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HIPPOCAMPAL STRUCTURE AND FUNCTION

IN THE ALBINO RAT

A DISSERTATION SUBMITTED TO THE GRADUATE DIVISION OF THE UNIVERSITY OF HAWAI'I IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF DOCTOR OF PHILOSOPHY

IN PSYCHOLOGY

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by

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HIPPOCAMPAL STRUCTURE AND FUNCTION
IN THE ALBINO RAT

by Ronald A. Fial

A dissertation submitted to the Graduate Division of the University of Hawaii in partial fulfillment of the requirements for the degree of Doctor of Philosophy

ABSTRACT

Theories concerning the function of the hippocampus in behavior have treated this structure as being homogenous in function. Inconsistencies in prior research have sometimes been attributed to differences in procedures or to extra-hippocampal damage resulting from the manipulation. While these factors have been shown to be potent variables, several writers have suggested that lesion location within the hippocampus is important and that smaller lesions are to be preferred to the generally large ones used in most earlier work. Recent work involving the organization of afferent and efferent pathways of the hippocampus has shown these to be well organized in terms of the various "fields" of the structure as distinguished by early anatomists.
In the present experiment 14 control operated (HC), 14 hippocampally lesioned (HE), 16 dorsal fornix lesioned (DF) and 16 lateral fimbria lesioned (LF) albino rats were tested in a series of five behavioral tasks in order to assess the relative behavioral effects of gross hippocampal lesions and lesions of the hippocampal efferent pathways. No group differences were found in open field activity levels, or in number of approaches to novel objects placed in an open field. In an orienting shift task with subjects under 23-hour water deprivation, HE, LF and DF groups had shorter initial latencies to drink than HC subjects, and the HE group made fewer orientations to light stimuli but not to tone stimuli. In a passive avoidance task the HE and LF groups made more errors than the HC group. In addition the HE group also made more errors than the LF and DF groups. HE and LF groups fought less than the DF and HC groups in a reflexive fighting task. The LF group also demonstrated shorter boxing-posture maintenance relative to the HC and DF groups at the end of the reflexive fighting session. All groups were similar to each other in a shock threshold rating situation, but the HE group had a lower jump threshold than the HC group.

The effects of the gross lesions were similar to those obtained in previous studies. Response perseveration, internal inhibition, and stimulus gating interpretations of hippocampal function were discussed and it was concluded that the gross lesion effects were best explained on the basis of an involvement of anxiety-defensive immobility mechanisms.
The pattern of results for the behavioral tasks changed markedly as a function of the efferent path lesion location. Unitary explanations were rejected, and it was concluded that anatomical differences in hippocampal projections are accompanied by separation of function. Two multi-functional interpretations were offered to account for the differences observed.
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INTRODUCTION

The hippocampal cortex is the largest component structure of the rhinencephalon or "smell brain" as named by the early anatomists. Along with the dentate gyrus, it makes up the structure called "Ammon's horn," because of its curved shape. The hippocampal cortex and the dentate gyrus are each formed in the shape of a U along the length of the horn, and the two U's are interlocked with each other (Figure 1). In the behavioral literature, references to the "hippocampus" refer to both the hippocampal cortex and the associated dentate gyrus. Early researchers believed that the hippocampus, along with other neural structures bordering the thalamus such as the amygdaloid nuclei, septal nuclei, and cingulate cortex, were primarily involved in the process of olfaction. Evoked potentials can be recorded from all of these areas after olfactory stimulation. But evoked potentials were also recorded in these areas after stimulation of many other sensory modalities (Gerrard, Marshal, and Saul, 1936; MacLean, Horowitz, and Robinson, 1952). Later evidence has indicated that while certain areas of the rhinencephalon (pre-pyriform, peri-amygdaloid, and olfactory tubercular areas) seem to be primary projection areas for olfactory sensation, most of the limbic cortex seems to have a more generalized function.

Herrick (1933) first suggested that the limbic system might influence processes involved in expressing emotion. Later, Papez (1937) originated a theory of emotion in which the hippocampus was
Figure 1-A. Idealized view of the hippocampus

Figure 1-B. Schematic diagram of the rat hippocampus (side view), with a section through the middle portion (from Douglas, 1967).
involved in the elicitation of emotional responses, with the cingulate cortex and hypothalamus involved in the experience of emotion and the integration of the emotional response respectively. MacLean (1949) in his theory of emotion, visualized the hippocampus as a control center for the limbic system. The hippocampus had the function of creating emotional arousal (sympathetic arousal), and the amygdala was to be reciprocally involved in the mediation of parasympathetic-like activities.

Although much of the limbic system seems to participate in the regulation of emotional behavior, no specificity of function at the level proposed by Papez (1937) and MacLean (1949) has been found. In particular, little or no change in overt emotional responsiveness has been found in animals with hippocampal lesions (Douglas, 1967).

The more recent interest in the hippocampus stems from two sources: (a) the work of Scoville (1954) indicating that bilateral lesions of the hippocampus result in an anterograde amnesia; and (b) the work of Isaacson, Douglas, and Moore (1961) indicating that rats with hippocampal lesions are faster than controls in learning to respond in a two-way active avoidance task.

In recent years, much research effort has been directed to the problem of functioning of the hippocampal cortex and its relationship to the behavior of organisms. According to Douglas (1967), within the years 1962 to 1967, more studies were published dealing with the effects of hippocampal lesions on behavior than in all previous years combined.
THE EFFECTS OF GROSS HIPPOCAMPAL LESIONS

Excellent reviews of the large body of hippocampal literature have appeared (Douglas, 1967; Kimble, 1968). Thus the present review will consider a sampling of studies stressing those behavioral measures most relevant to the proposed study. This material will be considered under two headings: (a) lesion-produced changes in untrained responses such as activity, food intake, etc.; and (b) lesion-produced effects upon the acquisition and performance of learned, experimenter-prescribed tasks.

Lesion effects on untrained behaviors

Somewhat contradictory results have been obtained from measurements of locomotor activity after hippocampal lesions. Gross hippocampal lesions have been found to increase nocturnal activity in rats in the home cage (Jarrard and Bunnel, 1968; Kirkby, Stein, Kimble and Kimble, 1967). But other investigators (Hostetter and Thomas, 1967; Spiegel, Hostetter and Thomas, 1966) have found no significant differences in activity between hippocampectomized and control subjects.

An increase in home cage water intake for hippocampectomized rats has been reported by Kimble and Coover (1966), but these authors found no corresponding increase in food intake for their subjects.

Blanchard and Fial (1968) placed rats in observation chambers and administered a series of footshocks. Post shock ratings of posture indicated a reliable decrease in immobility postures for hippocampectomized versus cortically lesioned controls.
Rats placed two at a time in a grid floor and repeatedly shocked exhibit pain-elicited aggressive responses toward each other. However, the subjects do not seem to actually inflict bodily damage upon each other (Azrin, Hutchinson, and Hake, 1963; Hutchinson, Ulrich, and Azrin, 1965). Blanchard and Blanchard (1968) and Blanchard, Blanchard, and Fial (1970) report decreased levels of reflexive fighting (as these responses have been called) in rats after hippocampectomy. The experimental animals not only fought less but also displayed significantly fewer of the "boxing" immobility postures which precede reflexive fighting with a high degree of regularity.

Hippocampectomized rats have been reported to have altered reactivity to shock. Using a shock-response rating procedure developed by Harvey and Lints (1965), Blanchard and Fial (1968) found a threshold decrease for jumping responses and a threshold increase for vocalization responses to foot shock for hippocampectomized rats.

Rats trained to run down a runway for food show less distractibility (slowing of running speed and exploring) when a novel stimulus is introduced during responding, if they have received hippocampal lesions (Wickelgren and Isaacson, 1963; Raphelson, Isaacson, and Douglas, 1965). These results were interpreted as indicating that hippocampectomized rats have a complete lack of distractibility. Riddel, Rothblat, and Wilson (1969) report similar results, but in their study hippocampectomized rats displayed a small but significant decrease in running time on the first trial of novel
stimulus introduction. This result was still in contrast to the many times larger decrease in speed exhibited by their control group upon novel stimulus introduction. Hendrickson, Kimble and Kimble (1969) investigated the "orienting response" dysfunction postulated by Raphelson et al. (1965) to account for the performance of their subjects. In a series of experiments, Hendrickson et al. found that following hippocampectomy, no orienting response impairment occurs to click or tone stimuli under ad-lib conditions of food and water deprivation, when a rat is not already orienting to a competing stimulus. But under 23-hr. water deprivation conditions with a water spout available in the test chamber, or under ad-lib conditions when the rat was already orienting to a stimulus, introduction of a novel stimulus resulted in a significant decrease in novel-stimulus orienting for hippocampal-lesioned versus control rats. Hendrickson et al. thus concluded that the hippocampus is involved in selective attentional processes in the rat.

These data indicate a quite varied effect of hippocampectomy upon untrained behaviors. Authors have reported changes in activity levels, water intake, the occurrence of immobility postures following shock, and changes in shock elicited aggression responses (reflexive fighting). In addition, changes in shock thresholds for at least two types of behaviors (jumping and vocalization) and shifts of attention from one active perceptual orientation to another have also been reported.
Effects of lesions on trained behaviors

Acquisition and performance in avoidance tasks is a sensitive indicator of hippocampal damage. But early investigators reported contradictory results for acquisition of an active avoidance response following hippocampectomy. This contradiction has been explained by distinguishing between two "types" of active avoidance tasks, the two-way active avoidance task and the one-way task. In the two-way task (shuttle box), the subject must repeatedly cross to the other side of a straight alley after a CS onset in order to avoid shock. Hippocampectomized rats have been reported to acquire this response pattern significantly faster than controls (Isaacson, Douglas, and Moore, 1961; Green, Beatty, and Schwartzbaum, 1967; Olton and Isaacson, 1968). In a one-way active avoidance task, the subject is usually placed in a start box, and must run to the goal box upon CS onset to avoid shock. The subject is then removed for an intertrial interval. Hippocampectomized rats have been reported to be poorer than controls in acquiring this response (Olton and Isaacson, 1968; Liss, 1968).

Two types of passive avoidance conditioning procedures have been commonly used to study the effects of hippocampal lesions. In the first type the subject is placed on a "safe area" surrounded by a charged electrical grid, and must learn to inhibit movement off of the platform to avoid shock. Teitelbaum and Milner (1963) and Blanchard and Fial (1968) have reported poorer performance in hippocampally lesioned rats trained in this type of task. In the
second type of passive avoidance conditioning, the subject is required to inhibit a previously trained response in order to avoid shock. Isaacson and Winklegren (1962) and Kimble (1963) have reported poorer performance for rats with extensive damage to the hippocampus.

Hippocampal lesions have also been reported to produce increased resistance to extinction in instrumental conditioning (Isaacson et al., 1961; Niki, 1962) and in classical conditioning (Niki, 1965). Also, poorer performance has been reported for discrimination reversal learning (Kimble and Kimble, 1965; Niki, 1967; Silveira, 1967; Thompson and Langer, 1963), Sidman avoidance (Ellen and Wilson, 1963) and Fixed Interval (FI-60) schedules of reinforcement (Beatty and Schwartzbaum, 1968), and after shifts in reinforcement schedules (Swanson and Isaacson, 1967). Finally, Winocur and Salzen (1968) found that hippocampectomized rats were poorer post-operatively on a transposition task, while Hostetter (1968) has reported that hippocampal lesions reduced the disruptive effect of electro-convulsive shock on consolidation of an avoidance response.

In the previously described studies of generally gross hippocampal lesion effects, the authors, in their interpretations of their results, generally viewed the entire hippocampal structure as homogeneous in function. The next section will present the results of studies indicating that the hippocampal formation may be differentiated into various anatomically distinct areas or "fields," both on the basis of its cellular organization and on the basis of the intrinsic and extrinsic neural connections of these areas.
The hippocampal cortex has been classified into seven layers by Lorente de Nó (1934) based on his own observations and the work of previous authors (see Figure 2-a). These layers are distinguished on the basis of the occurrence of cell types, presence and structure of dendritic processes, and the presence of fiber tracts of various origins and terminations. The prominent hippocampal pyramidal cells are arranged in the fifth layer, and send their axons through layer six to travel in the alveus (layer seven). The pyramidal cells send dendritic processes in two directions, toward both surfaces of the hippocampal cortex. The basal dendrites extend toward the alveus ending in layer six. The apical dendrites extend inward to receive connections from layers 4, 3, 2, and 1, where they terminate. Cajal (1901) divided the hippocampal cortex into an upper and lower "blade," after confirming earlier evidence that the fiber projections (called mossy fibers) from the dentate gyrus end in the upper but not the lower portions of this structure. Lorente de Nó (1934) divided the hippocampal cortex into fields in order to take into account its structure and connections. The division was made on the basis of size, appearance, location of the pyramidal cells, and changes in the layering from one field to another. His fields were called CA 1 (for cornu ammonis), CA 2, CA 3, and CA 4. In addition, he distinguished three subdivisions for field CA 1 and also for CA 3 (see Figure 2-b).
Figure 2-A. Layers of the hippocampal cortex (from Raisman et. al., 1965).

Figure 2-B. The four fields of the hippocampal cortex, as described by Lorente de Nó (1934).
Raisman, Cowan and Powell (1965) have reviewed earlier descriptions of the hippocampal connections. In that article, they also reported results of their own research, designed to investigate the possibility that the hippocampal afferent connections are organized in a fashion related to the four fields of Lorente de Nó.

They report that the hippocampus receives entorhinal projections via the alvear and perforant paths described by previous workers. It was discovered that the alvear pathway projects only to field CA 1, but that the perforant path is distributed to CA 1 and 2, and to a lesser extent to CA 3 and the dentate gyrus. Also confirmed was a septo-hippocampal projection running through the ventral-lateral part of the fimbria and terminating in fields CA 3 and 4 and to a small extent in the dentate gyrus. No trace of these fibers was found in the dorsal fornix or in field CA 1. Previous authors had suggested hippocampal afferents existed from the cingulate cortex, pyriform cortex, and induseum griseum. But the degeneration techniques used by Raisman et al. indicated that these areas project to entorhinal and subicular regions, and not to the hippocampus proper. Raisman et al. also found that a small number of afferent fibers may reach field CA 1 of the hippocampus from the hypothalamus and rostral midbrain, travelling in the medial forebrain bundle and the dorsal fornix.

This evidence suggests that the source and termination of hippocampal afferents is organized according to field. Raisman et al. also investigated the intrinsic hippocampal connections.

Within the hippocampus, CA 3 and 4 project axon collaterals to fields CA 1 and 2 (the Schaffer collaterals). The posterior part of
CA 1 projects to the subiculum, and possibly to the dentate gyrus. They were not able to show the mossy fiber projections from the dentate gyrus to CA 3 and 4, possibly because they used a different technique (Nauta) than earlier investigators (Golgi technique).

Raisman et al. have also traced the projections of various hippocampal commissural connections. They distinguish only two subdivisions (anterior and posterior) for field CA 1. CA 1 anterior does not seem to project to the contralateral hippocampus, but does receive commissural connections of indeterminate origin via the ventral hippocampal commissure. CA 1 posterior was found to project to its counterpart field across the same route. CA 2, 3, and 4 also project contralaterally, but it could not be determined if a one-to-one projection by field occurred. Commissural connections arriving at the dentate gyrus via the ventral hippocampal commissure were discovered, but the source could not be located. Thus there is evidence that the fields of the hippocampus are reciprocally connected to their contralateral counterparts, although the evidence is incomplete at this time.

In another study, Raisman, Cowan and Powell (1966) studied the hippocampal efferent connections as a function of the hippocampal fields. They found that fibers from CA 1 anterior run through the alveus to a point next to the midline, where they form the dorsal fornix. The fibers then project to the anterior thalamic nuclei, and the medial and lateral mamillary nuclei. CA 1 posterior projects via both the dorsal fornix and the dorsal part of the fimbria. These fibers may be traced to the septal and diagonal band nuclei, the nucleus
accumbens, and a post-commissural component projecting to the anterior thalamic nuclei and the mamillary nuclei. CA 2 projects through the fimbria and post-commissural fornix to the anterior thalamic nuclei and the mamillary bodies. Fibers from fields CA 3 and 4 travel in the alveus, fimbria, and precommissural fornix to the septal and diagonal band nuclei, the nucleus accumbens, and the area of the medial forebrain bundle. No extra hippocampal projection was found for the dentate gyrus.

The results are in agreement with the results of previous authors, especially the suggestion of Powell and Cowan (1955) that the fimbria may be related to the septum and rostral hypothalamus, and the dorsal fornix may be related to the mamillary nuclei.

In summary, the hippocampal cortex has been classified into various fields on the basis of cytoarchitectonic considerations. These fields have been investigated in various studies, and it has been found that the intrinsic and extrinsic projections are somewhat specific in terms of their points of origin, the anatomical locus of their paths, and their area of termination.
Raisman et al. (1966) offer evidence that the hippocampal cortex can be differentiated into various areas not only on the basis of its cytoarchitecture, but also on the basis of its efferent and afferent connections. They suggest an organization of the hippocampus into two systems. The first of these corresponds to the classical limbic circuit of Papez. Fibers of CA 1 project via the dorsal fornix to the anterior thalamic nuclei directly and also indirectly via hippocampal-mamillary and mamillo-thalamic tract connections. The connections are then relayed to the cingulate gyrus, then to the entorhinal area. From there fiber tracts arise terminating principally in field CA 1 and the dentate gyrus, with some overlap into fields CA 2 and CA 3 (CA 1 also has a medial septal projection). The second system involves fibers of CA 3 and 4 which project via the fimbria directly to the medial septal nucleus and the nucleus of the diagonal band, and also indirectly to these nuclei via a lateral septal-diagonal band relay. The diagonal band and medial septal nuclei again project back to fields CA 3 and CA 4.

It seemed reasonable to Raisman et al. that the degree of cellular organization seen in the hippocampus should result in a comparable degree of organization in the projections of the hippocampal fields. And this they have shown. In view of these findings, it seems reasonable to believe that there may be measurable behavioral effects due to the functioning of Raisman et al.'s two systems.
Because of the organization of the hippocampal fields, dorsal-anterior lesions mainly affect fields CA 1 and 2, while more posterior-ventral lesions mainly affect fields CA 3 and 4.

Lesion studies

The following survey will describe studies which involved using smaller hippocampal lesions of either anterior-dorsal or the more posterior-ventral hippocampus. Douglas (1967) has noted that those investigators who use small lesions often fail to find a passive avoidance deficit: "Many of the apparent contradictions in the literature may be traced to the use of small (usually electrolytic) lesions of the dorsal-rostral hippocampus . . . (p. 425)." Douglas notes that Snyder and Isaacson (1965) found that the magnitude of the deficit in performance following hippocampal lesions was a function of lesion size.

Probably the first study to show behavioral differences as a function of hippocampal lesion placement was that of Kimura (1958). Rats with posterior hippocampal lesions were poorer than controls in passive avoidance conditioning. Those with anterior hippocampal lesions showed no deficit. Thus, Kimura's evidence implies that deficits in passive avoidance (food deprivation-trained approach response) may arise from damage to CA 3 and 4 but not from areas CA 1 and 2.

Kaada, Rasmussen, and Kveim (1962) reported a failure to find the usual passive avoidance decrement following hippocampal lesions. Their lesions were apparently smaller lesions of the dorsal hippocampus and included some fornix damage.
Thomas and Otis (1958) found that rats with small rostral hippocampal lesions suffered a severe deficit in acquisition of an active avoidance response in a double grill box. Andy, Peeler and Foshee (1967), using a two-way active avoidance task (shuttle box) found no differences in acquisition, but on retention rats with dorsal lesions were poorer; whereas, rats with ventral lesions were no different from controls. Jarrard (1967) in a preliminary report, reports that using a one-way active avoidance situation, rats with posterior-ventral hippocampal lesions were much poorer than those with anterior dorsal lesions, although anterior and posterior lesioned groups were poorer than controls.

It had been shown previously that hippocampally lesioned rats were better than controls on the two-way active avoidance task but poorer than controls on the one-way task (Olton and Isaacson, 1968; Green, Beatty, and Schwartzbaum, 1967).

Roberts, Dember and Brodwick (1962) report results in which lesions of the hippocampus including the most anterior portion resulted in a lack of habituation of locomotor activity in a Y-maze. Rats with lesions sparing the most anterior hippocampus did not show significant experimental-control differences. Rats with lesions in the dorsal-medial hippocampus were found by Teitelbaum and Milner (1963) to be more active than normal and cortically lesioned controls in an activity cage. These rats also were poorer in a passive task involving inhibition of movement on a wooden platform surrounded by an electrified grid. Douglas and Isaacson (1964) found that anterior-dorsal lesions increased activity in an exploratory box (measuring light beam interruptions).
relative to controls. In an activity wheel, subjects with electrolytically produced lesions had significantly higher activity, but those with lesions produced by aspiration did not show this increase. It is possible that cortical damage resulting from the aspiration technique may have influenced this result. Jackson (1967) has also found that anterior dorsal lesions increase activity in a stabilimeter and also in a 6-unit Lashley maze. But rats with medio-dorsal lesions were even less active than controls in the stabilimeter.

Summary of lesion effects

It would then appear that anterior-dorsal hippocampal lesions produce increases in locomotor activity relative to controls in novel situations. For passive avoidance conditioning, the data are contradictory, Teitelbaum and Milner (1963) reporting poorer performance and Kimura (1958) and Kaada et al. (1964) failing to find such a deficit. This difference may result from differences in the tasks, in that the Teitelbaum task did not involve a trained approach response, and would perhaps be more sensitive to the increased activity levels reported for anterior-dorsal lesions. The data are not clear for the two-way active avoidance situation. One study (Thomas and Otis, 1958) reported an acquisition deficit while another study (Ande et al., 1967) reported no acquisition deficit. However, the Andy et al. rats were poorer on a retention test for this task. For the one-way active avoidance situation, Jarrard (1967) reports that rats with anterior-dorsal hippocampal lesions show slower acquisition than controls, but with a smaller impairment than that produced by posterior ventral lesions.
Ventral hippocampal lesions apparently do not increase activity and may actually decrease it (Jackson, 1967). Posterior hippocampal lesions seem to impair passive avoidance performance using a trained approach task (Kimura, 1958). Again the data are not clear for the active avoidance task, Jarrard (1967) reporting a deficit for a one-way situation, and Andy et al. (1967) reporting no difference in acquisition or retention of a two-way active task for rats with posterior-ventral lesions. It must be emphasized that these studies constitute very indirect evidence for behavioral differentiation of the hippocampal fields, since the dorsal versus ventral lesions are not "pure" lesions of CA 1 - 2 versus CA 3 - 4 (or CA 1 versus CA 3 - 4 as in the Raisman et al. 1966 distinction), and lesions vary in size and location from author to author.

Review of tractotomy studies

Because of the complex topographical relationships of the various hippocampal fields, it is difficult to make a lesion of a single field only. Thus while anterior-dorsal hippocampal lesions do great damage to fields CA 1 and 2, the most ventral portions of these fields are spared and damage is done also to fields CA 3 and 4. When we reconsider Raisman et al.'s (1966) two systems, we find that Field CA 1 anterior projects through the dorsal fornix bundle along with much of the posterior part of CA 1. Fields CA 2, 3, and 4 project fibers through the alveus and fimbria. These paths run closely together in the anterior hippocampal commisure, but the
pre-commissural fibers are quite separated spatially. Fibers of the
dorsal fornix run anteriorly along the midline just ventral to the
corpus callosum; whereas the fimbrial fibers gather into a bundle
more laterally in the lateral ventricles. It may be possible to
obtain some information about differential function in terms of the
various hippocampal fields by looking at the behavior of animals
which have received selective tractotomy of these two major efferent
pathways.

There have not been many studies which have examined behavioral
effects of dorsal-fornix and fimbria section. MacDougall, Van Hoesen
and Mitchell (1969) found that lesions of the dorsal fornix had no
effect on the acquisition of a DRL (20 sec.) responding schedule by
rats, but that combined dorsal-fornix fimbria lesions resulted in a
large and significant deficit. Septal lesions sparing supra-commissural
septum and pre-commissural hippocampal fibers produced deficits in
performance which were not as severe as the total fornix-fimbrial
lesions. The authors suggested that the two types of lesions represent
damage to a common system involved in response inhibition and timing
behavior.

Carey (1968) lesioned the fornix bundle and diagonal band nuclei
(Raisman's 2nd system) and found poorer DRL (22 sec.) performance.
However, rats with lesions of the post commisural fornix projecting to
the mammillary bodies (Raisman's 1st system) showed no DRL deficit
(there scores may have actually been better than those of the control
group). This result provides some direct support for Raisman et al.'s
2-system distinction.
Van Hoesen, MacDougall and Mitchell (1969) found that lesions of the dorsal fornix and fimbria at the level of the horizontal columns facilitated 2-way shuttle box performance and produced small deficits (significant for experimental versus normal control group at $p < .01$ but not significant between experimental and operated control) in passive avoidance utilizing a trained food-approach response. Septal and stria medularis lesioned groups performed better than controls on the active avoidance task and poorer on the passive avoidance task. This was interpreted as an indication that "... the fornix system is as important, or even more so, than the stria medularis system in accounting for the enhanced performance seen on active avoidance with septal lesions, but has little to do with the passive-avoidance deficit (p. 87)." They suggest further that the passive avoidance deficit found with hippocampal lesions may be due to damage to other areas, as has been suggested by McCleary (1966). Fischman and McCleary (unpublished study, cited by McCleary, 1966, p. 265) had sectioned the horizontal columns of the fornix in cats and found no passive avoidance deficit, but found poorer reversal of a position habit. McCleary (1966) noted that small lesions limited to the hippocampus (e.g., Kveim, et al., 1964) do not produce a passive avoidance deficit, but that larger, posterior hippocampal lesions "... potentially involving such adjacent structures as the stria terminalis or caudate ..." do produce a deficit. Rather than suggest that the deficit results from differential function of the hippocampal fields, he suggests that some of the effects reported for hippocampal damage may be due to inadvertent damage to nearby structures.
In addition to stria terminalis and caudate nucleus damage, McCleary implicated cortical damage resulting in studies using the aspiration technique.

Liss (1968) found that rats with small, rather dorsal hippocampal lesions and rats with lateral fornixotomy (sparing midline fibers from CA 1 and 2) were not different from controls in one-way active or passive avoidance tasks with massed trials (5 sec. ITI). However, when trials were distributed (10 min. ITI) they were poorer than controls on both tasks. Later testing on a two-way active avoidance task (shuttle box) showed both experimental groups significantly better than controls (p < .05). The two-way active avoidance results of the Van Hoesen et al. and Liss studies are in accord with each other. However, the passive avoidance results might be explained in two ways. One would be on the basis of the use of massed trials by Van Hoesen et al. which actually replicates the results with massed passive avoidance of Liss. Another explanation might involve the fact that Van Hoesen et al. used a complete dorsal fornix-fimbria lesion whereas Liss destroyed only the more lateral fimbrial connections.

In Liss' study, the striking correspondences of behavioral results between posterior lesions (involving CA 3 - 4) and lateral fornix-fimbrial (fibers from CA 3 - 4) section suggests that the mediation of posterior lesion effects may be via lateral fimbrial pathway.
Summary of tractotomy studies

Thus there exists some evidence that the behavioral effects of hippocampal lesions can be differentiated on the basis of hippocampal cytoarchitecture and corresponding sub-cortical efferent projections. If the effects of posterior-ventral hippocampal lesions (CA 3 - 4) are mediated via the lateral lying fimbria, then we would expect that fimbrial destruction would produce the same constellation of behavioral results as posterior-ventral lesions.

The evidence so far presented supports this idea. Both types of manipulation have been reported to produce deficits in a trained passive avoidance situation, and in a one-way active avoidance situation. Also, both manipulations have been reported by Liss to facilitate two-way active avoidance conditioning. Posterior-ventral hippocampal lesions have been reported to produce no change or a slight decrement in activity situations. No data is available for activity changes as a function of fimbrial section, and none for performance in passive avoidance situations without a trained approach response for either type of manipulation.

If the effects of anterior-dorsal hippocampal lesions (CA 1) are mediated via the medial-lying dorsal fornix, then it would be expected that dorsal fornix destruction would produce the same types of behavioral deficit or facilitation as anterior-dorsal hippocampal lesions. Very little data exists bearing on this point. Anterior-dorsal lesions have been reported to produce no deficits in a trained approach passive situation (Kimura, 1958; Kaada et al., 1964) and a possible deficit in a one-way active avoidance situation (Jarrard, 1967).
Poorer performance has been reported for an untrained passive
(Teitelbaum and Milner, 1963) and a two-way active avoidance situation
(Thomas and Otis, 1958). And an unequivocal increase in activity,
using Y maze and light beam interruption techniques, has been reported
for this manipulation (Roberts et al., 1962; Douglas and Isaacson,
1964). But no data have been reported for any of these learning tasks
or for activity levels following dorsal fornicotomy. The data on
dorsal fornicotomy are limited to the lack of differences on a DRL
schedule reported by McDougall et al. (1969), and the similar yet
more indirect evidence reported by Carey (1968).

Summary of above effects relating them to results of
gross hippocampal ablation

These data will now be related to the data for gross hippo-
campal lesions. For gross hippocampal, posterior-ventral and
fimbrial lesions the data show performance deficits in passive tasks
with a trained approach component, deficits in one-way active
avoidance, and facilitation of two-way active avoidance (shuttle box)
performance. For an untrained passive avoidance task, data is only
available for the gross lesions which show a performance decrement.
Gross lesions have resulted in an increase in activity levels
(Jarrard, 1967; Schmaltz and Isaacson, 1967) while posterior-ventral
lesions show no change or even a decrease in activity levels
(Jackson, 1967).

Since no data have been published for these tasks after dorsal
fornicotomy, the comparison must be limited to gross versus anterior-
dorsal lesions. The data are congruent for the untrained passive
avoidance task, both anterior-dorsal and gross lesions yielding poorer performance. The data are also similar for activity tasks, anterior-dorsal and the majority of gross hippocampal lesion studies reporting activity increases. Differences have been reported for three tasks: (1) Gross hippocampectomized poorer but anterior-dorsal lesioned not different from controls in a trained passive task; (2) Gross hippocampal subjects better but anterior-dorsal lesioned subjects poorer or not different in acquisition and poorer on retention for a two-way active avoidance task; and (3) Gross subjects poorer and anterior-dorsal lesioned subjects somewhat poorer (preliminary data, Jarrard, 1967) for a one-way active avoidance task.

Thus there is some evidence that smaller localized lesions (anterior versus posterior) of the hippocampus and of the hippocampal efferent fibers (dorsal fornix versus fimbria) may partition among themselves some of the effects of gross hippocampal lesions.

**Other evidence**

Several other lines of evidence indicate that the hippocampus is not a homogeneous structure. Jarrard (1967) in a preliminary report, indicated that carbacol injection into CA 1 resulted in an increase in drinking but that injection into CA 3 - 4 did not result in an increase.

Dithizone (diphenylthiocarbazole) injection results in very little staining of areas CA 1 - 2; whereas CA 3 - 4 are intensively stained (Fleischhauer and Horstmann, 1957), especially in the posterior portions of CA 3 - 4 (Purpura, 1959). These data indicate that some aspects of hippocampal differentiation may be chemical in nature.
Feldman (1962) has reported inhibition of evoked potentials in the postero-lateral hypothalamus (evoked potentials produced by sensory and lemniscal stimulation) when the hippocampus is stimulated. Thompson and Hawkins (1961) have reported that postero-lateral hypothalamic lesions produce deficits in avoidance CRs in the rat. However, mamillary lesions had no effect on this task. Rich and Thompson (1965) found large deficits in post-operative retention of an active avoidance task in rats with postero-lateral hypothalamic lesions. However, rats with postero-medial hypothalamic lesions were only slightly different from controls. These results provide very indirect evidence for differentiation of hippocampal function in that CA 1 projects fibers via the dorsal fornix to the mamillary bodies, and CA 2 - 3 - 4 may project to the postero-lateral hypothalamus via septum and medial forebrain bundle.
PURPOSE OF THE PRESENT INVESTIGATION

The present investigation had three main purposes:

A. To search for additional evidence indicating whether the behavioral effects of hippocampal function can be differentially related to the various cytoarchitectural fields. This was accomplished by comparing the behavioral effects of gross hippocampal destruction with the effects resulting from differential interruption of one or the other of the two major sub-cortical efferent projections originating from those fields.

B. To specify, differentially, the nature of the behavioral changes involved. Behavioral tasks were selected which have been shown in the past to differentiate hippocampally lesioned subjects from controls. Tasks were also selected which were maximally different in nature, in order to insure that a wide spectrum of behaviors was measured. Thus there was a higher probability of finding differential lesion effects than there would have been had a more homogenous set of tasks been chosen.

C. If some differentiation were indicated by the data, to use this information to account for some of the divergent results obtained with smaller, variously placed, hippocampal lesions, and perhaps contribute to a more comprehensive idea of hippocampal functioning.

Implicit in the above approach was the idea that damage to specific hippocampal fields and damage to the efferent projections from those fields might have similar behavioral consequences. This writer has found no data bearing on the question which would contradict this idea, and much data which support it.
SURGICAL AND HISTOLOGICAL PROCEDURES

Subjects The subjects were 60 male albino rats approximately 130 days old, from the colony maintained by the University of Hawaii Psychology Department. The subjects were randomly divided into four groups; (1) a gross hippocampal (HE) group of 14 rats, (2) a dorsal fornix (DF) group of 16 rats, (3) a lateral fimbria (LF) group of 16 rats, and (4) a control group (HC) of 14 rats.

Surgical procedures All surgery was done in one stage using 50 mg/kg sodium pentobarbital anesthesia. The subjects were anesthetized and mounted in a stereotaxic instrument. The scalp was retracted along with muscle and periosteum and the skull drilled to accommodate a lesioning electrode. Experimental group subjects were then electrolytically lesioned using the coordinate system of König and Klippel (1963) with exact coordinates and lesion current levels determined by pre-experimental trial placements in pilot subjects. HE subjects received bilateral lesions in the dorsal and posterior-ventral regions of the hippocampus (anterior 5.0 mm, lateral 4.8 mm, lesioned at vertical 0.9 and -3.3 mm with the electrode positioned at a 5 degree angle from the vertical towards the side of the lesion) using a current of 2.5 ma for 10 seconds. DF subjects received a midline lesion aimed at the dorsal fornix at the most rostral extent of the hippocampus (anterior 7.2 mm, lateral 0.0 mm, vertical 3.7, with the electrode positioned at a 25 degree angle from the vertical) using a current of 1.7 ma for 15 seconds. LF subjects received bilateral lesions aimed at the lateral fimbria approximately 1 mm
posterior to the hippocampal commissure (anterior 7.0 mm, lateral 4.0 mm, vertical 4.0 mm) using a current of 2.0 mA for 10 seconds. HC subjects had the lesioning electrode lowered and withdrawn bilaterally through the cortex to the dorsal border of the hippocampus, using the coordinates given for the HE group, however, no lesion was made. The lesion electrode consisted of a 0.5 mm diameter stainless steel needle, insulated except for 0.4 mm at the rounded tip with GE 9700 insulating varnish. Lesions were produced by passing direct current supplied by a constant current source from an anal electrode to the uninsulated tip of the lesion electrode (anode).

**Histology** Following the behavioral procedures, all subjects were anesthetized and perfused through the heart with physiological saline followed by 10% formalin. After fixing, the skulls were opened, and the brains were removed and sectioned through the area of the lesion at 50 μm using the frozen section technique. Every seventh section was mounted and stained with cresyl violet. Photographs were taken of representative sections for each of the experimental groups, showing typical destruction resulting from the lesions.

**Histological results** The DF lesions started immediately posterior to the hippocampal commissure and transected the dorsal fornix in all DF subjects. Cortical and corpus callosum damage was restricted to the unilateral lesioning needle tract. The area of destruction also included the most anterior-medial aspect of the hippocampus and subiculum. Appendix Figure A presents a photograph of a typical dorsal fornix section.
All LF subjects sustained bilateral lateral fimbria destruction ranging from 30 to 100 percent. In 7 of the subjects, the destruction extended into the adjacent alveus and hippocampal field CA 3. Two subjects also had unilateral damage to the lateral geniculate nucleus. Cortical and corpus callosum damage was restricted to the lesioning needle tracts with slight lesion incursions into the corpus callosum. Appendix Figure B presents a typical lateral fimbria section.

The HE subjects sustained bilateral damage to 80 to 95 percent of the hippocampus and dentate gyrus, sparing only small amounts of the most anterior and most posterior portions of the structures. All subjects sustained bilateral cortical and corpus callosum damage at the lesion electrode entry points. Slight unilateral destruction in the lateral and medial geniculate nuclei was noted in three subjects, unilateral amygdaloid damage in two subjects, and slight entorhinal damage in four subjects. No relationship was noted between these results and scores in the behavioral testing situations. Appendix Figure C presents a photograph of a typical hippocampal lesion.
EXPERIMENT 1 ACTIVITY MEASURE

A number of studies (Douglas and Isaacson, 1964; Kirkby, et al., 1967) have reported increased activity levels after hippocampal lesions. Although other investigators (Hostetter and Thomas, 1967; Spiegel, et al., 1966) have failed to find this effect, such a difference could be a potent variable in the other behavioral tasks used in the present investigation. Therefore the subjects in this experiment were tested in an open field activity box similar to those used by previous investigators.

Blanchard and Blanchard (unpublished data) have found that hippocampal lesioned rats make fewer approaches than controls to novel objects in a situation with which they are familiar. A similar measure was incorporated into the present experiment by measuring the number and duration of novel object approaches in a retest session.

**Apparatus** The apparatus was an 88 x 88 x 28.5 cm exploratory box. The bottom consisted of an off-white formica table top, and the top of the box was covered with a section of 0.6 cm$^2$ mesh chicken wire. The box was housed behind a one-way vision glass in a sound attenuated room, as were all of the other apparatus items used in the present study. Illumination was provided by a 60 watt incandescent bulb 1.5 meters above the floor of the test box. A random noise generator connected to a loudspeaker provided a 60 db (re 0.0002 microbar) "speech noise" (100-6000 Hz band limited white noise) in the box for the purpose of masking extraneous sounds. The floor of the box was
marked off into nine equal area squares by black lines. Room temperature was maintained at 24.5° C throughout this and all following procedures in the present study.

**Method** One week after surgery, each subject was placed in the open field box, and the number of squares crossed per minute for a 10-minute period was recorded. The subject was then removed to its home cage for a 10-minute period during which four objects were placed in the box. These consisted of a 3 x 3 x 4 cm 3-way brown bakelite electrical socket, a black 2.5 x 2.5 x 2.0 cm wood block mounted on edge on another block of the same size, a white 2.5 cm square plastic block, and a 6.5 x 3.5 x 2.5 cm DPDT ceramic base knife switch, each placed 31 cm from one of the box corners on a line bisecting the corner. The subject was then replaced in the box, and the number of squares crossed per minute was again rated for a 10 minute period. In addition, the amount of time spent approaching the objects and the number of approaches to the objects were recorded by the observer on electrically operated and sequenced timers and counters. Object approach was defined as the nose of the subject moving to within one-fourth inch of an object. The open field activity data was summarized for each experimental group for pre- and post-activity sessions, object approaches and duration of approaches for the 10-minute periods. In this and in all other procedures in the present study, the experimenter did not know the experimental condition of the subject or subjects being tested (i.e., all procedures were run blind).
RESULTS AND DISCUSSION

The means and standard deviations for the open field data are presented in Table 1. As this table shows, the HE subjects were slightly more active than controls in the initial 10-minute pretest. However, this difference was not statistically reliable ($t = .544$, $df = 26$, $p > .05$). It may be noted that these results are in agreement with those of many previous studies. Hostetter and Thomas (1967) found no open field activity differences nor activity differences in two maze learning situations for hippocampectomized rats. Spiegel, et al. (1966) repeated this experiment with posterior-ventral lesions and found the same lack of activity differences. On the other hand, Kirkby, et al. (1967) found reliably higher open field activity levels in hippocampectomized rats, and Douglas and Isaacson (1964) found that anterior lesions increase exploratory box activity (counting light beam-photo cell interruptions) and in an activity wheel. Bender, Hostetter, and Thomas (1968) addressed themselves to the inconsistent results of studies of this type. They reported that lesions of the hippocampus alone did not produce reliable open field activity increases, but that lesions which extended into the posterior cortex (entorhinal) produced a marked increase in activity. They concluded that such cortical involvement is necessary in addition to the hippocampal damage to produce the increase in open field activity sometimes reported to follow hippocampal lesions. The subjects in the present study sustained minimal entorhinal damage, and thus the present
Table 1. — Group means and standard deviations (SD) for number of line crossings during the activity measure

<table>
<thead>
<tr>
<th>Group:</th>
<th>HC</th>
<th>DF</th>
<th>LF</th>
<th>HE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First ten minutes</strong></td>
<td>Mean</td>
<td>89.78</td>
<td>79.00</td>
<td>74.44</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>23.18</td>
<td>21.67</td>
<td>34.63</td>
</tr>
<tr>
<td><strong>Second ten minutes</strong></td>
<td>Mean</td>
<td>42.50</td>
<td>37.31</td>
<td>39.12</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>36.65</td>
<td>26.24</td>
<td>40.48</td>
</tr>
</tbody>
</table>

Table 2. — Group means and standard deviations (SD) for number and duration of object approaches during second ten minutes of activity measure

<table>
<thead>
<tr>
<th>Group:</th>
<th>HC</th>
<th>DF</th>
<th>LF</th>
<th>HE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Number</strong></td>
<td>Mean</td>
<td>19.79</td>
<td>20.12</td>
<td>19.06</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>14.50</td>
<td>13.77</td>
<td>14.17</td>
</tr>
<tr>
<td><strong>Duration</strong></td>
<td>Mean</td>
<td>31.17</td>
<td>25.05</td>
<td>22.70</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>20.75</td>
<td>20.39</td>
<td>15.02</td>
</tr>
</tbody>
</table>
results tend to support the activity difference interpretation of Bender et al. (1968). The DF and LF groups suffered no cortical damage except for the needle track, and both groups have first activity session means that are slightly lower than those of the HC group. These scores indicate no differential effect on activity from dorsal fornix versus fimbria destruction. The lack of reliable activity differences between groups should thus enable a more clearcut interpretation of results in the other behavioral test situations used in this study.

The means and standard deviations for object approaches and durations are presented in Table 2. The HE, LF, and DF groups did, on the average approach the objects for a shorter duration than the controls, but the number of approaches measure did not show this effect. The approach differences were not statistically reliable, and provide no evidence for a distinction between dorsal fornix and fimbria.
Hendrickson, et al. (1968) have reported that hippocampal lesions reduce the tendency for rats to shift attention from one stimulus or task, to a novel stimulus. These authors have suggested that the hippocampus acts to inhibit the initial, ongoing response, permitting a shift of attention to the novel stimulus. They have also suggested that there is an important relationship between this mechanism and other effects seen with hippocampal lesions, such as passive avoidance deficits, poorer CER development, and impaired discrimination reversal. The present experiment was designed to examine the possibility that the "attention-shift" mechanism is topographically organized in terms of the hippocampal efferent tracts.

There is some preliminary evidence (Blanchard and Blanchard, unpublished data) that hippocampectomized rats stop exploring and eat faster than controls when placed in a novel open field situation. In this experiment, the subjects' latency to start drinking was also recorded, in order to test the generality of this effect, and to discover how it might be affected by dorsal fornix or fimbria section.

**Apparatus** Each subject was tested in its 17.5 cm wide x 24.5 cm long x 18.0 cm high home cage, which was moved to the experimental room and placed in an acoustical tile lined box open on the top and front. Each home cage had opaque side and rear walls, and a wire mesh bottom and front. A frosted 7 watt bulb was situated 4 cm from the cage front on the left side and a 10 cm diameter loudspeaker was placed 4 cm from the
cage front on the right side. A water bottle could be attached to the middle of the cage front. The water spout was insulated except for the tip and connected to a drinkometer circuit which recorded drinking activity on one channel of a Grass 7-channel polygraph. The experimenter was provided with buttons for the presentation of a 0.1 second light flash or a 0.1 second 1000 Hz pure sine wave tone via the light and loudspeaker respectively. The light flash consisted of an increase from a resting level (to correct for thermal lag) of 30 volts to a stimulus-on level of 80 volts RMS AC, providing a 22 foot-candle flash in the middle of the cage. The ambient light level was 5 foot-candles. The level of the tone was 75 db in the middle of the cage. Stimulus onset and offset were also recorded on the polygraph record.

Method Thirteen days after being tested in the activity measure, the subjects were placed on a 23-hr 45-min. water-deprivation schedule for 5 days prior to testing. They were deprived of all water for 24-hrs immediately prior to testing. At the time of testing, all food was removed from the rat's cage, the cage was properly positioned in the experimental apparatus, and the plexiglass cover attached, followed by the simultaneous attachment of the water bottle and the start of the polygraph. After the rat had been drinking for 15 seconds, a series of 10 tones and 10 light flashes were presented, at approximately 10 second intervals. Presentation of the tone and flash stimuli were alternated, and an attempt was made to present the first five tones and light flashes while the animal was drinking, and the second five while the animal was moving in the cage. To aid in scoring responses,
all trials were recorded by a videotape recorder, connected to a TV camera above the cage. The orienting response was defined as an alerted arrest of on-going behavior and/or abrupt head turning toward the source of the stimulus concomitant with or immediately following stimulus presentation (cf. Hendrickson, et al., 1969). The number of responses made was recorded by the experimenter during the trials, and verified the next day by playback of the videotape of the session and by consulting the drinking record on the polygraph. The amount of time each subject spent exploring before drinking a total of 5 seconds was also recorded.
RESULTS AND DISCUSSION

Table 3 presents mean number of orienting responses to light and tone stimuli by the experimental and control groups. Analysis of orienting shift trials when the subjects were drinking versus not drinking indicated no differences for any condition. Thus these results were combined for the present analysis. For the light stimuli, all three experimental groups differed from the control group in the predicted direction, making fewer orientations than the HC group. The HE group was reliably different from controls ($t = 2.11, df = 26, p < .025$, one-tailed $t$), but DF and LF differences from controls were not statistically reliable. For the tone stimuli, the differences were also in the expected direction, but were not statistically reliable.

Thus the Hendrickson et al. (1969) data is partially replicated by the present data for the HE group. Some procedural differences between the two studies may be relevant to the present failure to completely replicate the Hendrickson et al. results. In the earlier study click stimuli were used, while in the present study tone (sine wave) stimuli were used. However, the operated control subjects in this task responded to the tone on 60 percent of the trials, and those of Hendrickson et al. responded on 66 percent of the trials, so it cannot be argued that an ineffective sound stimulus was used. Hendrickson et al. also utilized an aspiration technique to produce hippocampal lesions, which produced large amounts of cortical destruction. In contrast, the electrolytic lesion technique utilized in the present study resulted in
Table 3. -- Group means and standard deviations (SD) for number of orientations to tone and light stimuli.
(10 possible)

<table>
<thead>
<tr>
<th>Group:</th>
<th>Tone Stimuli</th>
<th>Light Stimuli</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td>HC</td>
<td>6.00</td>
<td>5.36</td>
</tr>
<tr>
<td>DF</td>
<td>5.94</td>
<td>4.86</td>
</tr>
<tr>
<td>LF</td>
<td>5.31</td>
<td>4.50</td>
</tr>
<tr>
<td>HE</td>
<td>4.86</td>
<td>4.00</td>
</tr>
<tr>
<td>SD</td>
<td>2.42</td>
<td>2.13</td>
</tr>
<tr>
<td></td>
<td>2.43</td>
<td>2.82</td>
</tr>
<tr>
<td></td>
<td>2.15</td>
<td>2.13</td>
</tr>
<tr>
<td></td>
<td>1.79</td>
<td>1.11</td>
</tr>
</tbody>
</table>

Table 4. -- Group means and standard deviations (SD) for latency to drink in the attention task (number of seconds).

<table>
<thead>
<tr>
<th>Group:</th>
<th>HC</th>
<th>DF</th>
<th>LF</th>
<th>HE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>199.93</td>
<td>127.56</td>
<td>90.06</td>
<td>60.14</td>
</tr>
<tr>
<td>SD</td>
<td>106.55</td>
<td>78.90</td>
<td>90.92</td>
<td>77.85</td>
</tr>
</tbody>
</table>
minimal damage to cortex. Thus it is possible that cortical damage enhances the orienting shift deficit, providing a larger effect than was observed in the present study. The lack of DF and LF group differences suggests that the orienting shift deficit, or lack of "distractability," cannot be definitely ascribed to either DF or LF damage. However, since both DF and LF results were in the same direction as those of the HE rats, it is also not possible to interpret this lack of reliable results as indicating that hippocampal influence on the orienting-shift measure is mediated by an efferent pathway other than the dorsal fornix or lateral fimbria.

The mean latency to drink is presented in Table 4. All of the lesioned groups had reliably shorter drinking latencies than those of the HC rats (t = 2.13, 3.05, and 3.96, df = 28, 28, and 26, p < .05, .01, and .002 respectively for DF, LF, and HE groups compared with the HC group). There were no reliable differences between the DF, LF, and HE groups. There are several possible ways to interpret this finding. Kimble and Coover (1966) found that hippocampal lesioned rats drank 16 to 21 percent more water than unoperated controls at a postoperative time similar to that at which the present subjects were run. However, Boitano, Lubar, Auer and Furnald (1968) reported no increase in food or water intake for subjects with large hippocampal lesions. If hippocampal subjects drink more, they might be expected to make fewer orientations to other stimuli while drinking. Since the present results include deficits for HE subjects while not drinking, as well as when the subjects were
drinking, it is obvious that a postulated increase in drinking cannot entirely account for the observed deficit of lesioned rats in the orienting-shift task. Hendrickson, et al. also found orienting-shift deficits under ad-lib food and water conditions, which also suggests the inadequacy of an interpretation based on increased water intake.
EXPERIMENT 3 PASSIVE AVOIDANCE CONDITIONING

Hippocampally lesioned rats are consistently poor in passive avoidance performance (cf. Teitelbaum and Milner, 1963; Isaacson and Wickelgren, 1962). Kimura (1958) has reported a deficit in performance for rats with posterior-ventral hippocampal lesions, but no effect for rats with anterior-dorsal hippocampal lesions, in a trained-approach passive avoidance task in a straight alley. In the present experiment, a passive avoidance measure was taken for the HC, DF, LF, and HE groups in order to examine the possibility that the different effects found by Kimura might be mediated via the fornix and fimbria pathways. A safe-area passive task was used, utilizing a center safe area in an electrified straight alley. This task was chosen because it is minimally sensitive to the effects of lesion produced changes in appetitive drive levels, and thus affords a clearer interpretation of the passive avoidance results.

Apparatus Passive avoidance conditioning was carried out in a 28 cm high x 15.5 cm wide x 122 cm long straight alley with unpainted wood sides and ends, a plexiglass top, and grid floor. The grid floor was electrifiable except for a 20 cm section in the center. The alley was lighted by a 60 watt incandescent bulb in a ceiling fixture 1.4 meters above the apparatus. Shock was supplied by a Grason-Stadler model E 1064 scrambled shock source. A button was provided for the experimenter to depress while the subject was in the shock area, and which operated electromechanical counters and an event pen on a polygraph. The shock current was also recorded on the polygraph.
Method  Eleven days after the orientation task, subjects were tested individually in the passive avoidance alley. Each subject was placed in one end of the alley and allowed to explore for two minutes. At the end of this time, or as soon after that as the subject was on the shocked area, the floor was electrified (1.3 ma) for a period of 10 minutes. The subject was required to locate the non-shocked center section and remain there to avoid shock. The experimenter depressed the event pen button whenever the subject was receiving shock. An error was defined as any movement from the safe area which resulted in a shock for the subject.
RESULTS AND DISCUSSION

The mean number of errors for all groups is presented in Table 5. The HE group made reliably more errors than the HC, DF, and LF groups ($t = 5.28, 2.96, \text{ and } 3.11, df = 26, 28, 28, p < .001$, one-tailed; .01, and .01 respectively). This replicates the results reported by Blanchard and Fial (1968) for HE-HC differences. In addition the LF group also made significantly more errors than the HC group ($t = 2.50, df = 28, p < .02$).

Although the DF group's error mean was very similar to that of the LF group (32.37 as compared to 34.44), the DF group was not reliably different from the HC rats ($t = 1.62$). Thus while the present results clearly indicate that gross hippocampal damage produces higher passive avoidance error rates, and that damage to the lateral fimbria also results in more passive avoidance errors, it is not clear how dorsal fornix damage affects passive avoidance.

The finding of higher error rates for the HE group is compatible with the many previous reports of passive avoidance deficits for hippocampal damaged animals. Also, the finding of reliably more errors for the LF group, agrees with the report (Kimura, 1958), of passive avoidance deficits for rats with lesions restricted to the posterior portions of the hippocampus. Finally, the lack of reliable differences for the present DF group is congruent with the failure of Kimura (1958) and Kaada et al. (1962) to find passive avoidance decrements after damage to the anterior and dorsal portions of the hippocampus.
Table 5. — Group means and standard deviations (SD) for errors in passive avoidance conditioning.

<table>
<thead>
<tr>
<th>Group</th>
<th>HC</th>
<th>DF</th>
<th>LF</th>
<th>HE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>22.64</td>
<td>32.37</td>
<td>34.44</td>
<td>53.93</td>
</tr>
<tr>
<td>SD</td>
<td>10.44</td>
<td>20.14</td>
<td>14.67</td>
<td>19.54</td>
</tr>
</tbody>
</table>
However, it might again be noted that the mean difference of DF and HC groups was very similar to that of LF and HC groups. It is also of interest that, if the dorsal fornix has no function related to passive avoidance performance, then the present LF and HE groups might be expected to have similar passive avoidance scores. Since this was not the case, with the error scores of the HE group reliably greater than those of the LF subjects, it appears reasonable to regard the dorsal fornix lesions as a possible factor in passive avoidance, although the scores of the DF group were not reliably different from those of the HC group.
Reflexive fighting during footshock has been demonstrated in many animal species (Azrin, Hutchinson, and Hake, 1963; Azrin, Hutchinson, and Sallery, 1964). Blanchard and Blanchard (1968) and Blanchard, et al (1970) have reported that after hippocampal lesions, rats display reduced levels of reflexive fighting to footshock, and maintain the boxing posture characteristic of reflexive fighting during the intertrial interval for shorter periods of time when the footshock is terminated.

Since the Blanchard, et al. lesions tended to spare the most rostral and medial portions of the hippocampus, there remains the possibility that the lateral fimbria lesioned group in the present study would show a similar decrement in reflexive fighting, but that no effect would be seen after dorsal fornix section. In order to test this idea, the subjects were tested by pairs in the reflexive fighting task of Blanchard, et al. (1970).

**Apparatus** The apparatus consisted of a 25 cm long x 25 cm wide by 30 cm high plexiglass box, with black sides and top and a clear transparent front. Scrambled shock was deliverable as above through a grid floor.

**Method** Subjects were run by pairs, with each subject paired with another in its own group, approximately 18 days after passive avoidance conditioning. One of the subjects in each pair was daubed with a vegetable dye to aid the experimenter in identification. The pair of
subjects was placed in the plexiglass box, and after a one minute habituation period, a series of 20 shocks of 0.5 second duration at 2.3 ma were delivered. The inter-shock interval was 15 seconds. During each of the shocks the experimenter rated the response of each subject as (1) fighting, lunging, striking, or biting; (2) standing, and facing; or (3) no response other than running, jumping, or vocalization. After the series of 20 shocks had been completed, additional shocks were administered at 5 second intervals until the subjects faced each other and both had assumed the boxing posture (cf. Blanchard, et al., 1970). Shocks were then terminated and the duration of maintenance of this posture was timed to a maximum of 10 minutes. Termination of the boxing posture was defined as one of the animals in a pair moving such that one of its front legs touched the grid floor.
RESULTS AND DISCUSSION

Since the scores of the two rats in each pair were not independent, it was necessary to analyze reflexive fighting scores by pairs. Mean number of pair fighting responses out of a possible 40 are given in Table 6. The LF group had the lowest level of fighting, and fought significantly less than the HC ($t = 3.37, df = 13, p < .01$) or the DF ($t = 3.26, df = 14, p < .01$) groups. The HE group occupied an intermediate position between the HC and LF groups, and also fought significantly less than the HC ($t = 2.05, df = 12, p < .05$ one-tailed test) and the DF ($t = 1.92, df = 13, p < .05$, one-tailed test) groups. The results of the HE group replicate those of Blanchard and Blanchard (1968) and Blanchard, et al., (1970). Those of the LF group provide a new finding, that lateral fimbria destruction reliably decreases the incidence of reflexive fighting in response to foot-shock.

The data for duration of boxing posture maintenance at the end of the test session was also summarized by pairs in Table 7. The same pattern of differences was found. The LF group maintained the boxing posture for the shortest length of time, and was significantly different from the HC ($t = 2.23, df = 13, p < .05$) or the DF ($t = 2.58, df = 14, p < .05$) groups. The HE group also differed in the expected direction, with shorter average durations than the HC and DF groups, but these differences were not reliable.

In an earlier study, Blanchard, et al. (1970) found a reliable decrease in end of session boxing stance duration for the subjects with gross hippocampal lesions. The present finding of decreased fighting
Table 6. -- Group means and standard deviations (SD) for number of trials scored as fighting in reflexive fighting task. (score is total for pair, 40 possible).

<table>
<thead>
<tr>
<th>Group:</th>
<th>HC</th>
<th>DF</th>
<th>LF</th>
<th>HE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>25.14</td>
<td>24.87</td>
<td>11.87</td>
<td>17.29</td>
</tr>
<tr>
<td>SD</td>
<td>2.54</td>
<td>5.00</td>
<td>10.09</td>
<td>9.84</td>
</tr>
<tr>
<td>N =</td>
<td>7</td>
<td>8</td>
<td>8</td>
<td>7</td>
</tr>
</tbody>
</table>

Table 7. -- Group means and standard deviations (SD) for duration (minutes) of boxing posture maintainence after reflexive fighting.

<table>
<thead>
<tr>
<th>Group:</th>
<th>HC</th>
<th>DF</th>
<th>LF</th>
<th>HE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>4.10</td>
<td>4.11</td>
<td>1.10</td>
<td>2.45</td>
</tr>
<tr>
<td>SD</td>
<td>3.56</td>
<td>3.04</td>
<td>1.26</td>
<td>3.77</td>
</tr>
</tbody>
</table>
and boxing stance duration for LF subjects suggests that the mechanism involved is possibly the same as that for the Blanchard, et al. experimental subjects with gross lesions, and for the HE subjects in the present study. The direction of the behavioral changes for the fighting and for the boxing posture duration results coincide in both studies, and are statistically reliable except for the HE posture duration in the present study.

It is of particular interest that the LF scores showed a greater lesion effect than did the HE scores. This would seem to be an unlikely event in that the HE group suffered damage to all areas of the hippocampus, while in the LF group the damage is restricted to the fimbria, composed primarily of the efferent fibers from one portion of the hippocampus.

A possible explanation is that the effect is highly specific to areas CA 3 and 4 and that the gross hippocampal lesion diluted the effect of the CA 3 - 4 damage by adding effects from other areas of the hippocampus (CA 1 or dentate gyrus) which are antagonistic in some way to the reflexive fighting decrement. Another possibility is that the effect results from CA 1 functioning which is normally counteracted by CA 3 - 4 functioning. Then an LF lesion would produce the largest effect, removing the normal CA 3 - 4 counterbalancing function, while a larger, more total hippocampal lesion would destroy parts of CA 1 and 2, producing decrements in the effect. But this is a highly speculative interpretation, in view of the fact that the LF - HE reflexive fighting differences failed to reach an acceptable level of statistical significance.
Also noteworthy is the reliable difference between LF and DF fighting levels and also between their boxing posture maintenance durations. The almost identical HC (25.1) and DF (24.8) reflexive fighting means stand in contrast to the LF mean (11.8). Likewise, the HC (4.10) and DF (4.11) boxing posture duration means contrast with the LF mean (1.10). This may result from a zero or negligible effect of the DF manipulation on reflexive fighting scores. There is no data with respect to anterior dorsal hippocampal lesions and reflexive fighting, which might validate the idea that the effect on reflexive fighting of dorsal fornix section is similar to the effect of lesions affecting the area from which these fibers derive. However, the present data suggest that lesions restricted to the anterior-dorsal portions of the hippocampus (CA 1) would have no effect on reflexive fighting scores, and that such effects may be due entirely to cessation or alteration of normal CA 2, 3, and 4 functioning.
Blanchard and Fial (1968) have reported changes in the rats' sensitivity to footshock after hippocampal damage. By administering a series of shocks of various intensities, and rating the behavioral responses of the subjects, they discovered that hippocampectomized rats display lower jump thresholds than cortically lesioned controls. The subjects also displayed a higher vocalization threshold to shock than the control subjects. Since the passive avoidance and reflexive fighting tasks used in the present investigation involved responses to footshock, it was important to determine whether the experimental differences observed in passive avoidance conditioning and reflexive fighting might be attributable to changes in the subjects' reactivity to shock. If the modifications in shock thresholds found in hippocampectomized subjects also occurred after fimbrial or dorsal fornix destruction, then this information would be important for the proper interpretation of results in the present study, in addition to being of interest in their own right.

**Apparatus** Subjects were tested in a 23.5 cm wide x 28.5 cm long by 19 cm high Grason-Stadler model E 3125 B operant conditioning chamber, with a grid floor. Scrambled shock was supplied to the grid floor by a Grason-Stadler model E 1064 shock generator. A microphone under the grids connected through an amplifier to earphones allowed the experimenter to monitor sounds in the chamber.
Method  Nine days after reflexive fighting testing, the subjects were tested individually by administration of 10 groups of shocks, consisting of 12 shock intensities per group, for a total of 120 shock presentations. The intensities ranged from 0.05 to 2.0 ma, and the order of presentations was randomized within each group. The inter-shock interval was 10 seconds, and the shock duration was 0.1 second. Reactions to each shock were categorized by a trained observer as "jump," "flinch," "vocalization," or "no response" using the criteria of Harvey and Lints (1965). A second experimenter operated the shock generator, so that the observer did not know the intensity of the shock being administered.
RESULTS AND DISCUSSION

The proportions of jumps, flinches, and vocalizations for the four groups are plotted against shock intensity in Figures 3, 4, and 5. The group means of the computed threshold values (50% point of responding) for the three behavioral categories are also located on each graph. The HE group had a reliably lower jump threshold than the HC group ($t = 2.50, df = 26, p < .01$, one-tailed test). No other threshold value differences of the four groups were statistically reliable.

Thus the shock threshold data replicate the results for the lowered jump threshold in the HE group of the Blanchard and Fial study. This again supports the notion that the poorer passive avoidance condition scores of the HE group could not be attributed to a decreased sensitivity to shock, as these subjects were different from controls only in being more sensitive to shock, according to the jump criterion. Although all lesioned groups tended to vocalize at lower shock intensities than controls, the present findings did not include a reliable vocalization increase for the HE group as was found in the Blanchard and Fial (1968) study.

The lack of reliable LF or DF shock differences, as opposed to the replicated HE jump threshold difference, has two possible interpretations. First, the jump threshold effect may involve both DF and LF functioning such that both areas must be destroyed to observe the effect. This suggestion receives some support from the finding that both LF and DF subjects displayed lower jump thresholds than HC rats,
Figure 3. -- Proportion of jump responses at each stimulus intensity.
Figure 4. -- Proportions of flinch responses at each stimulus intensity.

### Mean Threshold and SD

<table>
<thead>
<tr>
<th></th>
<th>Threshold</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>HC</td>
<td>0.340</td>
<td>0.106</td>
</tr>
<tr>
<td>DF</td>
<td>0.273</td>
<td>0.103</td>
</tr>
<tr>
<td>LF</td>
<td>0.319</td>
<td>0.148</td>
</tr>
<tr>
<td>HE</td>
<td>0.286</td>
<td>0.069</td>
</tr>
</tbody>
</table>

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Stimulus Intensity (milliamperes)
Figure 5. -- Proportions of vocalization responses at each stimulus intensity.
although neither difference was reliable. Second, the output from the hippocampus which is involved in this effect may project over another as yet undiscovered pathway, possibly to the entorhinal area. This choice of arguments may also be applicable to the lack of LF-DF effects obtained in the orienting-shift task, where a similar pattern of results was obtained.
GENERAL DISCUSSION

The results of the present study included replications of previous findings of hippocampal lesion effects on the orienting shift-measure, passive avoidance, reflexive fighting, and sensitivity to shock utilizing the jump criterion. The lack of activity consequences of such lesions replicates some studies (Hostetter and Thomas, 1967), Speigel, et al., 1966) but not others (Douglas and Isaacson, 1964, Kirkby, et al., 1967). It therefore may be concluded that the results from rats with gross hippocampal lesions in the present study are clearly similar to those of previous investigators. In addition, the present results indicated one new finding, that latency to drink in a novel situation is reliably reduced after gross hippocampal lesions.

In attempting to provide a framework for the theoretical interpretation of these data, it is necessary to examine several contemporary theories of hippocampal functioning. One of the earlier theories is that of McCleary (1966). He has suggested that hippocampectomized rats perseverate in responding, when the response has a high probability of occurring in a particular test situation. For a deficit to become apparent, some change in the experimental conditions would have to occur which penalized subjects for continuing to respond (e.g., noxious stimulation, or the withdrawal of reward). The normal subject would stop responding under these conditions, while the hippocampally lesioned subject would be expected to continue responding inappropriately, resulting in a performance deficit. For
instance, the perseveration hypothesis can account for the HE group continuing to drink when a novel stimulus is presented, but fails to predict that these animals will have a shorter latency to start drinking. In reflexive fighting, the theory might either predict that the most probable response, reflexive fighting, would perseverate, or predict that the jumping and running responses to shock would perseverate. A similar problem occurs for the prediction of passive avoidance conditioning performance, indicating that the theory cannot make unequivocal predictions in these situations, but appears to readily make predictions only in those situations in which there is a clearly dominant response tendency.

Douglas (1968) has suggested that the hippocampus is involved in the control of sensory perception through inhibitory and facilitory control of inhibitory feedback loops in sensory systems. Two effects were postulated, (1) a non-specific gating mechanism which would act to exclude irrelevant stimuli during the process of concentration of attention (Pavlovian external inhibition), and (2) a specific gating which would act to inhibit reception of specific stimuli which have been associated with non-reinforcement (Pavlovian internal inhibition). The non-specific gating would function to protect memory traces from interference during consolidation, while the specific gating would function in the processes of habituation, extinction, passive avoidance conditioning, etc. Animals with hippocampal lesions would be expected to be deficient in these functions, being less able to focus their attention on relevant stimuli, and also would continue to respond to non-reinforced stimuli.
Either internal or external inhibition functions of the hippocampus appear to suggest that subjects with gross hippocampal lesions would have longer latencies to drink in a novel situation, rather than the reliably shorter latencies to drink obtained in the present study. In addition, an attention deficit would tend to make the gross hippocampally lesioned rat more distractable, rather than less distractable, as was found in the orienting-shift study. The Douglas theory does account for passive avoidance conditioning results, and since subjects with increased distractability would be expected to hold the boxing posture for less time, the theory is also able to account for the reflexive fighting results.

Internal inhibition has also figured prominently in a discussion of the hippocampal deficit by Kimble (1968). He has proposed that the major function of the hippocampus is to inhibit the activating systems of the lower brain upon repeated presentations of the same stimulus. This would permit the organism to shift its attention from that stimulus to another. Kimble suggested that "The organism with hippocampal damage would then be unable to inhibit or alter its responses (or attention) to initially prepotent environmental stimuli and would consequently display less flexible and less adaptive behavior" (p. 286).

Although this idea accounts for the HE group orienting-shift deficit observed in the present study, it does not explain the shorter latency to drink for the HE group. The present passive avoidance results would be obtained if the HE group continued to run and jump in the situation due to an impaired build up of internal inhibition.
The theory makes no prediction for the reflexive fighting situation, since it would seem that no "initially prepotent" environmental stimuli can be accounted for in this situation. Although the mechanisms are slightly different, the Kimble theory is similar to the response perseveration notion of McCleary (1966), and both ideas fail to make a prediction in a number of situations because of a difficulty in specifying either the prepotent stimulus (Kimble) or the prepotent response (McCleary) involved.

Blanchard and Fial (1968) and Blanchard et al. (1970) have proposed that the effects of hippocampal lesions on behavior may be due to a disruption of anxiety reactions and the defensive immobility reactions which they are postulated to produce. They have suggested that the hippocampus is involved in the regulation of species-specific defensive immobility reactions, and that the performance of hippocampectomized animals may be facilitated or inhibited depending on the effects of decreased active immobility reactions on performance in a particular behavioral task. The effects of gross lesions in the orienting shift task would be predicted on the basis that the attention shift involves an arrest of ongoing behavior, and that this immobility reaction is defective in hippocampal animals. Stimuli in a new situation are probably threatening to a rat, leading to a period of exploration. This effect would be reduced or absent in the hippocampectomized rat, producing shorter HE group drinking latencies. The passive avoidance deficit would appear because the HE rat is deficient in the immobility responses seen when a control rat attains the safe area, thus the HE rat would move off the safe area and score more
errors. The lack of defensive immobility postures (e.g., boxing) which have been shown to precede reflexive fighting (Blanchard et al. 1970) would also account for the poorer HE fighting scores observed in the present study.

None of the ideas suggested above predict an increased jump threshold to footshock in hippocampectomized rats. However, the anxiety-defensive immobility hypothesis seems to account most satisfactorily for the other results observed with gross hippocampal lesions in the present study, in that other theories either make the wrong prediction in some instances or fail to make any prediction at all.

We can now consider the behavioral effects obtained in the present study after more restricted lesions of the hippocampal efferent tracts. Like the HE group previously discussed, the DF group in the present study displayed a reliably decreased latency to drink, and a tendency to make more errors than controls in passive avoidance conditioning, although this difference was not statistically reliable. The DF group was not reliably different from controls in any other measure.

The LF group was similar to the HE group, and showed a reliably decreased latency to drink. It also made more errors than controls in passive avoidance conditioning, fought reliably less in the reflexive fighting task, and maintained the boxing posture for a reliably shorter time than the control lesioned animals.

In view of this pattern of results, the question arises as to whether unitary hypotheses such as those discussed above can account for the pattern of DF - LF differences. For example, both the DF
and the LF groups differ reliably from controls in latency to drink, but only one of these groups, the LF group, differs reliably from controls in reflexive fighting. It might be noted that the reflexive fighting scores for the DF group were extremely similar to those of controls, which would suggest that the lack of reliability of these differences reflects a genuine lack of effect of the dorsal fornix lesions on this measure. If the hippocampus is homogenous in its functioning and influences on behavior, as most theories of hippocampal dysfunction imply, then there appears to be no way to explain how disruption of this function in two different parts of the hippocampal output can have the same effects (for DF and LF group) in one behavioral situation (latency to drink), and no effect (DF group) and a very large, reliable effect (LF group) in another behavioral situation (reflexive fighting). Thus the data from the present series of experiments seem to preclude a comprehensive unitary interpretation of hippocampal function, and suggest that a partial localization will be called for in any multi-functional interpretation.

However, the specific content of such a "multi-functional" hippocampal theory depends on what interpretation of the DF passive avoidance results is accepted. An interpretation based on response perseveration would account for the passive avoidance deficit, but would be inadequate to handle the drinking latency results. Any such interpretation would also have to account for the lack of DF-HC differences in reflexive fighting. This pattern of results suggests a fear or anxiety mechanism, but one which is not associated with defensive immobility reactions. If we may assume that an anxiety
mechanism is mediated via the dorsal fornix, then the DF animals would be expected to habituate more quickly to novel stimuli and to drink more readily in a novel situation as a result of decreased anxiety. In the passive avoidance task, this decreased anxiety would make the DF subjects less concerned about the shock once in the safe area, and thus repeatedly step off onto the shocked grid. These animals would have intact defensive immobility reactions, however, and would not be expected to show a reflexive fighting deficit. It would also be assumed that a combined anxiety-defensive immobility function is mediated via the lateral fimbrial pathway. This would account for the decreased LF latency to drink in the same way as for the DF group. In passive avoidance conditioning, decreased anxiety and immobility reactions to shock would account for poorer performance. The decreased immobility reactions of the LF group would also impair its performance in the reflexive fighting measure.

Anxiety effects from the DF and LF groups would be expected to combine for the gross hippocampal lesioned group, thus the HE group would be expected to have shorter latencies to drink and higher passive avoidance errors than the LF or DF groups. However, these effects would not combine for the reflexive fighting situation, which would not be affected by decreased anxiety but by a decrease in immobility reactions (cf. Blanchard et al. 1970). Thus the HE group would be expected to be as different from control as was the LF group in reflexive fighting scores. In fact, the LF group in the present study was even poorer than the HE group, but this HE - LF difference was not reliable.
If the poorer passive avoidance conditioning performance of the DF group relative to the controls is not considered replicable, then the only DF lesion effect in the present tasks was a decreased latency to drink in the orienting-shift situation. The most parsimonious interpretation for this effect would be a DF lesion induced increase in thirst. Under these circumstances, the LF differences from the HC group might be accounted for by the Blanchard, et al. interpretation of an anxiety reduction and decreased immobility responses. These would produce poorer passive avoidance performance, shorter latency to drink, and less reflexive fighting for the LF group. The HE-HC differences would be accounted for since the effects would result from destruction of the corresponding LF mechanisms. However, the significant HE - LF drinking latency differences indicate that there must be an additional latency effect. This would be provided by the LF anxiety effect and the DF thirst effect adding together to produce the large HE difference.
SUMMARY

This study has addressed itself to the question of differentiation of hippocampal function by examining the behavioral effects of hippocampal, dorsal fornix and lateral fimbria lesions. The patterns of obtained results in several behavioral testing situations changed markedly as a function of the location of the experimentally induced lesions. Rats with gross hippocampal lesions generally showed the same pattern of behavioral effects as found in previous studies, including decreased levels of performance in orienting-shift, passive avoidance and reflexive fighting situations. They also had decreased thresholds for jumping to footshock, and no reliable changes in open field activity levels. It was also discovered that water deprived hippocampectomized rats show decreased latencies to drink in a novel situation. Various unitary theories proposed to account for hippocampal lesion effects were discussed, and it was concluded that the best predictions of obtained results were based on suggestions that the hippocampus is concerned with anxiety-defensive immobility mechanisms. Lesions of the dorsal fornix produced reliable decreases in latency to drink and a poorer but statistically unreliable performance in passive avoidance conditioning. Lesions of the lateral fimbria resulted in decreased latency to drink, poorer passive avoidance conditioning, and a large decrease in levels of reflexive fighting relative to operated control rats. The different behavioral results for the dorsal fornix and lateral fimbria lesioned groups indicate that these anatomical differences in hippocampal projections are
accompanied by a separation of function. These differences were dis-
cussed and two possible theoretical interpretations were offered to
account for the differences observed.
Figure A. — Typical dorsal fornix lesion.
Figure B. -- Typical lateral fimbria lesion.
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