, latency to drink, and the number of scoops of water taken. * $P < 0.002$, two tailed; ** $P < 0.028$, two tailed; ⁺ $P < 0.024$, two tailed. (Two similar symbols show the two figures that are compared.)

TABLE 5b

Passive-Avoidance Performance after DMHA and TSM Damage^a

Birds (DMHA)	TSM degeneration			Behavior score, number of shocked trials
	Severe	Moderate	Slight	
D74	0			17
D73	X			20
D69			X	20
D70			X	20
D71			X	20
D72			X	20

^aThis table gives estimates for the degeneration of the tractus septomesencephalicus for chicks with DMHA lesions and one of their passive-avoidance scores. There is little evidence to support a correlation between degeneration and recorded behavioral deficits. 0 represents damage to the main part of the structure; X represents damage to the posterior borders of the structure.

Section 3

Passive-avoidance task. From the data in Table 5a it may be seen that the control chicks acquired the ability to withhold the trained response to run down the alleyway to drink, whereas the DMHA-lesioned chicks continued to approach the water dish and to drink.

Cs drank on significantly fewer trials and obtained significantly fewer shocks than did DMHA-lesioned chicks ($P < 0.002$, two-tailed Mann-Whitney U test). By the tenth (final) trial on Day 1, Cs showed a significantly

increased latency ($P < 0.028$, two-tailed Mann-Whitney U test), although this difference did not prove significant on earlier trials. The latency would be expected to increase as the tendency to avoid shock supercedes that to drink (i.e., to continue with the trained response). On the second day (a further 10 trials), Cs took fewer scoops of water ($P < 0.024$, two-tailed Mann-Whitney U test) than did DMHA-lesioned chicks (scored for 6/8 Cs and 6/8 lesioned birds). This effect was also present on Day 1, but as a nonsignificant tendency.

Section 4

DRL-10 task. Cs show a higher frequency of rewards and a significant increase in the number of IRTs with values between 10 and 18 sec on the last DRL-10 session. This may be taken as a sign that intact chicks are starting to introduce pauses in between responses appropriate for a DRL schedule. On the last session, DMHA-lesioned chicks show fewer pecks with an IRT of more than 10 sec than would be expected from a random distribution of the number of responses emitted after a reward. There is a tendency for DMHA-lesioned chicks to peck more than Cs on the first session, but by the end of training they receive fewer rewards than expected (Table 6).

Median pecking rates on the CRF schedules are similar between pre- and postoperative sessions (4.1-4.9/min for Cs, 6.7-6.8/min for lesioned chicks).

TABLE 6
Pecking and Reward Rates on the First and Tenth DRL Sessions^a

	First DRL session			Tenth DRL session		
	Observed		Expected rewards	Observed		Expected rewards
	Pecks	Rewards		Pecks	Rewards	
DMHAs						
Mean	19.8*,** (18)	1.2 (1.3)	0.6 ⁺ (0.5)	7.9** (7.4)	1.5 (1.5)	1.7 ⁺ (1.8)
Cs						
Mean	12.7*,*** (12.1)	1.5 (1.7)	1.2 (1.1)	5.8*** (5.9)	2.1 (2.2)	1.8 (1.7)

^aThis table compares the means and medians (in parentheses) for the observed number of pecks and rewards which DMHA-lesioned ($N = 6$) and sham-operated chicks ($N = 6$) scored on their first and last DRL sessions. The expected reward rates have been calculated from the formula $\bar{X} = T/N$, where X = number of expected rewards, N = number of observed pecks, and T = total time of the session less the time spent feeding (after Duncan *et al.*, 1970).

* $P < 0.06$, two-tailed Mann-Whitney U test.

** $P < 0.05$, two-tailed Wilcoxon matched-pairs test.

*** $P < 0.05$, two-tailed Wilcoxon matched-pairs test.

⁺ $P < 0.05$, two-tailed Wilcoxon matched-pairs test (decreased proportion of observed: expected rewards). (The two symbols show the two figures compared.)

Table 6 shows the observed number of pecks delivered and rewards received for Cs and DMHA-lesioned chicks on their first and last DRL sessions. There is a tendency for lesioned birds to peck more than Cs on their first session ($P < 0.06$, two-tailed Mann-Whitney U test), but the number of pecks delivered on the last session is approximately equal between the two groups of birds. Thus, DMHA-lesioned chicks have slightly decreased the number of pecks which they delivered during the DRL training with respect to Cs. It should be noted that both groups of birds decreased the total number of pecks which they delivered between the first and last tests ($P < 0.05$, two-tailed Wilcoxon matched-pairs test).

At the same time that DMHA-lesioned chicks are decreasing their rate of response they make significantly fewer rewarded responses than expected by the last session if compared with the first session ($P < 0.05$, two-tailed Wilcoxon matched-pairs test). No such decrement in success is found with the performance of Cs. Indeed, more Cs continue to achieve a higher frequency of rewards than expected and this frequency is higher than that for the DMHA-lesioned chicks.

If the chick learns to withhold a response appropriately for a DRL schedule then the pattern of responding during the session will become nonrandom. Therefore, the calculated number of rewards might be expected to differ from the observed number on the last session. However, a comparison of the performance on the last with the first session is also essential, for chicks may not be responding at random on the first session (e.g., the delivery of burst responses—groups of pecks with IRTs of about 2 sec followed by a longer interval). Hence, such birds might be generating more rewards than expected from calculation on their first DRL session. The data show a non-significant tendency for an initial nonrandom response pattern for both groups of birds.

Messent (1973) calculates that if the delivery of pecks varies from 40 to 140 in a 10-min session, then the number of rewards which are generated will only rise from 15-22. Thus, a DRL schedule is very poor at discriminating a difference in performance on the basis of the number of rewards achieved in a session. Therefore, it is important to investigate whether there is a spacing of responses more appropriate to a DRL schedule or, more specifically, to examine the frequency distribution of IRTs (Fig. 2).

If a reward occurs at random intervals, then the frequency of responses should conform to a negative exponential distribution. The average interval (\bar{X}) and the total number of intervals (N) were therefore substituted in the formula $N[(e^{-x/\bar{X}}) - (e^{-y/\bar{X}})]$ to find the expected number of IRTs in any class interval of IRT from x to y (e.g., 2 to 4 sec) under the hypothesis of randomness (see Fig. 2 and Duncan *et al.*, 1970). Differences between the observed and the theoretical distributions might be found for different interval classes if a degree of learning (or extinction) was occurring. Intervals between

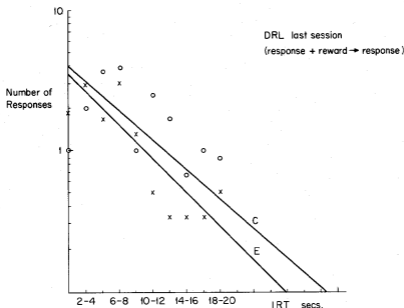


Fig. 2. Last DRL session. This is a semilogarithmic plot. Time intervals (IRTs in seconds) between a rewarded peck and the next peck are shown along the horizontal axis. The number of such intervals in the total score of pecks is shown along the vertical axis. The horizontal axis is measured in increasing IRTs of 2 sec. The vertical axis is a measure of the number of pecks occurring in these intervals. E, the expected number of pecks for DMHA; C, the expected number of pecks for Cs; x, the observed number of pecks for DMHA; o, the observed number of pecks for Cs. To calculate the expected number of pecks the following formula was used. $N(e^{-x/T}N - e^{-y/T}N)$, where N = observed number of pecks during the session; x and y = the time interval between pecks, e.g., 2-4, 4-6, 6-8 sec; and T = total time of the session less the time spent feeding (after Duncan *et al.*, 1970).

rewarded pecks have been considered separately from intervals after non-rewarded pecks, as information would be more easily acquired by a chick from a rewarded response. Therefore, the length of a succeeding interval is that most likely to show any change reflecting learning appropriate to a DRL-10 schedule.

There is a scatter of IRTs on the first session which do not significantly vary from expected values, irrespective of a response being rewarded or not.

Considering the two groups of birds separately, the DMHA-lesioned group responds with fewer IRTs longer than 10 sec than expected on the last DRL session. A comparison of this figure for each chick (negative with respect to the expected figure) with the performance of the first DRL session gives a

significant difference from Cs ($P < 0.042$, two-tailed Mann-Whitney U test. Cs > DMHAs: the observed less the expected number of IRTs greater than 10 sec, sessions 1-10). Thus, DMHA-lesioned chicks tend to continue to deliver pecks with shorter IRTs than Cs.

An examination of the IRTs of Cs on the last session after a peck has been rewarded shows that, with IRTs of up to 8 secs (e.g., 0-4, 4-8), the observed and the expected values do not differ significantly. But a significantly higher number of IRTs of 8-18 sec is observed than would be expected ($P < 0.042$, two-tailed Mann-Whitney U test).

As the data for DMHA-lesioned chicks deviates slightly lower than expected at the time that Cs deviate higher than expected, it may be concluded that some degree of learning of the DRL task has been achieved by Cs and not by the lesioned birds. The measure of learning that is taken is the greater ability of Cs to increase the pause in responding to the keys after receiving reward. Messent (1973) similarly reports that Cs were learning a DRL-10 schedule faster than were septal-lesioned chicks.

DISCUSSION

All the novel stimuli used tended to distract Cs and thus increase their latency to feed in a runway. Section 1 showed that the differences which were found between the experiments cannot be accounted for by the differences in the behavior conditioned by the experimental procedure alone. DMHA-lesioned chicks maintain a lower latency than sham-operated Cs, DLH-lesioned, and posterior-HA-lesioned birds in the presence of novel objects (e.g., alleys, BWS, grid).

DMHA-lesioned birds will approach and pass novel features and will approach food of a novel color, but will not feed from strange-colored foods, whereas Cs tend to remain in or close to the start box. Septal-lesioned chicks, like DMHA-lesioned birds, tend to be less easily distracted than Cs. For each of the batches of chicks used there was a significant tendency for Cs to show an increased latency to feed, whereas DMHA-lesioned chicks were not distracted by those novel features for as long as the Cs were. (Kendall coefficient of concordance across the three batches, 21 DMHA-lesioned, 21 C chicks, $P < 0.015$, two-tailed.)

The brain areas which contribute to this effect are largely hyperstriatal, toward the midline dorsal to the septum. They do not extend far anterior in the forebrain (Oades, 1976) nor do they extend far posterior in the hyperstriatum. Thus, the posterior-HA-lesioned and to some extent the DLH-lesioned birds serve as useful controls, for lesions in these areas do not render chicks significantly less distractible than sham-operated birds. Limited

lesions in the septal area resulted in these birds showing behavior similar to the DMHA-lesioned birds. However, the variable site (lateral-medial septum) and the often small size may account for the absence of a clear result emerging from the present data. The tendency for septal-lesioned chicks to continue with the trained response despite the presence of novel features in the runway requires support from further experiments.

DMHA-lesioned chicks can see the novel changes to the runway but do not pause so that they may examine, identify, and react to the novel feature to the same extent as Cs. This would be necessary for an alternative pattern to supercede the ongoing behavior (e.g., return to the start box). One of the major afferent visual pathways (thalamofugal) terminates in the hyperstriatum closely anterior and lateral to the site of the DMHA. It is believed that the DMHA lesion has not consistently damaged a large proportion of this pathway, but the precise position for this pathway in the chicken is currently being investigated. It is not clear what the role of this pathway in birds may be (Karten *et al.*, 1973; Hodos *et al.*, 1973), although V. Maier (personal communication) has found a specific pattern deficit following lesion. No published lesion study on the hyperstriatum reports gross visual deficits (Stettner and Schulz, 1967; Benowitz, 1972; MacPhail, 1971). The present results and those of Oades (1976) with a colored-food-choice task suggest that visual attention and related learning effects cannot be distinguished on the basis of these tests and that one of these roles may be implicated. The eliciting of an "orienting response" with hyperstriatal stimulation (Cohen and Pitts, 1967) could implicate a tectal control mechanism, but no deficits were observed with DMHA- or DLH-lesioned chicks on an optomotor task (Oades, 1975). The present results support the contention of Oades (1976) that such lesions make the chick less susceptible to changes in the environment, although one may not be able to distinguish between the possible mechanisms yet from these experiments.

The deficit of a reaction to novelty in a runway is similar to reports for hippocampally-lesioned rats (Wickelgren and Isaacson, 1962; Cohen, 1970). To show that this characteristic deficit of a DMHA lesion may have more than a coincidental similarity with damage to a mammalian limbic structure, two further tasks, on which rats with limbic lesions record behavioral deficits, were presented to DMHA-lesioned chicks.

For the first, a passive-avoidance task, DMHA-lesioned chicks retained the trained response to run to the water dish despite receiving a contingent electric shock, whereas the Cs did not continue to run. This was demonstrated over an experimental period of 10-20 trials according to three criteria: the number of drinking trials, the latency to drink, and the number of scoops of water taken. The results for these chicks are similar to those reported for hippocampally lesioned rats (Kimura, 1958; Kimble, 1963). Further, it is reported (Van Hoesen *et al.*, 1973; Stevens and Cowey, 1973)

that this lack of ability to withhold the trained behavior may be found in rats with lesions of the postero-ventral hippocampus and entorhinal cortex rather than of the more dorsal region.

Passive-avoidance deficits have also been reported for a range of septal mammals (Fried, 1972). There are close reciprocal connections with the hippocampus and there is said to be a close parallel with the mammalian and avian septum in anatomy and function (Ariens-Kappers *et al.*, 1936; Karten, personal communication, 1976). But no investigation has, as yet, been undertaken of the basis for hyperstriatal and septal roles in avian behavior, as have been attempted for the hippocampus and septum of mammals (Dalland-Evans, 1975). However, it is of interest to note that at this stage, although the site of the lesion is slightly more in the "hippocampal" (Van Tienhoven, 1962) than the hyperstriatal part of the DMHA, it seems that the behavioral deficit was not caused by large-scale degeneration of the tractus septomesencephalicus (Table 5 and Oades, 1976). The bulk of this tract passes through the septum, but slightly anterior to the bulk of the lesion.

Beatty *et al.* (1973) suggested that to learn to leave the drinking area may reflect an increase in fear and/or frustrative behavior as a result of receiving the electric shock (or surprise on novelty trials). It is possible that to return down the runway may reflect an increased responsiveness to positive reinforcement, now to be found in the security of the start box. This characteristic is one which has been attributed to the intact hippocampus and closely related structures (Beatty *et al.*, 1973). If hippocampal lesions brought about a change in responsiveness toward positive reinforcement, it seems unlikely that hippocampal mammals would be expected to acquire a DRL schedule as rapidly as intact mammals in the absence of CRF training (Isaacson, 1974).

Throughout testing all birds gave a mild jump when shocked, which suggests that the lesion does not interfere with the perception of the electric shock. No components of fearful behavior or signs of placidity were observed in DMHA-lesioned chicks in this experimental situation, as have been reported for amygdaloid mice (Slotnick, 1973) and archistriatal pigeons (Zeier, 1974).

It could be suggested that the DMHA and hippocampal lesions have brought about an increased responsiveness to the conditioned stimulus. However, this would not be consistent with reports that hippocampal mammals can acquire a passive avoidance if they are given experience of the electric shock prior to hippocampal lesion (Wishart and Mogenson, 1970).

As yet, there are no data from experiments using different training regimes to demonstrate that the DMHA lesion does not affect the responsiveness of chicks on these tasks, although a simple effect without reference to the animals' experience would seem unlikely from a parallel with hippocampally lesioned mammals. If DMHA-lesioned chicks showed differences in responsiveness from Cs these should be observed in the approach behavior

shown by these two groups toward food colored differently from the food color experienced; but this has not been recorded in home-cage (Oades, 1976) and runway situations (above). It might also be expected that if lesioned birds were more sensitive to fear and frustration the frequencies of components of behavior and vocalization would show marked changes in situations which allegedly evoke such behavior in intact controls (e.g., absence of expected food, introduction of a novel object). No marked increases or decreases were recorded in these situations, although "peeps" and "twitters" showed slight nonsignificant increases.

In addition to the runway-distractibility and passive-avoidance situations there is a third task on which DMHA-lesioned chicks and limbically lesioned mammals show a deficit. On a DRL-10 task, Cs were able to introduce more IRTs longer than 10 sec by the last training session than DMHA-lesioned chicks and more than would be expected from a random distribution of pecks. At the same time, lesioned chicks were showing a tendency to deliver fewer responses with IRTs longer than 10 sec than expected and to be pecking less than the relatively high number recorded on the initial session. Cs were learning to space their responses with longer intervals after a reward and were also achieving an increased number of rewards in contrast to lesioned chicks.

An emphasis is placed on the spacing of pecks rather than on the number of rewards, for the chicks of this preliminary study show a certain variability in responding. Zeier (1971) also remarked on individual variability while training archistriatal pigeons on a DRL task. His pigeons did not achieve a stable response rate until they had experienced four times as much of the task as the chicks reported here (20-min sessions for 2 weeks). More importantly, the DRL schedule is very poor at discriminating a difference in performance on the basis of the number of rewards achieved per session. Nonetheless, DMHA-lesioned chicks do receive significantly fewer rewards than would be expected from random pecking on the last session.

Much of the high initial pecking rate of DMHA-lesioned chicks showed short IRTs. Their total number of pecks declines over the next 10 sessions. The spacing of pecks does not improve so that the pecks which are emitted on the last session also have a short IRT. It would seem that if these chicks are delivering fewer pecks than earlier in their training they are delivering pecks with short IRTs in a nonrandom manner. An example of this pattern of responding would be the "burst" responses described by Sidman (1956) as those with IRTs of 2 sec or less. Zeier (1974) reports an initial increase of such responses from his archistriatal pigeons on a DRL-10 task.

Isaacson (1974) states that the deficit of hippocampal-lesioned mammals on DRL schedules has two aspects. There is a reduction in the ratio of reinforced responses to the total number of responses (which is shown by the DMHA-lesioned chicks in the present study). He also points to the exaggerated

rate of response of hippocampal-lesioned mammals. However, this effect does depend on the response rate shown under CRF conditions. Nonneman and Isaacson (1973) reported that hippocampal rats with low response rates under CRF conditions showed a much reduced increase of their response rate. In comparison, chicks (which have a low rate of response compared to rats), if they sustain a DMHA lesion, show a nearly significant higher response rate on their first DRL session. It should also be pointed out that irrespective of the species of experimental animal there is disagreement as to what is observed when hippocampally lesioned mammals are faced with a DRL task. A marked over-responding has been recorded by Rabe and Haddad (1967), Orbach *et al.* (1960), and Niki (1967), yet little or no impairment is reported by Ellen *et al.* (1964), Mishkin (1954), Correll and Scoville (1965), and Ellen and Aitken (1970).

There are only two other reports on the behavior of birds with hyperstriatal lesions on interval schedules known to the author. Reynolds and Limpo (1965) trained pigeons pre- and postoperatively on a multiple schedule of fixed interval (FI) and fixed ratio (FR) components. After lesion, their birds retained a normal and a sustained high rate of responding, characteristic of performance on the FR component. However, on the FI component there were high rates of responding which were suddenly broken by pauses. Performance on the FR component shows that hyperstriatal lesions do not impair the pecking response, render food an inefficient reinforcer, or affect the sensory discrimination. The abrupt breaks on the FI component show that this effect is not simply due to a disinhibition of responding. MacPhail (1971) may have caught the edge of this system with lesions in the ventral hyperstriatum and the more dorsal intercalatum superior. The hippocampus was left intact (unlike the present study.) Lesioned birds showed a slight deficit in inhibiting their response during the negative component of a variable interval red-green discrimination task. These data suggest that damage was sustained to the edge of an area which at the descriptive level is similar to the DMHA-lesioned chicks and mammals with slight hippocampal disruption (Clark and Isaacson, 1965).

Ariens-Kappers *et al.* (1936) and more recent unpublished work suggest that there is a structural and functional parallel between the mammalian and avian septum. Messent (1973) reported deficits in the abilities of septal chicks to space their responses appropriate to a DRL-10 schedule. This parallels reports from septal mammals (Ellen *et al.*, 1964; Carey, 1968). The hippocampus and septum of mammals are closely and functionally connected. Lesion of these structures appears to give rise to similar behavior on a DRL schedule. It is of interest that lesion of the avian DMHA and septum should also give rise to similar behavior on a DRL schedule, although as noted above, a possible functional similarity between these structures remains to be investigated.

Finally, it should be emphasized that the experiments reported here describe deficits which occur after DMHA damage, in the behavior over which the avian forebrain exerts control. They show that such birds are likely to continue with trained behavior when changes in the environment would suggest that changes of behavior would be appropriate. These results support the avoidance (Benowitz, 1972) and reversal learning deficits (Stettner and Schulz, 1967; Benowitz and Lee-Teng, 1973) reported from birds with damage to the hyperstriatum accessorium. They are suggestive of analogy with mammalian hippocampal studies with these particular tasks, but without investigation of the factors responsible the possibility that there is a common function cannot be confirmed (Campbell and Hodos, 1970).

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