MECHANISMS OF INFORMATION PROCESSING IN THE CHICK

Thesis by

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In Partial Fulfillments of the Requirements

for the Degree of

Doctor of Philosophy

California Institute of Technology

Pasadena, California

1973

(Submitted October 11, 1972)

Acknowledgments

I wish to thank Professor R. W. Sperry for useful suggestions, assistance with manuscripts, and for encouraging the pursuit of one's own interests amidst an atmosphere of diverse interests and free exchange of ideas.

I also thank Professors D. McMahon, J. Olds, R. Russell and A. van Harreveld for reviewing this thesis.

I am grateful to Drs. Geoffrey Magnus and Evelyn Lee-Teng, with whom I worked closely during my first three years, as well as other members of this laboratory from whom I have derived many ideas. Part I of the thesis was done in collaboration with Dr. Magnus, while Experiment III was done together with Dr. Lee-Teng.

I wish to thank my parents and my wife Brooke for encouragement and support (moral and otherwise) during the long course of my education. Brooke also assisted in the preparation of several parts of the thesis.

This work was supported by the California Institute of Technology and by the National Institutes of Health, Public Health Service (Grant MH-03372 awarded to Dr. R. W. Sperry and Training Grants GM-0086 and GM-02031).

ABSTRACT

The kinetics of neurobiological phenomena involved in the formation of a memory trace were examined in the first two experiments. Transcranial subconvulsive current was administered to large groups of chicks at various intervals following an aversive training experience. The resulting retrograde amnesia data indicate that immediately upon training a metastable mnemonic process becomes activated (STM) which then remains at a constant intensity. Within a minute STM induces a more permanent form of memory (Pre-LTM) to grow at a steadily declining rate, apparently as some restricted neural substrate of memory becomes exhausted. STM may continue to function as behaviorally accessible memory for the next few hours, during which time the behaviorally latent Pre-LTM trace undergoes a subsequent transition into permanent memory. An investigation of the retrograde amnesia resulting from a sequence of two training-current sessions provides support to the existence of these mechanisms and indicates that fractional engrams summate together in a simple fashion.

To examine the participation of different cerebral structures in information processing, chicks having various telencephalic lesions were tested in either a passive avoidance task or an appetitive discrimination. The hippocampus was found to be involved in reversal but not acquisition of the pattern discrimination, and in acquisition but not retrieval of the passive avoidance task. On the other hand, the amygdala seems to be important both for retrieval and acquisition of passive avoidance conditioning, but only for early stages of acquiring

the pattern discrimination. Frontal ablations resulted in a deficit to retrieval but not to acquisition of passive avoidance conditioning, and caused some motivational changes independent of chicks' learning ability in the performance of the appetitive task. A comparison of these results with those following lesions in homologous mammalian limbic system structures suggests that the information processing of both classes is based upon cerebral mechanisms which have remained unchanged despite their divergent evolution.

Mechanisms of memory processing and interhemispheric transfer were further studied in chicks having extensive unilateral ablations of the dorsal telencephalon, a region critical for visual learning. Although chicks were able to acquire a passive avoidance response equally well using either the eye ipsilateral or that contralateral to the surgery, subsequent extinction conditioning could be learned only through the ipsilateral eye. Since retinal projections cross completely at birds' optic chiasma, these results suggest that anatomically distinct systems, one bilaterally represented, the other lateralized, respectively mediate the acquisition and extinction of the aversive response. An inability of lateralized memory to transfer through the commissures is indicated by the absence of interocular transfer for monocularly learned extinction.

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Introduction

The means by which an organism modifies its behavior on the basis of relevant experience remains one of the most important yet elusive problems in neuroscience. The scope of the problem becomes apparent when one considers the complexity of the neural organization underlying information processing and the probable subtlety of any changes in this organization that would accompany learning. And to further complicate the matter, the classical work of Lashley (1950) has indicated the difficulty of localizing a memory trace on even a relatively gross level.

Yet despite these obstacles, considerable contributions have been made towards understanding physiological bases of learning and memory. Among the approaches which have been successfully applied, two that have been adapted in the present studies include investigating the involvement of various brain structures in memory storage and retrieval and studying the effects of either electrical stimulation or of various biochemical agents on learning. While the former method attempts, in a sense, to establish cerebral subroutines used in information processing, the latter is directed towards gaining insight into neurobiological phenomena underlying memory storage.

The idea that "complete information about the internal organization of the nervous system of a lower vertebrate would be of great assistance as our guide through the complicated structure of the central nervous system of mammals and of man" (van Gehuchten, 1895) led C. J. Herrick to devote much of his life towards studying the detailed

anatomical organization underlying the salamander's behavior. In an analogous vein, it would seem that fundamental mechanisms underlying learning and memory can be profitably investigated using vertebrates somewhat less sophisticated than mammals. Towards this end, the work described in this thesis examined mechanisms of memory storage in the chick, an animal which has an effective yet relatively restricted capacity to store and retrieve memory, and whose memory is a blank slate upon hatching.

In order to investigate the nature of neurobiological phenomena underlying memory storage, consolidation experiments have examined the effects of electrical or chemical brain stimulation on learning (see review: McGaugh, 1966; McGaugh & Dawson, 1971). These studies first developed from the observation that memory is laid down over a relatively long period of time, as indicated by the retrograde amnesia of human patients suffering brain damage (Russell, 1969). Subsequent systematic investigations in animals have shown that a semi-permanent memory trace is formed over a period of seconds (Chorover & Schiller, 1965; Lee-Teng & Sherman, 1966) or minutes (McGaugh, 1966) after training. Various inhibitors of protein or RNA synthesis or a chemoconvulsant drug can subsequently prevent the formation of long-term memory if given within several hours of training (Agranoff et al., 1967; Barondes & Cohen, 1967; Cherkin, 1966). On the other hand, an apparently independent short-term memory trace for an experience can be selectively impaired without affecting long-term memory (Kesner & Conner, 1972; Lee-Teng et al., 1970), and vice versa (Geller & Jarvik, 1968).

The first experiment described in Part I was directed towards clarifying the nature and interrelationship of the different mnemonic processes indicated by the multiplicity of these amnestic effects. Use of chicks' one-trial learning paradigm is particularly suitable for quantitatively investigating this issue, due to the availability of large numbers of genetically similar subjects and the simplicity of the experimental design (Lee-Teng & Sherman, 1966; Cherkin, 1966). In the second experiment of Part I, the manner in which fractional memory traces summate together was examined. This was done to gain insight into mechanisms which allow an animal to integrate a sequence of similar experiences, as well as to examine hypotheses concerning the mnemonic processes suggested by the results of the first experiment.

Traditionally, the intelligence of birds has been thought to be predicated upon a neuroanatomical organization quite different from that of mammals (Ariens-Kappers et al., 1936; Kuhlenbeck, 1938; Stettner & Matyniak, 1968). In the view of Ariens-Kappers, Huber & Crosby (1936), while the mammalian neocortex represented a proliferation of pallial portions of the reptilian telencephalon, the greater part of the avian cerebrum was thought to have evolved from an expansion of the basal ganglia (and was thus considered to be a "striatal" derivative). However, more recent embryological (Källén, 1962) and histochemical (Baker-Cohen, 1968) studies have demonstrated that only the most ventral portions of the bird's telencephalon are homologous with the basal ganglia; the preponderance of avian cerebral components once thought to be "striatum" are in fact derived from the embryological cell columns which give rise

to the neocortex in mammals (Källén, 1962; Nauta & Karten, 1971; see also Appendix A). Yet in contrast to the familiar laminated arrangement of neocortical units in mammals, the homologous cells in the avian brain appear to be arranged in a nuclear fashion (Karten, 1969; Nauta & Karten, 1971) having seemingly less convoluted interconnection (Jones & Levi-Montalcini, 1958) than in the mammalian brain. It would therefore seem possible that the pattern of cerebral interrelationships underlying memory processes might be more comprehensible in birds than in mammals.

Much attention has recently been directed towards examining the roles that sensory-specific components of the avian telencephalon play in information processing. Studies by Karten and co-workers indicate that ascending visual projections to birds' cerebrum are organized in a fashion similar to that of mammals (Karten, 1965; Karten & Hodos, 1970; Karten & Nauta, 1968). Furthermore, visual structures that are homologous in the two classes seem to play similar roles in information processing (Hodos & Karten, 1970; Hodos et al, in preparation; Diamond & Hall, 1969; Casagrande et al., 1972). Ascending auditory (Karten, 1969) and somatosensory (Delius & Bennetto, 1972) projections have also recently been traced in the telencephalon. However, with a few exceptions (e.g., Stettner & Schultz, 1967; Zeier, 1971), the functions of portions of the avian telencephalon which do not receive direct sensory afferentation have not been extensively investigated. Studies in mammals have shown those non-sensory specific components that comprise the limbic system to be intimately involved in learning (see reviews: Adey & Tokizane, eds., 1967; Grossman, 1967; Smythies, 1966), although the

bases for these functions remain unclear. It would therefore seem that investigating the functions of the limbic system in birds would be of value both for developing a more integrated view of how different parts of the avian brain participate in information processing and for allowing a comparative approach to be applied towards understanding the role of a system prominent in all vertebrate classes (Herrick, 1948; Riss et al., 1969).

The experiments of Part II were directed, then, towards determining which parts of the avian brain are involved in memory storage and retrieval, with particular emphasis placed on examining the functions of the limbic system. Experiments III and IV studied the roles that cerebral structures play, respectively, in avoidance learning and in appetitive conditioning. Experiment V studied similar questions using a somewhat different approach. Since retinal projections cross completely at the optic chiasma (Cowan et al, 1963) and the neocommissures are absent (Ariens-Kappers et al., 1936), birds can be regarded as "split-brain" animals for which each hemishpere might be expected to have some degree of autonomous functioning. Thus, several questions concerning the functional organization of the avian telencephalon were studied by combining brain lesions with interocular transfer studies.

Part I

MEMORY STORAGE PROCESSES FOLLOWING ONE-TRIAL AVERSIVE CONDITIONING IN THE CHICK[1]

EXPERIMENT I: The temporal course and interrelationship of early mnemonic processes following a training experience

Introduction

A central problem that faces us in attempting to understand how memory is stored is to clarify the nature and interrelationship of the various mnemonic processes that have been shown to result from a training experience (Cherkin, 1965, 1969; Kesner & Conner, 1972; Lee-Teng, Magnus, Kanner & Hochman, 1970; McGaugh & Dawson, 1971; Weiskrantz, 1966). In baby chicks, long-term memory for an aversive response task can be disrupted by electroshock within 45 sec of training (Lee-Teng & Sherman, 1966) or by a chemoconvulsant drug given within 24 hr (Cherkin, 1969). On the other hand, electroshock given a minute after training interferes with retrieval for 30 min but not at longer intervals (Lee-Teng et al. 1970).

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[1] The two experiments of Part I will be published together in Behavioral Biology. Dr. J. Geoffrey Magnus is second author.

These amnesic effects can be interpreted as indicating the existence of an electroshock-insensitive memory trace which forms during the first minute, a current-susceptible process which induces the growth of that trace, a permanent engram consolidated over a period of hours, and an independent, electroshock-sensitive memory trace present during the first few hours.

By using quantitative methods in the analysis of retrograde amnesia data, the present experiment examined the temporal course and interrelationship of processes giving rise to short- and long-term memory in chicks. From our results, it would appear that the current-insusceptible memory trace formed during the first 45 sec grows with simple exponential kinetics, possibly as some restricted memory substrate becomes saturated. A metastable process which induces this growth is activated immediately upon training and apparently continues at a constant intensity thereafter, perhaps serving as behaviorally accessible short-term memory for the next hour or so.

Methods

One hundred twenty newly-hatched White Leghorn cockerels were obtained daily from Kimber Farms, Pomona, Calif. (Strain k-137 and K-155). Upon arrival at the laboratory, chicks were briefly anesthetized in a mixture of 3% halothane (Ayerst Labs.) in air. Small fishhook electrodes were then bilaterally placed subcutaneously behind the eye and above the ear. The electrodes were attached to about 30 cm of fine insulated wire that were later connected to the pulse generator. Chicks remained in in-

dividual cartons throughout the experiment in a room maintained at 88°F and 40% humidity; the room light was on from 6:30 AM to 6:30 PM. Chicks were allowed at least 3.5 hr to adapt to their cartons before training began (2:00 PM).

Training procedure

Chicks were presented with a metal bead, 3 mm in diameter, attached to a thin wire and coated with methyl anthranilate, MeA. When this lure was placed about 1 cm from the beak, a chick would typically orient towards it, peck within 2 sec, and give a characteristic head-shaking (disgust) response after tasting the MeA. Chicks failing to peck within 5 sec (about 10%) or to shake their heads within an additional 10 sec (another 5%) were discarded. The onset of the disgust response was taken to indicate the beginning of memory formation, and training-current intervals (TCI's) were measured from this point (Lee-Teng & Sherman, 1966).

Experimental Groups

Series A

Chicks were shocked after training at intervals of 0, 2, 4, 6 or 10 sec, or 17 hr using a subconvulsive current (SCC) pulse of 12 ma, 60 Hz, 280 msec duration (Groups A_1 - A_6, Table 1). Time intervals were controlled by an oscilloscope-calibrated decade timer placed in series with the pulse generator and initiated at the onset of the head-shaking response by the experimenter.

Series B

Chicks were shocked at either 2, 8 or 10 sec after training using a briefer SCC pulse of 250 msec duration, 12 ma, 60 Hz (Groups B_1 - B_3, Table 1). TCI measurements were not automated.

Control groups

One Group (C_1) was trained but not shocked to establish the probability of pecking on Day-2 for chicks having no induced amnesia; another Group (C_2) was sham-trained using a dry lure not coated with MeA.

All chicks were tested for retention one day later (2:30 PM). Experimental groups were pseudorandomly mixed together and testing was blind. The indicant of induced amnesia was the fraction of chicks in a group that pecked at the lure within 5 sec of presentation in the retention trial (the peck score).

Results

Series A

Analysis of the retrograde amnesia gradient for this series indicates that the SCC-insusceptible memory trace grows exponentially with time after training.

The peck-score of trained chicks given current at 17 hr (A_6) did not differ from that of trained controls given no current (C_1) ($\chi^2 = 0.7$, $p > 0.6$). The peck score of these groups combined, which was 0.26, represents the probability of pecking in the absence of any amnesia. On the

Table 1

Experiment I: Retrograde amnesia induced by passing transcranial SCC at either of two pulse durations following the aversive training trial.

		Training		Retention test	
Group	N[a]	Lure used	TCI (sec)	Peck Score[c]	Fraction amnesia[d]
C_2	293	dry		0.96	(1.00)
A_1	226	MeA	0	0.90	0.92
A_2	209	MeA	2	0.81	0.79
A_3	219	MeA	4	0.76	0.72
A_4	209	MeA	6	0.70	0.63
A_5	267	MeA	10	0.64	0.50
C_1+A_6	505[b]	MeA		0.26	(0.00)
B_1	204	MeA	2	0.68	0.59
B_2	217	MeA	8	0.55	0.38
B_3	228	MeA	10	0.53	0.37

[a] N = final number of chicks in a group.

[b] Includes 295 chicks trained but not shocked and 208 chicks trained and given current after 17 hr.

[c] Peck score = fraction of chicks in a group that pecked at the lure within 5 sec in the retention trial.

[d] Fraction amnesia = (peck score of group minus peck score of $C_1 + A_6$) divided by (peck score of C_2 minus peck score of $C_1 + A_6$).

other hand, the peck-score of the sham-trained controls (C_2), which was 0.96, indicates the probability of pecking for chicks having no aversive conditioning. Peck-scores for other Groups (A_1 - A_5) were normalized between the limits determined by the control groups. The resulting normalized "fraction amnesia" values [c.f. "induced peck score" of Cherkin (1969)] reflect the fraction of memory formation disrupted by passing current at time TCI (Table 1). Current induced clonic or tonic convulsions in about 6% of the chicks. Whether or not chicks suffered convulsions did not affect peck scores (for Groups A_1 - A_5, $\chi^2 = 1.97$, df = 4, $p > 0.7$).

As shown in Fig. 1, the logarithm of the fraction amnesia (FA) decreases linearly with increasing TCI values. A least-squares analysis of these data indicates the relationship between FA and TCI to be empirically described by

$$\ln FA = -0.059\ TCI + \ln 0.91.$$

The last term in the above expression shows that some portion of the memory that forms does not depend upon the time at which current had been passed. That this TCI-invariant portion of memory is formed even when current is given immediately after training is shown by the peck score of Group A_1 being significantly below that of Group C_2 ($\chi^2 = 5.3$, $p < 0.05$). This phenomenon can be better understood if instead of FA we now consider its complement, or the "fraction of retrieval" (FR), a measure of the amount of memory that forms when storage processes have been disrupted at time TCI. Since $FA = 0.91\ e^{-0.059\ TCI}$, then $FR = 1 - 0.91\ e^{-0.059\ TCI}$, or

$$FR = 0.91\ (1 - e^{-0.059\ TCI}) + 0.09 \qquad (1).$$

Thus, FR can be considered as having two components; the magnitude of one

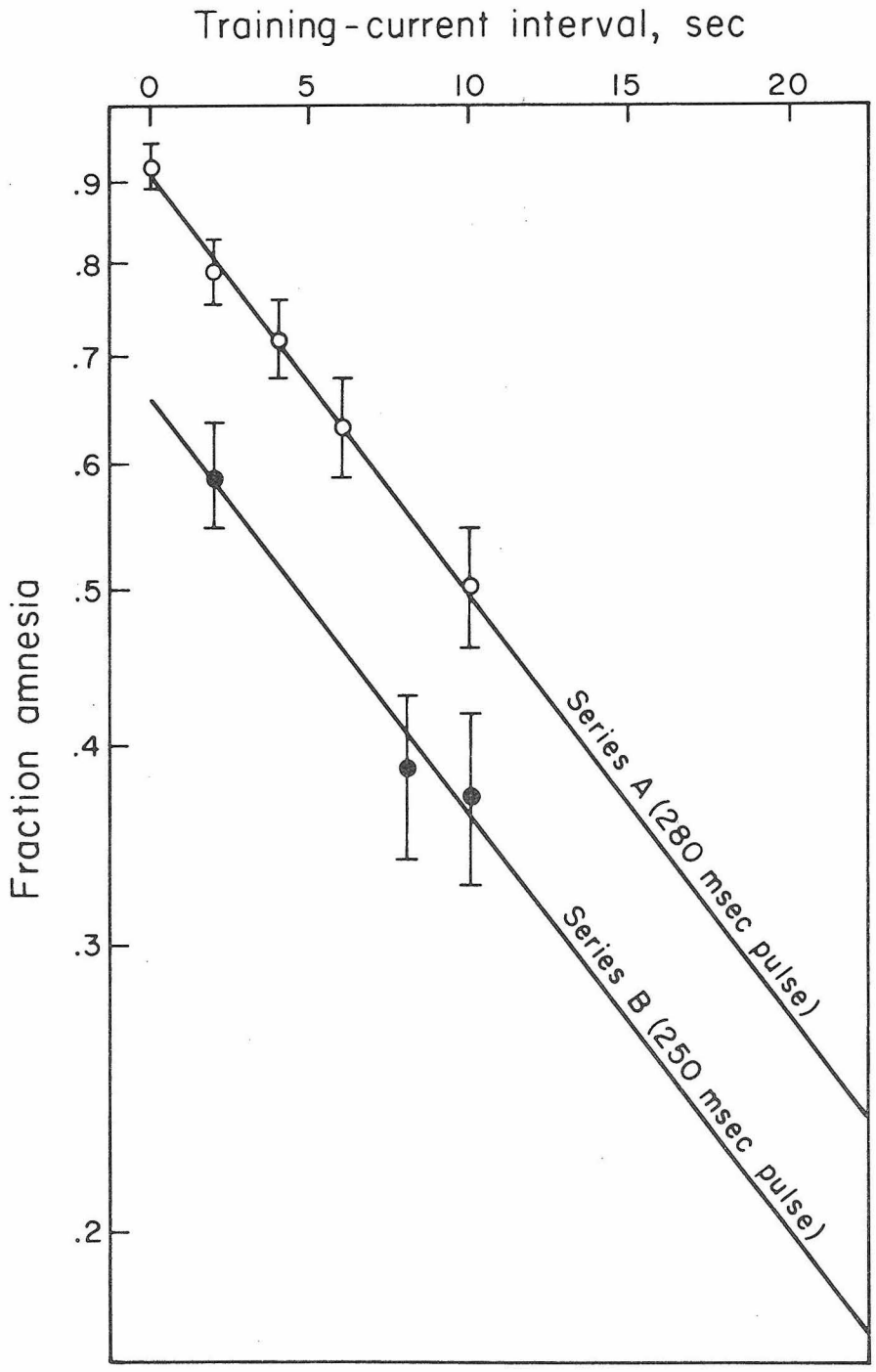

Figure 1. The exponential decline in the retrograde amnesia induced by SCC administered at increasing intervals following the training experience. The briefer SCC pulse used in Series B allowed a larger portion of memory formation to continue after passage of current than in Series A. Bars indicate plus or minus one standard deviation.

of these depends upon the time at which current is passed, while the magnitude of the second does not. Assuming a simple relationship between retrieval and memory[2], equation (1) suggests that the SCC pulse given at time TCI causes most of a first-order exponential growth process to halt while allowing a small part of it to continue unabated (i.e., the second term can be considered as $0.09\ (1 - e^{-0.059\ t})$, and t becomes very large).

Series B

Use of a briefer SCC pulse following the training trial increases the fraction of memory storage processes not affected by the passage of current. As was done with Series A, peck scores for groups in Series B were normalized between the score of the unshocked trained controls and that of the sham-trained controls (Table 1). A least-squares analysis of these normalized data indicates that for chicks shocked with a 250-msec pulse after training the fraction amnesia is empirically described by

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[2] The actual nature of the transfer function relating memory and behavior can be specified on the basis of results from Experiments I and II. We have observed that the retrograde amnesia gradients have first-order inetics, which we can reasonably assume to reflect the properties of the underlying memory formation processes. In this case, the relationship between the fraction amnesia, which we measure, and the fraction of the memory trace remaining to be completed (FIMT) must be such that the exponential kinetics of both are retained. Formally, if both are first-order functions, then $FIMT = FA^x$, where x is greater than 0. Consolidation studies often assume that the time course of memory growth is similar to that of the RA gradient, or that x is in the order of 1. If this is not the case, then the fraction of Pre-LTM induction that continues after SCC is given, as well as the rate-constant of Pre-LTM growth, would differ from the values suggested by our behavioral observations, although the basic relationships among the different mnemonic processes would still hold.

$$\ln FA = -0.060\ TCI + \ln 0.66$$

(Fig. 1). Additional data obtained using the same shock parameters as in Series B have been reported for TCI values up to 3 hr (Lee-Teng, 1969); these show little deviation from the above regression line ($\chi^2 = 2.8$, $df = 8$, $p > 0.80$).

The complement of FA, the "fraction of retrieval" can be expressed in this case by

$$FR = 0.66\ (1 - e^{-0.060\ TCI}) + 0.34 \qquad (2).$$

A comparison of equation (2) with equation (1) indicates that the nature of the exponential component of the memory formation does not depend upon the duration of the SCC pulse used. However, use of a 250-msec rather than a 280-msec disrupting pulse increases the relative amount of the memory formed that is unrelated to the time at which current is passed. In summary, then, the growth of FR as a function of time t after training is described by $FR = 1 - e^{-0.059\ t}$. Passage of SCC at time TCI halts 91% of the growth of FR using the 280-msec pulse or 66% of it using the 250-msec pulse.

Discussion

The foregoing results indicate that in the first minute after training an electroshock-insusceptible memory trace is formed by a mechanism having first-order exponential kinetics[2]. However, some portion of the memory formation process apparently continues undisturbed after SCC is given (Mah, Albert & Jamieson, 1972), as suggested by the component of the

"fraction of retrieval" not included in the exponentially increasing terms of Equations (1) and (2). A comparison of the two equations indicates that the magnitude of this undisturbed portion increases if a briefer disrupting pulse is used[3]. On the other hand, an electroconvulsive shock (ECS) longer and more intense than the pulses used here allows no significant memory formation to continue after disruption (Lee-Teng, 1969b).

The memory trace that grows within the first 45 sec does not appear to be the permanent form of memory storage, but rather an antecedent to it. Lee-Teng et al. (1970) have shown that at least 30 min is required before this trace develops into a behaviorally-manifest form, while studies by Cherkin (1969) indicate that a chemoconvulsant drug given within 24 hr after training can also disrupt the consolidation of permanent memory. It would therefore seem that the trace whose formation has been studied here might be appropriately termed Pre-LTM, a long-term memory precursor.

The rate at which Pre-LTM grows decreases continuously as the memory trace is formed. These first-order kinetics suggest that some restricted substrate of the memory trace becomes increasingly saturated as the growth of Pre-LTM proceeds. In conformance with this, one of us (Benowitz, 1972) has shown that in a population of trained chicks tested

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3 A reviewer has suggested that some part of the TCI-invariant portions of Equations (1) and (2) might be attributable to memory formation beginning as early as 0.5 sec prior to the time considered here. While this is plausible, memory formation would have to begin 1.63 sec earlier than we have considered to fully account for the undisturbed portion of memory formation in Series A and 7.01 sec earlier for Series B. Thus, although the estimates of the portion of undisturbed memory formation might be somewhat high, this would not be very significant.

several times under reinforcing conditions, the average peck score for the group does not improve from trial to trial and the peck-frequency histogram has a binomial distribution. These observations indicate that a single training experience causes a saturation of each chick's learning capacity, and are consistent with the notion that the amount of memory substrate remaining unsaturated is rate-limiting in the growth of Pre-LTM.

The process which induces the formation of Pre-LTM appears itself to remain at a constant intensity following the training experience. This is indicated by the rate of Pre-LTM growth depending strictly upon the amount of the trace remaining to be formed, as well as by the TCI-invariance of the portion of memory formation continuing undisturbed after current is given. Fig. 2 illustrates the temporal course of mnemonic processes effected by the training experience and the manner in which SCC affects these processes, as suggested by our results. Immediately upon training, a metastable induction process is initiated which then continues at a constant intensity. Prior to the passage of current, this causes Pre-LTM to grow at a rate that varies with the amount of memory substrate remaining unsaturated. In proportion to its intensity (Lee-Teng, 1969 a,b) and duration (McGaugh, 1966), SCC can allow the induction of Pre-LTM to continue in a segment of the memory substrate. Since the induction process is time-invariant, the fraction of it which is undisturbed by current is independent of TCI. Since this model can be regarded as SCC causing one fraction of the exponential growth of Pre-LTM to halt at TCI while allowing the remainder to continue, it is consistant with the observed exponential kinetics of Pre-LTM formation, the TCI-invariance of the undisturbed portion

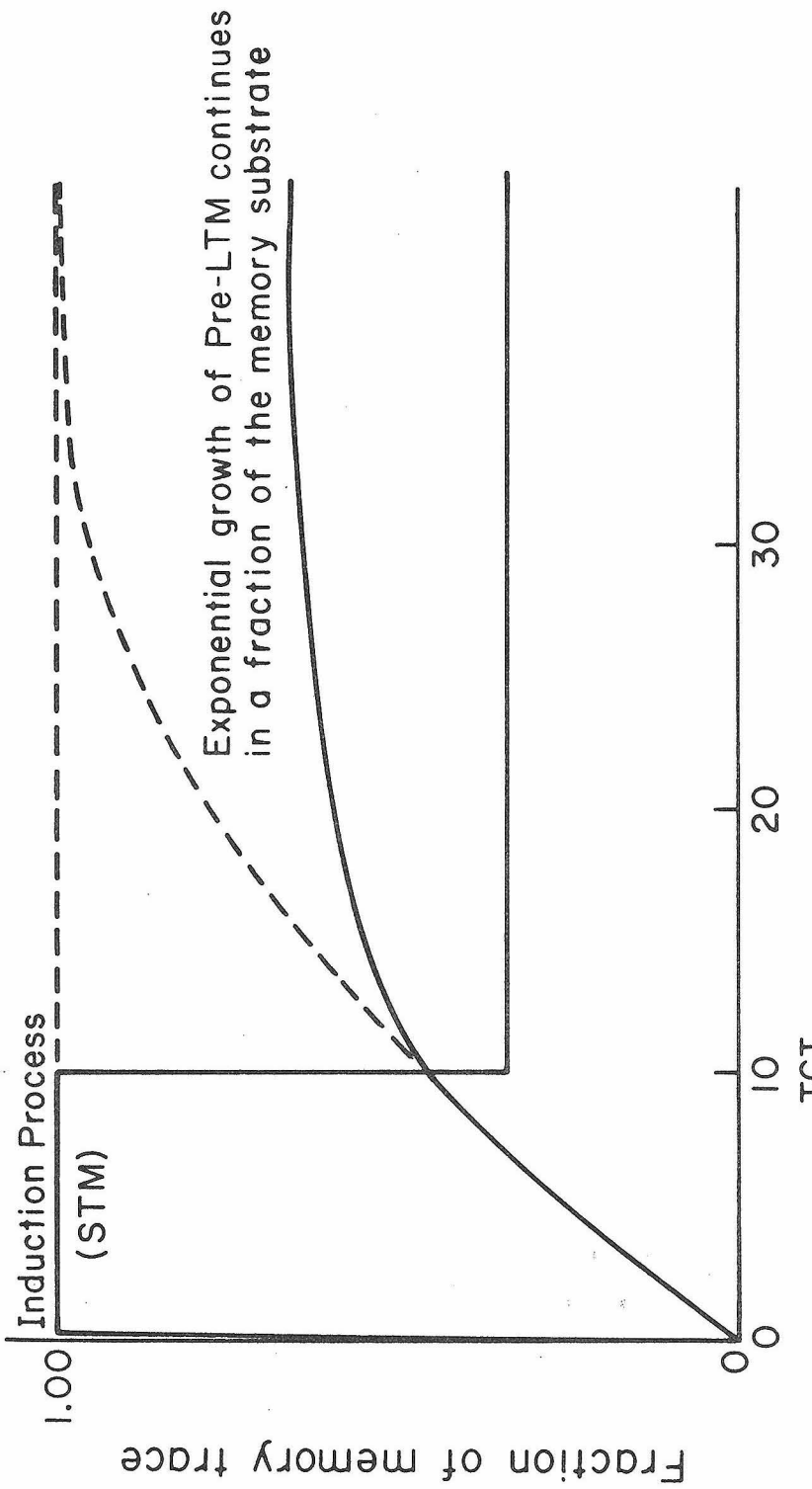

Figure 2. Schematic representation of the temporal course of the induction phase (STM) and the current-insusceptible memory trace (Pre-LTM) for the case in which the 250-msec SCC pulse is given 10 sec after training. STM is activated immediately upon training and begins to induce the exponential growth of Pre-LTM; passage of current allows Pre-LTM induction to continue undisturbed in a fraction of the memory substrate (see footnote 4). Dashed lines indicate the temporal course of STM and Pre-LTM if no current were given.

of memory that forms, and the presence of measurable learning in chicks given current almost immediately after training.

The mnemonic process which induces Pre-LTM growth may function as behaviorally accessible memory for the first hour or so after training. In this period, during which Pre-LTM is not in a retrievable state, an independent, SCC-sensitive trace has been shown to provide chicks with short-term memory (STM) (Lee-Teng et al. 1970). Our results indicate that the SCC-sensitive induction process continues at a constant intensity during the period in which Pre-LTM is formed, and it might conceivably continue to do so for longer periods. It thus seems possible that the induction process and the behaviorally manifest STM trace may be one and the same.[4] A similar relationship has recently been proposed by McGaugh & Dawson (1971). On the basis of reviewing the effects of amnesic agents on memory for aversive conditioning in mammals, these authors have suggested that "STM is essential for LTM. ECS impairs consolidation by speeding the decline of STM. The asymptote of LTM is determined by the duration of STM." Our model suggests that SCC does not speed the decline of STM, but rather acts to completely arrest one fraction of it (otherwise, saturation of the mem-

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[4] Although LTM induction is largely destroyed by SCC, a substantial amount of STM has nevertheless been shown to persist for a few hours after current is passed (Lee-Teng et al., 1970; Geller & Jarvik, 1968; McGaugh & Landfield, 1970). It would thus seem that for this notion (and for that of McGaugh & Dawson (1971)) to be consistent, one must postulate that SCC does not completely disrupt STM, but rather reduces it such that it can less successfully induce LTM. The reduction of STM shown in Figs. 2 and 3 should not be considered, then, to reflect the reduction of STM's behavioral potency, but rather of its capacity to induce LTM.

ory substrate would eventually occur for all vaues of TCI). The time scales of various species' memory formation also seem to differ greatly: the rise of STM seems to be immediate from the present results but requires more than 5 sec in rats; the rise of Pre-LTM occurs in the order of 30 min in rats, as contrasted with 45 sec here.

EXPERIMENT II: The integration of fractional memory traces

Introduction

In Experiment II, we examined the manner in which two fractional memory traces summate together (Chorover & Schiller, 1965; Kesner et al., 1970; Nachman & Meinecke, 1969; Wyers & Deadwyler, 1971). The formation of each memory trace was limited by administration of SCC shortly after training. While this study was initially intended to investigate the mechanisms by which an animal integrates a succession of similar experiences, it also allowed us to critically examine the hypotheses drawn in Experiment I. If the mnemonic processes described above were the only determinants of memory formation, then we might expect a second training to reactivate STM, which in turn would induce the growth of Pre-LTM to proceed at a rate depending only upon the amount of memory substrate remaining to be saturated. On the other hand, if memory actually formed after the first experience were not fully manifest behaviorally, then the second training might be expected to cause a disproportionate improvement in performance (Quartermain et al., 1970; Zinkin & Miller, 1967). Alternatively, memories from the two experiences might not become fully integrated together, possibly due to the use of different neural substrates for the registration of

each experience. Results from this experiment indicate that the two mnemonic processes described in Experiment I are the sole determinants of partial memory trace integration.

Methods

Series D

The first of two trainings was given to chicks interspersed among those of Series A described above. Chicks in Series D were given current 2, 4, or 6 sec following training, using the 280-msec SCC pulse. Seventeen hours later, these were trained again and given current at intervals after training as shown in Table 2 for Groups D_1 - D_4. Chicks that failed to peck on the retraining trial were discarded. All chicks were tested blind for retention 7 hr later.

Series E

Chicks trained together with those of Series B were given a 250-msec SCC pulse at 2 or 8 sec after training. The second training was given 6 hr later, with memory formation allowed to proceed for the intervals shown for Groups E_1 and E_2 in Table 2. Chicks failing to peck in the retraining trial were discarded. The retention test was given blind on the following afternoon.

Results

The second training caused the memory trace to continue growing exponentially from the point at which its formation after the first training had been halted.

Series D

As shown in Table 2 (top), the fraction amnesia values resulting from two partial memory formations are negatively correlated with the sum of the two training-current intervals, and do not depend upon the order in which the sessions were administered ($\chi^2 = 0.12$, $p > 0.7$). If, as suggested above, the second training were to cause Pre-LTM to continue growing from the point at which its formation had previously been halted, then the resultant FA after first TCI_1 and then TCI_2 sec of memory formation would be

$$FA = e^{-0.059\ (TCI_1 + TCI_2) + 2 \ln 0.91}.$$

It should be noted that in making this prediction we need consider that a portion of each of the two memory formations should be unaffected by the passage of current, as found in Experiment I. For example, we would expect the FA after first 6 and then 4 sec of TCI to be somewhat lower than that after 10 sec of continuous memory formation. The values of FA predicted using the above formula show little deviation from the four experimental values obtained for the selected populations of Series D (Table 2) ($\chi^2 = 1.16$, $df = 3$, $p > 0.7$).

Table 2

Summation of fractional memory traces. Chicks were given two training sessions, each followed by post-trial SCC (280-msec duration for Series D, 250-msec for Series E). Selected populations include only chicks that pecked in the second training session, while unselected groups include those that did not peck as well. Chicks not pecking on retraining were given an additional TCI_2 + 5 sec of consolidation. Predicted values assume the second training to cause the first-order growth of Pre-LTM to continue from the point at which its formation had been interrupted.

		Initial training	Retraining		Retention test		Fraction Amnesia	
Group	N_1[a]	TCI_1 (sec)	TCI_2 (sec)	N_2[b]	Peck score, Selected Pop.	Peck score, Unselected	Selected Pop.	Predicted
D_1	252	2	4	204	0.61	0.56	0.50	0.53
D_2	273	4	2	208	0.68	0.62	0.60	0.53
D_3	253	4	6	207	0.57	0.55	0.45	0.42
D_4	298	6	4	203	0.53	0.50	0.39	0.42
E_1	204	2	8	120	0.42	0.42	0.20	0.19
E_2	196	8	2	84	0.42	0.33	0.20	0.19

[a] N_1 = Size of population to exclusion of chicks not pecking on retraining.

[b] N_2 = Number of chicks that pecked on retraining and were thus retrained in the selected populations.

Series E

FA values experimentally obtained using a 250-msec pulse to disrupt each of two memory formations are shown in Table 2 (bottom). Predicted values in these cases would be

$$FA = e^{-0.060\ (TCI_1 + TCI_2) + 2\ \ln\ 0.66},$$

again considering that each of the partial memory formations would be incompletely disrupted by SCC. As with Series D, the two experimental values for the selected populations of Series E are quite close to predictions based on the assumption that the second training allows a resumption of the exponential memory trace growth.

In order to compare the memory resulting from a succession of two training-SCC sessions with theoretical predictions, we have used selected populations consisting only of chicks that pecked on retraining. This comparison is based upon the assumption that subgroups that did or did not peck do not differ from each other, but rather that excluded chicks represent the portion of a uniform population probabilistically expected not to peck. However, if after the first training the excluded chicks had a lower probability of pecking than those retained, the experimental FA values for selected populations would be too high an estimate of the memory resulting from the desired summation of fractional memory traces. Our experimental procedure allowed chicks that did not peck upon the second presentation an additional TCI_2 + 5 sec of exposure to the conditioned stimulus, during which time considerable aversive associations were probably formed (the strength of which could not be predicted by our model). Yet,

even if we were to combine the final retention test scores for chicks that had not pecked in retraining (and whose peck scores would be expected to be inflated) together with scores of the selected populations, FA values still do not differ from those predicted by our model at a conventional level of significance ($\chi^2 = 10.9$, df = 5, $0.10 > p > 0.05$; using selected populations, $\chi^2 = 2.0$, df = 5, $p \sim 0.8$).

Discussion

The foregoing results indicate that a second training experience causes Pre-LTM to continue growing from the point at which its formation had previously been interrupted. This summation of the two memory traces appears to occur in the absence of active mental associations (Quartermain et al., 1965; McGaugh & Alpern, 1966), insofar as passage of transcranial electroshock does not prevent it from taking place. This may perhaps be attributable to the use of a rather unique learning paradigm in which attentional and motivational factors do not seem to vary. While such conditions might not be expected for other learning situations and species (e.g. Wyers & Deadwyler, 1971; Kesner et al., 1970), our results suggest that under proper circumstances, physiological memory systems allow a succession of similar experiences to cause a continued modification of the same memory substrate.

The memory storage mechanisms discussed in Experiment 1 (Fig. 2) seem to underlie the continued exponential growth of the memory trace resulting from the second training experience (Fig. 3). It would appear

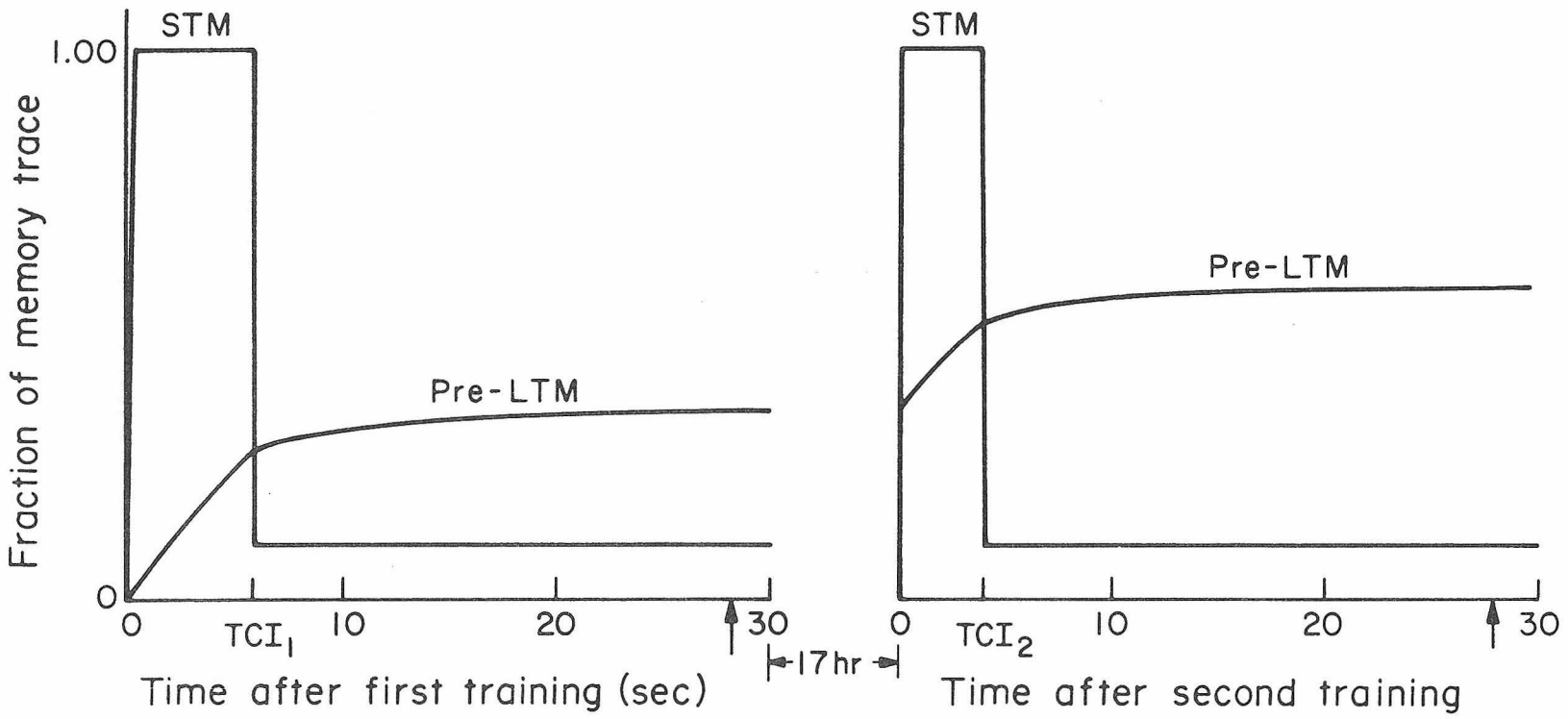

Figure 3. The growth of memory for a sequence of two training experiences. Memory formation after each training is interrupted by an SCC pulse of 280-msec duration. Upon the second training, STM is reactivated and induction of Pre-LTM begins at a rate determined by the amount of memory substrate remaining to be saturated. Passage of a second electroshock at time TCI_2 again allows a fraction of the induction process to continue undisturbed.

that upon retraining, STM is immediately reactivated and again continues at a constant intensity. This in turn induces the growth of Pre-LTM to proceed at a rate determined only by the amount of memory substrate still remaining to be saturated. Passage of suboptimal SCC disrupts most of the induction process but again allows some of it to continue the formation of the Pre-LTM in a segment of the memory substrate.

Summary and Conclusions

These experiments indicate that the growth of memory following aversive conditioning in chicks involves two early mnemonic phases. Initially, a metastable process is activated and seems to continue at a constant intensity. Within 45 sec, this induces the growth of an electroshock-insusceptible memory trace (Pre-LTM). The first-order kinetics describing the growth of Pre-LTM, along with the observation that a single training trial causes a saturation of chicks' memory capacity (Benowitz, 1972), indicate that some restricted substrate of memory becomes saturated as the memory trace grows. SCC halts most of the induction process but allows some of it to continue undisturbed. Within the first few hours after training, during which time the latent Pre-LTM trace becomes consolidated into long-term memory (Cherkin, 1969; Lee-Teng et al., 1970; McGaugh & Dawson, 1971), the mnemonic process which had induced the growth of Pre-LTM appears to function as behaviorally retrievable short-term memory. The results of the memory additions study support this model, and indicate that in the presence of a partial memory trace, a second experience simply results in the continued saturation of the Pre-LTM substrate.

Part II

ANATOMICAL ASPECTS OF MEMORY STORAGE AND RETRIEVAL

Experiment III: The effects of forebrain ablations on avoidance learning[5]

Introduction

The extensive ability of birds to learn has traditionally been thought to be predicated upon a neuroanatomical organization quite divergent from that of mammals (e.g., Ariens-Kappers, Huber & Crosby, 1936; Stettner & Matyniak, 1968). However, fundamental similarities in the prosencephalic structures of birds and mammals have recently been indicated (Kallen, 1962; Karten, 1969). Thus it is possible that a comparison of the neurological bases of learning in birds and mammals may be of relevance in understanding behavioral mechanisms of both classes. Recent studies have begun investigating the involvement of avian forebrain regions in processing sensory information and in mediating behavior (e. g., Hodos & Karten, 1970; Karten & Hodos, 1970; Lee-Teng & Sherman, 1969; Pritz, Mead & Northcutt, 1970; Stettner &

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[5] This experiment will be published in Physiology and Behavior, 1972

Schultz, 1967; Zeigler, 1963), yet the functional significance of much of the telencephalon remains to be determined.

In the present experiments, gross morphological units were selected for ablation as an initial attempt to discern which forebrain areas are involved in learning. The behavioral task involved learning to suppress an innate response tendency as a result of one-trial experience. The task is a suitable one for use here since retention of the one-trial experience is quite sensitive to the degree of impairment to memory mechanisms, as indicated by consolidation studies (e.g., Lee-Teng & Sherman, 1966). While several of the ablations included direct sensory-specific projection areas, some of the findings seem to reflect damage to structures homologous with parts of the mammalian limbic system.

METHODS

Subjects

A total of 375 one-day old White Leghorn cockerels (Kimber Farms, Pomona, Calif.) were used over a five month period. Upon arrival at the laboratory chicks were placed in individual cartons from which they were removed only during surgery. No food or water were provided since sufficient nutrients are provided from the yolk sac for the first few days after hatching. The experimental room was maintained at 88°F and 40% humidity. Room light was on from 6:30 AM to 6:30 PM.

Training task

The learning used throughout this study was that of suppressing,

usually after one training trial, an innate pecking response. A small metallic bead, 3 mm in diameter, mounted at the end of a wire and coated with methyl anthranilate (MeA) was used for training and testing for retention. Upon presentation of the lure, naive chicks typically orient towards it, peck, and give a characteristic disgust (head-shaking) response after tasting the MeA. In training, chicks were allowed 5 sec to peck at the lure and an additional 10 sec to shake their heads; those that failed to peck or shake during training were discarded (about 15%). Upon re-presentation of the lure, approximately 75% of trained chicks refrain from pecking and often avoid actively, while only 4% of mock-trained chicks fail to peck (Lee-Teng & Sherman, 1966). This behavioral task is particularly attractive for studying surgically induced learning deficits since gross behavioral impairments, which would probably result in a decrease in pecking, can readily be distinguished from specific learning deficits that result in an increase in pecking.

Surgery

Chicks were held in place between the ear bars of a small-animal stereotaxic apparatus. The beak was inserted into a funnel, through which vaporized halothane anesthesia (Ayerst Labs) or an alternative fresh air supply were administered by means of a foot pedal. Small incisions were made on either side of the median suture above areas to be ablated, leaving one side of bone flap uncut to allow replacement after surgery. Brain tissue was bilaterally removed by gentle suction. Gelfoam (Upjohn Co.) was inserted to replace ablated tissue, the bone

flap was closed and Gelfilm (Upjohn) was placed over incisions in cases where the ablations were near the midline. The skin was then drawn together, held in place, and collodion was applied. The mortality rate, almost exclusively due to excess anesthesia, was about 10%. A total of eight types of surgery, which are described below, were included in the study. Sham-operated controls followed a similar procedure which included anesthesia and incisions which caused superficial brain damage; bleeding from the large superficial blood vessels was allowed to continue for about the same length of time as in operated animals.

Histology

Following completion of behavioral testing one half of the chicks were sacrificed and perfused with chick Ringer's solution and Bodian fixative. Their brains were removed, dehydrated, embedded in paraffin and sectioned at a fixed plane (see Fig. 4) at 15 microns. One-fifth of the sections were retained and stained with cresyl violet. These were then projected and the extent of surgery was indicated on standardized brain outlines to allow comparison of ablations.

General procedure

Three separate studies are described. In the first, chicks were trained once before surgery and were later tested for retention and relearning. In the second study, postoperative acquisition was examined in groups similar to those which had demonstrated an ability to retain or relearn the avoidance in the first study. One group in the first

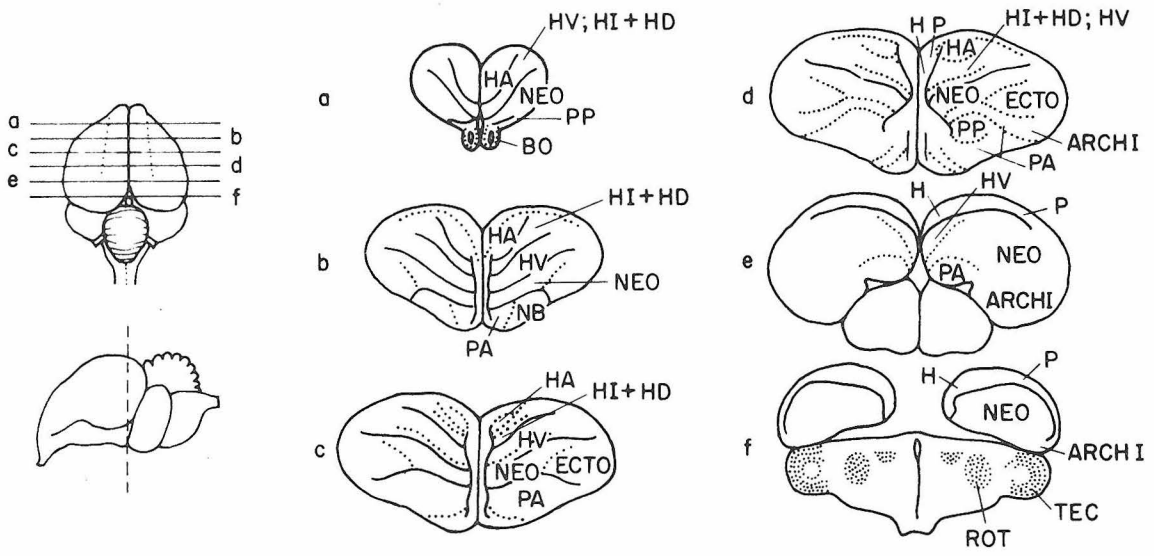


Figure 4. transverse sections through chick brain. Figures to the left show plane of section. ARCHI = archistriatum, BO = bulbus olfactorius, ECTO = ectostriatum, H = hippocampal area, HA = hyperstriatum accessorium, HD = hyperstriatum dorsale, HI = hyperstriatum intercalatus, HV = hyperstriatum ventrale, NB= nucleus basalis, NEO = neostriatum, P = prepyriform area, PA = paleostriatum augmentatum, PP = paleostriatum primitivum, ROT = nucleus rotundus, TEC = optic tectum.

study had shown no post-surgical retention or relearning, and the third study sought to localize this effect. At all times different groups of chicks were randomly mixed together and run with unoperated controls such that the experimenter did not know a chick's type of surgery or record on previous tests. The only exceptions to this were sham-operated controls, which were added afterwards. Preliminary studies had indicated that the different operations cause differing types of learning deficits, which suggested that it was unnecessary to control for non-specific behavioral impairments. Sham-operated controls were, however, included later for double checking.

Section A: Retention of the presurgically acquired response

Retention and relearning of a preoperatively acquired response were studied in four groups. The original intention of including the limited and extensive hyperstriatal ablations was to further study the nature of learning deficits reported for varying degrees of dorsomedial damage in birds (Hodos, Karten & Bonbright, in prep.; Lee-Teng & Sherman, 1969; Pritz et al., 1970; Stettner & Schultz, 1967; Zeigler, 1963). The frontal and lateral regions were selected for investigation arbitrarily.

Procedure

Chicks were given one training trial using the MeA lure at 2:00 P.M. of Day 1. Surgery was begun 3 hr later. The approximate extent of the four operations is shown in the top of Fig. 5. Two days after surgery operated chicks along with sham- and unoperated controls were tested for avoidance four times, at 9:00 A.M., 11:30 A.M.,

2:00 P.M. and 4:30 P.M. Testing was done using the MeA lure so that these were additional training trials for chicks that failed to avoid.

Results

As summarized in Table 3, the lateral ablations resulted in complete loss of retention and relearning, while less severe deficits were suffered by the frontal and extensive hyperstriatal groups. The number of trials in which a chick avoided the lure in the four Day-3 retention trials determined individual scores. These were then averaged for all chicks in a group. A one-way analysis of variance indicates the difference in retention and relearning among the different groups to be highly significant ($F = 12.1$, $n_1 = 5$, $n_2 = 133$, $p < .001$). The 14 sham-operated controls did not differ significantly from 59 unoperated chicks ($t = 1.59$, two tailed, $0.2 > p > .01$), and these two groups were pooled together to form the control group (C) in Table 3. The limited hyperstriatal chicks (LH) also did not differ from the controls ($t = 1.00$, $0.4 > p > .03$), and these may therefore be regarded as an operated control group for this experiment. Chicks having more extensive damage to the hyperstriatum (EH) averaged significantly lower than controls ($t = 3.0$, $p < .01$), as did the frontal chicks ($t = 2.8$, $p < .01$). By far the most severe loss of learned avoidance, however, was seen in chicks with lateral ablations (L). Despite successive retrainings, their pecking rate remained almost as high as that found in untrained chicks (Lee-Teng & Sherman, 1966).

A somewhat unexpected finding was that in each of the groups studied the retention scores measured on the first test of Day 3 were not

Table 3

Effects of forebrain ablations on retention and relearning of avoidance learning. Chicks were trained once prior to Day 1 surgery and tested four times on Day 3 under reinforcing conditions.

Group	N	Avoidances in four trials, Day 3	
		M	σ_m
Controls (C)	73	2.78	0.12
Limited hyperstriatal (LH)	17	2.46	0.34
Extensive hyperstriatal (EH)	16	1.87**	0.28
Frontal (F)	18	1.94**	0.33
Lateral (L)	15	0.27**,a	0.15 b

[a] Differs from other operated groups at $p<.01$.

[b] Experimental value of σ_m exceeds by more than 70% the value of σ_m which would result if all chicks within the group had the same probability of avoiding. In other groups, the experimental value of σ_m exceeds predicted value for a uniform population by 13%-48%.

** Significantly differs from the control group at $p<.01$ (two-tailed t-test).

improved upon by the three subsequent retrainings. That is, although failure to avoid on any trial presumably resulted in reinforcement, the average number of chicks in a group that avoided did not change from test to test. This strongly suggests that the Day 1 training trial caused a complete saturation of chicks' learning capacity for the task. Thus "relearning" trials can simply be used to get a statistically more accurate measure of postoperative retention.

Section B: Post-surgical acquisition of the response

Learning was studied in operated chicks that were not trained prior to surgery. By contrasting new acquisition with the retentional deficits found in Section A, it could be determined whether the areas studied were differentially involved in storage and retriefal mechanisms. This section also served as a control to ascertain whether avoidance found in Section A had in fact been due to retention or was instead a non-contingent effect of surgery. These two alternatives could readily be distinguished by seeing whether naive operated chicks all pecked during the training trial. Only groups that had demonstrated avoidance behavior in Section A were studied here. Lateral chicks had shown no relearning ability, and it was assumed that new acquisition would also be absent.

Procedure

This study followed the procedure of the first section except that there was no preoperative training. The first presentation

of the MeA lure on Day 3 served both to train naive chicks and to test for avoidance behavior in the absence of prior training. The subsequent three Day-3 presentations (at 11:30, 2:00 and 4:30) measured postoperatively acquired avoidance. Testing was again done with the MeA-coated lure so that these were reinforcing trials for chicks that did not avoid.

Results

Both extensive and limited hyperstriatal ablations impaired acquisition, while frontal ablations did not. The average number of avoidances in the three Day-3 retention tests are summarized for the different groups in Table 4. A one-way analysis of variance indicates significant between-group differences in post-surgical acquisition ($F = 4.23$, $n_1 = 4$, $n_2 = 144$, $p<.01$). The score of 20 sham-operated controls did not differ from that of 63 unoperated chicks ($t = 0.33$, two tailed, $p>0.8$) and these were pooled together to form the control group (C) in Table 4. All three types of untrained, operated chicks readily pecked at the lure in the first Day-3 presentation. This indicates that all of the postoperative avoidance observed in Section A had, in fact, been due to retention of the presurgical training experience rather than being a non-contingent effect of surgery. Following the training, both limited and extensive hyperstriatal groups scored significantly below controls in the three retention test trials (for LH vs. C, $t = 2.3$, $p<.05$: for EH, $t = 2.5$, $p<.02$). In contrast, however, frontal chicks appeared to avoid in the

Table 4

Acquisition for chicks first trained after surgery in Section B and a comparison with Day 3 performance of chicks in Section A trained prior to surgery. Scores indicate the number of avoidances in the last three Day-3 retention trials.

Group	N	Section B initial training given after surgery		Section A initial training before surgery
		Avoidances in 3 Day 3 trials		Avoidances in last 3 trials
		M_{II}	σ_m	M_I
C	83	1.84	.10	2.07
LH	18	1.28*	.23	1.82
EH	17	1.24*	.20	1.50**
F	31	2.19	.19 [a]	1.17**

*Significantly differs from the controls at $p<.05$ (two-tailed t-tests).

**Differs from controls at $p<.01$.

[a] See footnote b, Table 3.

retention trials more than controls, although this was not statistically significant ($t = 1.8$, $p \sim .07$).

In Section B, as in the previous section, scores did not improve from the first to the last retention tests for the LH, F and C chicks despite the fact that testing was done under reinforcing conditions. Thus in these cases the initial training appears again to have caused a saturation of chicks' learning capacity, and the reinforcement trials may be used for statistically more accurate measurement of one-trial acquisition. The EH group did, however, show a significant improvement (comparing the first and the last trials, $\chi^2 = 10.1$, $p<.01$; it follows from this that the actual impairment to one-trial learning was far more severe for EH chicks than is indicated by the data in Table 4).

To directly compare the effects of initially training before and after surgery, scores on the last three test trials of Section A, also shown in Table 4, are contrasted with the scores on the three test trials of Section B. A two-way analysis of variance for this data indicates no overall effect of initially training before vs. after surgery, but significant differences between the groups ($F = 3.27$, $n_1 = 4$, $n_2 = 263$, $p<.01$) and for group x time of training interaction ($F = 4.32$, $n_1 = 4$, $n_2 = 263$, $p<.01$). The Day-3 learning score of limited hyperstriatal chicks first trained after surgery was 30% lower than it had been when there was presurgical training. Control and EH chicks also showed somewhat better Day-3 learning scores if they had been trained before surgery. In contrast, however, the F chicks showed

an 87% improvement in Day-3 performance when no training preceded surgery. It would thus appear that the area included in limited hyperstriatal ablations is involved in storage but not retrieval mechanisms, while the more extensive hyperstriatal region is involved in both storage and retrieval. In contrast, removal of the frontal region impairs retrieval of presurgically acquired information while not affecting storage of previously unfamiliar information after surgery.

Section C: Localization of the region responsible for the complete loss of the avoidance response

This study sought to localize the region responsible for the total impairment to retention and relearning found in lateral chicks of Section A. The lateral area was subdivided two ways: mediolaterally, giving anterior and posterior subdivisions, and antero-posteriorly, giving medial and lateral subdivisions. The resulting four groups, each having one of these smaller regions removed, were trained presurgically and subsequently tested for retention and relearning.

Procedure

Areas removed in the four operations are shown in Fig. 6. The experimental design was identical to that of Section A: chicks were trained at least 3 hr before surgery on Day 1 and tested under reinforcing conditions four times on Day 3. Scoring was the same as in Section A.

Results

The posterolateral region was the most critical of the four subdivisions for retention and relearning (Table 5). One-way analysis of variance indicates the differences in post-surgical avoidance among the groups to be highly significant ($F = 32.5$, $n_1 = 4$, $n_2 = 155$, $p<.001$). The number of avoidances in 4 trials for the mediolateral groups (M) was, on the average, similar to that of the complementary far-lateral group (FL). Both of these scored significantly below controls of Section A ($p< 0.01$ in both cases), but higher than the L group of Section A ($p\sim 0.05$: $t = 2.0$ for M vs. L, $t = 2.2$ for FL vs. L). On the other hand, a comparison between the posterolateral (P) and anterolateral (A) subdivisions showed a more striking contrast: while the A group averaged 1.82 avoidances, the complementary P group averaged only 0.35, which is similar to the score for the L group of Section A. Thus the region included in the P ablations was the most critically involved in learning, while deficits seen with M and FL chicks may reflect their partial inclusion of the posterolateral region. Of these groups, only FL chicks showed a significant improvement in learning over the four test trials ($\chi^2 = 7.3$, $p<.01$).

In brief then, the statistically significant findings of the three sections ($p < 0.05$, two tailed t-tests) are that limited dorsomedial ablations (i.e., LH) impair postsurgical acquisition but not retention of a presurgically acquired response; extensive hyperstriatal ablations impair both acquisition and retention; frontal ablations do not affect postsurgical acquisition in previously naive chicks but damage

Table 5

Effects of ablating the four subdivisions of the lateral region on retention and relearning. Chicks were trained and tested as in Section A.

Group	N	Avoidances in four trials, Day 3		Significance of pair-wise contrasts			
		M	σ_m	FL	A	P	C [a]
Mediolateral (M)	21	1.00	0.28 [b]	NS	NS	NS	**
Far-lateral (FL)	22	0.91	0.20		*	NS	**
Anterolateral (A)	22	1.82	0.25			**	*
Posterolateral (P)	22	0.35	0.15				**

NS = not significant at $p<.05$.

* $p<.05$

** $p<.01$

[a] See footnote b, Table 3.

[b] i.e., the control group in Section A.

retention and relearning of a presurgically acquired response; postero-lateral ablations completely eliminate retention and relearning, while ablation of adjacent portions of the lateral telencephalon cause less severe damage. That these deficits are associated with the removal of specific brain tissue is indicated by the differential effects on learning caused by different types of surgery, by the similarity of sham- and unoperated controls and by the lack of learning deficits in operated groups found in two instances (retention in LH chicks, acquisition in F chicks).

Histological analysis

Ranges of the different types of surgery are summarized in Figs. 5 and 6. To construct these diagrams, histological reconstructions of individual chicks in each group were superimposed upon each other: areas removed in approximately 5/6 of chicks are shown by dark shading while areas removed in at least 1/6 of chicks are lightly shaded (these fractions were used to encompass the mean extent of surgery plus or minus one standard deviation). While both LH and EH operations removed hyperstriatum accessorium, -dorsalis, -intercalatus and overlying paleocortical tissue, EH ablations more frequently included hyperstriatum ventrale, parts of adjacent neostriatum (NEO) and medial portions of the hippocampal complex. Frontal ablations included the olfactory bulbs, nucleus basalis, nu. accumbens, parts of the medial septal nucleus, and frontal aspects of paleostriatum augmentatum, NEO and hyperstriatal region. Subdivisions of lateral

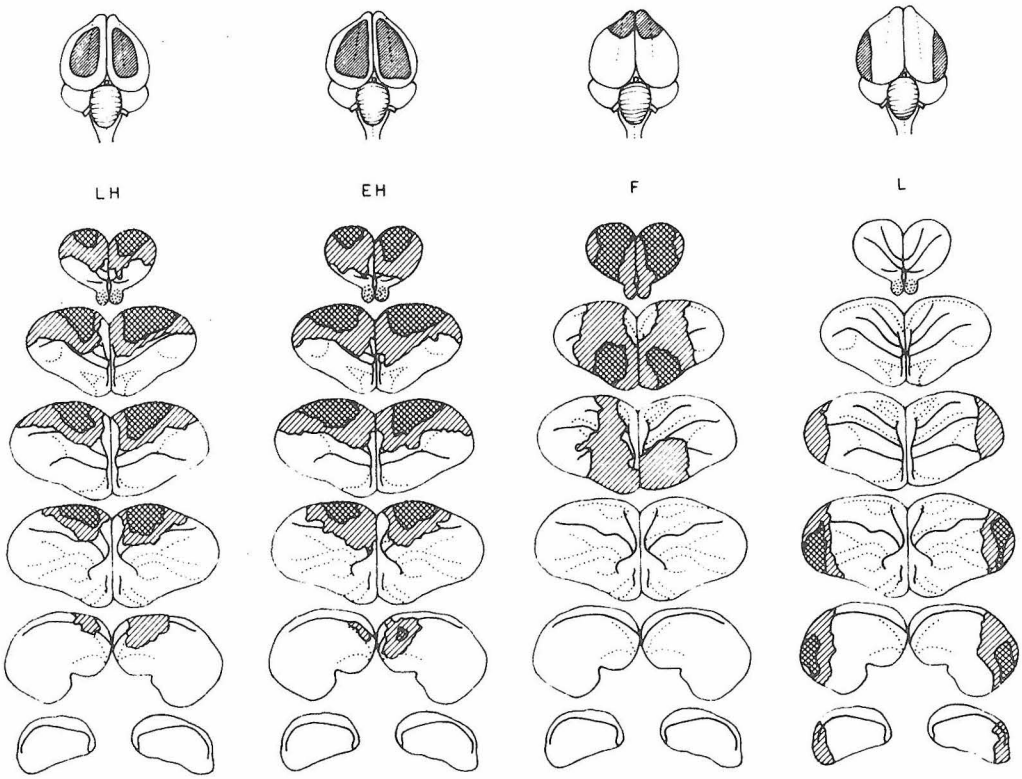


Figure 5. Top row: Dorsal views of ablations studied in Sections A and B. Below: Serial cross-sections showing ranges of ablations. Darkly shaded areas indicate tissue removed in approximately 5/6 of subjects, while lightly shaded areas were removed in 1/6 of subjects examined histologically.

ablations of Section A were: the mediolateral region (M), which included parts of caudal NEO and the ectostriatum (ECTO); the far-lateral region (FL), which included lateral NEO and parts of archistriatum (ARCHI); anterolateral, including rostral ECTO and lateral NEO; and posterolateral, which damaged caudal NEO and much of ARCHI. All four of these ablations also removed overlying corticoid tissue.

As shown in Fig. 7, areas within the ablated regions that were most critically involved in learning were discerned in the posterolateral and mediolateral areas. In these two groups and in frontal chicks, standard deviations around the mean learning scores were found to be very large (see f.n. 1, Tables 3-5). Such variations in learning scores were taken to reflect corresponding variations in extent of surgery. In these cases, brains of chicks showing the least learning were compared with those showing the most learning. In the mediolateral group, chicks that did not learn generally had more damage in posterior and lateral regions of ectostriatum and in caudal neostriatum (Fig. 7a). P and L groups were pooled along with two dorsal posterolateral controls (one of which showed learning while the other did not) to construct Fig. 7b. This figure indicates that chicks showing no retention or relearning tended to have more damage to the ventral posterolateral region, in the vicinity of the archistriatum. No such diagrams were constructed for F chicks since extensive frontal damage made reconstructions difficult to reference.

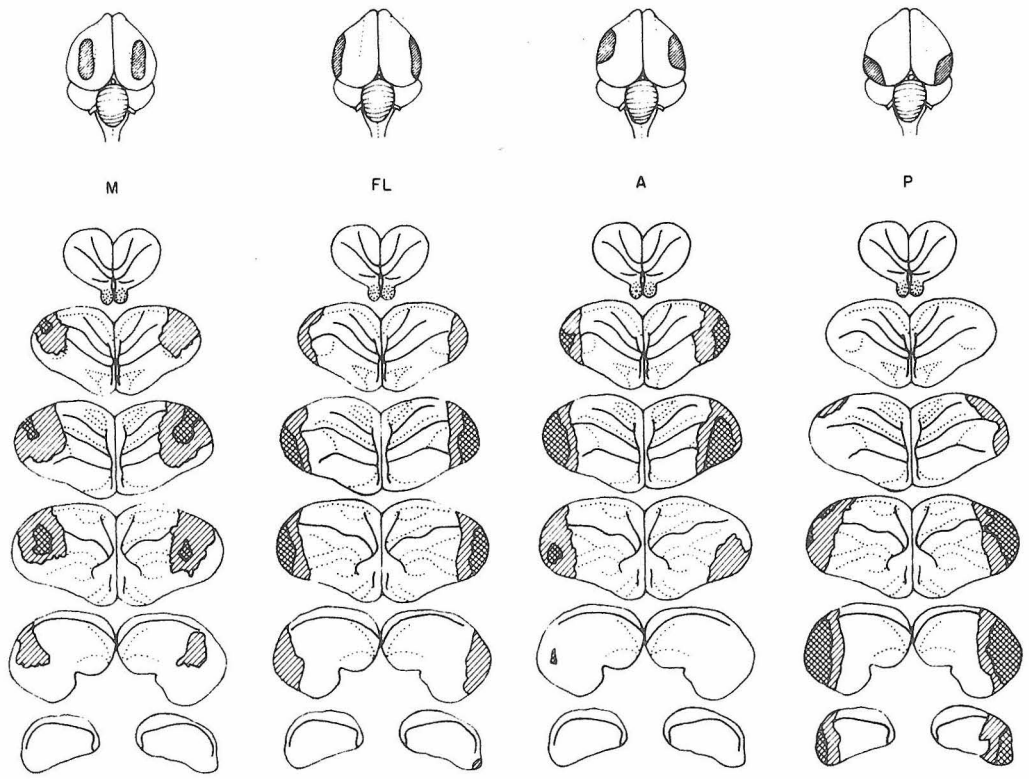
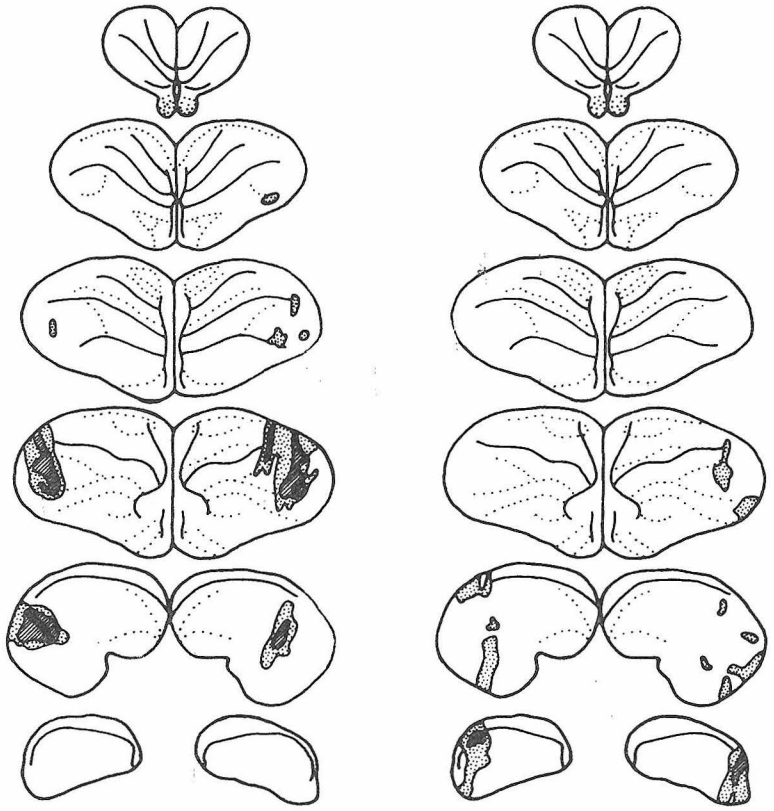


Figure 6. Top: dorsal views of the four subdivisions of the lateral region studied in Section C. Below: range of ablations, summarized from individual serial reconstructions as in Fig. 5.



(a) Mediolateral

(b) Pooled lateral, posterior lateral and dorsal-posterior lateral

Figure 7. Summary diagrams of histology indicate regions more commonly removed in chicks that showed no learning than in chicks that did learn. To construct these, histological reconstructions of four chicks that showed no learning were superimposed on each other and values ranging between 0 and 4 were assigned to regions depending upon the number of chicks having areas removed in common: the same was done for 4 chicks showing significantly higher learning. These two diagrams were then superimposed and values from the second diagram were subtracted from those of the first. A remaining value of 2 (shown by speckled areas) indicates a possible involvement in learning, while values of 3 (hatched areas) or 4 (black areas) present a stronger case.

DISCUSSION

The foregoing results indicate that several of the regions studied here are involved in distinctive aspects of learning. The contrasting effects of the limited dorsomedial (i.e. LH), posterolateral and frontal ablations are of particular interest in that each of these regions includes tissue homologous to a different component of the mammalian limbic system (Ariëns-Kappers et al., 1936; Källén, 1962). While interpretation of effects produced by removal of these and other regions is generally complicated by inclusion of more than one morphologically distinct structure in the ablations, the present findings and others suggest that telencephalic components of birds and mammals sharing a common ancestry play analogous roles in the behavior of the two classes.

The impairment to acquisition but not retention of avoidance learning found in the limited hyperstriatal group may reflect damage to the chicks' hippocampal complex, which lies medial and dorsal with respect to the hyperstriatum. Similar deficits have previously been found in dorsomedially ablated chicks (Lee-Teng & Sherman, 1969) and resemble the impairment to new acquisition but not to retrieval of passive avoidance responses in hippocampal mammals (Douglas, 1967). Consistent with the suggested functional similarity of the avian dorsomedial paleocortex and mammalian hippocampus is the finding that similar ablations in quail (Stettner & Schultz, 1967) and in chicks (Benowitz and Lee-Teng, 1972) damage the reversal but not the initial acqui-

sition of appetitive pattern discriminations, as is also the case for mammals suffering hippocampal damage (Douglas, 1967). These functional analogies conform with the homology of portions of the avian dorsomedial region (i.e. the avian hippocampus and parahippocampus of Ariens-Kappers et al.) and the mammalian hippocampal complex, as indicated by histochemical (Baker-Cohen, 1969) anatomical (Ariens-Kappers et al, 1936) and embryological (Kallen, 1962) studies. While the ablated area also receives thalamofugal visual projections (Karten, 1969), this visual pathway has been shown not to be of great importance for simple pattern learning in pigeons (Hodos et al, in prep). It thus seems more likely that the present limited hyperstriatal deficits primarily reflect hippocampal rather than thalamofugal visual system damage; the nature of the deficits supports this.

With regard to the posterolateral ablations, one plausible interpretation of the complete loss of retention and relearning is that part of this area is necessary for registering the motivational significance of the training. Indistinguishable from this is the possibility that suppression of the instinctive pecking tendency has been lost, in which case pecking may continue despite possible recognition. While it may alternatively be the case that this region is required for gustatory associations of the training, this seems unlikely since disruption of a known feeding network (Zeigler et al, 1969) in anterolateral chicks did not severely interfere with learning. Also, posterolateral chicks regularly showed a normal head-shaking response after tasting the training lure, although this does not of necessity indicate

telencephalic recognition of taste. The observed behavioral deficits are in conformance with the homology of portions of the archistriatum, which was included in the posterolateral ablations, and the mammalian amygdala (Ariëns-Kappers et al, 1936; Zeier & Karten, 1971). Severe deficits to acquisition and sometimes retention of comparable avoidance responses have been reported to follow amygdaloid damage in mammals (Brady et al, 1954; Hovrath, 1963; Ursin, 1965). It would therefore seem that the importance of the amygdala in mediating learned avoidance responses has also remained unchanged in birds and in mammals.

The impairment to retrieval and relearning, but not to new acquisition that results from ablating the frontal telencephalon suggests that removal of this region selectively alters associations or accessibility of presurgically stored information. While the extensiveness of the frontal ablations makes it difficult to attribute these findings to damage of any specific neural structure, these results are nevertheless suggestive of the learning deficits in septal mammals. Several studies have indicated that lesions of the mammalian septal region impair retrieval and relearning of various presurgically acquired avoidance responses but do not affect acquisition of the same behavior in previously untrained animals (Brady & Nauta, 1953; King, 1958; Moore, 1964; Zucker, 1965). However, while basal portions of tissue removed in the frontal ablations are homologous with the mammalian septum (Ariëns-Kappers et al, 1936; Källén, 1962), major portions of the chick's medial and lateral septal nuclei were excluded from the surgery. Thus the functional analogy of the mammalian and avian septal regions sug-

gested by the similarity of the present frontal and mammalian septal deficits can only be considered tenuous.

Deficits found in other operated groups may be related to damage of visual processing areas. Learning deficits in mediolateral chicks appear to correlate with the extent of damage to the primary visual projection area, the ectostriatum (Karten, 1969), which has also been reported for ectostriatal lesions in pigeons (Hodos & Karten, 1970). Impairments to both acquisition and retention following extensive hyperstriatal ablations are also found for discrimination learning in pigeons (Zeigler, 1963) and may be due in part to damage of ectostriatal efferents into ventral hyperstriatum (Hodos et al, in prep.; Karten, 1969) compounding the effects of destroying those structures included in the more limited dorsomedial ablations. The processing of similar types of information by homologous avian and mammalian visual pathways has recently been suggested (Diamond & Hall, 1969; Hodos et al, in prep.). Together with the functional analogies of the limbic system structures suggested above, it seems possible that neural mechanisms upon which learning is predicated in birds and mammals may to some extent have remained unchanged by divergent evolution. However, verification of this rests not only upon further investigating the behavioral functions of seemingly homologous forebrain components in the two classes, but also upon establishing the degree to which corresponding structures are truly homologous, in terms, e.g., of embryology, cell types, pattern of fiber connections, and histochemical properties (Campbell & Hodos, 1970).

EXPERIMENT IV: Contrasting effects of three telencephalic ablations on discrimination learning and reversal[6]

Introduction

Recent neuroanatomical (Karten, 1969; Nauta & Karten, 1970) and embryological (Källén, 1962) data have indicated striking similarities in the prosencephalic structures of birds and mammals. In general, however, it has not been determined whether forebrain structures that are homologous in the two classes play similar roles in learning. Of particular interest in studying comparative physiological bases of learning is the limbic system, which has been strongly implicated in mammalian learning (see reviews: Douglas, 1967; Goddard, 1964; Grossman, 1967), and which appears to be quite ancient phylogenetically (Riss, Halpern & Scalia, 1969).

The present study investigated the involvement in learning of three regions of the chick's telencephalon each of which includes tissue homologous to a portion of the limbic system. Included in the study were the dorsomedial telencephalon, part of which is homologous to the mammalian hippocampus (Baker-Cohen, 1969; Ariens-Kappers, Huber & Crosby, 1936), the posterolateral area, part of which is homologous with the amygdala (Ariens-Kappers, 1936; Zeier & Karten, 1971) and the frontal

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6 This experiment will appear in the Journal of comparative and Physiological Psychology. Dr. Evelyn Lee-Teng is second author.

telencephalon, of which the basal segment includes some septal tissue (Källén, 1962). Following ablations, chicks were trained for the acquisition and reversal of a simple pattern discrimination in an automated apparatus using temperature reinforcement (Lee-Teng & Butler, 1969; Zolman, 1968).

Method

Subjects

A total of 93 White Leghorn cockerels were used. These were obtained from a commercial hatchery when one day old. Chicks were housed with a free supply of food and water in individual units similar to the testing box but without the display keys. The room was maintained at 88° F and 40% relative humidity; room light was on from 6:30 AM to 6:30 PM.

Surgery

Surgery was done on the day chicks arrived. Chicks were held between the ear bars of a small-animal stereotaxic apparatus. Halothane anesthesia (Ayerst Labs) and an alternate fresh air supply were regulated using a foot pedal. Leaving one side of the soft bone flap attached, small triangular incisions were bilaterally placed above the area to be removed. Brain lesions were made bilaterally using gentle suction. Gelfoam (Upjohn) was then inserted in the ablated region, the bone flap was closed and Gelfilm (Upjohn) was placed over the incisions. The skin was drawn together and collodion was applied. Recovery was generally quite rapid as evidenced by casual observation and by the be-

havioral testing the next day. The mortality rate was about 8%, all apparently due to excess anesthesia during surgery. The extent of surgery for the three groups is shown in Figure 8.

Apparatus

The apparatus used for training has been illustrated and described in detail (Lee-Teng & Butler, 1971). Briefly, the chick was put on a wire mesh platform in a small grey-walled box that had a transparent roof. The box, set under a heat lamp, was connected to either a warm or cold air circuit, depending on the positions of two solenoid-activated vanes. On one wall of the box were two adjacent ground-glass keys for stimulus presentation and peck detection. In the base condition, two visual stimuli were back-projected onto the two keys, the heat lamp was off, 54° F cold air flowed through the box, while 95° F warm air (the optimal ambient temperature for baby chicks) bypassed through a shunt duct. Air flow rate in either circuit was set at 1.2×10^{-2} $m^3 sec^{-1}$. Normally, a peck at the key showing the positive stimulus started a 5 sec. reward period during which warm air replaced cold air to flow through the box, the heat lamp turned on, and the visual pattern turned off. A peck at the key showing the negative stimulus started a 2 sec. time-out period during which the visual stimuli were off, but the cold temperature condition did not change. At the end of either the reward or the time-out period, the visual stimuli reappeared and the next trial began. Thus chicks were essentially able to control their own rate of response. The position of the positive stimulus changed between

the two keys according to a balanced pseudorandom schedule that recycled every thirty-two trials. Programming and recording were automated.

Training Procedure

Starting on Day 2 chicks were given a sequence of pretraining, acquisition and reversal training. There were two daily 18 min. sessions, one in the morning and the other in the afternoon, separated by 4 hours. All chicks were first pretrained to a positive stimulus that consisted of a 1.5 mm black dot centered on a 10 mm illuminated disc; the negative stimulus was simply no pattern (i.e., the key showed no back-projected pattern). When a chick reached a criterion level of twelve consecutive correct responses, it was put on the "acquisition" training in the next session.

Chicks were required to discriminate between a horizontal bar (the positive stimulus) and a vertical bar. Unpublished data from this laboratory have shown that there is no innate preference for either orientation. After a chick again reached the criterion of twelve consecutive correct responses, reversal training, in which pecking at the vertical bar was now rewarded, was begun in the following session. Training was terminated after chicks attained criterion level in reversal or when training was not completed after 14 sessions. The latter was generally caused by a very low pecking rate or by a chick's acquiring a position habit.

Over a three month period, weekly groups, each comprised of several types of operated chicks or unoperated controls, were run.

Histology

Following completion of behavioral testing animals were sacrificed and perfused with chick Ringer's solution and Bodian fixative. The brains were sectioned at 15 microns along a constant angle. One-fifth of the sections were retained and were stained with cresyl violet. These were then projected and drawn to allow comparison of the ablations.

Data Analysis

The results of testing were analyzed in three separate ways to fully describe the behavioral changes resulting from surgery. The usual analysis of the number of trials to criterion was supplemented by an analysis of the shapes of the learning curves in order to investigate more specific functional deficits. In addition, since the training procedure allowed chicks to control their own rates of trials presentation, the number of trials per session was analyzed as an independent measure of "motivational" changes.

Analysis of the Learning Curves

To establish whether any of the regions studied were particularly important for early stages of acquisition or reversal of the discrimination, a numerical index of learning curve curvature was determined in each group as follows: For each chick, the number of correct responses in a block of 20 trials was plotted against the block number for both acquisition and reversal. A line was drawn between the point for the first block and the point for the block in which criterion

was attained. The number of points falling above the line was then divided by the total number of points between these two limits. These fractions were averaged for all chicks having the same surgery. Values greater than 0.5 represent an overall convexity of the learning curves, while values less than 0.5 represent concavity.

Results

The experimental results are summarized in Table 6 in terms of trials to criterion, shape of the learning curves and number of trials per session.

None of the operated groups differed significantly from the unoperated controls in the number of trials needed to learn the elementary discrimination used in pretraining, nor in the number of trials per session.

Acquisition

All three operated groups approximated the controls in the number of trials needed to acquire the discrimination. However, the learning curve index of curvature for the posterolateral group was significantly below that of the controls ($t = 2.84$, $p < .01$). This indicates that posterolateral surgery selectively interferes with early stages of acquisition without ultimately affecting chicks' ability to learn the discrimination. The index of curvature for the frontal group's learning curve was almost identical to that of the controls, while that of the dorsomedial group was slightly higher. The frontal

Table 6

Pretraining, Acquisition, and Reversal in Operated and Control Groups

Operation	N	Mean Trials to Criterion			Learning Curves Index of Curvature[a]		Mean Trials per Session		
		Pre-training	Acquisition	Reversal	Acquisition	Reversal	Pre-training	Acquisition	Reversal
Controls	45	16.1	112.3	239.9	.42	.51	72.5	118.0	132.3
Dorsomedial	21	16.3	118.9	371.8**	.50	.43	54.5	110.7	127.8
Posterolateral	14	10.7	118.1	279.9	.24**	.57	105.4	118.8	140.0
Frontal	13	19.7	98.1	301.8	.41	.48	55.1	70.1**	100.9**

[a] Values greater than 0.5 represent a net convexity of the learning curves, values below 0.5, a net concavity.

** $p < 0.02$ (two-tailed t-test) compared with controls; where not stated otherwise, $p < 0.10$ with respect to controls.

group's trials per session, however, was significantly below that of controls, (t = 3.93, p < 0.01), suggesting a motivational deficit independent of learning ability for this group. The other two groups did not differ from controls in this regard.

Reversal

Significant reversal learning deficits were found for the dorsomedial group (compared with the controls, t = 2.66, p < .01, two-tailed); the other two operated groups did not statistically differ from controls in the number of trials needed to reverse the discrimination. While the index of learning curve curvature for none of the operated groups differed significantly from that of the controls, the results were nevertheless of interest: the posterolateral group, which had shown the most concave learning curve in acquisition, exhibited the most convex learning curve in reversal; on the other hand, the dorsomedial group, which had shown the most convex learning curve in acquisition, had the most concave in reversal. Contrasting the changes of the curvature index for these two groups in acquisition and reversal suggests striking differences between the functions of the posterolateral and dorsomedial areas (t = 2.99, two-tailed p < .01).

As had been found in the acquisition, the frontal group again had significantly fewer trials per session than the controls (t = 2.50, p < .02) during reversal training, while the posterolateral and dorsomedial groups did not. This indicates, once again, an apparent "motivational" deficit not affecting the learning ability for the frontal

group.

The chicks were weighed daily. On the whole, operated chicks gained weight steadily, going from an average of 37.6 g on Day 1 to 46.5 g on Day 5, which was indistinguishable from the controls. However, several individuals in the posterolateral group did appear to be aphagic, although the weights of the group as a whole did not differ significantly from the controls.

Histological Analysis of the Operated Chicks

The ranges of the three different types of surgery are shown in the serial cross-sections of Figure 8. These diagrams were constructed by superimposing the histological reconstructions of all chicks given the same type of surgery. Areas included in at least 5/6 of the chicks are indicated by the doubly-hatched zones, while areas included in at least 1/6 of the chicks are singly-hatched. These limits were selected to approximate the mean extent of surgery plus or minus one standard deviation. The frontal ablations generally included some of the medial septal nucleus, the olfactory bulbs, nucleus basalis, nucleus accumbens and frontal aspects of the paleostriatum augmentatum, neostriatum and the hyperstriatal complex. Dorsomedial ablations removed most of the so-called Wulst, which includes much of the dorsal hyperstriatal complex and overlying paleocortical regions, including the hippocampus and parahippocampus. The posterolateral ablations included the archistriatum, some caudal neostriatum and the overlying corticoid areas, including the periamygdaloid cortex. Within the range afforded by the small variabil-

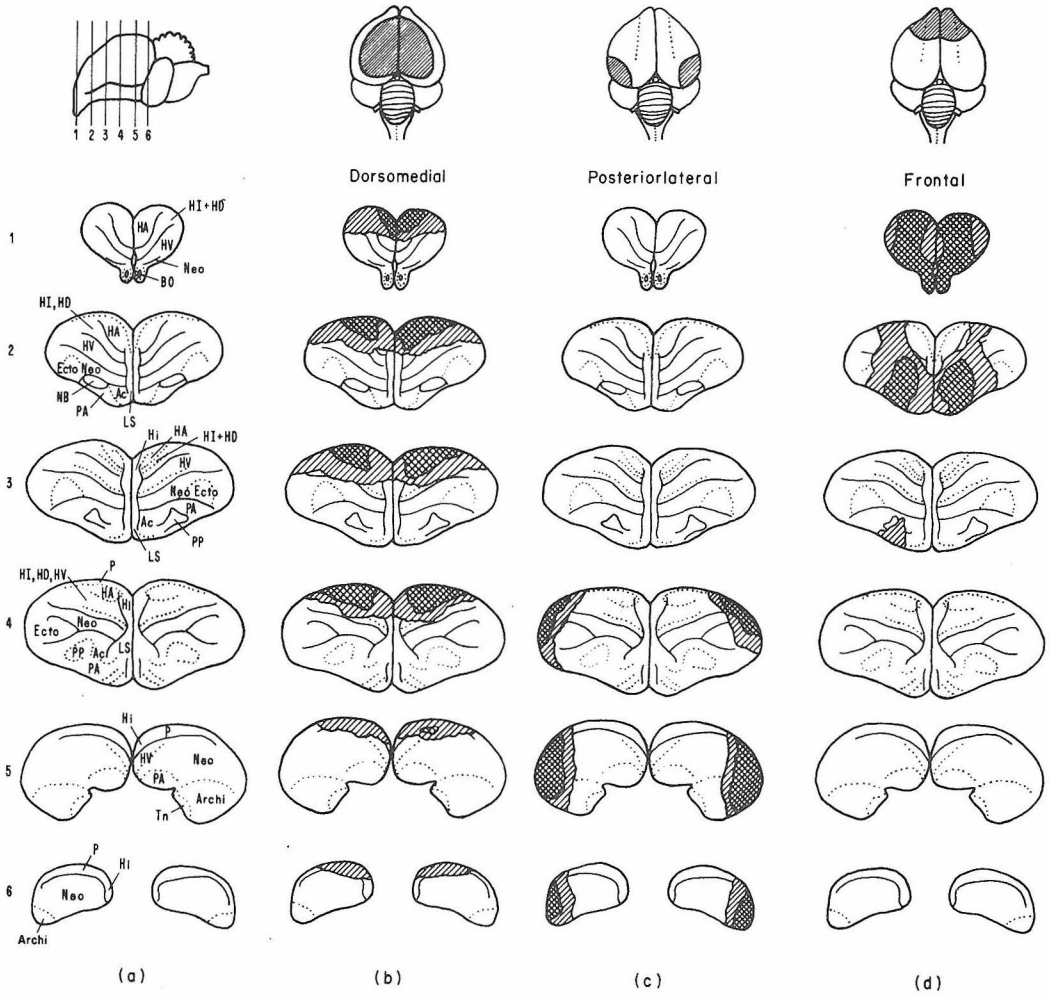

Figure 8. Serial cross-sections through the brain of a 4-day old chick showing normal structures and extent of lesions (Ac = Nu. Accumbens, Archi = Archistriatum, BO = bulbus olfactorius, Ecto = Ectostriatum, Hi = hippocampal area, HA = hyperstriatum accessorium, HD = hyperstriatum dorsale, HI = hyperstriatum intercalatus, HV= hyperstriatum ventrale, MS = medial septal nucleus, NB = nucleus basalis, Neo = neostriatum, P = parahippocampal area, PA = paleostriatum augmentatum, PP = paleostriatum primitivum, Tn = nu. taeniae. See text for details.

ity in extent of surgery, no within-group correlations could be found between the size of lesion and the consequent performance deficits.

Sham-operated controls were not included in the study, as preliminary results had indicated that each of the different ablations causes highly distinctive behavioral changes. The present results confirm this. Furthermore, for each of the three methods of analysis, at least one operated group does not differ from unoperated controls. Thus all of the statistically significant behavioral deficits described above are controlled for and may be attributed to loss of specific brain tissue. In brief, these findings were that dorsomedial ablations impair reversal of the discrimination but not the original acquisition; posterolateral ablations affect early stages of acquisition but do not increase the number of trials required to reach the criterion level, nor do they impair reversal training; frontal ablations decrease the rate of responding but do not influence learning in terms of trials to criterion or shapes of the learning curves ($\underline{p} < .02$ in all cases; where differences are considered insignificant, $\underline{p} > 0.10$).

Discussion

The foregoing findings indicate that the three telencephalic regions investigated contribute towards distinctive aspects of the chick's discrimination learning. While the dorsomedial telencephalon is apparently not required for initially acquiring the discrimination, it does seem to contribute towards the reversal learning. On the other

hand, the posterolateral region appears to be involved in mediating early stages of acquisition, although its removal does not affect the number of trials needed to acquire or reverse the discrimination. The frontal area seems to participate in regulating motivational aspects of performance although it may not be of importance for learning itself.

The three regions studied here each contains tissue homologous with a portion of the mammalian limbic system. The hippocampus and parahippocampus, which are included in the dorsomedial ablations, have been shown from embryological (Källén, 1962), anatomical (Ariens-Kappers et al., 1936) and histochemical (Baker-Cohen, 1969) data to be homologous to the horn of Ammon and associated mammalian hippocampal structures. However, since the dorsomedial region (as well as the regions included in the outer two operations) had initially been selected for this experiment as a gross morphological unit found previously to be involved in avoidance learning (Benowitz, 1972), the surgery has included non-limbic structures as well. Thus, projections of the thalomofugal visual system (Karten, 1969) were damaged by the dorsomedial ablations. However, this system has been shown not to be of importance for simple visual learning (Hodos, Karten & Bonbright, personal communication), and the present learning deficits, which confirm the results of a comparable study of dorsal paleocortical ablations in the quail (Stettner & & Schultz, 1967), are most likely due to the loss of the non-visual, paleocortical structures of the dorsomedial region related to the hippocampus; the specificity of the deficits supports this. With regard to

the posterolateral ablations, parts of the archistriatum included in the surgery are homologous with the amygdala of mammals (Ariens - Kappers, Huber & Crosby, 1936; Zeier & Karten, 1971). The specific mammalian homologies of most of the remainder of the posterolateral region are as yet unknown. Of the tissue removed by the frontal ablations, the basal structures lying ventral and medial with respect to the ventricle are homologous with parts of the septal region of mammals (Kallen, 1962). However, much of the medial septal nucleus was excluded from the surgery.

In conformance with the anatomical homologies, the behavioral deficits found here resemble those reported to follow lesions in homologous limbic system structures in mammals. As was the case with the present dorsomedial ablations, hippocampal lesions in mammals cause deficits to the reversal but not the acquisition of spatial (Kimble & Kimble, 1965; Thompson & Langer, 1963), tactual (Teitelbaum, 1964) and visual discrimination (Douglas & Pribram, 1966; Silveira & Kimble, 1968) problems, although there is evidence to the contrary for visual discrimination reversal in monkeys (Mahut, 1971). On the other hand, amygdaloid lesions in mammals do not affect the number of trials needed to acquire (Schwartzbaum, 1965; Ursin, 1965) or to reverse a visual discrimination problem (Douglas & Pribram, 1966; Ursin, 1965). However, as was found by the analysis of posterolateral chicks' learning curves, Douglas and Pribram (1966) have shown amygdaloid ablations in monkeys to interfere with early stages of discrimination learning but not reversal (These

authors also found an increase in the number of trials to criterion in acquisition). While other results from the learning curve analyses generally did not attain statistical significance when compared with the controls, the changes caused by the dorsomedial and posterolateral ablations were nevertheless consistent with contrasting hippocampal and amygdaloid deficits described by Douglas and Pribram. In part, the lack of significant differences with respect to the controls may be due to an insensitivity of the "index of curvature" for measurement of performance deficits in early stages of learning. Nonetheless, a comparison of the changes in this index between acquisition and reversal for the dorsomedial and posterolateral groups indicates a striking contrast in the roles these two regions play in acquisition and reversal. With regard to the frontal ablations, the motivational deficit seen in these chicks might be attributable to a deficit in thermoregulatory ability, or perhaps a greater excitability or ability to be distracted by other stimuli. Septal lesions in rats have been found to increase the length of time required to learn a maze without affecting the number of learning trials (Thomas, Moore, Harvey & Hunt, 1959), although whether these deficits are attributable to the same source as those of the frontal chicks is not clear. In any case, comparison of the frontal ablations with mammalian septal lesions is difficult since the present surgery has excluded much tissue that is septal and included much that is not.

Together with previous studies based upon chicks' one-trial avoidance learning, the present results suggest a functional similarity

between components of the avian and mammalian limbic systems. Dorsomedial ablations in chicks cause deficits to new acquisition but not to retention of a presurgically acquired passive avoidance response (Benowitz, 1972; Lee-Teng & Sherman, 1969), which has also been shown for passive avoidance behavior in mammals (see review: Douglas, 1967). In contrast to the relatively small contribution of the posterolateral region towards chicks' appetitive learning, ablation of this region causes severe deficits to the retention and acquisition of an avoidance response in chicks, which is similar to, although somewhat more severe than, amydaloid deficits in mammals (Goddard, 1964; Ursin, 1965). It would therefore appear that functions of the phylogenetically ancient limbic system (Riss, Halpern & Scalia, 1969) have to some extent remained unchanged in birds and in mammals despite 300 million years of divergent evolution (Bock, 1969). In such a case, it seems possible that the functions subserved by this system are fundamental to the structure of learning in both classes.

EXPERIMENT V: Bilateral memory for acquisition but not extinction of monocularly presented aversive conditioning.

Introduction

Mounting evidence indicates that memory traces are often laid down in only one cerebral hemisphere in mammals (Doty, Negrao & Yamaga, 1972; Gazzaniga, 1963; Kaas, Axelrod & Diamond, 1967). It has been suggested that the corpus callosum plays an active role in this regard, both by suppressing the unnecessary establishment of bilateral engrams and by allowing an untrained hemisphere access to unilaterally stored memory (Doty et al, 1972). In light of this hypothesis, it is of interest to determine the mechanisms underlying interocular transfer in birds, a class lacking the callosum (Ariens-Kappers, Huber & Crosby, 1936). Although the avian retina projects initially to the contralateral brain stem (Cowan, Adamson & Powell, 1963), birds trained monocularly to perform a response generally show full retention when tested with the untrained eye (Catania, 1965; Cherkin, 1970; Levine, 1945 b; Meier, 1971; Mello, 1968; Moltz & Stettner, 1962). Is memory encoded unilaterally under these conditions and later retrieved by the untrained eye, or is information from each eye somehow projected bilaterally to the forebrain and processed by the two hemispheres in parallel?

The avian dorsal telencephalon, including the hyperstriatal and hippocampal complexes, is critically involved in the acquisition of both approach and avoidance responses (Benowitz, 1972; Zeigler, 1963). While

this region does not include the primary telencephalic recipient of ascending projections from the optic tectum, it may serve in the subsequent analysis of efferents from the visual projection area, the ectostriatum (Hodos, Karten & Bonbright, in preparation; Karten & Hodos, 1970). Thus, by contrasting the learning that takes place using either the eye ipsilateral or that contralateral to a unilateral ablation of the dorsal telencephalon, the present experiment sought to decide between the above two alternatives: i.e., whether memory is encoded only in the hemisphere contralateral to the trained eye, or whether ascending visual information reaches both hemispheres. Yet another less likely alternative could also be tested, namely, that ascending visual information is initially processed in the contralateral hemisphere and subsequently transferred and stored in the ipsilateral side. These possibilities were investigated using chicks' one-trial avoidance learning, a task shown by Cherkin (1970) to transfer interocularly.

Another question considered here was whether or not learning to extinguish the aversive response transfers interocularly. Sechzer (1964) has shown that in cats having visual input restricted to one hemisphere and the corpus callosum split, interocular transfer of a pattern discrimination depends upon the nature of the reinforcement used in conditioning. Studies in both birds and in mammals (see below) indicate that anatomically distinct systems may mediate an aversive conditioning and its reversal (e.g., Douglas, 1967). The question that was asked, then, was whether or not the possible use of anatomically different mechanisms for acquisition and extinction could result in a

difference in interocular transfer for these two tasks?; and, accordingly, is memory stored unilaterally or bilaterally for the extinction?

Materials and Methods

Subjects

Three hundred forty eight White Leghorn cockerels, obtained when one day old from a commercial hatchery, were used in the experiment. Chicks were maintained in individual cartons in a constant temperature (88° F) and humidity (40% RH) room. Food and water were not provided since adequate nutrients are available from the yolk sac through the first four days after hatching.

Surgery

Surgery was done on the day chicks arrived. Operations included either unilateral (N = 77) or bilateral (N = 45) removal of the dorsal telencephalon, including the entire hyperstriatal complex (i.e., hyperstriatum accessorium, -intercalatus, -dorsale and -ventrale) and adjacent medial and dorsal paleocortical structures (i.e., the hippocampus, parahippocampus, prepyriform cortex). The surgery, done under halothane anesthesia, has been described (Benowitz, 1972). Small incisions were made on the skull above the region to be ablated. Tissue was removed using gentle aspiration. Gelfoam (Upjohn) was inserted to replace ablated tissue, the bone flap was replaced, and Gelfilm (Upjohn) was placed over the incisions. The mortality rate was about 10%, resulting from an excess of anesthesia. While still under anesthesia, the right

eye was closed in 37 of the chicks suffering ablation of the right hyperstriatum, the left eye in 40 similarly operated birds. Eyes were kept closed by applying a drop of collodion over the closed lid. Recovery from surgery was generally quite rapid, as evidenced by casual observation and by the unimpaired performance during training and testing the next day. The right eye was also closed in 68 briefly anesthetized unoperated controls on Day-1.

Training and Testing

Chicks were trained in the afternoon of the day following surgery. The training procedure has been described in detail (Lee-Teng & Sherman, 1966). Briefly, chicks were trained to inhibit their instinctive peck response by allowing them to peck at a small metallic bead mounted at the end of a wire and coated with an unpalatable liquid, methyl anthranilate (MeA). Chicks not pecking within 5 sec of presentation or not shaking their heads in a characteristic disgust response within an additional 10 sec were discarded (about 15% of the chicks). The training procedure was followed for 158 chicks having no surgery and both eyes open (Group C_1), 68 having no surgery and the right eye closed (C_2), the 77 unilaterally ablated chicks (Groups E_1 and E_2) and 45 bilaterally ablated chicks (E_3).

Two and a half hours later, chicks were tested for retention of the avoidance response. Five test trials, each separated by a 5 min inter-trial interval, were given under extinction conditions. Each trial consisted of a 5 sec presentation of a dry lure resembling that

used in training. The indicant of retention was the number of trials on which a chick did not peck at the lure.

A half hour after the test session, chicks were again anesthetized in a 3% halothane-in-air mixture. In one fraction of chicks in each group, the eye that had been closed during the first retention test was opened with a drop of acetone to dissolve the collodion, while the eye that had been open was then closed. In the remainder of chicks, no changes were made in the eye open. This procedure was not followed for bilaterally ablated chicks nor for unoperated controls having both eyes open (although 40 of the Group C_1 chicks given a second retention test were given anesthesia between test sessions).

Two hours later, chicks were tested a second time, using the same procedure as described above: 5 presentations of the dry lure, each separated by 5 min inter-trial intervals were given.

Following the behavioral testing, approximately one-fourth of the operated chicks were sacrificed. These were perfused with saline and formalin. The brains were sectioned at 15 microns, retaining one fifth of the sections. Sections were stained with cresyl violet, projected and drawn on to standardized brain outlines to allow comparisons of the ablations.

Results

None of the operations affected naive chicks' tendency to peck during the training trial (the fraction of untrained chicks avoiding

the lure for the bilaterally ablated, unilaterally ablated and unoperated groups was respectively 0.12, 0.11 and 0.10; these do not differ significantly: $\chi^2 = 0.21$, $p > 0.8$). Thus, the avoidance seen in the subsequent retention tests in all cases reflects a learned rather than a non-contingent, surgically induced aversive reaction.

Retention of the acquired response

Although the first five retention trials were presented under extinction conditions, chicks' aversion to the lure seems not to have diminished over the course of these trials. In the unoperated controls, for example (Group C_1, N = 158), the average probability of avoiding the lure in each of the trials was respectively 0.68, 0.72, 0.69, 0.68, 0.61. These values do not significantly differ from each other ($\chi^2 = 1.69$, $p > 0.8$). The five trials can therefore be pooled together to give a statistically reliable measure of chicks' one-trial learning. The average number of avoidances for the different groups is summarized in Table 7. A one-way analysis of variance indicates highly significant between-group differences in learning ($F = 19.4$, $n_1 = 4$, $n_2 = 343$, $p < 0.001$).

Although unilaterally ablated chicks showed a significant deficit, the ability of these chicks to learn was the same regardless of whether they were trained with the eye ipsilateral or contralateral to surgery (comparing the two unilaterally ablated groups with each other, $t = 0.26$, $df = 75$, $p > 0.8$; comparing these groups with controls, $t = 3.09$, $df = 196$, $p < 0.01$ for E_2 vs. C_1; for E_1 vs. C_1, $t = 3.23$, $df = 193$,

Table 7

Retention of the passive avoidance response. Operations were done on Day-1. Chicks were trained in the afternoon of Day-2 and given five retention trials (separated by 5 min each) under extinction conditions $2\frac{1}{2}$ hr later.

Group	N	Ablation	eye open	Avoidances in 5 test trials		Significance of pair-wise comparisons[b]			
				M	σ_m	C_2	E_1	E_2	E_3
C_1	158	none	both	3.33	0.14	NS	**	**	**
C_2	68	none	L	3.26	0.23		NS	NS	**
E_1	37	RDT[a]	L	2.24	0.29			NS	**
E_2	40	RDT	R	2.35	0.30				**
E_3	45	BDT[a]	none	0.89	0.19				

[a] RDT = right dorsal telencephalon, BDT = bilateral dorsal telencephalon

[b] For all 5 groups, $F = 19.44$, $p < 0.001$. Two-tailed t-tests were used for comparisons.

NS = $p > 0.10$

** $p < 0.01$

$p < 0.01$). Bilaterally ablated chicks scored significantly below both unilaterally ablated chicks and controls (Table 7). Unoperated chicks learned the response equally well using either one eye or both (comparing C_2 with C_1, $t = 0.46$, $df = 224$, $p > 0.6$).

Several lines of argument indicate that performance deficits in the various groups reflect damage to specific mechanisms associated with learning. The similar tendency of all chicks to peck at the lure during the training trial indicates that surgery does not affect chicks' arousal level, motivation or vision; this is further supported by the inverse correlation between extent of surgery and the amount of pecking at the lure in the subsequent retention trials. It has been shown previously (Benowitz, 1972) that random brain damage does not produce these effects .

Extinction of the aversive conditioning

Binocularly trained controls (C_1) showed a 46% reduction in avoidance behavior in the second set of test trials as compared with the first ($t = 4.88$, $p < 0.001$). This effect is seen regardless of whether or not chicks are given anesthesia between the two test sessions (comparing 40 Group C_1 chicks given anesthesia with 35 that were not, $t = 0.15$, $p > 0.8$). It would therfore seem that although no extinction had been observed over the course of the previous five retention trials, the earlier testing under non-reinforcing conditions nevertheless did result in an extinction manifest $2\frac{1}{2}$ hr later. A two-way analysis of variance of the scores on the first and second retention tests for all

groups indicates significant learning differences between tests ($\underline{F}$ = 34.01, $\underline{p} < 0.001$) and between groups ($\underline{F}$ = 9.92, $n_1 = 7$, $n_2 = 452$, $\underline{p} < 0.001$. Accordingly, a one-way analysis indicates significant between-group differences in the extinction of the learned response between the two test sessions ($\underline{F}$ = 3.84, $n_1 = 8$, $n_2 = 226$, $\underline{p} < 0.01$).

Monocularly trained controls showed no interocular transfer of the extinction. Unoperated chicks trained monocularly and tested both times with no changes in the eye left open (C_2-s) exhibited an extinction similar to that of binocularly trained controls (comparing the scores on the two tests, $\underline{t}$ = 3.50, $p < 0.001$). In contrast, however, unoperated chicks trained under extinction conditions with the left eye open and subsequently retested 2½ hr later with the right eye open (C_2-d) showed no extinction (comparing this group's scores on the two tests, $\underline{t}$ = 0.14, $\underline{p} > 0.8$).

Unilaterally ablated chicks showed an extinction of the response if the non-reinforced conditioning in the first test session had been presented to the eye ipsilateral to surgery. This was manifest regardless of which eye was used in the second retention test (for E_2-s and E_2-d pooled together, the extinction of the response between the two test sessions was significant at $\underline{p} < 0.02$ (t = 2.57); contrasting the extinction of Groups E_2-d and E_2-s shows this effect to be independent of the eye used to retrieve the extinction: $\underline{t}$ = 0.38, $\underline{p} > 0.6$). In contrast, however, unilaterally ablated chicks trained to extinguish the response with the eye contralateral to surgery showed no significant extinction; this effect did not depend upon which eye was used in

Table 8

Extinction of the avoidance response. The second set of retention trials was given 2 hr after the first. Testing was done under extinction conditions at all times.

Group	N[a]	Ablation	Eye open		Difference between two test scores		Significance of between-group comparisons of extinction $(M_I - M_{II})$[d]						
			first test	second test	$M_I - M_{II}$	σ_m	C_2-s	C_2-d	E_1-s	E_1-d	E_2-s	E_2-d	E_3
C_1	76	none	both	both	1.45	0.19	NS	**	*	**	NS	NS	**
C_2-s	36	none	L	L	1.50	0.25		**	*	**	NS	NS	**
C_2-d	29	none	L	R	0.07	0.33			NS	NS	(*)	(*)	NS
E_1-s	17	RDT[b]	L	L	0.47	0.43				NS	NS	NS	NS
E_1-d	16	RDT	L	R	0.04	0.59					NS	NS	NS
E_2-s	17	RDT	R	R	1.23	0.50						NS	(*)
E_2-d	17	RDT	R	L	1.00	0.37							(*)
E_3	27	both	both	both	0.33	0.24							

[a] chicks were randomly selected from the larger populations shown in Table 7.

[b] see footnote a, Table 7

[c] i.e., mean of first 5 trials minus mean of second set.

[d] for all 8 groups, $F = 3.84$, $p < 0.01$. Two-tailed t-tests used for comparisons.

NS: $p > 0.10$; (*): $0.10 > p > 0.05$; *: $p < 0.05$; **: $p < 0.01$.

the second test session (for Groups E_1-s and E_1-d pooled together, the scores on the two test sessions do not differ: $\underline{t} = 0.69$, $\underline{p} > 0.4$; contrasting the scores of Groups E_1-s and E_1-d shows that this lack of extinction does not depend upon the eye open during the second test: $\underline{t} = 0.45$, $p > 0.6$). Extinction in bilaterally ablated chicks was significantly below that of both unoperated controls ($\underline{p} < 0.01$ comparing E_3 with both C_1 and C_2-s), and unilaterally ablated chicks trained with the eye ipsilateral to surgery ($\underline{p} < 0.05$ for E_3 vs. E_2-s and E_2-d combined together).

In summary then, unilaterally ablated chicks acquire the avoidance response equally well whether using the eye ipsilateral or contralateral to surgery. The learning is somewhat less effective, however, than that of unoperated chicks. In contrast, only those chicks trained through the eye ipsilateral to surgery learn to extinguish the response. This effect is seen regardless of which eye is used to test for the monocularly acquired extinction. Acquisition and extinction in bilaterally ablated chicks is below that of either unoperated controls or chicks with unilateral ablations. In unoperated groups, acquisition and extinction were not affected by whether training proceeded with both eyes or with only one eye open. However, monocularly acquired extinction did not transfer interocularly.

Histological analysis

As indicated by the summaries of the individual ablations, the mean extent of surgery of the bilaterally ablated group was approximate-

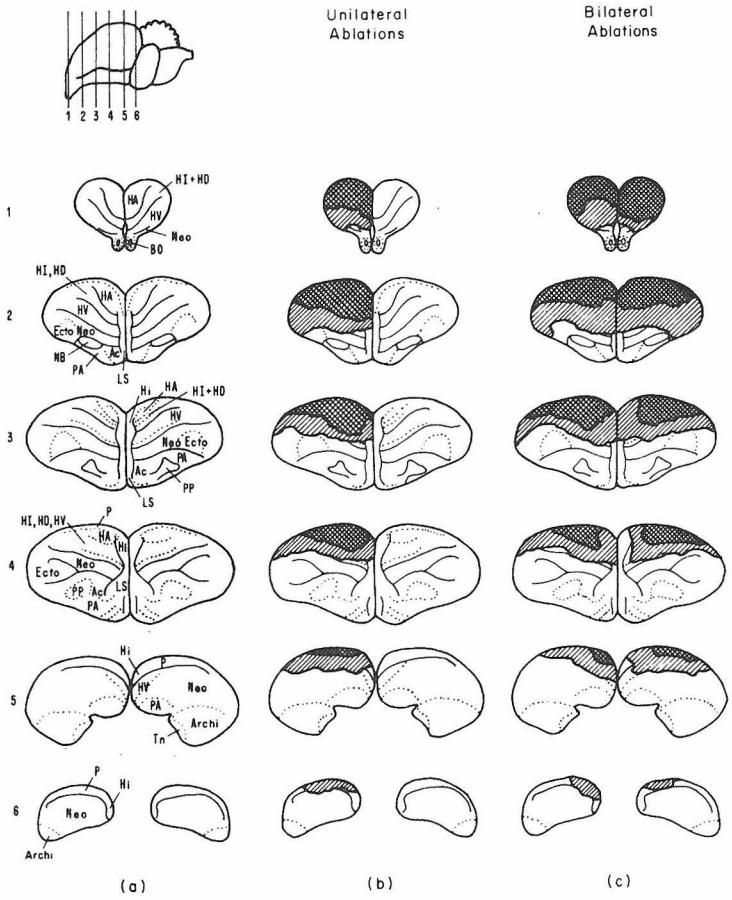

Figure 9. (a) Serial cross-sections through the brain of a 4-day old chick (Ac = Nu. Accumbens, Archi = archistriatum, BO = bulbus olfactorius, Ecto = ectostriatum, Hi = hippocampus, HA = hyperstriatum accessorium, HD = hyperstriatum dorsale, HI = hyperstriatum intercalatus, HV = hyperstriatum ventrale, MS = medial septal nucleus, NB = nucleus basalis, Neo = neostriatum, P = parahippocampal area, PA = paleostratum augmentatum, PP = paleostriatum primitivum, Tn = nu. taeniae). Figures (b) and (c) indicate mean extent of surgery (shown by hatching) plus or minus one standard deviation for unilaterally and bilaterally ablated chicks.

ly twice that of unilaterally ablated chicks. To construct the summary diagrams shown in Fig. 9, histological reconstructions for individual subjects within either group were superimposed upon each other. Boundaries indicate areas removed in at least 5/6 and no more than 1/6 of the chicks in each group. These limits were used to estimate the area included in plus or minus one standard deviation around the mean extent of surgery. Ablations generally included the entire hyperstriatal complex, the hippocampus, parahippocampus and frontal aspects of the neostriatum. Within both the unilateral and bilateral groups, the extent of damage to the frontal neostriatum and to the ventral hyperstriatum vary among subjects. As far as was possible, however, no within-group correlations could be found between extent of surgery and behavioral deficits.

Discussion

The severe impairment to one-trial learning resulting from bilateral dorsal telencephalic ablations confirms earlier reports on the importance of this region for visual learning in birds[7,8] (Benowitz, 1972;

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[7] Acquisition of this task is affected neither by sham surgery nor by extensive frontal ablations that include the olfactory bulbs, nu. accubens and frontal aspects of the hyperstriatal complex, paleostriatum and neostriatum (Benowitz, 1972).

[8] The present bilateral ablations were generally more severe than the extensive hyperstriatal (EH) ablations reported previously (Benowitz, 1972). The former include more hyperstriatum ventrale and hippocampus, as well as more frontal neostriatum. A comparison of the first retention trial for these two groups shows no differences in learning deficits ($\chi^2 = 0.33$, df= 1, $p > 0.50$). This

Pritz, Mead & Northcutt, 1970; Zeigler, 1963). Although each retina projects exclusively to the contralateral tectum and thalamus (Cowan et al., 1963), chicks with unilateral ablations of the dorsal telencephalon acquire the response equally well using either eye. Thus the effects of aversive conditioning presented through either eye seem to become spread centrally to the two hemispheres and stored bilaterally. In contrast, however, extinction learned monocularly does not spread similarly, but is confined to the contralateral hemisphere. This is indicated by the inability of unilaterally ablated chicks to extinguish the response if trained with the eye contralateral to surgery.

The interocular transfer of chicks' one-trial learning (Cherkin, 1970) seems, therefore, to be based upon the establishment of engrams in both hemispheres. As is seen for the extinction conditioning, memory that is stored in only one hemisphere can not be retrieved with the ipsilateral eye in chicks (Doty & Negrao, 1973). Chicks' commissural system would therefore appear to mediate the bilateral projection of ascending information under certain motivational conditions, but does not allow for the interhemispheric communication of unilaterally stored memories (Doty & Negrao, 1973; McCleary, 1961). It has been suggested (Doty et al., 1972) that the presence of the neocommissures in mammals overcomes the unnecessary duplication of engrams in the two hemispheres while giving both sides access to important information that has been

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similarity indicates that there is little effect of training and testing one vs. two days after surgery.

stored unilaterally. However, even in the absence of direct interhemispheric communication, when both eyes are open and different engrams are stored in the two hemispheres of pigeons, the side having the appropriate response encoded can assume dominance (Levine, 1945 a). And in the absence of the dorsal telencephalon on one side, the intact hemisphere dominates regardless of which eye is tested. This is indicated by the unilaterally lesioned chicks' retrieval of the aversive response and later its extinction, both of which are presumably stored in the intact hemisphere, using either eye.

The differential processing of visual information during acquisition and extinction suggests that anatomically distinct systems mediate these two learning processes. Chicks' archistriatum is critically involved in the avoidance learning (Benowitz, 1972), just as the amygdala, which is homologous with part of the archistriatum (Ariens-Kappers et al., 1936; Zeier & Karten, 1971), mediates similar learning in mammals (Goddard, 1964; Ursin, 1965). While the anterior commissure (AC), which interconnects the archistriata of the two hemispheres (Zeier & Karten, 1971) is not important for interocular transfer of appetitive discriminations in pigeons (Cuenod & Zeier, 1967), it may have facilitated transfer of the avoidance conditioning. In support of this, studies in cats have shown the AC to be of importance for the transfer of aversive, but not appetitive conditioning (Sechzer, 1964). On the other hand, the hippocampus has been shown to be particularly important for learning to extinguish a response in mammals (Douglas, 1967), and

studies by the writer indicate that chicks' hippocampal complex plays a role in learning that is, in some respects, analogous to that of its mammalian homologue[9] (Benowitz, 1972; Benowitz & Lee-Teng, 1972). The inability of unilaterally lesioned chicks to acquire the extinction using the eye contralateral to surgery, as well as the lack of interhemispheric transfer of the extinction seen in unoperated chicks, might then be attributable to the involvement of the hippocampus in learning the extinction and to the apparent lack of direct commissural connections between the hippocampi of the two hemispheres. In support of this, it might be noted that learning not to avoid the depth of a visual cliff apparatus does not transfer interocularly (Zeier, 1970). In requiring the reversal of a prepotent response tendency, this learning paradigm might be expected to involve the hippocampus (Douglas, 1967; Kimble, 1968). On the other hand, the hippocampus is not essential for learning the responses which have been shown to transfer (Benowitz, 1972; Benowitz & Lee-Teng, 1972). These tasks include appetitive pattern discriminations under certain conditions of stimulus presentation and one-trial aversive conditioning (Catania, 1965;

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[9] Preliminary studies by this writer indicate the hippocampus to be critically involved in extinction of the aversive response. The number of experimental subjects is small. However, if chicks pecking at 5 sec are considered to indicate $\frac{1}{2}$ avoidance, hippocampal chicks show a significant lack of extinction as compared with controls over three non-reinforced test trials given $2\frac{1}{2}$ hours apart ($N = 10$, $\chi^2 = 6.04$, $df = 2$, $p < 0.05$), despite the fact that the initial acquisition of the aversive response was similar to that of controls ($\chi^2 = 0.10$, $p > 0.7$).

Cherkin, 1970; Levine, 1945 a, b; Meier, 1971; Mello, 1968). While the dorsal supraoptic decussation (DSOD) has been shown to be critically involved in the interocular transfer of pigeons' appetitive learning (Meier, 1971), it is possible that either the avoidance learning and its extinction involve pathways other than this, or that use of the DSOD is differentially facilitated according to motivational aspects of the learning.

Summary and Conclusions

In the introdu ction several reasons had been suggested why basic mechanisms of memory storage and retrieval might be profitably investigated in the chick. Accordingly, the foregoing studies have examined the properties of neurobiological phenomena underlying the formation of a memory trace and the manner in which various brain structures participate in chicks' information processing. In many instances the results from these experiments provide a simple model of mechanisms which, in a modified form, are incorporated into the intelligence of more complex vertebrates.

The two experiments of Part I examined the nature and interrelationship of various neurobiological representations of memory effected by a training experience. From these and other experiments in chicks a simple model of memory storage mechanisms emerges. It would seem that immediately following a training experience, a metastable memory trace is activated which is then maintained at a constant intensity. Within 45 sec of training, this induces the growth of a semi-permanent memory trace which is insusceptible to electroshock. The kinetics describing the growth of the semi-permanent memory trace, along with the observation that a single training experience causes saturation of chicks' capacity to learn the response task (Benowitz, 1972) indicate that some restricted neurobiological substrate becomes saturated as the memory trace is formed. Over a period of hours, the electroshock-insensitive trace, which is not itself expressed behaviorally, undergoes one

or more subsequent transitions into long-term memory (Cherkin, 1969; Lee-Teng et al, 1970); the trace is therefore referred to as Pre-LTM. During this transitional period, the ongoing activity of the process which induced the growth of Pre-LTM appears to serve as short-term memory[10] (McGaugh & Dawson, 1971). Upon a second training, the induction process is re-activated and causes the formation of Pre-LTM to continue at a rate determined by the amount of memory substrate remaining to be saturated. Results from the memory additions study demonstrate that even in the absence of conscious associations, engrams for a succession of similar experiences are recorded, in a sense, in register with each other.

The manner in which various cerebral structures contribute towards chicks' learning was examined in Part II. While some of the results reflect the participation of sensory-specific structures in information processing, the more interesting findings demonstrate the importance of telencephalic components that do not recieve direct sensory afferentation. Among the latter class of structures that were investigated, chicks' hippocampal complex seems to be involved in the encoding of memory in situations for which a response tendency has already been established. Ablation of this structure selectively impairs the rever-

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[10] Although transcranial electroshock disrupts the mechanism inducing the the formation of Pre-LTM, less extreme methods of temporarily halting ongoing electrical brain activity do not (Soltysik, 1972). Thus although short-term memory is not stored by means of reverberating electrical circuits, it may be represented by means of some metastable configurational changes, perhaps occuring on neuronal membranes.

sal of a pattern discrimination without affecting acquisition of the original problem, impairs the acquisition but not retrieval of a passive avoidance response, and seems also to be important for extinction processes. On the other hand, chicks' archistriatum appears to participate in recognizing the significance of stimuli in proportion to their motivational importance and novelty. Ablation of this region, which includes the amygdala, destroys chicks' ability to acquire or retrieve a passive avoidance response and impairs early stages of acquiring an appetitive discrimination. Another region studied, the frontal telencephalon which contains part of the septal region, seems to be involved in the regulation of chicks' motivational level, although not the ability to learn, in the performance of a pattern discrimination. Destruction of this area also alters motivational associations of a pre- but not of a post- surgically acquired passive avoidance response.

As with mammals, the ascending visual pathways to birds' telencephalon seem to be essential for memory storage and retrieval. Destruction of the ectostriatum, which receives projections from the optic tectum via the nucleus rotundus (Karten & Hodos, 1970; Revzin & Karten, 1967), severely impairs pigeons' appetitive discrimination (Hodos & Karten, 1970). Results from the anterolateral and mediolateral groups in Experiment III suggest this to be the case for chicks' avoidance learning as well. Yet the results discussed above suggest that cognition is related not only to the telencephalic projections of ascending visual information, but also to complex associative or affective factors which seem to involve components of the limbic system[11]. Accordingly, differ-

ences in affective or associative factors can result in the use of anatomically distinct cerebral mechanisms for the processing of information in various learning situations. For example, the foregoing results have shown that while the initial learning and subsequent reversal of a pattern discrimination would seem to have comparable visual and even reinforcement properties, differences in associative factors result in the use of anatomically distinct systems for the analysis of these two learning situations (Douglas & Pribram, 1966). On the other hand, part of the brain essential for the registration of an aversive experience is not particularly important for aquiring or reversing a pattern discrimination (Ursin, 1965). And the results of the unilateral ablations and interocular transfer experiments indicate that the mechanisms used in acquiring the aversive response task differ from those used in learning the subsequent extinction.

A comparison of the results from Part II with deficits resulting from damage to structures in the mammalian nervous system indicates that homologous components of birds and mammals play similar roles in the two classes' information processing (Benowitz, 1972; Benowitz & Lee-Teng, 1972). Furthermore, the behavioral significance of various visual structures of the two classes' telencephala also seems to be similar (Hodos et al., in preparation; Casagrande et al., 1972; Hodos

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[11] Preliminary investigations on the participation of chicks' limbic system in several formalized behavioral tasks assessing motivation are reported in Appendix B.

& Karten, 1970; Diamond & Hall, 1967). The ensemble of these analogies suggests that functions subserved by various cerebral components in the bird represent fundamental elements of all higher vertebrates' information processing.

Appendix A

Embryogenesis of the chick's telencephalic nuclei and the homologization of these nuclei with respect the the mammalian forebrain.

Using the nomenclature of Kallen, letters a, b, c and d refer to the original ventricular positions of cell columns in the neural tube: d = pallial cell column (dorsal); a, b and c are subpallial, c being the most dorsal of these. Numerals I, II, III, IV refer to order of proliferation within columns. Subscripts: d = dorsal, v = ventral, l= lateral, m = medial, ext = external, int = internal. While common names of avian nuclei are those of Ariens-Kappers, Huber & Crosby, the suffix 'striatum' is frequently a misnomer.

Structure in avian telencephalon	Cellular derivative	Mammalian homologue
medial septal nucleus	a	medial septal nucleus, nu. septo-hippocampalis
nu. diagonal band	a_{ext}	nu. diagonal band
archistriatum mediale	part of b^{II}	nu. medialis amygdalae
	c^{I}_{ext}, caudal	nu. centralis amygdalae
part of paleostriatum augmentatum	c^{I}_{int}, caudal	nu. corticalis amygdalae
archistriatum posterior	d_v	nu. basalis amygdalae, claustrum
lateral septal nucleus	b_m	lateral septal nucleus
area parolfactoria	c_m	

Structure in avian telencephalon	Cellular derivative	Mammalian homologue
	b^{I}	
nu. accumbens	b^{II}	tuberculum olfactorium, nu. accumbens, head of caudate
	c_{v}^{II}	
nu. basalis	c_{ext}^{I}, rostral	bed of stria terminalis, nu. tr. olfactorius lat.
paleostriatum augmentatum	c_{d}^{II}	caudate-putamen
paleostriatum primitivum	c_{int}^{I}, rostral	globus pallidus
hyperstriatum accessorium, hippocampus, hippocampus p. dorsalis, prepiriform area	$d^{I} + d^{II}$	hippocampal complex, other paleocortical zones
hyperstriatum intercalatus, -dorsale	$d_{d''}^{III}$	
hyperstriatum ventrale	$d_{d'}^{III}$	
neostriatum	d_{v}^{III}	Neocortex
ectostriatum	d_{1}^{III}, rostral	
archistriatum anterior, -intermedium	d_{1}^{III}, caudal	

References: Ariens-Kappers et al, 1936; Jones & Levi-Montalcini, 1958; Kallen, 1951 a, b, 1962; Kuhlenbeck, 1938; Northcutt, 1969; Zeier & Karten, 1971.

Appendix B

Motivational changes following forebrain ablations in the chick

To further investigate the behavioral significance of telencephalic regions studied in Experiments III and IV, several additional behavioral tests were given. These tests were developed from casual observations on the instinctual tendencies of precocial chicks. No comprehensive interpretation of the roles played by various telencephalic structures emerges from combining these results and those from Experiments III and IV. Further observations and restriction of lesions to more discrete anatomical units will be necessary to accomplish this. However, these data do provide some information on the behavior of normal chicks and place constraints on interpreting the functions subserved by the different regions.

Chicks similar to those described in Experiments III and IV were used 2 to 4 days after hatching. The behavioral tasks are described here.

Visual cliff Chicks were placed on a narrow platform, about 1" above a large sheet of transparent plexiglass. On one side of the platform lay a black and white checkerboard pattern directly beneath the transparent sheet, while on the other side a similar pattern lay 3' below the surface of the plexiglass. The translucent patterns were illuminated from below using a fluorescent lamp. To induce chicks to make a choice between the two sides of the apparatus, a mild foot shock was delivered after chicks had remained on the platform for 5 sec. Two trials were

given to each chick.

Following Chicks were placed between two concentric cylinders (inner diameter = $1\frac{1}{2}$', outer = $2\frac{1}{4}$') having very high walls to prevent distraction. A green foam rubber object connected to an arm leading to a motor was moved between the cylinders for 2 min. In the first minute the object moved in one direction at a constant speed while in the second minute its direction was reversed each time the chick stopped following it. The total number of seconds in which a chick followed is reported.

Pecking activity and Pecking Preference Chicks were placed in a small cubicle similar to that described in Experiment IV. Two windows had back-projected patterns presented simultaneously: a small triangle and an irregular polygon having many sharp edges. Patterns were randomly alternated between the two windows after each peck. No external rewards were given for pecking,but a high response rate was observed nevertheless. The number of pecks at each of the patterns was recorded automatically. The total number of pecks to both patterns in 10 minutes, as well as the ratio of pecks to the jagged pattern divided by the total number of pecks, are reported.

As summarized in Table 9, both the dorsomedial and frontal ablations caused severe impairments to chicks' aversion of depth, while the posterolateral ablations did nd. None of the ablations affected chicks' following, pecking activity or preference for the jagged pattern over the triangle.

Table 9

Results of the Behavioral Testing

Operation	Structures included in surgery[a]	Visual cliff[b]	Following (sec)	Pecking	
				total	ratio
Dorsomedial	hippocampus, hyperstriatum ventralis, dorsalis, access.	0.42**	26.5	178.1	0.65
Posterolateral	Archistriatum (incl. amygdala), posterior neostriatum, overlying corticoid zones	0.03	28.4	135.0	0.74
Frontal	Frontal portions of neostriatum, hyperstriatum ventrale, septal nuclei	0.42**	29.6	157.7	0.67
Unoperated	(none)	0.13	26.8	180.8	0.66

[a] The surgery is similar to that described in Experiment IV. A more complete description of structures damaged is reported there.

[b] Probability of going off deep side.

** compared with controls, $p < 0.01$ using a two-tailed t-test. Where significance is not indicated, $p > 0.2$.

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