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Effects of Lesions in the Hippocampal Rudiment on Somesthetic and Visual Discrimination in the Albino Rat

Thomas W. Planek
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EFFECTS OF LESIONS IN THE HIPPOCAMPAL RUDIMENT
ON SOMESTHETIC AND VISUAL DISCRIMINATION
IN THE ALBINO RAT

Thomas W. Planeck

A Dissertation Submitted to the Faculty of the Graduate
School of Loyola University in Partial Fulfillment of
the Requirements for the Degree of
Doctor of Philosophy

June
1965
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I wish to take this opportunity to thank Dr. M. B. Arnold for her advice and consideration during the period of my research. My thanks also to Rev. V. V. Herr and S. E. Dollard for their help during my graduate school career.

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Finally, I wish to express my gratitude to my wife, Belle, for her patience and reinforcement during my five years in graduate work.
Life

Thomas W. Planek was born November 1, 1936 in Chicago, Illinois. He attended Quigley Preparatory Seminary and graduated in 1955. After attending St. Mary of the Lake Seminary for one year, he entered Loyola University in 1957 as a junior majoring in psychology and received the Bachelor of Arts degree in 1959. He entered the Graduate School of Loyola University in the same year under a National Defense Education Act grant and completed his coursework in 1962. In 1963, he served as acting director of a State of Illinois Grant in the Behavior Laboratory, Loyola University. Since then, he has been assistant manager of the Research Department of the National Safety Council.
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CHAPTER I.
INTRODUCTION

Memory is basic to the learning and retention of all problems that are presented to both humans and animals. It has been the focus of investigation for both psychologists and neurophysiologists. Psychologists have been concerned with the memory process as it relates to problems of learning, recall, reproduction and relearning; while neurophysiologists have been considering the storage of memory within the brain and its neural transmission. Though progress in each of these disciplines has been made, the intermeshing of the psychological and the neurophysiological components of memory had, until recently, seldom been accomplished in a testable theory.

Arnold's approach (1960) has integrated existing research in the areas of psychology, neurology and physiology to formulate a theory of brain function. Within this theory, she proposes a definitive memory circuit.

As a psychologist, her starting point is behavioral. However, she includes under the term "behavior" both behavior observable by others and experience which is not directly observable by others. This phenomenological approach combines an analysis of "what goes on behaviorally" with a description of "what goes on neurophysiologically to produce specific behavior."

In suggesting well defined brain circuits for the mediation of behavior, Arnold has made possible empirical verification of her phenomeno-
logical theory. She has substituted testable explanations of psychological behavior for untestable descriptions.

It is the purpose of this dissertation to test one of the hypotheses that has been generated by Arnold's theory. Although her conclusions are based on sound research, a number of the hypotheses involving physiological mediation of behavior have yet to be tested. A major aspect of physiological mediation of behavior is her proposed memory circuit. Though psychologically, memory operates in such a way that recall from all appropriate sense modalities occurs in a well-coordinated manner, the physiological mediation of recall has yet to be empirically verified.

The technique to be used in this study is the placement of a specific lesion designed to interrupt recall in a specific modality. The emphasis is on the placement of the lesion in definite structures rather than on the amount of brain tissue damaged.

For example, much of the research literature on brain lesions has involved destruction of primary and association cortices. Confusion in interpretation of the results of such studies has been the rule rather than the exception. Arnold's method differs from the approach represented in the brain lesion experiments that have hypothesized general impairment of function stemming from molar ablation of cortex.

In Arnold's integrative approach, a definite brain circuit is described to be interrupted by lesions at various points. If they result in a deficit and support the hypothesis, that aspect of the theory becomes the stepping stone to guide further investigation. If the results prove
to be negative, further analysis of the problem and of the neurophysiology of the brain is necessary.

The validity of the theory as a whole, therefore, is not demonstrated by one experiment, neither is it invalidated by a single study. Rather, the sum total of evidence must be gathered before a final judgment about it can be made.

This study will be the third in a series designed to test one phase of Arnold's hypothesis, namely, that the hippocampus and the hippocampal rudiment serve memory recall in the various modalities. Specifically, this study will aim to determine whether or not the hippocampal rudiment is needed for somesthetic recall in a tactual discrimination problem.

Because this study arises simultaneously from both physiology and psychology and is, therefore, a shared problem, it requires techniques, skills, and prior knowledge from both disciplines. Since it rarely happens that one investigator has fully mastered all the techniques of two disciplines, and this is the case in the present study, competent help from the neighboring discipline of physiology was enlisted at successive stages of preparation of brain slides, and evaluation of histological results. Discussion of the specific applications of method, both psychological and physiological, will be deferred until such time as it becomes directly relevant to the study at hand.
CHAPTER II.

HIPPOCAMPAL STRUCTURES AND THEIR FUNCTIONS

Interest in the hippocampus has increased with the years and today holds the attention of many investigators. Its significance lies in its anatomical position, surrounding the thalamus, and its numerous connections in cortical and subcortical structures. A description of the phylogenetic development of hippocampal structures will aid the reader in appreciating its role in the architecture of the brain.

Phylogenetic development. Embryologically, the hippocampus is derived from the medial wall of the cerebral hemisphere or hippocampal primordium. Together with the pyriform cortex, formed from the lateral wall of the hemisphere, it serves as the cerebrum of primitive vertebrates. As the dorsal cortex develops into neocortex and grows longitudinally as well as transversely, the posterior parts of the hemisphere are pushed downward. Consequently, the originally straight primordium (see Fig. 1A) is bent down, curving around until its posterior end points anteroventrally in the temporal region of the hemisphere (see Fig. 1B). At the same time, the transverse growth of the neocortex forces the primordium of the pyriform cortex down laterally until it reaches the ventral position in the rat (see Fig. 1C) and finally the anterior ventromedial location in man. (Zeman & Innes, 1963)

As the neocortices envelop the brain, the corpus callosum is developing between them within the lamina terminalis. (Peele, 1961) The corpus callosum influences hippocampal positioning as it grows at an angle through
Fig. 1. (A) Diagram of medial surface of cerebral hemisphere of the frog with primordium hippocampi shaded. (Redrawn from Zeman and Innes.) (B) Diagram of medial surface of cerebral hemisphere of marsupial (opposum). (Redrawn from Zeman and Innes) (C) Diagram of medial surface of cerebral hemisphere of the rat. (Redrawn from Zeman and Innes.)
the hippocampal primordium and the fornix. The corpus enlarges so that only a hippocampal arch remains above it on its anterior and dorsal aspects. This arch elongates to form the indusium griseum or hippocampal rudiment as the major portion of the hippocampus is pushed into the temporal lobe. Due to the corpus callosum's development, fibers to and from the hippocampus are found above and below the corpus callosum and also incorporated within it. (Green, 1960)

**Neural connections.** Hippocampal structures usually are said to include the hippocampus proper with the gyrus dentatus (Ammon's horn) and its embryological rudiment (indusium griseum); also the surrounding cortical areas (hippocampal, retrosplenial and cingulate gyrus), and amygdaloïd complex, and subcallosal septal and frontotemporal juxtallocortex.

Kolliker (1896) considered these structures olfactory in nature and so called the whole complex "rhinencephalon." Though many investigators have pointed out since then that these structures have no connection with the olfactory bulb or tubercles and have no olfactory function, the name is still being used for the above structures.

In an effort to clarify the function of "rhinencephalic" structures, Pribram and Kruger (1954) divided them into three basic systems, according to their anatomical connections (Table 1).

### History of Hippocampal Function

Throughout the phylogenetic scale, the hippocampus migrates extensively within the brain, but its original connections with the brain stem remain. This fact led Herrick (1933) to conclude that the hippocampal
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primordium serves to correlate afferent impulses from a variety of brain stem visceral centers and to relay them secondarily to the neocortex. Herrick's view was the first to emphasize the type of function in which the hippocampus is currently thought to participate, viz. the correlation of cortical and subcortical functioning.

Herrick's description of hippocampal function was not widely accepted since the popular view then was that the hippocampus plays a major role in olfaction. This position was proven untenable by the work of Swann (1934, 1935) and Allen (1940, 1941). These investigators showed that animals with the hippocampus removed could both relearn and retain an olfactory discrimination. Brodal (1947) summarized these findings and demonstrated that the hippocampus and its connections could not be considered a "nose brain".

**Hippocampus and emotion.** In 1937, Papez formulated the first theory in which the hippocampus plays a pivotal role in emotion. On the basis of the work of Cannon (1927), Bard (1934), Penfield (1933), Ranson (1934) and Herrick (1933), Papez described a cortical circuit that could mediate emotion: according to his theory, emotion can be aroused by afferent stimuli coursing through the subthalamus and mammillary body to the anterior thalamic nucleus and the cingulate gyrus (which is the "receptive region" for emotion). Emotion can also be aroused by cortical processes from the frontal lobes or by excitation of the hippocampus. The connection with the gyrus cinguli is established via the medial forebrain bundle (from the frontal lobe) or the fornix (from the hippocampus). Thus Papez considered the hippocampus as one area which could relay excitation to the gyrus.
cinguli and so initiate emotion.

Later work seemed to confirm that the hippocampus was active in emotion. Kluver and Bucy (1938) removed the greater part of both temporal lobes of macaque monkeys and found that these monkeys did not seem to recognize their surroundings ("psychic blindness"). They were hyperactive and mouthed everything ("oral tendencies") rather than using their hands; they showed no aggressiveness but indiscriminate and greatly increased sexual activity. Of all these changes, only the lack of aggressiveness and—perhaps—the increased sexual activity could be said to indicate an emotional change.

Spiegel, Miller and Oppenheimer (1940) found that bilateral rhinencephalic lesions in cats and dogs produced sham rage rather than the "tameness" reported by Kluver and Bucy. Bard and Mountcastle (1948) reinvestigated the experimental production of sham rage and found that bilateral hippocampal lesions resulted in sham rage, provided that the amygdala with pyriform cortex was also removed bilaterally. Placidity or "tameness" resulted from decortication, but only when the whole hippocampus rhinencephalon and at least part of the cingulate gyrus remained undamaged.

Others investigating rhinencephalic lesions have found respectively hypersexuality in cats (Green, Clemente and DeGroot, 1957; Schreiner and Kling, 1953), fearlessness in monkeys (Thomson and Walker, 1951) and a variety of affective behavior in man (Bard, 1928; Gray, 1942; Torzian, 1958). These data and others (Cobb, 1943; Erickson, 1945; Forbes, Cobb and

1. According to Kluver and Bucy, unilateral or partial bilateral temporal lobectomy did not produce the same change.
Cattell, 1923; Crosby and Humphrey, 1941; Dusser de Barenne and McCulloch, 1938, Fulton, Pribram and Stevenson and Wall, 1949) offer evidence that an injury to the rhinencephalon modifies certain basic patterns of behavior common to several species of animal from the rodent through carnivores up to and including primates (Schreiner and Kling, 1953). This conclusion, however, is so vague that the role of the hippocampus in emotion remains unclear.

In an attempt to develop Papez' theory in greater detail, MacLean (1949) suggested that the rhinencephalon is a "visceral brain" which maintains the connection with lower autonomic centers and dominates vegetative life, in contrast to the neocortex which is the center of "intellectual" functions.

In an attempt to deemphasize the olfactory connotation of the term "rhinencephalon," now known to be inappropriate, MacLean chose the term "limbic lobe." He proposed a division of the limbic lobe into an area concerned with "self-preservation," and another concerned with the "preservation of the species." The former includes the frontotemporal portion of the limbic lobe and is involved in obtaining and assimilating food; while the latter includes the hippocampus, cingulate gyrus and parts of the septum, and mediates the experience and expression of emotion.

The formulations of Papez and MacLean were indeed noteworthy. Papez postulated a definite area in the brain which mediated emotion. MacLean showed the limbic lobe including hippocampal system and cingulate gyrus to be the correlation center for impulses from the neocortex. Both theorists have contributed to the increase of interdisciplinary work among neorol-
ogists and psychologists. However, their work has not suggested how sensory impulses are integrated and transformed into emotion; neither do they explain the role of the limbic system in this integration. (Arnold, 1960)

Hippocampus and memory. Hippocampal structures have also been thought to have a memory function. Theories ascribing a memory function to the hippocampus have been neither as complete in scope nor as detailed in presentation as the theories of Papez and MacLean. They have been based on memory deficits or amnesia in human beings following injury to the hippocampus and hippocampal connections. Examples of more recent theories of this type are found in the writings of Nielsen (1958) and Milner (1954).

Nielsen (1958) maintains that a hippocampal circuit is active when the "memory of one's individual life experiences" is operating. He distinguishes memories of "life experiences" from "retentive memory of acquired knowledge," which he says requires the proper cortical association areas and a diencephalic component. Nielsen states that memories of experiences are stored in cortical association areas but also sequentially in the hippocampi; so that individual recall will be structured temporally in the sense that one remembers one event as preceding another in time. Bilateral and, at times, unilateral hippocampal damage can produce total or partial amnesia that disrupts recall from the present backward in time.

Milner (1954), in discussing memory, distinguishes between short-term storage and long-term storage. The hippocampus is necessary to retain experiences for a period of time until a cortical neural change occurs that will permanently store a memory. The process in which the hippocampus acts is referred to as "consolidation." Through the consolidation process, the
trace of an experience is retained by the individual even though the experience is no longer the object of attention. Thus, long-term memories have established a cortical linkage which has become autonomous of the hippocampus. Short-term memory depends on the hippocampus for consolidation. Hippocampal ablation, therefore, leaves long-term memories intact but gravely inhibits the building of new associations, even though attention to stimuli is not affected. Milner's findings that bilateral hippocampal lesions interrupt short-term memory in man have been supported by the work of Penfield and Milner (1958) Glees and Griffith (1952), Scoville (1954) and Walker (1957).

Pribram (1961), in reviewing these findings, suggests that the inability to execute complex sequences of action is the common denominator explaining the inability of patients to carry out tasks demanding short-term memory. He further points out that when the plan of events is written out on a piece of paper, hippocampectomized patients are able to overcome their short-term memory impairment. He proposes that the hippocampus is part of the individuals planning mechanism.

Recently a theory of hippocampal function has been presented by McLardy (1959) based on the morphological characteristics of the mammalian brain. He assigns to some hippocampal neurons the work of detecting and coding intensity gradients and to others the work of detecting and coding complex temporal patterns within the organism. In the detector-coder process, innate reaction patterns are released, dependent upon the neural set stimulated. The notions McLardy presents have yet to be fully developed by him and await verification.
In all these theories of assigning a memory function to the hippocampus, it was this structure alone that was considered. The function of the hippocampal rudiment (indusium griseum) has been neither investigated by researchers nor explained by theorists.

In summary, the hippocampus has been seen as active in emotion by Papez and MacLean, in memory by Nielsen, and by Milner and Penfield, in planning behavior by Pribram and in the general correlation of neural impulses by MacLean, Herrick, McLardy, Green and Arduini (1953) and Kaada (1951).

Arnold's theory of hippocampal function. Arnold has worked out a theory of brain function based on a review of the available evidence and a phenomenological analysis of human experience. Briefly, when something is experienced, it is also appraised; but before the appraisal can lead to action, relevant past experiences must be recalled together with the action taken; next, the result of this action has to be appraised, and action appropriate to the present situation must be planned (imagined) and its consequences appraised.

According to Arnold, the function of appraisal is mediated by some rhinencephalic structures, the function of memory recall by others. Consequently, she divides the so-called rhinencephalon into two systems: the hippocampal rudiment and fornix; the latter comprises the subcallosal, cingulate, retrosplenial and hippocampal gyri and the island of Reil (Arnold, 1960). The hippocampal system mediates the initiation of memory recall, and the initiation of action; the limbic system, the appraisal of objects and actions, and the registration of affective memory.
According to Arnold's theory, each sense impression, each action or action impulse is registered as a trace or disposition in a cortical association area. Just as each sense has a primary sensory area, so each sense has an area for the registration of these sense impressions. Once an impression is so registered, later contact with the object will reactivate the registered pattern via sensory projections to the association cortex as well as the primary sensory cortex: the object will be seen and recognized. But objects or situations can also be recalled: in this case, an appraisal that this would be "good to know" initiates a nerve impulse relayed from the limbic cortex to the hippocampus and from there via fornix and midbrain to the sensory thalamic nucleo and the cortical sensory areas. Thus, the hippocampal system serves as a switchboard connecting with the association areas and so reactivating the original impression in the proper pattern and temporal sequence.

In this way, Arnold's theory accounts for the temporal sequence of "life's memories" without having to postulate that these are stored in the hippocampus, as Nielsen (1958) does. This theory also accounts for the loss of "recent memory" after damage to both hippocampi without having to postulate that the hippocampus is necessary for memory "consolidation," as Milner (1954) does: with such lesions, the recall of visual or auditory experiences would no longer be possible; long-term memories, on the other hand, have been recounted repeatedly, thus registered also as motor memories, and are unaffected because motor memories are mediated by the hippocampal rudiment.

Arnold's theory can also account for the deficit in planning, noted by
Pribram (1961). In the lesions reported by Milner and others, the amygdaloid complex was destroyed bilaterally, together with part of the hippocampi. According to Arnold, the amygdaloid complex is the starting point of an "imagination circuit" which makes it possible to imagine and plan action. When the plan to be followed is written out, patients with such lesions have no difficulty.

This theory postulates that memory is not a unitary function, but can be analyzed into various memory modalities, each with their separate cortical representation in the association areas nearest the primary sensory areas. According to Arnold, the hippocampal circuit serves recall of sense impressions from many modalities at once. Upon perception, an object is appraised as "good to know" (via the neighboring limbic cortex); next it is identified by recalled similar situations via the hippocampal memory circuit. This implies that impulses from various association areas are relayed to neighboring limbic areas and from there to the nearest point in the hippocampal circuit.

In the modalities of vision and audition, the nearest limbic region is the hippocampal gyrus which connects with the hippocampus; in olfactory, motor, taste and somesthetic modalities, the nearest limbic regions are the subcallosal and cingulate gyri, which connect with the hippocampal rudiment. Since the hippocampal rudiment, as well as the hippocampus, have independent connections with the fornix, transection of the rudiment,

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2. We are using the term "hippocampal rudiment" in preference to the more common term "indusium griseum" to remind the reader that this structure has a similar embryological origin as the hippocampus itself.
depending on the location of the lesion, should impair olfactory, motor, somesthetic or taste memory, but should not affect visual or auditory recall. In contrast, transection of the hippocampus, depending on its location, should affect either auditory or visual and auditory recall but should not impair recall in olfactory, motor, taste and somesthetic modalities. Thus, Allen's (1940, 1941) dogs could relearn and retain an olfactory discrimination after removal of the better part of both hippocampi.
CHAPTER III.
REVIEW OF RELATED LITERATURE

The research that is most relevant to this experiment involves the hippocampus and its various connecting structures as they relate to memory, and the effect of brain lesions on somesthetic discriminations. The following discussion will attempt to describe particular investigations, report results, and when appropriate, provide a possible interpretation in terms of the theory that is being tested in this experiment.

As is apparent from the review of theories of hippocampal function, the role of this structure is not clear. Arnold's integrative approach is plausible because it provides a consistent interpretation of different and, at times, seemingly contradictory results.

**Memory Deficits with Hippocampal Lesions**

Moore (1962) compared the effects of cingulate lesions in cats with the effects of control lesions, i.e., septal lesions, and septal-hippocampal lesions. The control lesions permitted perfect retention of an auditory conditioned avoidance response (CAR). Septal lesions impaired retention in nine out of eleven subjects, three of which were not retrainable. Septal-hippocampal lesions impaired seven out of seven animals, two of which could not relearn. Lesions in the cingulate cortex produced retention deficits in five out of six animals, all of whom relearned.

In the septal group, the three animals that failed both to retain or relearn the conditioned avoidance response received bilateral lesions that
maximally (90 per cent to 100 per cent) destroyed the fornix. In terms of Arnold's theory, bilateral destruction of the fornix would eliminate learning and memory in all modalities since the fornix is the chief efferent system of the hippocampus and hippocampal rudiment.

The septal-hippocampal lesions produced an inability to retain and relearn the conditioned avoidance response to an auditory stimulus in two animals. One of these animals had maximal fornix damage; while the other had moderate to heavy (50 per cent to 90 per cent) bilateral fornix damage plus maximal bilateral damage to the stria terminalis. The stria terminalis constitute a primary efferent of the amygdaloid complex which, for Arnold, mediates imagination. Though the amygdala itself was undamaged, motor innervation resulting from the impulse to imagine (Arnold, 1960) was definitely interrupted and the animal was unable to imagine what to do in response to the auditory stimulus.

In Moore's cingulate animals, which showed less impairment than the other subjects, and which were able to relearn in fewer trials than they needed before operation, the lesions sometimes involved the anterior limbic region, sometimes the retrosplenial area, and sometimes both regions. The defect in animals with damage primarily to the anterior cingulate region could be explained as inability to appraise appropriate action, which is mediated by this limbic region. Since, in the cingulate lesions, the damage was subtotal, and it is likely that rudiment damage was, therefore, also subtotal, the relearning that occurred would be explained in Arnold's formulation as owing to remaining tissue which could mediate it.
Isaacson, Douglas and Moore (1961) demonstrated that partial hippocampal ablation reduced both latency and trials to criterion in rats' learning a CAR to a buzzer. Since the lesions were small and both dorsal and rostral sections of the hippocampus remained, the auditory memory circuit can be assumed to be functioning. The reduction in latency and trials to criterion may be explained in terms of the elimination of interfering memories.

Recent studies have been done with rats on the effects of hippocampal ablation on behavior by Niki (1962) and Kimble (1963). Niki found after bilateral ablation of the hippocampus (mostly in the dorsal hippocampal area), little effect on the learning or retention of an avoidance response to an auditory conditioned stimulus. Deficits were found in maze performance when visual discriminative stimuli were used. Since much of the hippocampus was intact, and its connection with the fornix was undamaged, auditory and visual recall was still possible. The visual deficit can be explained in terms of partial interruption of fibers from the visual association areas via the hippocampal gyrus to the dorsal hippocampus.

Kimble produced in his subjects complete bilateral lesion in the hippocampus with heavy destruction of the fimbria. His results showed the hippocampal animals to be deficient in their ability to reach criterion in a Y maze on a successive brightness discrimination although they performed as well as controls on a simultaneous discrimination. Further, the hippocampal animals traced the same path over and over again in an open field maze and made a greater number of errors in learning problems in the Hebb-Williams maze one and maze six.
In analyzing these results, we find that deficits occurred on those problems that involved motor as well as visual memory. In the successive discrimination, both arms of the Y maze were of the same brightness, either both white or both black. Some animals were trained to go right when the paths were white and left when the paths were black. To control for right or left preferences, other animals were trained to go right when the paths were black, and left when the paths were white. This problem involves remembering that white means going right (or vice versa). According to Arnold's theory, impulses from the visual association area would have to be relayed via the hippocampal gyrus to the hippocampus and from there via the fornix and midbrain back to the visual association area and also to the motor (prefrontal) association area. In other words, there would have to be a double associative connection. In the simultaneous problem, the animals were trained on a Y maze with one arm white and one arm black. White meant the correct turn for one group of animals, black was correct for another group. In this discrimination, only visual memory is involved. It is conceivable that the lesion was far enough ventral to allow the relay of impulses serving visual recall but not the double relay necessary for visual and motor recall.

**Somesthetic Discrimination Studies**

Research in this field has focused on the primary somatosensory area and the somatosensory association area, i.e., the posterior parietal and occipital areas adjoining the primary somatosensory area. Rose and Mountcastle (1959) comment that "systematic analysis has not proceeded very
far perhaps because of the confusion which was created by the finding that simple somesthetic discriminations are still possible or can be relearned after removal of the first somatic field." Perhaps the confusion would clear up if it were realized that somatosensory experience is possible as long as the thalamic somatosensory nuclei are intact; and that discrimination, as indicated by the animal's response, always implies recalling which stimulus indicates food or water, i.e., it implies memory. According to Arnold's theory, somesthetic impressions are registered in the parieto-occipital association cortex and recalled via the hippocampal circuit. As long as these structures are intact, somesthetic discrimination can be relearned even after removal of the primary somatosensory cortex (post-central gyrus), though there will be a retention deficit because the sensory experience (now mediated only by the thalamus) will be different.

Several studies support this interpretation. Smith (1939) found that rats lesioned in the postcentral region relearned a roughness discrimination in approximately the same number of trials it took them to learn it. Since the experience of roughness was now mainly mediated by the thalamus, the retention deficit is the result of having to adjust to the different somesthetic experience. The rats could relearn because the somatosensory association cortex and the hippocampal system were intact.

Zubek (1951) made a more extensive ablation of the primary somesthetic area in rats. Again, these lesions produced a retention deficit, though all animals relearned. Also, lesions in somatosensory area II (an area bordering on the posterior insula) produced an impairment in relearning a
roughness discrimination. The posterior insula, according to Arnold, is
the limbic area receiving relays from the lateral surface representing face
and forelegs. Thus, sensations from face and forelegs could no longer be
adequately appraised as pleasant or unpleasant so that the response was
inappropriate.

Allen (1947) ablated both the somatosensory areas I and II and their
surrounding cortex in dogs. After these lesions, the animals failed to
relearn a negative conditioned response using a tactile stimulus. They had
been trained to lift their foreleg when stroked lightly on the back, once
a second, and to withhold this response when stroked three times per second.
After the lesion, the dogs lifted the foreleg every time their back was
stroked. Since somatic area I was destroyed, the dogs still felt the strok-
ing, but diffusely, because the sensation was now mediated by the thalamus
alone. They still feel the touch, but could no longer localize it, thus
could no longer distinguish one stroke from three. When trained in a
similar manner using an auditory stimulus, the same animals quickly relearned
the conditioned response.

That the parieto-preoccipital area is concerned with somesthetic dis-
crimination has been confirmed by a number of studies. Ruch, Fulton and
German (1938) reported that in both man and chimpanzee, damage to the
posterior parietal lobe (sparing the primary sensory area in the postcentral
gyrus) reduces the ability to discriminate weight and roughness. Blum,
Chow and Pribram (1950) showed that parieto-temporo-occipital lesions in
the rhesus monkey resulted in deficits in both visual and somesthetic dis-
crimination. Since the visual deficit was permanent while the somesthetic
deficit was not, these workers concluded that these two functions could be dissociated. In a series of studies (Chow, 1952; Mishkin, 1954; Mishkin and Pribram, 1955; Riopelle and Ades, 1951; Riopelle and Ades, 1953; and Pribram and Barry, 1955) it was found that inferotemporal ablations affect visual discrimination, while parieto-preoccipital resections affect somesthetic discriminations.

In the Pribram and Barry study, two rhesus monkeys had inferotemporal ablations and three rhesus monkeys had large lesions of the parieto-preoccipital area. These parieto-preoccipital lesions extended from the lunate sulcus posteriorly to the intraparietal sulcus anteriorly. Laterally, their extent was variable; medially, the entire precuneus and the posterior portion of the cingulate gyrus was destroyed.

All animals were given pre-operative training in three discriminations in a modified Wisconsin general testing apparatus. The visual discrimination consisted of the simultaneous presentation of two square masonite plaques: one with a plus sign painted on it, the other with a circle. The animal learned to choose one or the other to obtain a food reward. In the tactual discrimination problem, the visual stimuli figures of the plus sign and circle were cut from masonite and glued to plaques. During tactual training, the animals were prevented from visually observing the stimuli. The weight discrimination consisted of two different weights attached to the masonite plaques. Post-operatively, the animals were trained on a length discrimination using a three inch strip of .05 dowelling as the positive stimulus and a two inch strip as the negative cue.
The parieto-preoccipital animals postoperatively showed impaired somesthetic discriminations and were unable to learn the length discrimination; they showed no impairment on the visual tasks. The animals with inferotemporal damage failed the visual task, but showed no somesthetic decrement. The authors suggest that problem complexity and intricacy of procedure may account for the failure of the parieto-preoccipital animals to learn the length discrimination. This conclusion is questionable on the grounds that the inferotemporal animals learned the length problem in fewer trials than either of the tactual or weight discriminations. A more probable explanation for the failure to learn the length problem could be that length discrimination involves not only tactual memory but motor memory. To estimate length, it is necessary to move the hand along the strip. This implies recall of the extent of movement in addition to recalling the feel of the strip. Apparently, a recall in two modalities ("active touch") requires more intact association cortex than does simple tactual recall. Since the representation of hand and arm is on the lateral side, close to the lateral fissure, there was apparently sufficient association-cortex left to make the simple tactual discrimination possible.

Wilson's study (1957) of the effect of lesions similar to those of Pribram and Barry (1954) offers further support for the conclusions that the parieto-preoccipital area is crucial for normal somesthetic discrimination, while the inferotemporal area is necessary for normal visual discrimination; and that these two memory modalities can be dissociated. Although their results show some correlation between deficit manifested and the complexities of the tests as defined by the number of trials taken to
learn pre-operatively, the exact role of test complexity needs clarification. In Arnold's formulation, test complexity depends on the complexity of function, e.g., a problem whose solution depends on the coordination of memories and action impulses from many sense modalities.

In Wilson's study, the parietal animals that showed greatest deficit on the formal somesthetic tests also had the most difficulty orienting themselves and other objects accurately in space. This observation has been corroborated by Pribram and Barry (1956), Glees and Cole (1953) and Ettlinger and Wegener (1958). This impairment apparently was not due to an incapacity for receiving tactile stimuli since the parietal animals responded to objects placed in the hand as consistently as inferotemporal or normal animals. Rather, the author felt that an impairment of discriminative functions was generated by the lesions. Arnold's explanation would be that an absence of somesthetic memories makes it difficult if not impossible to position limbs and body correctly. If we cannot remember what it feels like to make a movement, there will be no somesthetic cues to give direction to the next movement.

Further support for Wilson's observation about disorientation in parietal animals comes from Bates and Ettlinger (1960). They found that animals with parietal lesions (between the intraparietal and lunate sulci on the lateral surface, extending inferiorly to the upper part of the superior temporal gyrus and medially, to include the whole precuneal gyrus) showed an abnormality of: posture, spontaneous movement (reaching and jumping), visual placing and the fright reaction. Although reaching for these animals was most inaccurate, the movement of the hand to the mouth
with a food object was quick and faultless in all parietal animals from the beginning. This seeming paradox in reaching behavior is explainable in Arnold's terms by the fact that hand to mouth reaching had been practiced so often by the animals that it had become a motor memory, and therefore, automatic. It is through motor memory, which goes into action as soon as the cue is given, that habitual or complicated skills are faultlessly reproduced.

The animals with parietal damage reported in this study showed a great deal of impairment when they had to reach for either of two cue boxes set eight inches apart and distinguish them by touch, both in the dark and when it was light. The same animals showed little impairment when the cue boxes were set side by side. In interpreting these data, the authors suggest a selective motor retardation or poverty of movement, which was more pronounced in darkness. More simply, Arnold would account for this behavior by the difficulty of knowing what limb to move or how far to move it when the animal has somesthetic sensations from the limbs but no somesthetic memories to direct the movement. Vision would help, but cannot make up for the somesthetic memory deficit.

The results of a later study by Ettlinger and Kalsbeck (1962) confirm the fact that parietally damaged animals have more difficulty with tactual problems that involve reaching. Also, it is interesting to note that no parietally damaged animal showed defects on the testing of the visual fields, eye or head movement, or the reaction to sounds. This would argue against a simple motor retardation and not be at variance with Arnold's interpretation.
The above reports have suggested that damage of the somesthetic association area produces a greater deficit in learning tactual discriminations than does damage of the primary somesthetic area. According to Arnold, this is so because the association area is necessary for the registration of tactual and other somesthetic impressions. She further maintains that the association area has a localized representation of somesthetic memories from various parts of the body quite similar to the representation in the motor and primary somatic area. These somesthetic memories are revived (recalled) via the hippocampal circuit.

Up to now, two studies have been completed that were designed to test this theory. Fagot (1962) did the first study, investigating the role of the hippocampal rudiment in learning and retention of an olfactory discrimination. After having the hippocampal rudiment transected bilaterally at the genu of the corpus callosum, rats could no longer discriminate between the odor or extract of pine (which led to water reinforcement) and oil of hyacinth (which did not lead to water). These animals were unable to relearn the discrimination in many more trials than were necessary for normal animals. Incomplete transection of the rudiment produced a learning or retention deficit though relearning was possible. Lesions in neighboring structures produced no deficit.

Gavin (1963) found that albino rats with a bilateral transection of the hippocampal rudiment posterior to the motor area were unable to learn or retain a motor discrimination problem. The problem involved alternation in a T maze for a water reinforcement. In this case, the animal had to remember which way it had turned on the preceding trial in order to make
the correct response. These two studies indicate that the hippocampal rudiment does play a role in recall of olfactory and motor cues.

The logical follow-up to this research is to interrupt the rudiment at a point posterior to the lesions of Fagot and Gavin, to discover whether this will result in impairment of a somesthetic discrimination.
The purpose of this study is to determine the role of the hippocampal rudiment in the recall of somesthetic (tactual) memories. In Arnold's theory, recall of somesthetic memories is mediated by connections from the primary somatosensory areas and limbic sensory areas (posterior cingulate gyrus) to the hippocampal rudiment. From the rudiment, fibers run via the fornix to the midbrain reticular formation (Nauta, 1956) then to the sensory thalamic nuclei and back to the sensory, association and limbic cortices (French, Verzeano and Magoun, 1953). A bilateral lesion of the rudiment at a point caudal to the primary and limbic somatosensory cortex should interrupt these connections and thereby prevent recall of somesthetic memories. The proper locale for the placement of the lesion in the rat brain (Krieg, 1955) seems to be at the splenium of the corpus callosum, immediately before the fibers turn underneath the corpus in their course to the fornix.

In adapting the method about to be discussed for the study of a tactual discrimination, an attempt was made to provide as unambiguous a tactual deficit, where one exists, as possible. To obviate difficulties in interpretation of results, four of the five following recommendations from Wilson (1957) guided the design of this study:

1. It must be shown that the deficit reflects a loss in ability to use somesthetic cues and does not merely reflect a difficulty in orientation in space or in manipulation of the stimulus objects.
2. The effects of brain lesions upon retention as opposed to the effects on initial learning must be established to determine whether or not any performance decrements that may occur can be attributed solely to amnesia for specific somesthetic habits. (Though the mechanism of recall in learning and retention is identical in Arnold's theory, this guide was followed.)

3. "Double dissociation of function" (Teuber, 1955) must be shown, both to demonstrate that a given lesion affects somesthesia alone and to show that the tests used are valid indicators. Thus, the effects of a given lesion upon at least two tests specific to different modalities must be studied, and the effects of at least two lesions upon the same test must be studied.

4. In order to insure an adequate sampling of behavior within a modality, several tests that are presumed to measure the same function should be given. In this way, factors of order, difficulty, and interval between operation and test can be evaluated and some indication of consistency of effect obtained.

Recommendation number four was not followed in this study since factors of order, difficulty and time should have little or no effect on the interpretation of results. If the lesion in question is effective, neither learning nor retention should take place. It is left to succeeding experiments to attempt to demonstrate the consistency of any positive results that evolve from this study, not simply on one other tactual discrimination problem, but on many more.
5. Histological verification of lesions should be available in order to specify the relation between the locus and extent of lesion and a given performance as exactly as possible.

**Method**

**Subjects**

The subjects were divided at random into two groups, **learning** and **retention**. The learning group was operated on before training, while the retention group received training before the operation and was retested after the operation. The animals in both groups were taught somesthetic and visual discriminations simultaneously. The training for the somesthetic discrimination occurred on an elevated Y maze, while the visual discrimination was learned in a bar-pressing apparatus. All the animals were allowed a seven day recovery period after the operation. The control group of unoperated animals was given an eight day rest period to coincide with the recovery period of the operates.

The final separation of the animals into groups depended on the histological results. An attempt was made to aim for bilateral rudiment lesions at the level of the splenium of the corpus callosum for all animals in the operated groups. Those animals which were found to have either incomplete transections of the rudiment or lesions in other structures were used as controls.

**Apparatus**

**Tactual Discrimination:** A Y-shaped, elevated-path apparatus similar to that used by Smith (1939) was designed for this experiment. The starting
platform, 12 inches in length, led to a forked path, the arms of which presented the surfaces to be discriminated. (see Fig. 2) The correct and incorrect runways were constructed as separate units that could be assembled on a table to form the complete apparatus. The first 18 inches of each path ran horizontally and led to a 14 inch long incline of 45 degrees. At the end of each incline was a platform 10 inches in length, on which the animal received reinforcement. The correct pathway was covered with a smooth rubber finish, while the incorrect path was covered with corrugated rubber. The coverings were removable from the main structure, and were alternated randomly. The floor-boards of both runways, as well as the supports on which they were laid, were tapered for a distance of four and one-half inches. In assembling the apparatus, both runways were placed on a table with the tapered portions brought together and placed in contact with the starting platform.

Visual Discrimination: The test chamber and other components used in the visual bar press discrimination was manufactured by Foringer and Company (Catalog #1102TC, 1133EMI, and 1102H). A speaker located between the house lights provided white noise from a generator manufactured by Grason Stadler (#455B).

The discriminative stimulus consisted of a small flashing light directly above the bar. The onset of the flashing light was controlled electrically by general purpose timers and in combination with a sequence alternator panel presented the "continuous light, flashing light" phases for random durations of time. The minimum duration for each phase was 17 seconds.
During the training-light phase, the green trigger of the apparatus
was a shape filled with sand with clear manhole cover. During the
continuous light phase, the green trigger shone through the grid flow
of the slot's chamber. The feed was set at 20 minutes or approximately 30
minutes during the study. The standard Paradigm stock food was
and used. After

Fig. 2. Tactual discrimination apparatus
During the flashing light phase, a bar press triggered the appearance of a dipper filled with one-tenth cubic centimeter of water; during the continuous light phase, a bar press triggered shock through the grid floor of the test chamber. The shock was set in intensity at approximately .32 milliamperes during the study. The standard Foringer shock generator #1154 and scrambler #1155 were used.

Training

Deprivation: The procedure for all groups was the same. Two days before training, the subjects were placed on a water deprivation schedule during which they were given three ounces of water per day immediately after running. The animals had ad lib access to food. This schedule was maintained throughout the experiment.

Tactual discrimination: On the third day of deprivation, the subjects were allowed to explore the maze and drink from the dipper on the goal platform. For the next week, the animals were trained to run up the single, smooth incline to obtain water. During the early part of training, the $\alpha$ of the incline was set at 45 degrees and the animal was trained for one more day. The corrugated path was then introduced to form the Y and testing began. The smooth and corrugated paths were alternated randomly.

During the entire training and testing periods, the subjects were run in a darkened room. A small six watt light painted blue was lit, so that the experimenter could observe the animals. (An experiment described in the Experimental Controls section was conducted to assess the likelihood of the subjects responding to visual rather than tactual cues in the semi-darkness.) All animals were run 20 trials per day until they reached the
criterion set at 90 per cent accuracy for 54 or more correct responses in 60 trials. If an animal reached criterion before the 20 trials for the day were completed, running was stopped on a trial divisible by five.

**Visual discrimination:** The subjects were shaped to the bar press under light-on conditions. Once the animal learned to press the bar to obtain water, it was exposed to the "continuous light, flashing light" discrimination for 15 minutes daily, until criterion was reached. When the light was flashing, water reinforcement was available; when it was on continuously, reinforcement was unavailable and a shock of a .32 milliamps intensity was administered when the bar was depressed.

As in the tactual problem, all animals were run until they reached a criterion of 90 per cent correct responses for a period of three successive days, i.e., nine out of ten or better correct bar press responses for three 15 minute sessions.

**Experimental Controls**

**Tactual discrimination:** To demonstrate that visual cues were not affecting the response behavior of the subjects, a group of laboratory animals, not part of the experiment, were brought to criterion in the tactual maze. The corrugated path or incorrect path, was then covered with a transparent piece of heavy plastic and the animals were rerun in the maze. If visual cues were operating in the darkened experimental room, the animal's level of correct responses should not be significantly reduced. The results of the test are shown in Table 2. All of the animals were given more trials in the relearning phase with the plastic cover than they had taken to reach criterion without the cover. None of the animals reached criterion in the
Table 2

Number of Trials for Laboratory Control Animals to Reach 90 Per Cent Criterion Performance on Roughness Discrimination

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Total Trials</th>
<th>No. of Correct Responses During Last 60 Trials</th>
<th>Achieved Criterion</th>
<th>Total Trials</th>
<th>No. of Correct Responses During Last 60 Trials</th>
<th>Achieved Criterion</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC-1</td>
<td>80</td>
<td>56</td>
<td>Yes</td>
<td>180</td>
<td>39</td>
<td>No</td>
</tr>
<tr>
<td>TC-2</td>
<td>100</td>
<td>54</td>
<td>Yes</td>
<td>160</td>
<td>35</td>
<td>No</td>
</tr>
<tr>
<td>TC-3</td>
<td>100</td>
<td>54</td>
<td>Yes</td>
<td>140</td>
<td>32</td>
<td>No</td>
</tr>
<tr>
<td>TC-4</td>
<td>120</td>
<td>57</td>
<td>Yes</td>
<td>160</td>
<td>31</td>
<td>No</td>
</tr>
<tr>
<td>TC-5</td>
<td>80</td>
<td>58</td>
<td>Yes</td>
<td>140</td>
<td>29</td>
<td>No</td>
</tr>
<tr>
<td>Total</td>
<td>480</td>
<td>279</td>
<td></td>
<td>780</td>
<td>166</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>96</td>
<td>56</td>
<td></td>
<td>156</td>
<td>33</td>
<td></td>
</tr>
<tr>
<td>S.D.</td>
<td>9.66</td>
<td></td>
<td></td>
<td>9.66</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
relearning phase. A "t" test for differences between correlated means was computed at this point, and showed a difference between mean trials well beyond the .01 level of confidence.

Though the above results indicate that visual cues did not seem to be affecting learning behavior in the experimental situation, precautions had to be taken to control for other sensory cues. Auditory cues were minimized by covering the paths of the maze with rubber rather than with sandpaper as Douglas had done. Also, by running the animals in an open Y maze, the possibility of producing footfall echoes and other floor cues experienced in the closed maze (Sheppard, 1959) was reduced. Both olfactory and kinesthetic cues resulting from possible minute variation in height or angle of the horizontal discrimination pathways were greatly reduced since the cue stimulus paths were removable and alternated from side to side unsystematically. Alternation of cue stimulus was guided by Gellerman's tables (1933).

**Visual Discrimination**: In the visual apparatus, the house light and stimulus light were on at all times. The use of a flashing light discrimination in the test chamber rather than a "light on-light off" discrimination eliminated the possibility of producing a painful, or at least unpleasant, sensory stimulation caused by sudden pupillary contraction at the onset of a light in the dark chamber which might serve as additional somesthetic cue. The electrically controlled apparatus produced some noise which, however, was masked by additional white noise in the test box. Also, since four boxes were in operation at the same time, the sounds produced were sufficiently inconsistent to rule out this extraneous stimulation as
a secondary cue.

**Operations:** The lesions were produced using a clean operating technique. The animal was anesthetized with a mixture of ether and air. The average time for the anesthetic to take effect was 15 minutes.

To begin surgery, the scalp on the dorsal surface of the skull was shaved and incised at the midline. The skull was then cleared of galea and periostium, to expose the bregma. The Krieg Atlas (1946) for the rat brain was used to locate the points at which the trephine holes were drilled. For this lesion, the holes were drilled at 54.5 millimeters according to Krieg, or 2.5 millimeters posterior to the bregma, so that a rectangular opening approximately four millimeters by one-half millimeter was made laterally across the midline. A bipolar electrode was inserted into this opening at depths of 3.8 mm on the midline and 3.0 mm immediately to the left and right of the midline. The Krieg stereotaxic instrument (Model #51200, Stoelting) was used to secure the animal and fix the point of the lesion and electrode depth. A lesion maker (Model #LM3) manufactured by Grass Instruments provided the current through the bipolar electrode. The poles were approximately one-third millimeter in diameter and separated by approximately two millimeters.

After the lesion was produced, the wound was closed with bone wax and the scalp was sutured. The animal was returned to his cage. During recovery, it was given ad lib access to food and water.

**Histology**

In preparation for histology, the animal was perfused with formalin solution by use of a 30 gauge needle and syringe. The brain was then
excised in toto and placed in buffered (saturated CaCl$_2$) formalin solution for three days. The solution was replaced; the brain was grossly trimmed and returned to the solution. All specimens were refrigerated during the time following perfusion.

Before cutting, the specimen was trimmed again, washed and dehydrated in ascending concentrations of ethyl alcohol. It was then transferred to three changes of xylene and embedded in paraffin.

The embedded tissue was cut at 10 microns with a rotary microtome. Each section was stained with hematoxin and eosin for general morphologic study. Luxol fast blue and Cresyl Violet stain was used on some cuts for combination nerve fibers and cells.
CHAPTER V.

RESULTS

Histology

The extent of damage to critical brain areas will be discussed in terms of the drawings shown in Figures 3, 4, 5, and 6. Only structures relevant to this study are shown and labeled in these figures. The abbreviations used to label the structures are listed alphabetically in Appendix I. The marginal millimetric calibrations given in Figure 3 are to be used to set the level of the brain slices presented in Figures 4 through 15.

Complete Rudiment Transections

The results of the histological study indicate that six animals received lesions interrupting the hippocampal rudiment bilaterally at the level of the splenium of the corpus callosum. Except for one subject, the corpus callosum was also transected by the lesion. In all of the bilateral animals, some cortical, cingulate and hippocampal damage occurred. The damage to these structures was not heavy except for subject HR-4.

The lesion to animal HR-1 (See Fig. 7) extends from approximately 4000 μ well beyond 3000 μ. Fig. 7-A reveals bilateral damage to the rudiment with unilateral callosal interruption and some depression of the hippocampus. The extent of the lesion increases as it proceeds posteriorly to completely sever the corpus callosum. Fig. 7-B shows damage to the hippocampus and cingulum, including bilateral damage of surrounding structures down to and including the superior fornix.

Animal HR-2 (see Fig. 8) received a bilateral lesion of the hippo-
Fig. 3. Outline of a sagittal cut of the rat brain approximately 580 microns (μ) from the midline. (Redrawn from Konig and Klippel, 1963)
Fig. 4. Outline of a coronal cut of the rat brain anterior to level at which the hippocampal rudiment begins to perforate the splenium on route to the fornix. (Redrawn from König and Klippel, 1963)
Fig. 5. Coronal outline of the rat brain from a level within the critical area at which the rudiment, still supracallosal, begins in the next 500 microns to take a downward course through the splenium. (Redrawn from Konig and Klippel, 1963)
Fig. 6. Coronal outline at which the hippocampal rudiment is no longer represented supracallosally. (Redrawn from König and Klippel, 1963)
Fig. 7. Animal HR-1. A bilateral hippocampal rudiment transection.
Fig. 8. Animal HR-2. A bilateral hippocampal rudiment transection.
campal rudiment which extends from approximately 2400 \( \mu \) and includes the splenium of the corpus callosum to approximately 3200 \( \mu \). Considerable unilateral cortical, cingulate, and hippocampal damage is present in this animal and the superior fornix is also injured.

Figure 9 shows a bilateral rudiment transection in animal HR-3. The extent of rudiment damage is from approximately 3200 \( \mu \) to 2600 \( \mu \). The photographs, from two levels, show damage which includes fornix fibers and is mainly unilateral in extent. Much of the damage shown to structures below the fornix level is due to artifacts, not the lesions.

Animal HR-4 received extensive bilateral damage of all structures down to the brain stem. (see Fig. 10) The obliteration of structures extends from approximately 1600 \( \mu \) beyond the 3000 \( \mu \) level and is the most extensive lesion in any animal.

Figure 11 depicts an abscess that functionally interrupts rudiment fibers bilaterally in animal HR-5. It depresses the hippocampus and extends in the same degree anteriorly and posteriorly from the site of the cut shown in Fig. 11, approximately 1000 \( \mu \).

The lesion to animal HR-6 interrupts the rudiment unilaterally at the 4200 \( \mu \) level, as well as the hippocampus and the structures directly above and below it. (see Fig. 12) It extends further posteriorly until it begins to bilaterally damage the rudiment at approximately the level of 3200 \( \mu \).

**Partial Rudiment Transections**

Five animals received lesions on the midline that failed to interrupt the hippocampal rudiment bilaterally at the level of the splenium. Three
Fig. 9. Animal HR-3. A bilateral hippocampal rudiment transection.
Fig. 10. Animal HR-4. A bilateral hippocampal rudiment transection.
Fig. 11. Animal HR-5. A bilateral hippocampal rudiment transection.
Fig. 12. Animal HR-6. A bilateral hippocampal rudiment transection.
Fig. 13. Animal HA-3. An example of a bilateral rudiment lesion that fails to extend to the splenium of the corpus callosum.
Fig. 14. Animal RP-1. An example of a midline lesion posterior to where the rudiment turns downward into the splenium of the corpus.
Fig. 15. Animal N-1. An example of slight cortical brain damage.
of these animals, RA-2, RA-3, and RA-4, received bilateral lesions that interrupted the rudiment completely at a point anterior to the splenium; another, RP-1, was lesioned at a point posterior to it. The fifth animal, RU-5, received a unilateral lesion of the rudiment at the level of the splenium. Figures 12 and 13 depict animals from this group. In Fig. 13-A, animal RA-3 shows a definite bilateral rudiment interruption at the truncus of the corpus callosum anterior to the splenium; however, Fig. 13-B shows clearly the reappearance of rudiment fibers at a point further posterior, toward the splenium.

Fig. 14 shows a possible bilateral lesion to animal RP-1 slightly posterior to where the hippocampal rudiment turns downward to perforate the splenium of the corpus callosum.

Cortical Damage

Five animals, N-1 through N-5, received lesions that did slight damage to cortical fibers, but failed to injure the indusium at any level. Fig. 15 shows this type of damage for animal N-1. The rudiment and surrounding structures are intact.

Discrimination Learning and Retention

Somesthetic Discrimination

The results of the somesthetic tactual discrimination are presented in Table 3. There is no learning or retention deficit in the tactual discrimination in any of the animals regardless of the type of lesion; "t" tests computed between the lesion groups revealed no significant differences in mean learning or retention scores. The mean preoperative learning score and its standard deviation for the tactual discrimination is based on the
Table 3

Number of Trials to Reach 90 Per Cent Criterion Performance on Tactual Discrimination for Lesioned and Intact Animals.

<table>
<thead>
<tr>
<th>Type of Lesion</th>
<th>Animal No.</th>
<th>Preoperative Learning Retention</th>
<th>Postoperative Learning Retention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete</td>
<td>HR-1</td>
<td>--</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>HR-2</td>
<td>--</td>
<td>90</td>
</tr>
<tr>
<td>Rudiment</td>
<td>HR-3</td>
<td>--</td>
<td>75</td>
</tr>
<tr>
<td></td>
<td>HR-4</td>
<td>--</td>
<td>80</td>
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<tr>
<td></td>
<td>HR-5</td>
<td>80</td>
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<tr>
<td></td>
<td>HR-6</td>
<td>80</td>
<td>--</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td></td>
<td>345</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td></td>
<td>86</td>
</tr>
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Table 4

Number of Sessions to Reach 90 Per Cent Criterion Performance on Visual Discrimination for Lesioned and Intact Animals.

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</table>
scores of all the animals that were intact at the time of learning. Therefore, scores for each lesion group do not appear separately in the Table.

Visual Discrimination

Table 4 presents the results of the visual discrimination. The wide variation in both pre and postoperative learning scores may be attributed to idiosyncratic responses to shock following a bar-press during the continuous light phase of the discrimination. Some animals froze after being shocked and refused to press the bar for an entire session. Others continued their trial and error behavior in spite of the shock conditions. As can be seen in Table 4, all animals reached criterion on the visual discrimination both before and after lesioning. Again, the preoperative learning mean and standard deviation is presented for all intact animals across groups.

"t" tests computed between group scores on the visual discrimination reveals no difference between lesion groups and the intact group in either learning or retention. However, impairment to specific animals within the lesion groups appears to be present. There is a slight learning deficit in animals HR-1 and HR-4 and a slight retention deficit in animals HR-5 and HR-6, when compared with the scores of the intact animals. In the incomplete rudiment group, animal RA-1 shows some learning deficit; while in the cortical group, animal N-5 shows some retention impairment. Whether these individuals deficits are chance fluctuations or due to the lesions is subject to speculation.
These results do not seem to confirm Arnold's theory regarding tactual memory. And though the visual discrimination deficit could be the result of incidental damage to the hippocampus or the commissure, it is too slight to allow any conclusions.

There are a number of possible explanations for the failure of hippocampal rudiment lesions to produce a tactual learning deficit:

1. The hippocampal rudiment does not function in the manner expressed by Arnold.

2. Efferent fibers from the hippocampal rudiment do not all turn around the splenium to join the fornix but perforate the corpus callosum anterior to it.

3. Connections flow into the hippocampal-fornix circuit via another route besides the rudiment.

The first explanation about rudiment function is questionable, since the data of Fagot and Gavin both support Arnold's theory. Olfactory discriminations were lost after the rudiment was sectioned at the genu of the corpus callosum, while motor discriminations were lost after a rudiment section caudal to the motor area. Presumably, loss of ability, either to learn or retain these discriminations, is indicative of memory loss. The fact that the subjects in both Fagot's and Gavin's studies were not run on more than one sense modality discrimination clouds the issue as to the differential effect of rudiment lesions. However, the failure to have
"double dissociation of function" does not affect the major results, assuming the controls for each study were reliable.

Another possibility in interpreting these results rests in the phylogenetic development of the hippocampal rudiment. There is evidence that rudiment fibers cross through the corpus callosum to enter the septum pellucidum where they join in the distribution of the subcallosal fornix fibers. Other fibers perforate the corpus at the splenium, to join the superior fornix (Olson and Magee, 1961). There are also rudiment fibers which pass around the genu of the corpus callosum to reach the septum and become part of the precommissural fornix (Ariens Kappers, Huber and Crosby, 1936, pp. 1430). However, it is not certain whether these are afferent or efferent rudiment fibers.

If, in fact, Arnold's interpretation is correct, and the rudiment does mediate memory in the manner she suggests, the failure to obtain positive results may be attributed to the existence of a secondary group of fibers that flow into the hippocampal circuit via a structure other than the hippocampal rudiment. An analysis of the tactual discrimination problem used will help to provide the basis for this interpretation.

The tactual discrimination between corrugated and smooth rubber in the two arms of the maze was mediated by the animal's fore and hind legs. The sensory representation of the hind legs (if it is homologous with the representation in higher mammals) would be located on the dorsal surface of the parietal cortex and so could be expected to feed into the posterior cingulate gyrus and the adjoining stretch of the hippocampal rudiment. But head and forelegs may be represented on the lateral surface, as in
higher mammals, and so might have connections with the insula rather than the posterior cingulate gyrus. The insula is connected through the claustrum (perhaps a homologue of the hippocampal rudiment?) with the hippocampus. Since this connection is intact, we could expect that touch impressions from the forelegs, indicating smooth or corrugated floor, could be used for discrimination even when touch memories from the hindlegs can no longer be recalled.

This is admittedly speculative, for the function of the claustrum is unknown. But we do know that the claustrum has monosynaptic connections with the insula and the hippocampus (Rae, 1954) so that it could be part of the postulated memory circuit. The only way to test this notion would be to lesion the posterior insula and claustrum in addition to a bilateral complete lesion of the hippocampal rudiment and repeat this experiment; or to devise a touch discrimination which employs the forelegs only, and compare performance (a.) after lesioning the hippocampal rudiment at the splenium, and (b.) after lesioning the claustrum.

**Summary**

This study investigated one aspect of Arnold's hypothesis that the hippocampal system, including hippocampus and hippocampal rudiment, is necessary for recall of memory in the various sense modalities. Specifically, this experiment was designed to determine the effect of a complete bilateral lesion of the hippocampal rudiment on the learning and retention of somesthetic and visual discriminations. It was hypothesized that such a lesion at the level of the splenium of the corpus callosum would interrupt recall of the somesthetic sensations, but not of the visual sensations.
To test this hypothesis, 21 albino rats were divided randomly into two groups, some for the study of learning, and others for the study of retention. The tactual problem involved a discrimination between corrugated and smooth rubber paths of an elevated Y-shaped maze similar to the type used by Smith (1939). Water was used as the reinforcement. The visual problem involved a discrimination between a continuous light and a flashing light in a bar-pressing apparatus. A bar press during the continuous light phase would result in shock; while a bar press during the flashing light phase produced a water filled dipper.

The animals were further separated into groups on the basis of the following postoperative results:

1. Complete bilateral transection of the hippocampal rudiment at the splenium.
2. Partial transection of the hippocampal rudiment.
3. Cortical damage without rudiment interruption.
4. Unoperated and therefore cortically intact.

The results indicate that the six animals with a complete bilateral rudiment transection were able to learn or relearn both discrimination problems as were all the animals in the other groups.

In discussing these findings, an interpretation was presented based on the representation of the forelegs and hindlegs in the brain and the neural connections that mediate such representation.

It was suggested that recall of foreleg and head sensations may be mediated over a circuit running from the lateral parietal cortex, via the insula and claustrum to the hippocampus, while hindleg and trunk sensations
are mediated via the posterior cingulate gyrus and hippocampal rudiment.
CHAPTER VII.

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CHAPTER VIII.
APPENDIX

INDEX OF ABBREVIATIONS

A - Alveus hippocampi
C - Cingulum
CFD - Commissura fornicis dorsalis (Commissura hippocampi dorsalis)
CP - Commissura posterior
FH - Fimbria hippocampi
FLC - Fissura longitudinalis cerebri
FO - Fornix
FS - Fornix superior
GCC - Genu corpus callosi
GD - Gyrus Dentatus
H - Habenula
HI - Hippocampus
HR - Hippocampal rudiment (Indusium griseum)
PF - Polus frontalis
RP - Recessus pinealis
S - Subiculum
SCC - Sulcus corporis callosi
SH - Sulcus hippocampi
SLL - Stria longitudinalis lateralis (Lancisi)
SLM - Stria longitudinalis medialis (Lancisi)
SM - Stria medullaris thalami
SPCC - Splenium corporis callosi
SR - Sulcus rhinalis
TCC - Truncus corporis callosi
The dissertation submitted by Thomas W. Planek has been read and approved by five members of the Department of Psychology.

The final copies have been examined by the director of the dissertation and the signature which appears below verifies the fact that any necessary changes have been incorporated, and that the dissertation is now given final approval with reference to content, form, and mechanical accuracy.

The dissertation is therefore accepted in partial fulfillment of the requirements for the Degree of Doctor of Philosophy.

May 28, 1968
Date

Magda W. Smith
Signature of Adviser