ACQUISITION AND REVERSAL OF A TWO MANIPULANDA DIFFERENTIATION IN SHAM, NEOCORTICALLY, AND HIPPOCAMPALLY LESIONED RATS

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Sham, neocortically, and hippocampally lesioned rats were examined in the acquisition of a two manipulanda differentiation under conditions which insured that either the absolute number of reinforced or non-reinforced responses each S emitted on each manipulandum during each experimental session were equated. Acquisition performance was not differentially affected by the three lesions, nor by equated reinforced or non-reinforced responding. Reversal performance did not differ for the neocortically and hippocampally lesioned Ss, but both appeared to be facilitated when compared to sham control Ss. As in acquisition, equated reinforced or non-reinforced responding did not differentially affect performance of the three lesion groups.

INTRODUCTION

The hippocampus, nestled in the inner folds of the temporal lobe, has been subjected to more intense experimental investigation within the past decade than in all previous years combined (after Douglas, 1967). The early neuroanatomical investigations of Papez (1937) which first linked the paleocortical structures of the rhinencephalon with emotional behavior were supported by the contemporary experimental investigations, performed by Kluver and Bucy (1939), of the effects of temporal lobectomy on behavior. MacLean (1954, 1955, 1957, 1958) formalized this orientation, postulating a dichotomy between the phylogenetically older paleocortex and the more recent neocortex. The former was presented as involved in a variety of emotional and visceral functions, while the latter was thought to be concerned with more cognitive functions. However, as the hippocampus was subjected to more intense experimental investigation a more complex picture of hippocampal function emerged. Before summarizing the results of more recent investigations of the role of the hippocampus in behavior and, concurrently, reviewing the physiological theories of hippocampal function which have emerged from those data, a general description of the hippocampus and its' interconnections with other brain structures will be provided.

The hippocampal formation, composed of the hippocampus (Ammon's horn), the hippocampal gyrus, the fascia dentata and the fornix, lies along the medial and ventral border of the temporal lobe where it is wrapped

around the posterior surface of the thalamus. The hippocampus, reminiscent of the common sea horse Hippocampus hippocampus from which its name is derived, is the major structural component of the hippocampal formation. Gross description of the hippocampus was provided by Lorente de No (1934, cited in Douglas, 1967) who divided it into four segments: CA1, located proximal to the subiculum, CA2, CA3, and CA4, lying in the fold of the granule cell layer of the fascia dentata. The most generally accepted cytoarchitectonic description of the complex internal structure of the hippocampus was provided by Cajal (1955). Starting from the ventral surface above CA2, then proceeding vertically, seven major layers are evident: the ventricular ependyma, alveus, stratum oriens, stratum pyramidale, stratum radium, stratum lacunosum, and stratum moleculare. A detailed exposition of the internal morphology of the hippocampus and hippocampal formation is provided by Meissner (1966).

Two major afferent pathways serve the hippocampus: the alvear path through the fornix system and the perforant path through the subiculum. The fibers of the fornix arise primarily in the septal area and the intralaminar nuclei of the thalamus (Green & Adey, 1956). The system is more involved than this, however, for inputs also reach the hippocampus from the ascending reticular activating system of the midbrain and thalamus (Green & Arduini, 1954) and from the hypothalamus as well (Feldman, 1962). The perforant path reaches the hippocampus by way of the entorhinal cortex and subiculum. The temporoamonic tracts pass from the entorhinal cortex through the subiculum to the hippocampus proper. The entorhinal cortex, in turn, receives its afferents from considerable areas of the neocortex (Green, 1964). In addition, there is

evidence for direct fibers from the cingulum attaining the hippocampus via the perforant pathway (Adey, 1961).

Fibers passing into the fimbria constitute the main efferent pathway of the hippocampus. These fibers cross to the contralateral hippocampus via the hippocampal commissure, or enter the fornix and project variously to the septum, hypothalamus, anterior thalamus, and rostral portions of the brain stem (Green & Adey, 1956). The hippocampus also gives rise to efferent fibers to the entorhinal area via the temporoamonic pathway. In addition, Gloor (1960) presents evidence for primary hippocampo-amygdaloid fibers as well as secondary amygdalo-hippocampal connections. An intensive review of the literature pertaining to the neuroanatomical investigation of the hippocampal afferent and efferent systems may be found in Green (1964) and Stumpf (1965).

In an attempt to assess the contribution of the hippocampus to the physiological substrata of behavior, researchers have examined the effect of hippocampectomy upon a wide variety of behaviors. One of the most studied classes of behavior falls under the general notation of avoidance conditioning. Interest was sparked in this paradigm by the relatively early study of Kimura (1958) who found that rats with bilateral posterior hippocampal lesions were deficient when compared to neocortically lesioned and sham operated subjects (Ss) in their ability to withold a well-practiced, food motivated approach response following the introduction of punishment (electric shock) of the consumatory response. The essential characteristics of this experimental paradigm are prototypic of what is generally referred to as passive avoidance conditioning. Such deficits in passive avoidance have since been replicated under

varying conditions in numerous studies (e.g. Isaacson & Wickelgren, 1962; Kimble, 1963; Teitelbaum & Milner, 1963). Those studies which have failed to replicate these findings have reported restricted lesions involving only the dorsal portion of the hippocampus (Poitano & Isaacson, 1966; Kvein, Setekliev & Kaada, 1964), or have employed a response of low probability (Kimble, Kirkby & Stein, 1966; Winocur & Mills, 1969). It is possible to construct various explanations of the underlying nature of the observed hippocampectomy-induced deficit; one such suggestion is that hippocampal lesions in some way vitiate the aversive effect of the punishing stimulus. Such a position would lead to the prediction that hippocampectomized Ss would be deficient in a wide range of shock motivated behaviors. This does not appear to be the case, however, for hippocampectomized Ss are not necessarily retarded when compared to control Ss in their ability to master a variety of active avoidance tasks.

In the typical one-way active avoidance paradigm Ss is required to move from one compartment of a shuttlebox to a second in the presence of a warning signal to avoid an aversive stimulus is delivered and S must then perform the response in order to escape from it. Following completion of the trial S is returned to the original compartment of the shuttlebox and the procedure repeated. Although Niki (1962) reported that destruction of the hippocampus had no effect on this variation of avoidance conditioning, more recent investigations have indicated that there is a lesion-induced deficit which, however, appears to be of a lesser relative magnitude than that found in the passive avoidance paradigm (McNew & Thompson, 1966; Olton & Isaacson, 1967).

If the above avoidance paradigm is modified so that S is not returned to the original compartment of the shuttlebox following each trial but instead must return to it as the response in the following avoidance trial the task is reterned two-way active avoidance. When compared to neocortically lesioned and sham operated Ss, hippocampectomized Ss appear to be facilitated in the acquisition of this response (Isaacson, Douglas & Moore, 1961). It has been suggested that facilitation is due to the presence of a passive avoidance component involved in the two-way active avoidance paradigm which interferes with acquisition in control Ss (S is required to return to the compartment which has most recently been associated with aversive stimulation). Hippocampectomized Ss which have been demonstrated to be relatively impervious to the effect of the contingencies necessary for the instatement of the passive avoidance response are not so hampered and consequently acquire the two-way active avoidance response more readily (Douglas, 1967). The slight deficit seen in the hippocampectomized Ss' acquisition of the one-way active avoidance task can also be related to the deficit hippocampectomized Ss manifest in passive avoidance. Here, however, the hippocampectomized Ss' tendency not to avoid the compartment associated with aversive stimulation retards acquisition relative to control Ss (Olton & Isaacson, 1967).

A second formulation of the underlying nature of the hippocampal contribution to behavior which, like the aversive stimulus position, relates to the passive avoidance deficit suggests that hippocampal lesions enhance the reinforcing properties of appetitive stimuli or,

alternatively, holds that the hippocampal lesion in some fashion elevates drive level relative to non-hippocampectomized Ss under equal levels of deprivation. Jarrard (1968) has briefly reviewed the literature which supports this position and points out that in addition to the intimate connections of the hippocampus with structures important for physiological homeostasis, behavioral evidence indicates that hippocampectomized Ss are more active in both novel and non-novel situations, increase their response rate for food and water, and show slower extinction of a food-motivated running response. Although hippocampectomized Ss have not been found to eat more food than control Ss, they have been found to drink more water. It has been argued that the increased drive hypothesis has generally been abandoned (Douglas, 1967). However, the arguments marshalled against this position have stressed the findings of the avoidance conditioning paradigms and reasoned that because hippocampectomized Ss do not appear to be more sensitive to the drive-inducing properties of aversive stimuli than do intact Ss, it is inappropriate to posit that the reinforcing or drive reducing properties of appetitive stimuli might differentially affect hippocampectomized and normal Ss. Such a critique is cogent only if the theorist holds that a unitary or one-process theory of hippocampal function will explain the whole spectrum of lesion-induced behavioral anomalies. Whether it is possible to formulate a one-process theory of hippocampal function has yet to be demonstrated.

The deficit seen in the passive avoidance performance of hippocampectomized Ss has also been viewed as a manifestation of a general tendency towards response perseveration or, alternatively, an inability to inhibit responses. A considerable body of evidence is available in support of such a position. Correll (1957) found that cats subjected

to bilateral hippocampal stimulation during the acquisition and extinction of a food-motivated straight alleyway running response showed no difference in rate or acquisition when compared to control Ss but did require a greater number of trials in extinction. This finding has been reliably replicated in rats following hippocampal destruction (Jarrard & Isaacson, 1965, Raphelson, Isaacson & Douglas, 1966). A closer examination of the phenomenon indicates that the interval between extinction trials is an important variable to be considered, for while the increased resistance to extinction is demonstrable when trials are spaced, the hippocampal lesion deficit disappears in the massed presentation of the extinction trials (Jarrard & Isaacson, 1965; Jarrard, Isaacson & Wickelgren, 1964). Both Peretz (1965) and Douglas and Pribram (1966) have reported that hippocampectomized Ss show shorter response latencies and a greater number of responses to extinction than do control Ss. Increased resistance to extinction has also been demonstrated in the two-way active avoidance paradigm (Isaacson, Douglas & Moore, 1961).

However, Schmaltz and Isaacson (1967) have presented slightly divergent findings concerning the performance of hippocampectomized Ss in extinction. They ran hippocampally lesioned and control Ss to complete extinction in as many 30-minute free operant sessions as were required for the attainment of their stringent criterion. No difference was found between the experimental and control Ss in the total number of sessions required for extinction. In addition, the hippocampectomized Ss showed shorter response latencies in only the first extinction session; no differences between groups were found for any of the subsequent sessions.

Kaplan (1967) has reported that hippocampectomized Ss show faster extinction of a freezing reaction taken as indicative of a classically conditioned emotional response.

The general inability of hippocampally lesioned Ss to inhibit responses has been widely demonstrated in a number of other situations. Ellen and Wilson (1963) found hippocampectomized rats impaired in their ability to inhibit one type of bar press and adopt a second following a change in the response requirements for reinforcement. Both Niki (1965) and Swanson and Isaacson (1967) have demonstrated a hippocampal lesion-induced deficiency in yielding to stimulus control following the initiation of S^D-S^Δ training. However, the latter authors also demonstrated that hippocampectomized Ss could readily acquire the discrimination provided they were not subjected to a long past history of continuous reinforcement for responding prior to the initiation of discrimination training. Clark and Isaacson (1965) found that hippocampectomized Ss were less efficient than control Ss on DRL schedules of reinforcement. A follow-up study by Schmaltz and Isaacson (1966) presented findings analogous to those of Clark and Isaacson (1965), indicating that hippocampally lesioned Ss could perform well on DRL schedules if not first subjected to prolonged crf training.

The apparently critical role of past learning in the demonstration of hippocampal lesion-induced deficits in discrimination and DRL performance suggested to some that the hippocampus was not involved in the inhibition of behavior in general, but was more specifically necessary for the inhibition of well practiced responses. The demonstrations by Kimble, Kirkby and Stein (1966) and Winocur and Mills (1969) that

hippocampectomized Ss showed no deficit in their ability to inhibit an unlearned escape response from a small, elevated perch when the response was punished lead to their formal statement of that position. However, Isaacson, Olton, Bauer and Swart (1966) and Teitelbaum and Milner (1963) have presented contradictory data, indicating that hippocampectomized Ss are deficient in withholding a naturally occurring response involving a step-down from a platform to an electrified grid. The former authors, who shook the platform to increase the probability of response occurrence, suggested that the escape response employed by Kimble, Kirkby and Stein (1966) was too weak or improbable in nature to adequately reveal a hippocampal lesion-induced deficit.

Inhibitory deficits of hippocampectomized Ss have also been widely examined within the context of exploration and spontaneous alternation paradigms. Roberts, Dember and Brodwick (1962) compared exploration rates of hippocampectomized and control Ss in T- and Y-mazes and found no differences in initial rates, but a more rapid decrease in exploration rate in control than in lesioned Ss. An additional analysis revealed that Ss with small hippocampal lesions showed a moderately, but significant, slower exploration rate decrease than controls, and that Ss with massive hippocampal destruction showed no rate decrease whatsoever. Leaton (1965) studied opportunity for exploration as a reinforcer of a T-maze turning response and found evidence for acquisition in normal and sham operated Ss while hippocampectomized Ss were unable to overcome perseverative tendencies and consequently showed no acquisition effect. Forced training was instituted in the second phase of the experiment and measures of running speed were taken. The hippocampectomized Ss showed slower habituation to the reinforcer, indexed by a slower decline in

running speed over trials than control Ss. Kirkby, Stein, Kimble and Kimble (1967) examined perseveration of a T-maze response as a function of goal-box confinement. With short confinement periods (50 seconds) hippocampal lesioned Ss showed perseverative behavior while control Ss spontaneously alternated their responses on successive trials. With longer confinement periods (10 and 50 minutes) both hippocampectomized and control Ss demonstrated spontaneous alternation. A supplementary analysis revealed hippocampectomized Ss' perseverate responses per se rather than responses to specified locations.

Studies of the effect of hippocampal lesions upon maze learning have yielded rather consistent results. In general, the hippocampal lesion-induced deficit is slight, if present at all, in very simple mazes, but as maze complexity increases the lesion-induced deficit in acquisition becomes increasingly more manifest. These findings have been attributed to the hippocampectomized Ss' inability to inhibit the reentry of previously explored blinds and the greater frequency of blinds in progressively more complex mazes (Kaada, Rasmussen & Kvien, 1961; Kimble, 1963; Kimble & Kimble, 1965). Hosteller and Thomas (1967) have demonstrated that the hippocampal deficits in maze learning cannot be attributed to enhanced thigmotaxis. The hippocampal lesion-induced changes in spontaneous alternation and maze performance suggested to Kimble and his co-workers (Kimble, Kirkby & Stein, 1966; Kirkby, Stein, Kimble & Kimble, 1967) that hippocampectomized Ss suffer from a reduced rate of information acquisition. This position is incomplete, however, for it fails to account for the unimpaired acquisition rates hippocampectomized Ss demonstrate in alternative learning paradigms.

Although hippocampectomized Ss appear deficient in their ability to withhold responses in successive or go-no go discrimination problems (Kimble, 1963), numerous studies have demonstrated that they do not differ from control Ss on a wide variety of simultaneous discrimination problems (Allen, 1940, 1941; Brown, Kaufman & Marco, 1969; Grastyan & Karmos, 1962; Hirano, 1966; Kimble, 1963; Kimble & Zack, 1967; Swann, 1934, 1935; Teitelbaum, 1964; Webster & Voneida, 1964). When hippocampectomized Ss are required to reverse such a discrimination, a pronounced deficit in shifting responding from that which was previously reinforced to that which is newly reinforced is regularly observed (Brown, Kaufman & Marco, 1969; Kimble & Kimble, 1965; Rabe, 1963; Stutz & Rocklin, 1968; Swanson & Isaacson, 1967; Teitelbaum, 1964; Thompson & Langer, 1963).

In an attempt to explain the changes observed in positively reinforced behavior following hippocampectomy in terms of the loss of a single process contributing to such behavior in the intact organism, Douglas and Pribram (1966) developed a sophisticated neurophysiological theory of "problem solving." Although the authors were initially concerned with the hippocampus, they found it necessary to include in their theory a second limbic system structure, the amygdala, in order to account for the behavior of which hippocampectomized Ss are capable. Each of these structures is postulated to be intimately involved in two distinct processes underlying problem solving or discrimination learning: the hippocampus-centered "error-evaluate" process and the complementary amygdala-centered "reinforce-register" process. The terms are indicative of the function of each: the reinforce-register process is depicted as

increasing the future probability of a response which has been followed by reinforcement; the error-evaluate process is postulated as decreasing the future probability of a response which has not been followed by reinforcement. During discrimination learning in the intact organism both these processes or systems are cooperative as behavior is brought under stimulus control.

The proposed neuronal system underlying the error-evaluate process involves hippocampally mediated inhibition in a Renshaw-like mechanism within afferent systems which serves to "gate out" non-reinforced stimuli. In the absence of the hippocampus non-reinforcement cannot alter behavior and discrimination learning must be accomplished by the remaining reinforce-register system. The effect of reinforcement termed "impellence" is incremental over reinforced training, constant in size, and related to the magnitude of reinforcement and the effort required for its production. At the primary neuronal level, impellence is depicted as involving normally occurring collateral inhibitory processes in afferent systems. The work of Dewson, Nobel and Pribram (1966) and Spinelli and Pribram (1966) is taken as direct evidence for the existence of these proposed systems.

To summarize the Douglas-Pribram theory: It has been suggested that the hippocampus is a key structure in an error evaluating system which mediates the effect of non-reinforced responses during learning. Organisms with hippocampal disruption are rendered relatively insensitive to the effects of non-reinforcement and are therefore required to learn appetitively motivated tasks via the remaining reinforcement

sensitive amygdaloid system. Although the theory is a posteriori in construction, Douglas and Pribram (1966) do present some data confirming predictions made from the theory.

The present experiment focuses upon the hippocampus and its proposed involvement in situations involving non-reinforced responding. An experimental paradigm in which manipulation of reinforcement and non-reinforcement contingencies generates differential predictions concerning the behavior of hippocampectomized rats has been developed from the theory in question. In both the acquisition and reversal phases of a position discrimination, equation of the absolute number of reinforced responses to each of two to-be-discriminated manipulanda combined with differentiation between the two in terms of the absolute number of non-reinforced responses would be predicted from the theory to retard both acquisition and reversal in hippocampectomized Ss when compared to neocortically-lesioned and sham operated controls. However, when the absolute number of non-reinforced responses to the manipulanda are equated and the number of reinforced responses differ, any hippocampal lesion-induced deficit would be predicted to be of a significantly lesser magnitude if present at all. Positive results would constitute support of the Douglas-Pribram theory (Douglas, personal communication, 1968).

METHOD

Subjects

The Ss were 60 male Long-Evans rats approximately 125 to 175 days old at the start of training.

Apparatus

A total of four experimental chambers were employed. One was constructed in the laboratory while the remaining three were commercially obtained. The chamber constructed in the laboratory was a converted ice chest with a sheetmetal partition dividing it into two compartments. One compartment contained a pellet dispenser and related reinforcement delivery equipment; the second compartment, with the inclusion of a hardware cloth floor, measured 28.5 mm. by 28 mm. by 23 mm. high and served as the experimental space. A Ralph Gerbrands Company rat lever was situated along the verticle center line of one wall, 2.25 mm. above the hardware cloth floor. Reinforcement was delivered to a food cup situated 5 mm. above the manipulandum. An exhaust fan provided ventilation, and a 20 VDC bulb located in the center of the ceiling provided illumination during experimental sessions. The commercially obtained chambers were all Lehigh Valley Electronics Model 1316 small cubicles. A metal food cup was located along the verticle center line of one wall and rested on the grid floor. Two Lehigh Valley Electronics Model 1352 rat levers were mounted on the same wall, one on each side of the food cup. The center point of each manipulandum was 3 mm. above the

floor and 5 mm. from the nearest side wall. Illumination was provided by a 20 VDC bulb located 2 mm. above the center of the plexiglass ceiling. All manipulanda were calibrated so that a weight of approximately 20 grams would activate the response circuitry. All experimental operations and contingencies were controlled by automatic electro-mechanical programming equipment. Reinforcement consisted of 45 mg. Noyes rat pellets. A plexiglass cover, measuring 3 mm. by 7 mm. by 14.5 mm. high, was available to cover either manipulandum in the two manipulanda chambers, thereby forcing Ss to respond on the uncovered manipulandum when the conditions of training so required.

Experimental Design

The 60 Ss were assigned in equal numbers to the 6 cells prescribed by the first two factors of a 3 x 2 x 2 experimental design involving repeated measures as the third factor. Animals subjected to hippocampal, neocortical, or sham lesions (factor A) were assigned to conditions of differentiation training which insured that for each daily session either the number of reinforced responses or the number of non-reinforced responses (factor B) emitted on each of two manipulanda were equal, and were then tested in both the acquisition and reversal (factor C) of a two manipulanda differentiation.

Procedure

Upon receipt from the supplier all Ss were placed on ad lib food and water. Following recovery from the rigors of shipment a mean base weight derived from five consecutive days weighing was established

for each S, and Ss were reduced to 85% of these values and maintained at that level for the duration of pretraining.

The goal of the pretraining phase of the experiment was to establish in each S a bar press response free of any procedurally-induced left or right position preference. To accomplish this pretraining was conducted in the single manipulandum chamber. Subjects were first magazine trained and then shaped to press the manipulandum by the delivery of food reinforcement. Special care was taken to insure that no S received a disproportionate amount of training under crf and low FR reinforcement schedules. The reinforcement ratio was gradually escalated and pretraining was terminated upon each S's demonstration of stable responding under the requirements of an FR 10 reinforcement schedule. Subjects were then returned to ad lib food maintenance.

Following recovery of lost weight Ss assigned to the appropriate cells of the factorial design were subjected to bilateral hippocampal removal, bilateral removal of the neocortex overlying the hippocampus, or bilateral sham operations in which the dura overlying the neocortex removed in the neocortical lesions was exposed. Following recovery from surgery a mean base weight derived from five consecutive days weighing was again established for each S, and Ss were reduced to 85% of these values and maintained at that level for the duration of the experiment.

Subjects were then returned to the single manipulandum chamber and retrained to respond under the conditions of the FR 10 reinforcement schedule. With few exceptions reestablishment of control of responding by the FR 10 schedule was accomplished during one session of approximately

45 minutes' duration. In no instance did this retraining require more than three daily sessions. Following completion of retraining in the single manipulandum chamber Ss were advanced to the two manipulanda chambers in which the experimental operations were conducted.

During preliminary training in the two manipulanda chambers the right manipulandum was first covered with the plexiglass cover provided for forced training. Responding on the left manipulandum was first maintained by a crf schedule of reinforcement, and then by intermittent reinforcement. The reinforcement ratio was escalated one step following every tenth reinforcement until 10 reinforcements on an FR 10 schedule were delivered. Subjects were then removed from the chamber, the plexiglass cover moved to the left manipulandum, and the preliminary training regimen repeated. Those Ss which failed to earn 10 FR 10 reinforcements on either manipulandum within a 5-minute period during which that schedule was in effect repeated the preliminary training regimen the following day. With few exceptions pretraining required no more than one session approximating one hour in duration; in no case were more than 4 daily sessions required. On the day following the completion of preliminary training the experimental procedures were initiated.

In discrimination acquisition responses on one manipulandum were reinforced onan FR 5 schedule and responses on the second were reinforced on an FR 9 schedule. Of the 10 Ss in each of the two hippocampal and sham lesion groups, 6 were assigned to one chamber and 4 to a second. Within each group the relationship between manipulandum and reinforcement schedule was counterbalanced. The two groups subjected to neocortical destruction were assigned to the third chamber and the relationship between manipulandum and reinforcement schedule also counterbalanced. The first portion of each daily experimental session consisted

of a 5-minute free choice period during which both manipulanda were exposed for responding and reinforced on the appropriate schedules. At the conclusion of the test period, which served to monitor the formation of the discrimination, the number of responses emitted and the number of reinforcements earned on each manipulandum were recorded. The forced training portion to the experimental session was then initiated.

The purpose of forced training differed for each of the two hippocampal, neocortical, and sham lesion groups. One each of the hippocampal, neocortical, and sham lesion groups was run under the condition prescribing the equation, for each S, of the absolute number of reinforced responses emitted on each manipulandum during each daily session. The second hippocampally, neocortically, and sham lesioned groups were run under the conditioning prescribing the equation, for each S, of the absolute number of non-reinforced responses emitted on each manipulandum during each daily session. It should be noted that as a result of the utilization of an FR 5 and an FR 9 schedule of reinforcement, Ss which emitted an equal number of reinforced responses on each manipulandum also emitted twice as many non-reinforced responses on the FR 9 manipulandum as on the FR 5 manipulandum. Conversely, Ss which emitted an equal number of non-reinforced responses on the two manipulanda also emitted twice as many reinforced responses on the FR 5 manipulandum as on the FR 9 manipulandum.

Subjects assigned to the reinforced responses equated procedure fulfilled a dual requirement during each complete experimental session. These requirements were: (a) each S earn an equal number of reinforcements

on the FR 5 and FR 9 manipulanda, and (b) a total of at least 50 reinforcements be earned on each of the two manipulanda. If S did not earn the minimum 50 reinforcements on either manipulandum during the 5-minute free choice period, the forced training portion of the session involved responding on both manipulanda. At the end of the free choice period the required number of make-up reinforcements to be earned on each manipulandum was determined, one manipulandum was covered, and S was allowed to respond on the second until the required number of make-up reinforcements for that manipulandum had been delivered. The cover was then moved to the second manipulandum and S was allowed to respond on the first until the requirements for that manipulandum had been fulfilled. For example, if S earned 40 FR 5 reinforcements and 25 FR 9 reinforcements during the free choice period, S would be required to earn an additional 10 FR 5 reinforcements and 25 FR 9 reinforcements during the forced training period. As a result, S would have earned an equal number of reinforcements (50) on each manipulandum during the course of the experimental session. The order in which the manipulanda were covered alternated across daily sessions.

If S earned 50 or more reinforcements on either, or both manipulanda during the free choice period, the forced training period involved responding only one manipulandum. At the termination of the free choice period the difference between the number of reinforcements earned on the two manipulanda was determined, and the manipulandum on which S had earned the greater number of reinforcements was covered. The S was then allowed to respond on the second manipulandum until the

required number of make-up reinforcements was delivered. For example, if S earned 65 FR 5 reinforcements and 20 FR 9 reinforcements during the free choice period, the FR 5 manipulandum would be covered during the forced training period and S would be allowed to respond on the FR 9 manipulandum until 45 reinforcements had been delivered. This fulfilled the requirement that S emit an equal number of reinforced responses, in this instance 65, on each manipulandum during each experimental session.

Subjects assigned to the non-reinforced responses equated procedure fulfilled a different dual requirement during each experimental session. These requirements were: (a) each S earn twice as many reinforcements on the FR 5 manipulandum as on the FR 9 manipulandum, and (b) a minimum of 60 reinforcements be earned on the FR 5 manipulandum and, consequently, a minimum of 30 reinforcements be earned on the FR 9 manipulandum. The free choice period and subsequent forced training proceeded in a manner analogous to that described above for the Ss assigned to the reinforced responses equated regimen. In the present condition, if S failed to earn the minimum 60 and 30 reinforcements on both the FR 5 and FR 9 manipulanda, respectively, during the free choice period, the forced training period would insure that these minima were earned. For example, if S earned 40 FR 5 reinforcements and 25 FR 9 reinforcements during the free choice period, he would be required to earn an additional 20 FR 5 reinforcements and 5 FR 9 reinforcements during the forced training period. The S would have therefore earned the minimum 60 and 30 reinforcements on the FR 5 and FR 9 manipulanda during the course of the experimental session.

If S earned more than 60 reinforcements on the FR 5 manipulandum and/or more than 30 reinforcements on the FR 9 manipulandum during the free choice period, the forced training period involved only one manipulandum and served to insure that the ratio of reinforcements earned of the FR 5 manipulandum to those earned on the FR 9 manipulandum was 2 to 1. For example, if S earned 70 FR 5 reinforcements and 40 FR 9 reinforcements during the free choice period, S was required to earn an additional 10 FR 5 reinforcements during the forced training period. As a result, S earned a total of 80 FR 5 reinforcements and 40 FR 9 reinforcements during the course of the experimental session, and fulfilled the requirement that the ratio of FR 5 to FR 9 reinforcements be 2 to 1. If S earned 90 FR 5 reinforcements and 10 FR 9 reinforcements during the free choice period, S was required to earn an additional 35 FR 9 reinforcements during the forced training period. The S therefore earned a total of 90 FR 5 reinforcements and 45 FR 9 reinforcements, and the ratio of FR 5 to FR 9 reinforcements was again the required 2 to 1.

Discrimination training was terminated when S attained a criterion of at least 90% responding on the FR 5 manipulandum in 9 of 10 consecutive free choice periods. Upon completion of this requirement S was subjected to one half again as many daily sessions as were required for attainment of the criterion and then moved to the discrimination reversal phase of the experiment. If it became statistically impossible for S to satisfy the criterion within 40 days of training S was considered to have failed to satisfy the requirements for discrimination and was then moved to the discrimination reversal phase of the study.

Discrimination reversal training was instituted for each S on the day following termination of the acquisition portion of the experiment. In reversal training the relationship between reinforcement schedule and manipulandum was reversed for each S. Training in reversal proceeded in the same fashion for the two groups as described in the acquisition phase above. Reversal training was terminated when each S met either the criterion of acquisition or failure established for the acquisition phase of the study.

Surgery

All Ss were operated under 40 mg/kg Nembutal anesthesia supplemented with .30 cc. of atropine injected interperitoneally. All operations were performed while S was held in a Baltimore stereotaxic instrument. A dissecting scope was employed to assist in the visual guiding of neocortical and hippocampal removal. In all operations the skull was exposed by means of a midline incision, bilateral trephine holes were placed lateral to the midline and posterior to bregma. The holes were enlarged with rougures to expose the neocortex overlying the dorsal and lateral portions of the hippocampus. In the sham operated Ss surgery was terminated at this point. For those Ss sustaining neocortical removal the dural was cut and the neocortex overlying the hippocampus was aspirated off, with care taken not to damage the hippocampus. For those Ss subjected to hippocampectomy the operation proceeded until the thalamus was exposed. In addition, hippocampal removal was expended anteriorally as far as the hippocampal commissure as well as extended around the posterio-lateral surface of the thalamus. Care was taken not

to damage the thalamus. Following completion of surgery Ss were returned to their home cages and maintained on a water and tetracycline solution for three to five days.

Histology

Following the termination of the experiment all Ss were sacrificed with a lethal dose of Nembutal anesthesia and intracardially perfused with saline followed by a 10% formalin solution. All brains were then removed from the brain cavities and those of the Ss in the neocortical and hippocampal groups were infiltrated with, and embedded in, celloidin and sectioned at 15 u. Every tenth section was retained, mounted on a slide and stained with thionin. In addition, for 5 Ss in each lesion group the section following the thionin section was stained with Wile and then mounted.

RESULTS

Tracings of representative cross sections through the hippocampal and neocortical lesions are presented in Figures 1 and 2, respectively. Hippocampal destruction regularly involved at least 75% of that structure and in all instances resulted in the complete separation of the dorsal and lateral aspects of the hippocampal formation. Specific damage to the thalamus was minimal and, when evident, was typically unilateral in nature. The neocortical lesions did not encompass a volume of tissue comparable to that removed in the hippocampal lesions, but did approximate the neocortical destruction incurred by the hippocampectomies. Hippocampal damage resulting from the neocortical lesions was minimal and, if present, usually unilateral in nature. Gross examination of the intact brains of those Ss subjected to sham operations revealed no discernible neocortical damage.

The sessions required to attain criterion in acquisition and reversal by those sham, neocortically, and hippocampally lesioned Ss trained under the reinforced responses equated regimen are presented in Table 1. The trials to criterion for Ss assigned to the non-reinforced responses equated condition are presented in Table 2. Inspection of these tables suggests there is an inverse relationship between performance in acquisition and reversal. It appears that Ss who readily attain criterion in acquisition are retarded in reversal and, to a lesser degree, Ss who are retarded in acquisition appear to perform well in reversal. To test the possibility of such an inverse relationship, all Ss were ranked

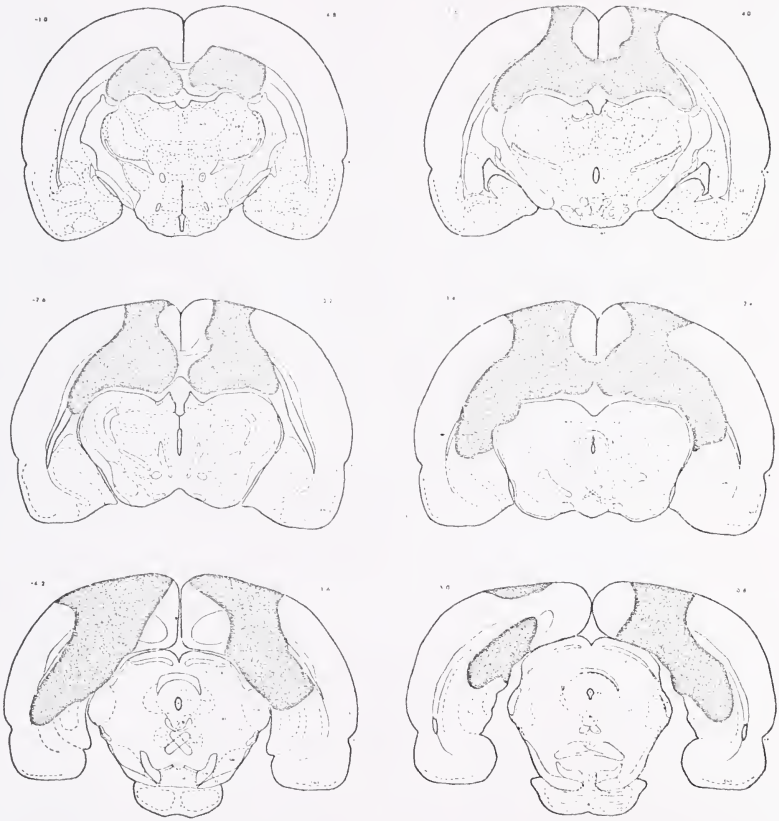

Fig. 1.—Tracings of representative cross sections through the hippocampal lesion (After Pellegrino and Cushman, 1967).

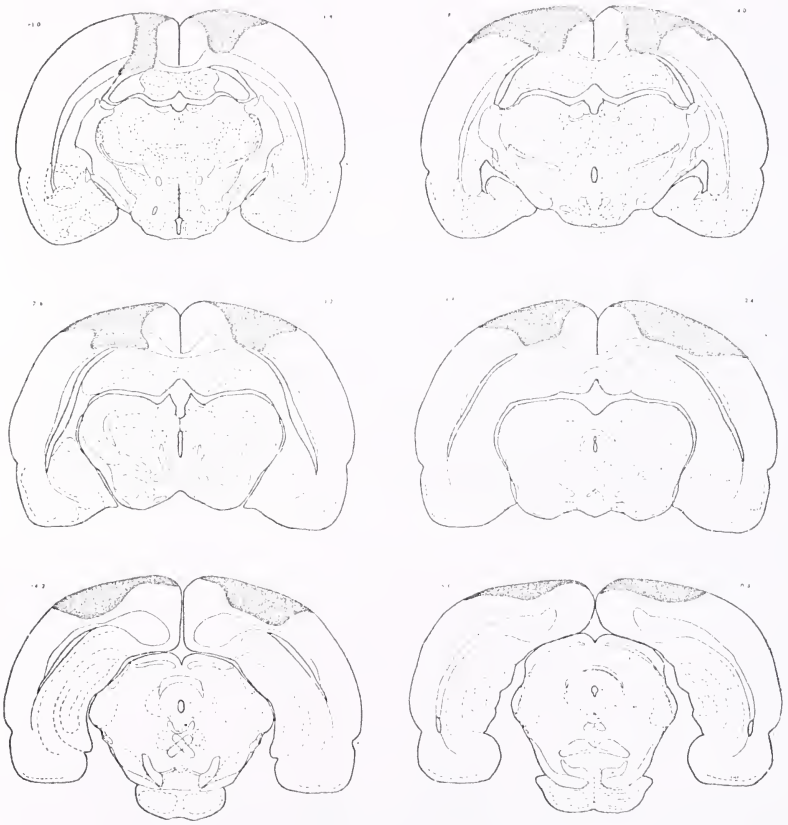

Fig. 2.—Tracings of representative cross sections through the neocortical lesion (After Pellegrino and Cushman, 1967).

TABLE 1

TRIALS TO CRITERION IN ACQUISITION AND REVERSAL FOR SHAM, NEOCORTICALLY, AND HIPPOCAMPALLY LESIONED Ss ASSIGNED TO THE REINFORCED RESPONSES EQUATED CONDITION

	Acquisition	Reversal
Sham Control	10	14
	40	11
	10	24
	11	40
	9	40
	10	16
	10	26
	11	26
	10	40
	10	40
Neocortical Lesion	40	14
	14	17
	32	12
	10	10
	10	14
	10	23
	10	19
	10	13
	14	14
	19	40
Hippocampal Lesion	11	22
	40	10
	12	12
	30	15
	10	16
	9	40
	17	12
	25	13
	13	13
	9	15

TABLE 2

TRIALS TO CRITERION IN ACQUISITION AND REVERSAL FOR SHAM, NEOCORTICALLY, AND HIPPOCAMPALLY LESIONED Ss ASSIGNED TO THE NON-REINFORCED RESPONSES EQUATED CONDITION

	Acquisition	Reversal
Sham Control	21	12
	10	32
	15	13
	13	16
	9	24
	10	40
	9	22
	10	32
	18	16
	11	22
Neocortical Lesion	10	17
	11	19
	40	15
	13	13
	12	12
	10	15
	10	20
	10	16
	31	14
	9	13
Hippocampal Lesion	11	14
	11	25
	19	14
	16	14
	10	12
	9	17
	9	40
	10	17
	10	14
	10	12

from low to high on the number of sessions to attain criterion in acquisition, and from high to low on the number of sessions required to attain criterion in reversal. A Spearman rank order correlation coefficient (r_s) was then computed (Siegel, 1956), and found to be significant ($r_s = .47$, $t = 4.0567$, $df = 59$, $p < .001$). Spearman rank order correlation coefficients were also computed in the same manner for the sham, neocortical, and hippocampal lesion groups (see Table 3), and for the three lesion groups when further divided on the basis of the reinforced versus non-reinforced responses equated dimension (see Table 4). The former analysis indicates the inverse relationship between performance in acquisition and reversal is present in only the sham control Ss; the latter analysis reveals that while the sham control Ss under the non-reinforced responses equated condition do show this relationship, their counterparts under the reinforced responses equated condition do not. In addition, the finer grain analysis indicates that the hippocampectomized Ss under the reinforced responses equated condition also manifest this relationship, albeit to a lesser degree.

The sessions required to attain criterion in acquisition and reversal for sham, neocortically, and hippocampally lesioned Ss under the reinforced responses equated requirement are presented graphically in Figures 3 and 4, respectively. Analogous data for Ss under the non-reinforced responses equated condition are presented in Figures 5 and 6. An analysis of variance assessing the effects of the three lesion conditions, the two response-reinforcement contingencies, and acquisition and reversal upon performance as indexed by the trials to criterion measure was performed (Winer, 1962). Since the trials to criterion

TABLE 3

SPEARMAN RANK ORDER CORRELATION COEFFICIENTS (r_s) FOR SHAM, NEOCORTICAL, AND HIPPOCAMPAL LESION GROUPS*

Sham Control	$r_s = .51$
	$p < .05$
Neocortical Lesion	$r_s = .10$
	$p > .05$
Hippocampal Lesion	$r_s = .36$
	$p > .05$

*See text for ranking procedure.

TABLE 4

SPEARMAN RANK ORDER CORRELATION COEFFICIENTS (r_s) FOR SHAM, NEOCORTICAL, AND HIPPOCAMPAL LESION GROUPS UNDER EQUATED REINFORCED OR NON-REINFORCED RESPONDING DURING TRAINING*

	Reinforced Responses Equated	Non-Reinforced Responses Equated
Sham Control	$r_s = .42$	$r_s = .76$
	$p > .05$	$p < .01$
Neocortical Lesion	$r_s = .06$	$r_s = .29$
	$p > .05$	$p > .05$
Hippocampal Lesion	$r_s = .68$	$r_s = .35$
	$p < .05$	$p > .05$

*See text for ranking procedure.

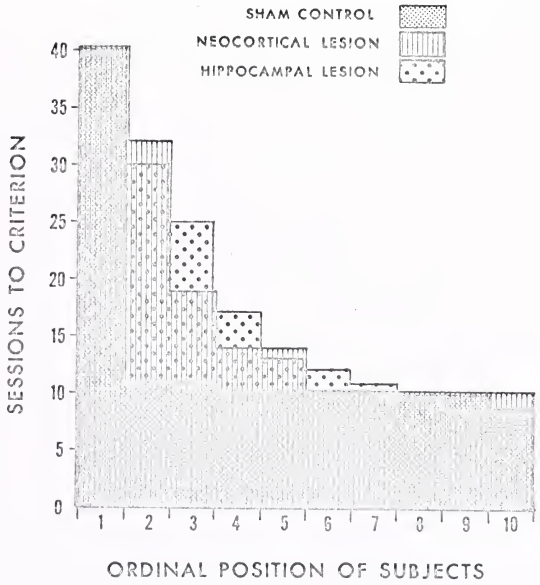

Fig. 3.—Number of sessions for Ss in each lesion group to attain criterion in acquisition: Reinforced responses equated.

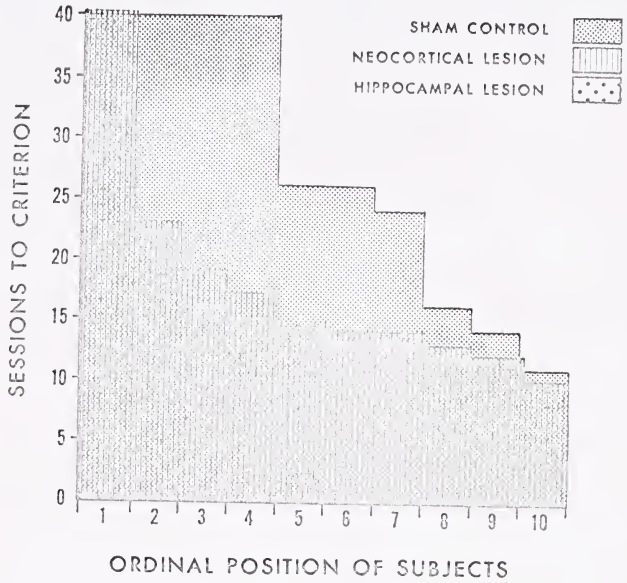

Fig. 4.—Number of sessions for Ss in each lesion group to attain criterion in reversal: Reinforced responses equated.

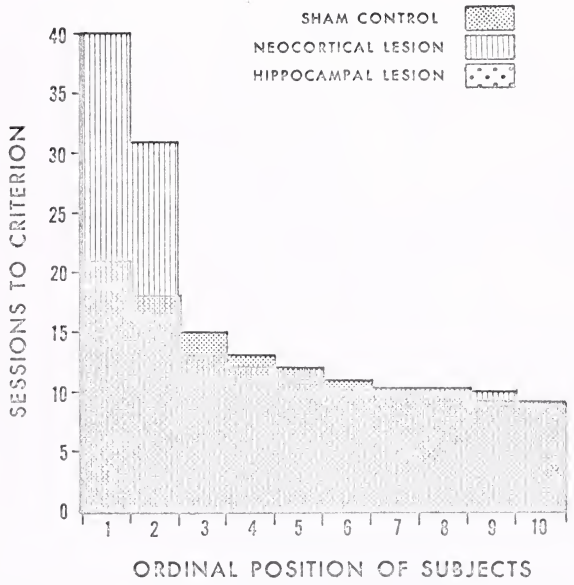

Fig. 5.—Number of sessions for Ss in each lesion group to attain criterion in acquisition: Non-reinforced responses equated.

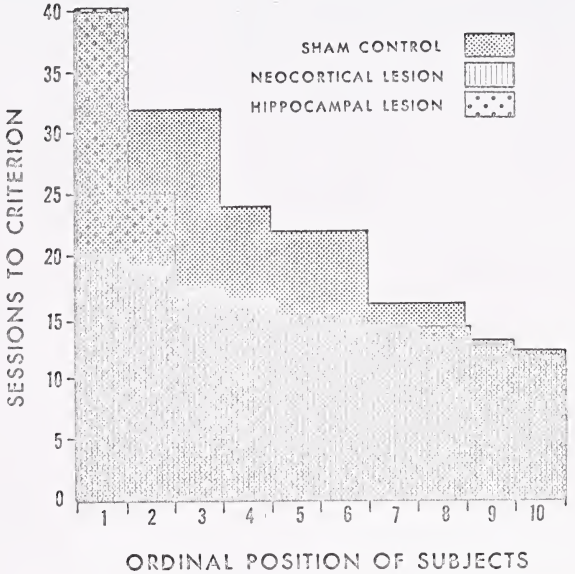

Fig. 6.—Number of sessions for Ss in each lesion group to attain criterion in reversal: Non-reinforced responses equated.

measure produced slightly skewed data, these values were subjected to a square-root transformation and a second analysis of variance performed upon the resultant data. The results of these two analyses are presented in summary fashion, in Tables 5 and 6, respectively. A comparison of the two tables reveals little inconsistency in the results of the two analyses.

An examination of Figure 7, which depicts the cumulative percentage of Ss in each lesion group attaining criterion in successive five session blocks, suggests that the neocortically and hippocampally lesioned Ss are slightly facilitated with respect to the sham control Ss in discrimination performance. The analyses of variance indicate that this difference is not large enough to be statistically reliable. However, the results of the analyses do reveal a significant interaction between this factor and the acquisition and reversal phases of training which must be examined before it can be concluded that the various lesion conditions have no effect on discrimination learning.

A comparison of the effects of equating either reinforced or non-reinforced responses during discrimination training is depicted in Figure 8. Neither level of this factor, nor this factor's interaction with the lesion dimension, were indicated by the analyses of variance as differentially affecting performance in the discrimination task.

Inspection of Figure 9, which represents performance in the acquisition and reversal phases of the study, suggests that Ss attained criterion more rapidly in acquisition training than in reversal training, and this is verified as a significant difference by the analyses of variance.

TABLE 5

ANALYSIS OF VARIANCE ON TRIALS TO CRITERION

Source	MS	df	F
Between Ss		59	
Lesions (A)	117.11	2	2.23
Response-Reinforcement Contingency (B)	151.89	1	2.90
A x B	2.79	2	0.05
Ss Within Groups (Error)	52.46	54	
Within Ss		60	
Acquisition-Reversal (C)	795.69	1	7.93**
A x C	417.62	2	4.16*
B x C	1.86	1	0.02
A x B x C	83.21	2	0.83
C x Ss Within Groups (Error)	100.35	54	

*$p < .05$

**$p < .01$

TABLE 6

ANALYSIS OF VARIANCE ON SQUARE-ROOT TRANSFORMATION OF TRIALS TO CRITERION

Source	MS	df	F
Between Ss		59	
Lesions (A)	1.23	2	1.7
Response-Reinforcement Contingency (B)	1.38	1	1.91
A x B	0.02	2	0.03
Ss With Groups (Error)	0.72	54	
Within Ss		60	
Acquisition-Reversal (C)	10.96	1	6.61*
A x C	5.42	2	3.27*
B x C	2.01	1	1.21
A x B x C	0.04	2	0.02
C x Ss Within Groups (Error)	1.66	54	

*$p < .05$

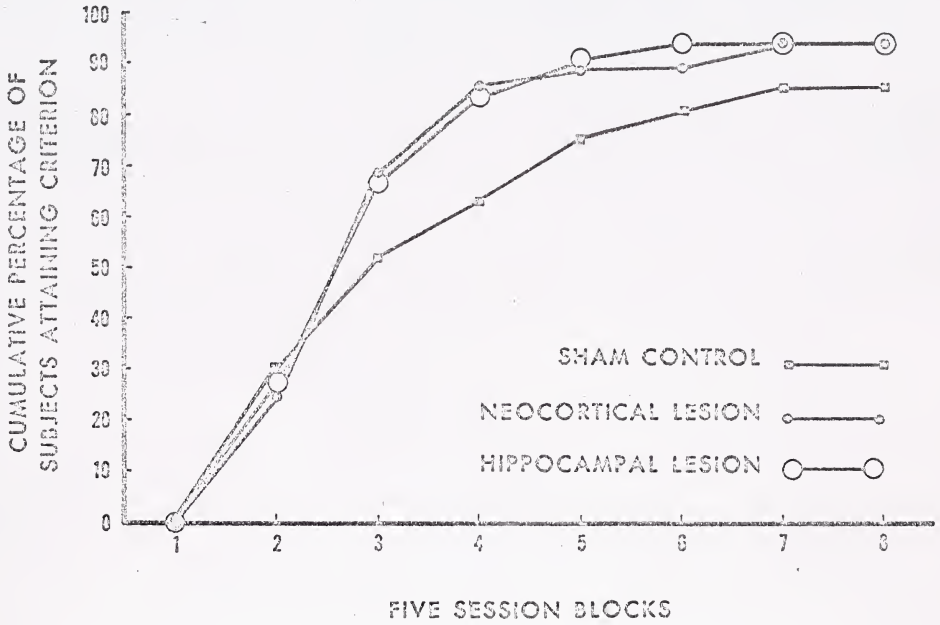

Fig. 7.—Cumulative percentage of Ss in each lesion condition attaining criterion in successive 5 session blocks.

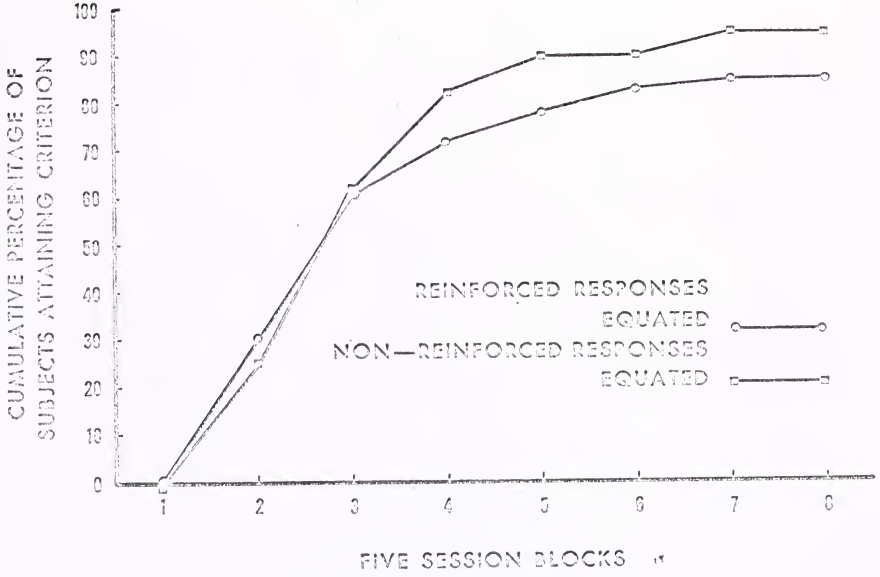

Fig. 8.—Cumulative percentage of Ss in each response-reinforcement condition attaining criterion in successive 5 session blocks.

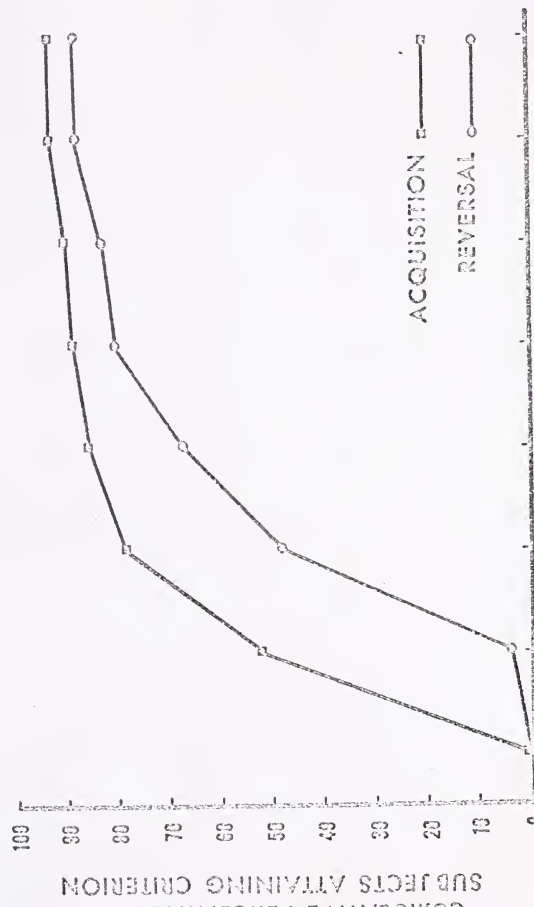

Fig. 9.—Cumulative percentage of Ss attaining criterion in acquisition and reversal in successive 5 session blocks.

As indicated previously, a significant interaction between the lesions factor and the acquisition and reversal phases of training was revealed by the analyses of variance. The components of the interaction are depicted in Figure 10, which presents the performance of the three lesion groups in acquisition training, and in Figure 11, which presents their performance in reversal training. The mean trials to criterion for Ss in each of the three lesion groups is presented for acquisition and reversal in Figure 12. Examination of these figures suggests that the three lesion groups did not differ in the acquisition phase of training, but that in the reversal phase the neocortically and hippocampally lesioned Ss, while not differing among themselves, did attain the criterion more rapidly and in greater numbers than the sham control Ss. A posteriori comparisons between the cell means involved in this interaction were performed utilizing the Studentized range statistic (Winer, 1962). The results of the comparisons are presented, in summary fashion, in Table 7. The results support the above observations and reveal, in addition, that sham control Ss attained criterion significantly faster in acquisition than in reversal, but that such a difference is not present in the neocortically and hippocampally lesioned Ss. Neither the remaining first order interaction (lesions by response-reinforcement contingency) nor the single second order interaction (lesions by response-reinforcement contingency by acquisition-reversal) attained significance.

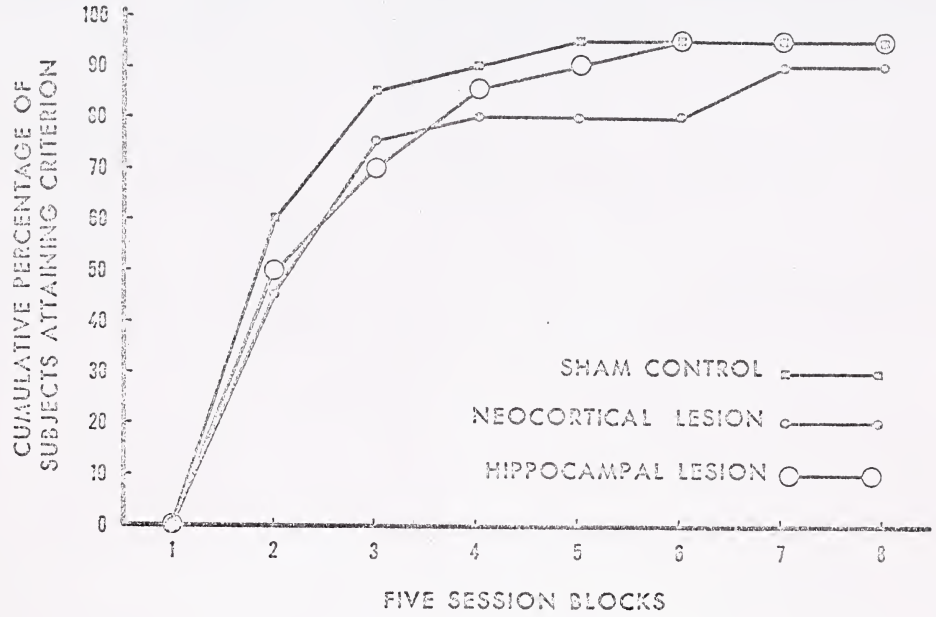

Fig. 10.—Cumulative percentage of Ss in each lesion condition attaining the acquisition criterion in successive 5 session blocks.

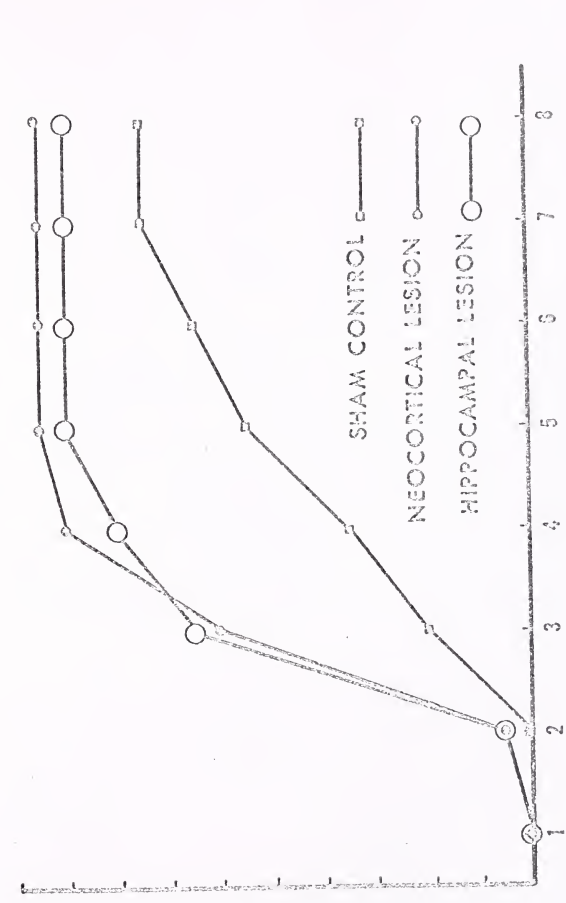

Fig. 11.—Cumulative percentage of Ss in each lesion condition attaining the reversal criterion in successive 5 session blocks.

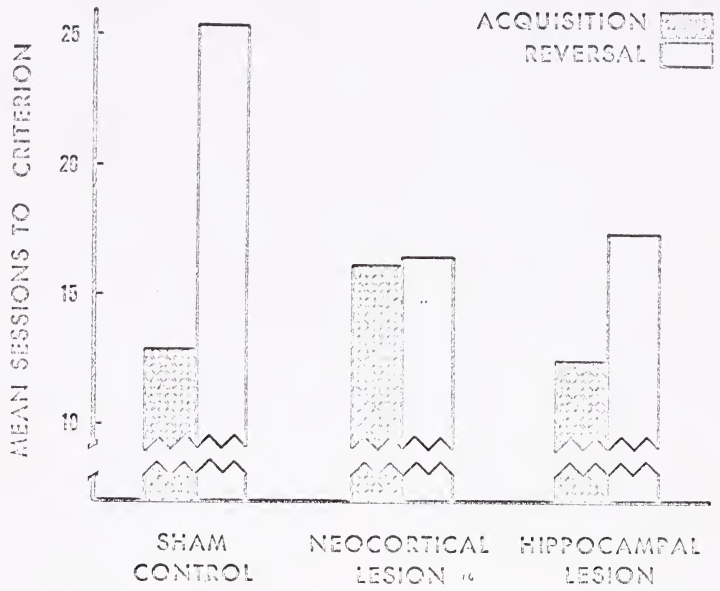

Fig. 12.—Mean trials to criterion in acquisition and reversal for Ss in each lesion group.

TABLE 7

STUDENTIZED RANGE STATISTIC A POSTERIORI TESTS

Acquisition	
Sham vs. Neocortical Lesion	3.09
Sham vs. Hippocampal Lesion	0.86
Neocortical vs. Hippocampal Lesion	0.83
Reversal	
Sham vs. Neocortical Lesion	20.29**
Sham vs. Hippocampal Lesion	13.94**
Neocortical vs. Hippocampal Lesion	0.23
Sham Control	
Acquisition vs. Reversal	19.77**
Neocortical Lesion	
Acquisition vs. Reversal	0.01
Hippocampal Lesion	
Acquisition vs. Reversal	2.30

**$p < .01$

DISCUSSION

This study examined the performance of sham, neocortically, and hippocampally lesioned rats in the acquisition and reversal of a two manipulanda differentiation as affected by certain manipulations of response-reinforcement contingencies. For one-half the sham, neocortically, and hippocampally lesioned Ss, these manipulations insured that each S emitted an equal number of reinforced responses on the two manipulanda during a complete experimental session. As a result of this procedure, each S also emitted twice as many non-reinforced responses on one manipulandum (the FR 9 manipulandum) as on the second (the FR 5 manipulandum). For the remaining Ss, the experimental manipulations insured that each S emitted an equal number of non-reinforced responses on the two manipulanda during a complete experimental session. As a result of this procedure, each S emitted twice as many reinforced responses on the FR 5 manipulandum as on the FR 9 manipulandum.

The Douglas-Pribram theory of hippocampal function (Douglas & Pribram, 1966; Douglas, 1967) is a vigorous attempt to integrate the wide variety of behavioral changes following hippocampal disruption, and leads to clear-cut predictions of the behavioral effects of the manipulations performed in this study. Specifically, the theory predicts retarded acquisition and reversal in hippocampal Ss when compared to sham and neocortical control Ss under the treatment condition which specifies equation of the number of reinforced responses emitted

on the two manipulanda, less or no retardation in hippocampal Ss under the condition which served to equate the number of non-reinforced responses emitted on the discriminanda, and, indirectly, no difference between sham and neocortical Ss under either of these two treatment conditions. These predictions are contradicted by the results of this experiment.

The trials to criterion data do not reveal any differences between the three lesion groups in acquisition performance. This finding is in general accord with the results of recent studies of the role of the hippocampus in discrimination learning. However, such studies have not examined the effect of differential densities of reinforced and non-reinforced responding to the alternative discriminanda upon discrimination learning in hippocampectomized Ss. The present findings, although inconsistent with predictions derived from the Douglas-Pribram model, indicate that such differences have no significant effect on acquisition performance in either sham, neocortically, and hippocampally lesioned Ss.

The performance of the three lesion groups in reversal also is inconsistent with predictions derived from the Douglas-Pribram for, as in acquisition, the two response-reinforcement contingencies do not differentially affect in either sham, neocortically, or hippocampally lesioned Ss. In addition, the reversal data appear to be inconsistent with the bulk of the data on the performance of hippocampectomized and control Ss in discrimination reversal; namely the hippocampally lesioned Ss are typically reported as retarded in discrimination reversal when compared to neocortically lesioned and sham control Ss who usually

do not differ from each other. The results of the present study indicate that it is neocortically and hippocampally lesioned Ss who do not differ from each other, and both appear facilitated when compared to sham operated Ss on the trials required to attain criterion in reversal. A comparison of the sessions to criterion data with alternative performance measures, such as trials to successive criteria and the absolute per cent of FR 5 responding for successive days, revealed that all three measures depicted acquisition and reversal performance in a similar fashion.

A possible explanation of this disparity is offered by the relationship between acquisition and reversal performance as revealed by the Spearman rank order correlation coefficient. The sham operated Ss, who require a significantly greater number of trials to attain criterion in reversal when compared to the neocortically and hippocampally lesioned Ss as indicated by the Studentized range statistic, are also the Ss who manifest a significant inverse relationship between trials to criterion in acquisition and reversal. In addition, a greater number of Ss in the sham control group attained the criterion in 11 or less sessions (15 of 20 Ss) than in either the neocortical (11 of 20 Ss) or hippocampal (12 of 20 Ss) lesion groups. When the three lesion groups are partitioned in terms of the response-reinforcement contingencies, similar phenomena are observed. These findings suggest that some Ss possess an initial position preference which, when in accord with the requirements of the initial differentiation, facilitates acquisition and retards reversal. Moreover, it appears that a greater proportion of Ss in the sham operated group fall into this category

than in either of the two other lesion groups. It may be assumed, then, that the poorer performance of the sham control Ss in reversal is an artifact resulting from a failure to completely control for initial position preference, and that if this had been done the differences between the three lesion groups in reversal would be eliminated.

The experimental procedures employed in this study differ in a number of details from those in which hippocampal lesion-induced deficits in discrimination reversal are commonly observed. It is possible that the lack of a lesion-induced deficit in the present study may be attributable to one or more of these differences. One such modification involves the utilization of an overtraining procedure following attainment of criterion in acquisition. Investigations of the effect overtraining upon reversal in the T-maze indicate that Ss subjected to, on the average, at least 1.3 (Macintosh, 1962) and 3 (Publos, 1956) times as many overtraining trials as were required to reach criterion in the acquisition of a discrimination perform better in reversal than Ss without overtraining (the overtraining reversal effect). An examination of Reid's (1953) data indicates that when overtraining involves less trials than were required to attain criterion the overtraining reversal effect is not seen. It is difficult to compare those procedures and the present one, for different responses and procedures were employed. However, since this study employed only one-half as many overtraining sessions as were required to attain the acquisition criterion it is unlikely that the overtraining reversal effect was operative. Despite this, an examination of the overtraining reversal phenomenon does add to an understanding of the results of this study.

Macintosh (1965) notes that it is presumably justifiable to regard reversal learning as consisting of two parts: (a) extinction

of a tendency to select the former S^D, and (b) acquisition of a tendency to select the new S^D. Stage (a), extinction, is usually regarded as continuing so long as S scores below chance level, and stage (b), acquisition, is typically depicted as commencing as soon as S begins to perform above chance level. It should be noted that implicit in the formulation described by Macintosh is the generally accepted assumption that learning is a continuous, rather than discontinuous, process. Whether this is indeed the case has not yet been completely resolved. The present discussion is concerned primarily with the effect of attentional factors upon discrimination reversal rather than with the underlying nature of the learning process.

It is often reported that overtraining of a runway response results in reduced resistance to extinction (Wagner, 1963). It is logical to assume, therefore, that this phenomenon is what underlies the overtraining reversal effect; namely, overtraining facilitates extinction of responses to the former S^D in the formulation described by Macintosh. This is not the case, however, for overtraining in the discrimination paradigm regularly increases resistance to extinction of responses to the former S^D (Macintosh, 1962). As Macintosh (1965) points out, overtraining facilitates reversal of a simultaneous discrimination not because of, but in spite of its effect on extinction. This finding would appear to negate an extension of the frustration (Lawrence & Festinger, 1962) and generalization decrement (Kimble, 1961) explanations of extinction to the overtraining reversal effect and, by implication, to reversal training in general.

Macintosh (1965) reports that existent evidence indicates overtraining facilitates reversal by shortening runs of incorrect responses during the middle of the reversal. This suggests that overtraining reduces Ss' tendencies to respond to irrelevant cues during reversal. There are two possible explanations for this: Either overtraining effectively enables Ss to "adapt out" cues along irrelevant dimensions; or overtraining allows ample opportunity for Ss to learn to attend to the relevant cue dimension. Macintosh presents evidence which indicates that it is the latter alternative which underlies the overtraining reversal effect, and points out a distinction between research utilizing visual and spatial cues. Studies which have involved a simultaneous visual discrimination (brightness, pattern, etc.) invariably produce the overtraining reversal effect, while those studies which employ a spatial discrimination (left turn versus right turn in T-and Y-mazes, etc.) frequently do not. A major reason for this, Macintosh contends, is that the rat (the commonly used experimental organism) is primarily spatially oriented and, as a consequence, spatial cues have a high priority even without overtraining. Since the rat is already attending mainly to spatial or position cues, overtraining would not be expected to have much effect on performance in reversal. Conversely, the lower the relevant cue dimension is on the Ss "attending hierarchy," the more valuable overtraining would be expected to be in firmly establishing the relevant cues in a position of dominance. The magnitude of the overtraining reversal effect should be inversely related to the probability that S will attend to the relevant cue at the beginning of discrimination training, and to the number of irrelevant cues involved in the discrimination.

The preceding discussion is markedly similar to the Douglas-Pribram conceptualization of the role of the amygdala in discrimination learning; namely, the registration of the effects of reinforcement or, alternatively, the direction of attention to the aspects of the task (relevant cues) associated with reinforcement. However, the above formulation of the function of attentional factors in discrimination learning does not incorporate a process analogous to that attributed to the hippocampus; the gating out of stimuli associated with non-reinforcement. It will be recalled that an alternative to the attentional model indicates that the overtraining reversal effect can be attributed to the opportunity for Ss to effectively "adapt out" irrelevant cues (Spence, described in Macintosh, 1965). Perhaps an explanation of the overtraining reversal effect involves both these processes. The Douglas-Pribram model suggests that this is so. In addition, the results of this study may not be as inconsistent with the Doublas-Pribram model as was first indicated. The differentiation required in the present study involves spatial cues, a dimension thought to be high on the rat's "attentional hierarchy." If this is the case, the role of the hippocampus, that of gating out irrelevant stimuli, would be minimal in the intact S, and Ss without the hippocampus should not be greatly impaired. Perhaps a hippocampal lesion-induced deficit would be evident in the present paradigm if a task involving a non-spatial differentiation, or, more probably, a differentiation between a large number of equally salient cues was employed. Support for this possibility is provided by Pribram (1969), who reports that hippocampectonized monkeys show retardation in discrimination learning, provided there are

a large number of non-rewarded alternatives in the situation (pg. 137).

A second procedural innovation employed in the present study involves the response selected for study. Those studies, reported previously, which have demonstrated the hippocampal lesion reversal deficit have typically employed an instrumental response requiring some form of gross locomotion on the part of S. In contrast, this study utilized an operant response, a bar press, which, unlike the typical instrumental response, requires a minimum of locomotion, takes a short time to execute, requires relatively little effort, and leaves S in the same place ready to respond again. Although it is generally assumed that the behavioral principles and neurophysiological mechanisms underlying what appear to be analogous tasks in the two experimental approaches do not differ in any critical aspect, a thorough comparison of these two procedures has not yet been attempted. However, a recent study by Means, Walker, and Isaacson (1969) indicates that the effect of hippocampal disruption upon go-no go performance may be response-specific. Although it is typically reported that hippocampectomy interferes with this behavior when examined in an instrumental paradigm, such as an alleyway (Brown, Kaufman & Marco, 1969), Means et al. report that hippocampal ablations facilitate performance in this task when a bar press response is utilized. Findings such as these question the trans-situational nature of the pattern of behavioral disruption observed following hippocampal destruction and, consequently, any formulation which attempts to account for these effects with global concepts

such as "perseveration," "gating," or "inhibition" without further refinement or qualification.

The third major difference between this and contemporary investigations of the role of the hippocampus in discrimination learning involves the schedules of reinforcement associated with the two to-be-discriminated responses. The research reported previously has typically provided continuous reinforcement for "correct" responses and withheld reinforcement for "incorrect" responses. In the present study concurrent operants were utilized: Both responses were reinforced, one on an FR 5 schedule and the other on an FR 9 schedule, and the formation of the discrimination was based on a relative, rather than absolute, differential in reinforcement density. The experimental analysis of concurrent ratio schedules indicates that with unequal FR requirements, responding tends to be maintained only by the schedule with the smaller FR requirement; with equal FR requirements, responding can be maintained by either one, and shifting from one schedule to the second occasionally occurs (Catania, 1966, Herrnstein, 1958). In a study which is only superficially comparable to the one reported here, Douglas and Pribram (1966) examined the effects of probabilistic reinforcement upon the formation of a discriminated panel press in monkeys. As in the present study the discrimination rested upon a relative differential in reinforcement density; one response was reinforced 70% of the time and the second reinforced 30% of the time. Their results, in contrast to those of the present study, indicated that hippocampectomized Ss are retarded with respect to control Ss

in their ability to acquire a discrimination under such conditions. Unfortunately, no data are presented on the performance of these Ss in discrimination reversal. The reasons for these apparently contradictory findings are unknown, but these studies reveal that insufficient attention has been directed towards an elaboration of the effects of schedules of reinforcement on discrimination formation and reversal in hippocampectomized Ss.

Another difference between this and other studies of hippocampal function involves the spacing of test trials. Most research in this area has employed a discrete trial procedure and the related technique of massed training trials during each daily experimental session. In the present study the equivalent of test trials, the five minute free-choice periods, were widely spaced for they occurred at the beginning of each daily experimental session. No direct evidence is available concerning the effect of this factor on discrimination learning and reversal in hippocampectomized Ss. However, there is evidence that the interval between trials does influence the behavioral effects of hippocampal disruption. As reported previously, Kirkby, Stein, Kimble and Kimble (1967) have demonstrated that the lack of T-maze spontaneous alternation commonly reported in hippocampal Ss can be reestablished by lengthening the intertrial interval from 50 seconds to 10 minutes. Although their explanation of this phenomenon, a postulated lesion-induced reduced information acquisition rate, has been generally abandoned on the premise that such preparations do not show deficits in a number of alternative learning tasks, no adequate explanation has been

formulated. Little or no additional research has been directed towards an understanding of this finding, and until this phenomenon is investigated in greater detail such an explanation of the results of the present experiment cannot be fully evaluated.

A fifth major departure of this experiment relative to previous research is the utilization of a forced training technique. As a function of fulfilling the requirements of the response-reinforcement contingencies, this procedure insured that each S was fully exposed to the conditions of reinforcement throughout both acquisition and reversal training. In addition, it can be assumed that this innovation most probably maintained the strength of the FR 9 response at a relatively higher level than discrimination studies which have not employed forced training on, and reinforcement of, the "incorrect" response. Isaacson, Olton, Bauer and Swart (1966) present evidence which indicates that the hippocampally lesioned S's inability to withhold a response in the passive avoidance task is directly related to the strength of that response. It is also possible that the ease with which hippocampectomized Ss can inhibit one response and initiate an alternative is dependent upon the relative strength, or probability, of those two responses.

These findings, which demonstrate that hippocampal Ss are capable of inhibiting an established response and initiating another, stand in marked contrast to the bulk of the data on the performance of such Ss when faced with similar tasks. It is not surprising that task variables, some of which have been discussed above, have the potential to profoundly influence the behavioral effect of physiological manipulations. What is surprising is that no concerted effort has been made

to explain such findings within the contexts of present formulations of hippocampal function, or to revise these formulations so that they may incorporate these results. All too often findings such as have been discussed here are neglected or dismissed as aberrent. Perhaps a detailed examination of the manner in which experimental manipulations can change or counteract the effects of physiological manipulations will provide increased insight into the role of neurophysiological systems in the intact organism.

The unexpected facilitation of discrimination reversal performance resulting from the neocortical damage sustained by the control Ss is most likely attributable to uncontrolled position preferences, as was discussed previously. Other experimentation on the effects of hippocampal ablation has typically involved analogous neocortically lesioned control Ss, and has regularly reported that such Ss do not differ from their unoperated counterparts. There are exceptions to this however, for Means, et al. (1969) have found that destruction of the neocortex overlying the hippocampus leads to a retardation of performance in the go no-go task; and Olton and Isaacson (1967) have reported that damage of this area, as well as this area plus the hippocampus, lengthens response latencies in avoidance and escape tasks.

In the present experiment neocortical destruction involved considerable portions of the rat neocortex comparable to Broadman's area 7, which is involved in somesthesis, particularly the integration of information on weight and the state of muscles and joints; areas 17

and 18, the visual projection and association areas, respectively; area 25, the entorhinal cortex; and area 37, which receives somesthetic and optic association fibers and, in man, is thought to be involved in the recognition of body image, individuality and continuity of personality, and of the self in relation to the environment (Kreig, 1957). Since a considerable portion of the hippocampal research has involved some damage of these areas it is possible that commonly observed hippocampal lesion deficits are in actuality a function of an interaction of the hippocampus and the neocortex which overlies it. Within this context, Douglas (1967) has observed that electrolytic lesions restricted to the hippocampus frequently do not produce the deficits seen in ablation studies involving neocortical destruction. It is also possible that the facilitated performance shown by the hippocampectomized Ss in the present study is fully accounted for by the effects of neocortical destruction. Questions such as these point to the relative primitiveness of our understanding of the role of the hippocampus in behavior, and to the importance of further research in this area.

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BIOGRAPHICAL SKETCH

Michael Arnold Milan was born on January 25, 1938, at New York, New York. He attended public school in New York state and graduated from Fayetteville-Manlius High School, Fayetteville, New York, in June, 1956. In May, 1960, he received the degree of Bachelor of Arts from Syracuse University. From 1960 until 1962, he served in the Adjutant General Corps of the United States Army. Following his release from the Army he was employed as a psychological research assistant with the Veteran's Administration Hospital in Syracuse, New York. In September, 1963, he enrolled in the graduate school of the University of Florida and received the Master of Arts degree in December, 1965. He served as a research assistant for Dr. H. S. Pennypacker and as an interim instructor with the Department of Psychology while matriculating for the Doctor of Philosophy degree in psychology.

This disseration was prepared under the direction of the chairman of the candidate's supervisory committee and has been approved by all members of that committee. It was submitted to the Dean of the College of Arts and Sciences and to the Graduate Council, and was approved as partial fulfillment of the requirements for the degree of Doctor of Philosophy.

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