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## Effects of Lesions in the Hippocampal Rudiment on T-Maze Single Alternation in the Albino Rat

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EFFECTS OF LESIONS IN THE HIPPOCAMPAL RUDIMENT  
ON T-MAZE SINGLE ALTERNATION  
IN THE ALBINO RAT

by

Sister Helen Gavin

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

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## LIFE

Sister Helen Gavin, C.S.J. was born in Chicago, Illinois, on May 30, 1931.

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## Chapter I

### Introduction

Memory needs no justification and little introduction as a topic for psychological research. Probably because memory profoundly influences the quality of action, experimental investigation of this function has attracted research workers ever since psychology became a discipline in its own right. Since every study presents its own specific line of development, however, the background that led both to choice of the rationale and eventually to this particular study of a proposed structural basis for motor memory will be clarified through a brief introduction.

As psychology has come of age, many investigators have become increasingly dissatisfied with an approach to the study of psychological problems that not only starts, but stops with application or alteration of a stimulus, followed by measurement of a response. Investigators of memory, for example, want to know, not only that memory occurs or does not occur (as inferred from response measures), but also (1) how memory operates, including knowledge of how memory meshes with other functions; and (2) which precise structures mediate memory. Study of the process of memory, without reference to neural structures, is primarily, of course, a psychological problem which can be studied in a variety of ways. In contrast, study of which structures mediate memory equally claims the interest, not only of psychology, but of the basic disciplines of neuroanatomy and neurophysiology, as well as the applied area

of psychiatry.

The question of which structures are specifically needed for memory to go on at all, then, provides a meeting ground for a number of disciplines. Since study of proposed neural structures could lead to a more thorough knowledge of psychology, it is well worth study. The fact that such knowledge will at the same time contribute to more than one discipline makes study of a structural basis for psychological functions such as memory all the more inviting. The problem, then, of studying one proposed neural link for mediating memory will be the focus of the present study. How this particular study came to be chosen, however, still remains to be shown.

As a starting point in the search for structures that mediate a function such as memory, either of two kinds of approach could be employed. The first approach, very commonly used in neurophysiological research, starts with assessment of results of previous investigations and then uses the previously gathered results as a basis for further study. Following this line of approach, one might also choose to investigate a structure, to see what particular kind of behavior it serves, irrespective of what others have found. An error in lesion placement may also lead to suggesting structures to damage or stimulate in further studies. This first approach, then, leads to damaging or stimulating a structure (usually on one of the bases cited) and then to interpreting the findings psychologically. This approach claims certain strengths and advantages as well as disadvantages.

One point in favor of this approach is that it has certainly provided a vast and serviceable heritage of information for students of brain function, whatever their theoretical perspectives might be. Through this approach

domains of the brain, having relevance for a variety of further studies, have been mapped. But on what basis does one choose to stimulate or destroy this rather than some other structure?

A disadvantage of this approach is that the clues it offers as to which functions a structure might serve are radically limited by previous experimental and clinical investigations of brain structures. Since, unlike a more directly physiological function such as breathing, a psychological function like memory offers no overt clues as to which structures might mediate it, this first approach may be potentially wasteful of research efforts. It also appears as if an approach of this kind is destined to end with isolated findings, since it precludes proposal of ways for linking structures with one another in terms of related functions, at least until such a time as much more information will be available.

A still more serious objection accrues from starting by damaging a structure in order to see whether a given function will be impaired as a result. This objection is that the damaged structure may serve not only or even mainly the function that is found defective. For example, the many studies done during the past decade on the reticular system in mediating sleeping and waking provide no basis for indicating other functions this system may serve.

Clearly, a more desirable approach would incorporate information gained through use of the first approach, and could also propose clear-cut suggestions as to what kinds of structures to look for in the brain.

An alternative to starting with damage to a structure, founded first on neurophysiological research and the results of previous experiments, would be

to begin instead with an analysis of the psychological processes that lead from perception to action, and then to assess neurophysiological research studies. With a prior psychological analysis, one is in a better position than before to know what structures could mediate the function in question, and related functions as well.

Exemplifying this second approach, a 1960 formulation of M. B. Arnold, which is relevant to the structural basis for memory, invites study. On the basis of a psychological analysis, followed by assessment of neurophysiological research studies, this formulation proposes definite structures for mediating memory. A psychological analysis can distinguish different functions (though these are not rigidly separated) in the sequence of activities leading from perception to action, to which each function may contribute.

Arnold's approach, on which this study will be based, starts like any other psychological formulation, with what is observed--seen, said, done. The first step in this psychological analysis is to inquire: what is the common sequence of activities that leads to the observed action? No matter what is eventually done, a person must first perceive something, must appraise it as good, bad, or indifferant, must like or dislike it in some way before he will approach or avoid it. Not what is done (whether it be climbing a wall, learning a maze, writing a composition) is important for this purpose, but that the psychological activities leading to the observed action occur in a particular sequence.

By achieving knowledge of the sequence of activities that leads to action, one is by that fact in a better position than before (1) to understand the way in which various functions contribute to the sequence that leads to action at

succeeding states; and (2) to suggest which kinds of connections appear to be needed in the brain for mediating a particular function or activity. At this point study of the neurophysiological research investigations already available may be profitably assessed, with the aim of pinpointing specific structures that appear likely to mediate a given function, while connecting with related ones.

As a result of applying the procedure of psychological analysis, followed by assessment of neurophysiological research studies. Arnold (1960) concluded that the hippocampal structures (hippocampus and hippocampal rudiment) appear to have the kinds of connections that could serve memory. From the structures proposed by Arnold, and following the rationale of this second approach to neuropsychological functioning, the present study took rise.

Applied to the present study of a structural basis proposed for motor memory, Arnold's formulation commends itself on several counts. First, by its emphasis on study of real processes and their structural bases, it is consistent with a philosophic position of realism. Both in their operation and in their structural basis, real psychological functions, not abstractions to be substituted for them, can be known.

Second, in specifying which structures appear as if they could mediate particular functions, Arnold's formulation may lead to greater economy of research efforts than has been achieved so far.

Third, in specifying definite structures and proposing how they could mesh, this formulation also satisfies the scientific canons of explicitness and clarity. Because it is explicit and clearly drawn, the formulation lies open to experimental test, a means needed for gaining certain knowledge of

structural bases for psychological functions.

For two immediate reasons the function of memory was chosen for study:

(1) because memory profoundly influences the quality of action; and (2) because the clear-cut proposal of structures for serving memory invites experimental test.

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

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 brain surgery, preparation of 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

### Proposed Functions of the Hippocampal Structures

#### Early Background of Hippocampal Functioning

Until very recent times the hippocampus (and its anterior continuation, the hippocampal rudiment) has been more elusive and baffling in its functions and relationships to other structures than the sea horse of classical mythology for which it was named. For nearly three hundred years after Arantius (in 1587) identified and christened the hippocampus proper in the human brain (Papez, 1937), it remained a silent structure. Only in the past quarter of a century have proposed functions for the hippocampus been investigated on a large scale.

In the nineteenth century Ferrier did the first experimental study of the hippocampus reported in the literature (Papez, 1937). After destroying the hippocampus in the monkey, Ferrier reported a depressive effect upon the sense of touch. This early record implicating the hippocampus in cutaneous sensibility is the only known report of its kind.

In contrast to the single conclusion that the hippocampus serves touch, a function very commonly associated with the structure has been olfaction. In the early literature (Broca, 1878) the hippocampus was believed to form part of a widespread system, called "limbic lobe" because it surrounds the hilus of the brain hemispheres. In this system, Broca included the olfactory tubercle



the hippocampus and hippocampal rudiment, the cortex near the olfactory striae and the prepyriform area, the hippocampal, parasplenic, cingulate, and subcallosal gyrus. Other regions in Broca's limbic lobe include the frontotemporal cortex, a band of tissue between the prepyriform area, and the orbitofrontal and anterior temporal neocortex. (Arnold, V. II, p. 31) Broca did not distinguish the hippocampus from the other structures in the limbic lobe in terms of function. Because the hippocampus was included with olfactory structures in the limbic lobe, it is no wonder that the hippocampus came to be associated with the sense of smell. The fact that the structure of the hippocampus is similar to that of parts of the brain that serve the sense of smell also lent support to its being considered as the cortical receiving area for the olfactory sense. The early functional linking of the hippocampus with the olfactory structures has carried over in modern use of the term "rhinencephalon" meaning "nose brain" (Peele, 1961), a term coined by Kölliker and now applied to a number of non-olfactory structures, usually including the hippocampus.

The interpretation that the hippocampus serves olfaction could have been avoided if only a fact of history had been associated with the purported olfactory function. The overlooked fact of history was a result of a comparative study done by Broca (1878), who found that the hippocampus is common to all mammals. Broca remarked particularly on the presence of the hippocampus in the dolphin, an animal that lacks olfactory nerves, bulb, and tract, and hence cannot smell (Smith, 1944). Whether later morphologists reflected upon Broca's observation or were even aware of it cannot be determined on the basis of available fragmentary evidence. Whatever the case

may have been, a growing number of morphologists, including Cajal, Dejerine, Turner, G. E. Smith, and Kölliker came to impute an olfactory function to the hippocampus, a view that was in vogue for many years.

In the meantime, the results of certain investigations of the brain that could have led to further knowledge of functions of the hippocampal structures were passed by. Perhaps because of selective attention paid to the hippocampus as serving olfaction, studies that in a later age would have been seized upon and carefully followed through, were appraised at the time they were reported as simply curious facts.

The first early study of importance to discovering functions of the hippocampal structures was not seen as relevant at the time it was reported. (Brown & Schäfer, 1888) After ablating the temporal lobes in monkeys, these investigators noted "severe intellectual depression," characterized by apparent inability to recognize common objects by sight. The investigators reported that the sensory functions of vision, audition, taste, touch, and smell were evidently intact (p. 318). Understanding of the sensory impressions needed for recognizing objects by sight or sound, however, appeared to be impossible without first tasting, smelling, or touching the object in question. Since many structures besides the hippocampus are involved in temporal ablation, and because an olfactory function was already imputed to the hippocampus, the fact that little attention was accorded Brown and Schäfer's study is not astonishing. Brown and Schäfer may themselves have obscured and retarded recognition of the role of the hippocampus in producing the observed results by attributing the functional loss in their animals to probable vascular changes. Fifty years after publication of this study, when much more was known about brain

functioning than was true at the time of Brown and Schäfer's study, findings similar to theirs (Klüver & Bucy, 1938, 1939) came at a time more propitious, apparently, for further investigation of the possible role of the hippocampus and hippocampal gyrus in such functional anomalies.

The second relevant early finding, a case history of a man who had manifested extraordinary memory loss and apathy, reached scholarly circles in a 1900 report by the Russian neurologist Bechterew (Penfield, 1959). Post-mortem examination of the subject's brain revealed lesions of the mesial surface of both temporal lobes, involving damage to the hippocampus in both hemispheres. In this instance, as in that of Brown and Schäfer, fifty years elapsed before strikingly similar cases reached the clinical literature (Griffith, 1952; Grunthal, 1947). Probably because more information about brain functions became available during this fifty-year period, these later reports led to widespread investigation of the role of the hippocampus in psychological functions.

#### Later Formulations of Hippocampal Functioning

Herrick (1933) was the first to suggest that the hippocampus, elusive sea horse, might serve functions in addition to the sense of smell. Calling the hippocampus, together with other structures of Broca's classic limbic lobe, a "nonspecific activator of all cortical activities" from learning and memory to emotion (Herrick, p. 14, 1933). Herrick presaged all the major theoretical positions and experimental investigations that have involved the hippocampus ever since. Herrick, however, proposed no circuits and did not attempt to explain how the hippocampus mediated "adjustive, intelligent" behavior. How olfactory structures might be related to the hippocampus, which was still

believed to serve the sense of smell, was not delineated in Herrick's brief statement. Yet his insights, though not explicitly correlating structures and functions, were well-timed, penetrating, and prophetic. They inaugurated the beginning of systematic study of the hippocampus and its possible functional kinship with other non-olfactory structures.

Just a few years after Herrick's statement on hippocampal functioning appeared, Papez (1937) suggested that the hippocampus was part of a circuit for mediating emotion. In Papez's formulation, the hippocampus became the discharging structure for the emotions.

At about the same time Papez's formulation appeared, Kluver and Bucy's experimental reports on temporal excision in the monkey began to claim widespread attention (1938, 1939). These investigators reported marked emotional changes after ablating some of the structures (in the temporal lobes) that Papez proposed for serving emotion. On the basis of the proposed circuit of Papez and the experimentation of Kluver and Bucy, other research workers now began to investigate proposed functions of the hippocampus and neighboring structures in the temporal lobe.

In the meantime the view that the hippocampus serves olfaction became passe. This change seems to have been owing at least in part to the research of Allen (1940, 1941) with dogs. On the basis of his studies, Allen concluded that the hippocampus is not needed for the sense of smell. Probably because of suggestions that the hippocampus might serve other functions (made directly by Herrick and Papez, and indirectly by the research of Kluver and Bucy), the majority of research workers in the 1940's abandoned the notion that the hippocampus serves as projection cortex for the sense of smell. Brodal's

careful review of hippocampal studies (1947) consolidated the conclusion that the hippocampus does not serve the sense of smell. In the decade or so that followed Brodal's report, ample further evidence confirmed this view (Green & Arduini, 1954; Kaada, 1951, 1960; MacLean, 1949). But if the hippocampus does not serve olfaction, the functions it does serve were still far from clear. During the past fifteen years, however, much more information regarding the structure has come to light.

A decade after Papez proposed his famous circuit for emotion, including the hippocampus as one of the structures in it, MacLean (1949) published a highly stimulating article bearing relevance to hippocampal functioning. MacLean proposed that the hippocampus serves as a correlation center for every form of internal and external perception, that it gathers impulses from every sensory area. For MacLean, the hippocampus serves emotion in its "visceral" or survival aspects, inferring this from its primitive cortical structure. In proposing that definite non-olfactory functions are mediated by the hippocampus, and that the hippocampus gathers impulses from the sensory areas, MacLean's report leads in a direct line to the theoretical formulation from which the present investigation takes rise. Between MacLean's report and the expression of the theory on which this study will be based, however, came a decade of intense further study of the hippocampus on the part of numerous investigators.

In the period that followed publication of MacLean's article, a number of investigators of different persuasions began to consider the hippocampus to be a center for collecting impulses from the neocortex and for relaying them to subcortical structures (e.g., Green & Arduini, 1953; Kaada, 1951; Nielsen, 1956, 1958). Now investigations of the hippocampus and of structures believed

to be related to it in function increased in a way that hints of geometrical progression.

Based on experiments and clinical observations of the past decade, a number of roles relevant to the present study have been proposed for the hippocampus. Kaada, Gloor, Penfield and Milner, Nielsen, McLardy, and Pribram have all recently proposed functions for the hippocampus. For Kaada (1951) the hippocampus is part of a general forebrain suppressor system. For Gloor (1956) the hippocampus serves as modulator of functional patterns. Penfield and Milner (1956, 1958) in their clinical orientation see the hippocampus as part of the interpretive cortex, serving memory functions. For Nielsen (1958) the hippocampus is the site of memory traces. In a hypothetical application of the logic of computer systems McLardy (1959) views the hippocampus as detector-coder of information from the temporal lobes. Finally, Pribram (1961) conceives the hippocampus to be part of a planning mechanism.

The reasons for basing the present study on Arnold's 1960 formulation are fully discussed in Chapter 1. It will suffice here, therefore, to compare this formulation with those that appeared in the 1950's. Arnold's formulation resembles other recent proposals on hippocampal functioning in: (1) being buttressed by experimental and clinical findings; (2) being amenable to further experimental and clinical investigations; (3) contending that the hippocampus gathers impulses from the neocortex (e.g., Gloor, Green and Arduini, McLardy, Nielsen); (4) proposing that the hippocampus is part of a larger circuit (not always specified by others) with both cortical and subcortical connections; and (5) suggesting that the hippocampus serves memory (e.g., Nielsen, Penfield and Milner, McLardy, and Pribram). In the cases of McLardy and Pribram, no direct

reference is made to the hippocampus in mediating memory. The fact that they see the structure as a detector-coder of information (McLardy) and as part of a planning mechanism (Pribram) implies memory.

Arnold's formulation differs from the other approaches of the past decade in some ways, too: in (1) starting with analysis of psychological activities that lead from perception to action; and (2) only afterwards, considering neurophysiological research studies in terms of the psychological processes; (3) distinguishing between the hippocampal and limbic structures in functions served; and finally (4) proposing how the hippocampal structures and functions could mesh with other structures and functions.

#### Arnold's Theory of Hippocampal Functioning

Since the present study was suggested by Arnold's formulation, it will be considered in some detail. For Arnold the hippocampal system serves recall. Structurally, the hippocampal system includes the hippocampus and the hippocampal rudiment. According to Arnold, the hippocampal structures lead via the fornix to the brainstem and back to the cortical association areas. (See Fig. 1) In this formulation the limbic system consists of the subcallosal, cingulate, retrosplenial and hippocampal gyri, and the Island of Reil, but does not include the hippocampus and hippocampal rudiment, since different functions are proposed for the two groups of structures (Arnold, Vol. II, p. 32, 1960) For Arnold the limbic system serves the function of appraisal of objects, situations, and actions.

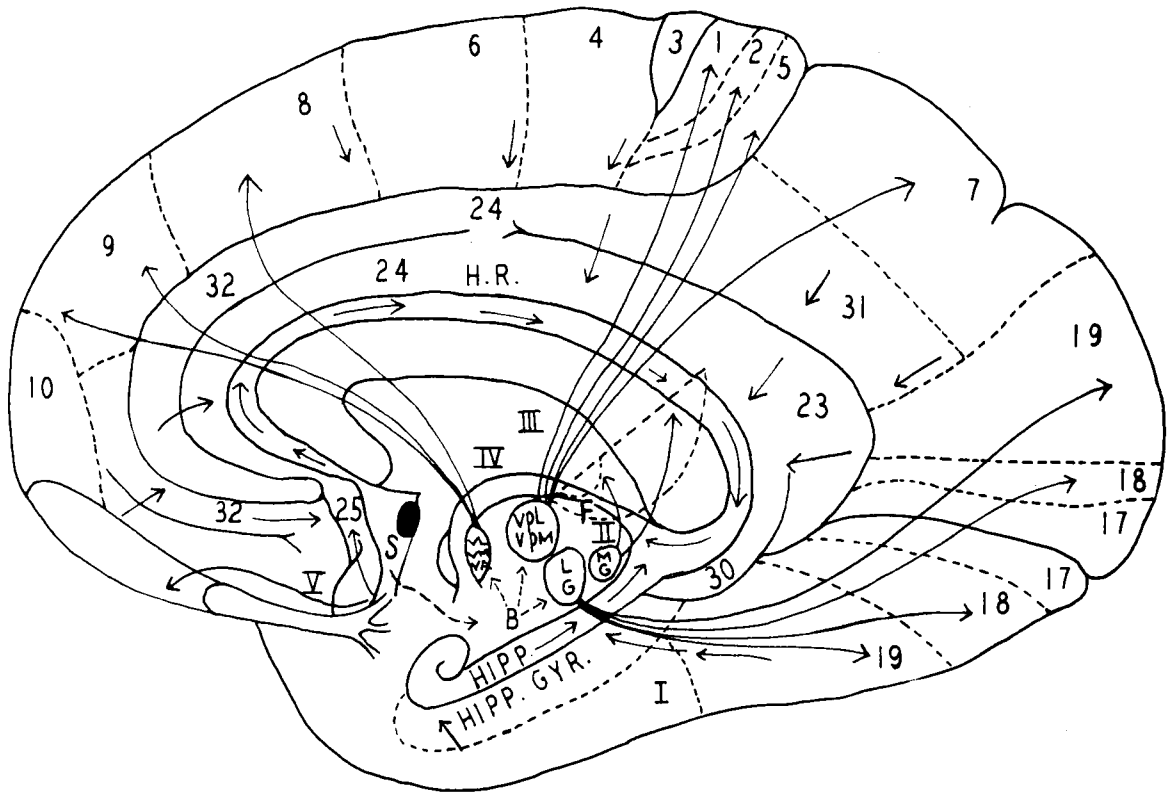
In this view, memory, served by the hippocampal system, is not a unitary function, but is modality-specific. Structurally, this is so because sensory and motor impressions for each of the different modalities are registered

Fig. 1. Memory circuit proposed in Arnold's formulation, with pathways followed after impulses have been registered, leading to recall.

Arrows indicate direction of conduction. I Visual system; II Auditory system; III Somesthetic system; IV Motor system; V Olfactory system. Arabic numerals represent Brodmann areas. B. Brain stem; Hipp. hippocampus; Hipp. Gyr. hippocampal gyrus; H. R. hippocampal rudiment; F. fornix; LG lateral geniculate nucleus; MG. medial geniculate nucleus; S. septal area; VA. anterior ventral nucleus; VM. ventromedial nucleus; VL. ventrolateral nucleus; VPL. ventroposterolateral nucleus; VPM. ventroposteromedial nucleus.

Source: Redrawn from M. B. Arnold, 1960, *Emotion and personality*, v. II, p. 69, Columbia Univer. Press, New York, without sensory projections and appraisal pathways.





separately in the various sensory and motor areas. Under the proper circumstances, the separately-registered sensory and motor impressions can be recalled via a memory circuit leading via the hippocampal structures.

According to this formulation, when something is sensed through some modality, it is first of all appraised as good to know. In the modalities of vision and audition, appraisal is mediated by the hippocampal gyrus (limbic region which serves these modalities, and which borders on the primary sensory areas). In the other modalities, too, appraisal is mediated by the respective neighboring limbic areas.

Appraisal of sensory impressions in the modalities of vision and audition produces an impulse to recall similar situations (mediated by connections from limbic cortex to hippocampus and from there via fornix, midbrain, and sensory thalamic nuclei back to the various cortical association areas). At the same time the effects of previous situations, good or bad, are recalled (mediated by connections from the limbic cortex to the hippocampus and from there via fornix, mammillary body and anterior thalamic nucleus to the cingulate gyrus). The modality-specific connections for visual and auditory memory lead from the hippocampal gyrus (visual and auditory limbic cortex) to the hippocampus. After fibers leave the hippocampus they join with other fibers (from the rudiment) to make up a common fornix path, shared by all modalities. (Fig. 1)

Impulses from the olfactory, motor, touch, and taste modalities, according to Arnold's formulation, are also registered separately and have no direct connections with the visual and auditory registration areas. In these other modalities impulses are believed to reach the fornix by way of the hippocampal rudiment, anterior continuation of the hippocampus, which courses over the

dorsal surface of the corpus callosum. Thus, in this view damage to the hippocampus (resulting in severing of visual and auditory connections) would not be expected to interfere with memory in the other modalities. On the other hand, bilateral damage to the hippocampal rudiment, depending on the exact location of the lesion, would interfere with olfactory, motor, touch, and taste memory but would leave visual and auditory memory unimpaired. According to this formulation, selective loss would occur because impulses from the septal area (following the rudiment) up to the posterior cingulate gyrus connect with the fornix from one direction (serving olfactory, motor, touch, and taste memory), while impulses from the hippocampus proper (serving auditory and visual memory) connect with it from another direction. The impulses would reach the hippocampal system at different points (leading to selective loss of memory) because of the location of registration areas and limbic areas relative to the hippocampal structures. In the formulation proposed, it is from these areas that the hippocampal structures receive impulses. Following the logic of this formulation, memory in the various modalities would be selectively impaired, depending upon the point at which the hippocampal system is damaged.

If this formulation is correct, olfactory recall would be lost when the hippocampal rudiment is bilaterally transected at the genu of the corpus callosum (See Fagot, 1962). If the hippocampal rudiment is bilaterally transected behind the motor area, both olfactory and motor memory would be lost. (See Fig. 1) Damage to the hippocampal rudiment at the level of the somatosensory cortex, would in this formulation prevent impulses from several modalities---smell, motor, touch, and taste---from reaching the fornix,

therefore preventing memory in these modalities. It would not affect visual and auditory memory, however, for which functions connections from the hippocampus to the fornix are proposed. Only bilateral interruption of the connections would affect memory, for the hippocampal commissure assures bilateral relays to fornix and association cortex as long as the hippocampus or the rudiment are intact on one side.

In the present investigation the aspect of Arnold's theory that applies to motor recall will be tested after transecting the hippocampal rudiment at the level of the motor cortex. The hypotheses Arnold's formulation suggests for testing motor memory, to be studied in the albino rat, may be expressed simply and explicitly:

1. Rats having surgery that transects the hippocampal rudiment (including the induseum griseum and longitudinal striae) will not be able to learn a motor discrimination task, for this can be learned only if motor recall is possible.

2. Rats having surgery that transects the hippocampal rudiment (including the induseum griseum and longitudinal striae) will not be able to retain a motor discrimination, for this can be retained only if motor recall is possible.

Before considering this problem in greater detail, recent and relevant research studies will be reported and appraised.

## Chapter III

### Motor Learning Studies: Review of the Related Literature

Arnold's theory, which this investigation is designed to test, suggests that learning in different modalities may be selectively impaired, depending upon site of damage in the hippocampal structures. In preparing to test this theory, then, relevant studies will be appraised and reported, not in terms of type of behavior or task (e.g., approach or avoidance task, alternation problem, discrimination learning), but according to the modalities employed in the task (e.g., visual, auditory, motor learning).

In the experimental laboratory (as in life) tasks that specifically require motor memory, the focus of this investigation, are extremely rare. Conceived in a general way, of course, all laboratory tasks are motor, in the sense that their performance involves some form of motor activity. But few of these are motor tasks in the sense that motor memory is needed for their solution.

Because of the rarity of studies that specifically test motor learning, this review of the literature will extend its scope to include studies for which motor memory is not specifically required, if they involve the hippocampal rudiment or other structures proposed as serving motor memory. This extension of the related literature will also highlight the differences between tasks that specifically require motor memory and learning and those that do not. On the basis of this review it should also be possible to

determine whether the hippocampal rudiment, as Arnold suggests, is a likely structure for mediating motor recall.

In deciding what function a structure mediates two lines of evidence will be taken into account: what happens when the structure (1) is stimulated; and (2) is ablated. In the psychological literature what happens under these two conditions is commonly tested through performance in laboratory tasks.

#### Stimulation of the Hippocampal System

Electrical stimulation of a single brain structure will not, of course, duplicate the effect of its being stimulated in the natural course of events. When the visual or the motor cortex is stimulated, the result is not vision of a natural situation, or production of a movement as it is usually made. But the results of pertinent stimulation experiments must at least be consistent with a theory to be tested and be explainable by it. Two recent stimulation studies have relevance for the present investigation.

The first of these studies (Flynn & Wasman, 1960) reports that some (but not all) cats stimulated in the hippocampus during learning trials required less time to learn to lift a paw to avoid shock announced by an auditory signal than did cats stimulated in the neocortex (precise locus not specified). Testing whether learning had occurred came after stimulation was over. Judging from the cats' performance after stimulation, learning in at least some animals had apparently occurred during stimulation of the hippocampus. Was the hippocampus, then, not needed for learning this particular performance?<sup>1</sup>

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<sup>1</sup>During stimulation of the motor cortex, spontaneous movement is impossible, and during stimulation of the visual cortex, normal sight is prevented. Analogously, stimulation of the hippocampus would prevent normal use of this structure.

How can these results be accounted for in terms of the formulation to be tested?

The fact that only some cats learned could be explained in terms of auditory recall as it is related to spread of stimulation from the hippocampus. If a cat is able to remember the sound and what it led to, it is likely that the spread of stimulation is slight. A connection from the auditory area to the various cortical association areas is necessary for such learning, according to Arnold's theory.

But how explain why those cats who learned at all learned faster than cats stimulated in the neocortex? This phenomenon may have occurred when the hippocampus was stimulated in the area receiving impulses from visual cortex, thus excluding interfering impressions.

A second research team (Weiskrantz, Mihailovic, & Gross, 1962) found that monkey subjects exhibited seriously impaired performance in alternating between identical plaques to obtain a food reward, with trials given during stimulation in the region of the central sulcus. In contrast, stimulation in the hippocampal gyrus region produced only a slight adverse effect on alternation. In terms of Arnold's formulation the defective performance that occurred during stimulation of the central sulcus could be attributed to effects of stimulation spreading to the motor cortex and rudiment, thus cutting off most connections from motor registration areas to the fornix. The slight impairment that occurred during stimulation of the hippocampal gyrus could be explained as the result of some spread of current to the fornix, thus interfering with recall to some extent. It should be remembered, however, that the exact locus of hippocampal stimulation is rarely given in these research

reports. For this reason, any attempt at an explanation of the results can be little more than an educated guess.

#### Interruption of the Hippocampal Circuit

Since some investigations involving structures in Arnold's proposed circuit for motor memory are directly relevant to the study at hand, while others, since they involve learning in other modalities, are less directly relevant, the ablation literature will be divided into three classes: (1) learning in sensory modalities; (2) motor learning; (3) olfactory learning: a study to test one aspect of Arnold's general hypothesis. This review will concentrate primarily on the literature of the past five years. Reviews of the earlier literature are available elsewhere in survey form (Brodal, 1947; Jasper, 1956) and as specifically applied to the problem at hand (Arnold, 1960).

Learning in sensory modalities Moore (1962) recently reported impairment or loss of a well-established habit of avoiding shock announced by an auditory signal in a shuttle box after damaging cats in the septal region, in the septal-hippocampal regions combined, and in the cingulate region. Lesser impairment was associated with the cingulate injuries than was true of the other kinds of lesions.

In terms of the theory being tested, the severe loss of ability to avoid shock following large lesions in the septal region could be attributed to destruction of precommissural and some postcommissural fornix fibers. Arnold's formulation would predict that such severe damage to the fornix connections would be critical for learning and retention in all modalities, since it suggests that impulses serving all modalities traverse the fornix. The



somewhat more severe loss following the combined hippocampal-septal lesions could be attributed to damage to further connections in the proposed memory circuit, including those proposed for auditory recall (coming from the hippocampus).

In Moore's cingulate animals, which showed lesser 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. In terms of Arnold's formulation, substantial damage to the posterior cingulate region would lead to poor performance because the animals could not recall the sensation connected with shock (because of impulses leading from the somatosensory area to the rudiment being interrupted) or appraise it as bad and so experience it as pain (because of posterior cingulate damage). 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.

In another shuttle box experiment in which an auditory signal announced shock, Isaacson, Douglas, and Moore (1961) reported no learning impairment following hippocampal damage. Since a portion of the hippocampus remained dorsally and rostrally, connections from the hippocampus to the fornix still remained. In Arnold's terms, then, auditory learning and memory (relevant to this shuttle box task) could still occur. The fact that the hippocampal

rudiment was intact could also explain why this task could be learned; touch and pain were both relevant modalities, and in terms of the proposed memory circuit, might be expected to be intact.

A further relevant and provocative study was done by McCleary (1961). McCleary reported that cats learned one task but not another following septal-subcallosal lesions. After cingulate lesions the reverse picture occurred in learning the same two tasks.

In the first task, called "active avoidance" in the 1960 nomenclature of Mowrer, cats were trained to avoid shock in the same kind of shuttle box situation described earlier: sound announced oncoming shock. In this task McCleary found that cingulate animals performed poorly but septal-subcallosal animals performed well.

Impaired performance of the cingulate animals could be explained as an appraisal for action defect; in Arnold's terms, the animals could not appraise what to do. In the same formulation of Arnold the slight septal damage in the animals that learned the same task could be explained because only precommissural fibers were damaged but postcommissural fornix fibers were left intact; thus learning to respond to an auditory signal would still be possible.

Connections from hippocampus (for auditory memory) were not disturbed, and the fornix fibers arriving at the septal region were not sufficiently disturbed to prevent the passage of impulses. This explanation is consistent with the apparently contradictory evidence of Moore who, using septal cats, trained them in the same shuttle box task. The discrepancy can be explained in terms of the theory to be tested. In McCleary's animals the septal lesions were far less severe than was true of Moore's. The more anterior placement of lesions

in McCleary's subjects also left postcommissural fornix fibers intact, which would be sufficient to mediate learning of the shuttle box task.

The same animals that learned to avoid shock announced by a sound (McCleary, 1961) failed to learn another task, called "passive avoidance" by McCleary, in the Mowrer (1960) nomenclature. Passive avoidance required cats to learn to avoid a food dish that had formerly delivered an electric shock. In this task the (septal-subcallosal) cats that had learned to avoid shock announced by a sound ("active avoidance") did poorly in "passive avoidance." The cingulate cats, in contrast, did poorly in "active avoidance" but well in the "passive avoidance" task.

An analysis of functional requirements for the passive avoidance task, compared with the kind of lesions, can account for the apparently discrepant findings. Both the defect in septal-subcallosal animals and the apparent success in cingulate animals can be explained satisfactorily in terms of functional loss. In determining how this can be, it is important to consider what goes on in animal learning. Animals, of course, depend upon sensory experiences to recall associated memories. These associated memories, in turn, prompt recall of others. On seeing a food box in the passive avoidance task the animal does not recall as would a human being that "food" was in the box. He only remembers sensory experiences such as sights, smells, tastes, etc.

In terms of functions needed for the passive avoidance task poor performance of McCleary's septal-subcallosal animals could be explained as an olfactory memory loss, owing to the fact that the anterior septal region, from which the hippocampal rudiment takes rise, was extensively damaged (McCleary,

Personal Communication, 1963). In terms of Arnold's proposed circuit impulses from olfactory regions travel to the fornix by way of the hippocampal rudiment; hence, damage to the hippocampal rudiment would lead to olfactory memory loss. Applied to McCleary's "passive avoidance" task, animals with septal-subcallosal lesions could still be expected to smell the food (a moistened food mash) when a sliding door in the training cage opened to reveal it. But they could no longer recall that this smell had been followed by unpleasant consequences in the past (the shock) because the connection between the orbital area (olfactory registration) and the hippocampal rudiment and fornix was interrupted. Thus they would appraise the smell as good to investigate, would do so, and again be shocked.

The cingulate animals (at least those with complete rudiment transections as the cingulate level) would in Arnold's terms suffer the same olfactory agnosia present in the septal animals with complete rudiment transections. But in the case of the cingulate animals there would also be a defect in appraising action<sup>1</sup>---the same functional defect postulated to explain the failure to take appropriate action in the "active avoidance" test. Since staying away from the food box is "successful" behavior in the passive avoidance task, however, the functional defect would not be manifest in animals that stayed away from the food box. Both cingulate and septal animals, then, in Arnold's terms, must have suffered functional defects that could account for the discrepant behavioral results. These results testify that success in response terms does

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<sup>1</sup>i.e. they could no longer appraise the smell as good to investigate and so would not take appropriate action.

not always signify adequacy of behavior.

A final study (Peretz, 1960) illustrates the importance of analyzing both the functions needed for a task and site of damage as well. Peretz lesioned rats in the anterior cingulate region, at the genu of the corpus callosum. This damage sometimes resulted in transection of the hippocampal rudiment further forward in the brain than the motor area. After such damage rats did well in a T-maze task, requiring them to run down an alley of a particular color, in order to receive a food reward.

In a second task, however, a shuttle box situation in which the animals were placed in a black compartment and had to learn to run into an open white compartment in order to avoid shock, rats with such anterior cingulate damage did poorly. This task provided no signal to announce shock. The animals simply had to run to the next compartment within ten seconds after being placed in the box or be subjected to a shock. How account for the discrepancy in performances on the two tasks by animals with similar lesions?

In the T-maze task the animals had clear-cut visual cues. In terms of Arnold's formulation, with definite discriminative visual cues and with damage that came nowhere near the circuit proposed for visual memory, the animals might be expected to perform well, which in fact they did. In the shuttle box task, however, where no discriminative visual cues could lead to appropriate action, impairment appeared. According to Arnold's formulation, the impairment in this task could be interpreted as an appraisal for action defect, occurring in a situation with reduced cues. With repeated trials these animals overcame the defect, probably because some anterior cingulate tissue (for mediating appraisal) still remained.

Motor learning Very few studies specifically test motor memory after damage to regions that Arnold suggests serve that function. An occasional study, however, approximates this kind of test.

For instance, Thomas and Otis (1958) found that hungry rats with bilateral posterior cingulate lesions did poorly in learning to run a Lashley 8-cul Type III maze. Damage extended to the caudal hippocampus, spread dorsally to the posterior cingulate region, and transected the hippocampal rudiment at the splenium of the corpus callosum. In terms of Arnold's formulation the defective performance could be interpreted as owing to transection of both the motor memory circuit (posterior level of hippocampal rudiment) and of connections leading to the hippocampus via the retrosplenial area, proposed for mediating visual learning. These lesions interfered with both motor and visual learning.

Also using a Lashley Type III maze, another research team (Thomas, Moore, Harvey & Hunt, 1959) found that rats trained in maze learning after being lesioned in the septal area of the brain showed no impairment. Since the lesions transected only precommissural fornix fibers, and postcommissural fibers would be sufficient for mediating learning, this result is consistent with what Arnold would predict.

With rats as subjects, Kaada, Rasmussen, and Kveim (1961) studied learning and retention of a maze habit after making lesions in the hippocampus, fornix, and overlying cortical areas. These workers found that rats with subtotal, though sometimes bilateral, hippocampal and fornix lesions tended to do poorly in maze learning and retention. Rats with damage to the overlying cingulate region showed more impairment than did rats with hippocampal or subtotal

fornix lesions which lacked cingulate damage.

In Arnold's terms the defective maze performance found in rats with overlying cingulate damage could be attributed to failure to recall the satisfaction of taste (eating) because of transection of the circuit proposed for memory in that modality. Visual memory could also suffer interference (because of damage to the hippocampal connections), and to the fornix connections as it leaves the hippocampus. The fact that the fornix lesions were subtotal, of course, indicates that the functional loss could not be attributed to damage to that structure alone. Hence, in animals in which the damage was primarily in the fornix (as it leaves the hippocampus) or in the hippocampus proper, only visual memory would suffer interference. Thus learning and relearning could still occur via the hippocampal rudiment, as its fibers reach the fornix from another direction. (See Fig. 1).

In Arnold's terms the results of Kaada and his coworkers (1961) would be entirely expected; damage to the hippocampus or fornix as it leaves the hippocampus would prevent impulses from only one direction from reaching the fornix. Those impulses that travel by way of the hippocampal rudiment (for motor and taste memory) would not be stopped in the absence of rudiment transection. The fact that animals with damage to both the posterior cingulate region (with underlying rudiment) and hippocampus or fornix did poorly in the maze task supports this interpretation.

In a final experiment, lesions were made in the cingulate region near the motor area in rats. After this the rats received training in a Lashley Type III maze. Though the subjects learned the maze, an occasional rat did very poorly. Since the lesions made in this experiment closely approximate lesions

intended in the study of motor memory and the hippocampal rudiment, this experiment deserves detailed analysis.

The heart of the matter is how to account for good learning in some cingulate animals and for relatively poor learning in others, in terms of the theory to be tested. Cingulate damage was incomplete in these animals. Hence appraisal for action, which requires some intact anterior cingulate connections, could still occur. Moreover, the circuit proposed for visual learning (relevant to the Lashley Type III maze task)<sup>1</sup> was intact. Intactness of both visual memory and of ability to appraise appropriate action could account for the performance of the good maze learners. With both visual and motor learning possible, no learning defect would be expected.

Study of the best and poorest learner revealed only one slight difference in lesion. In the best learner the lesion was placed just ahead of the motor region; the hippocampal rudiment was not transected. In the poorest learner the lesion was placed somewhat further back in the brain, and the hippocampal rudiment was bilaterally transected at the motor level.

In both the animals--the best and the poorest learner---the part of the memory circuit Arnold proposes for visual learning (relevant to this task) was intact. The present task is not a test, therefore, for judging the presence or

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<sup>1</sup>In the Lashley Type III maze the animal is confronted at each choice point with two alleys of different lengths. Visual cues can aid in learning appropriate turns, since the animal can see that the two alleys are different in appearance.



absence of motor memory in an unambiguous fashion. The poor learner's performance, however, suggests that complete rudiment transection at the motor level of the brain could be crucial in accounting for his poor performance, particularly if the animal happened to be one that profits little from visual cues. This is a reasonable possibility since Honzik (1936) has pointed out that rats do not all use the same cues to the same extent. Since visual cues were not controlled in this experiment, however, and since it is also feasible that the slow animal would have been a very slow learner whether lesioned or not, the results must be interpreted cautiously. They suggest, however, that if motor learning is the only relevant modality available to the poor learner in learning this task, the hippocampal rudiment is crucial. Of course, the fact that only one animal is used as a basis for this possible conclusion calls for marshalling of further data, before a more conclusive statement is made.

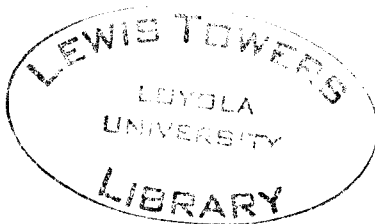
Finally, in a recent study of four human beings (Whitty & Lewin, 1960) it is reported that amnesia and confabulation followed cingulectomy. Since reporting remembered events is a motor situation in which verbal recall is employed, damage to the hippocampal rudiment at the motor level could in terms of Arnold's formulation account for the results. Until anatomic data are available, however, the interpretation is simply suggestive.

Olfactory learning Fagot (1962) did the first study designed to find out whether the hippocampal rudiment is needed for learning and retention in a particular modality--olfaction. After transecting the rudiment bilaterally at the genu of the corpus callosum, Fagot reported that rats could no longer discriminate between the odor of extract of pine (which led to water reward)

and oil of hyacinth (which led to no reward). Control operated animals, however, could make such distinction.

Fagot's study supplies evidence for the phase of Arnold's hypothesis that deals with olfactory recall. Every animal with bilateral transection of the hippocampal rudiment was unable to learn or retain the olfactory discrimination while every animal without such lesions was able to learn and retain.

Of the other relevant studies, none contradicts Arnold's theory, but none provides evidence as to whether or not the hippocampal rudiment is needed for motor memory. To answer this question, the present investigation aims to discover whether bilateral transection of the hippocampal rudiment at the level of the motor cortex will or will not prevent learning and retention of a motor habit.



## Chapter IV

### Purpose, Problem, Method

A simple statement of procedure would afford little evidence of the steps that led to the experimentation. Thus, before stating the procedure some time will first be devoted to considerations that led to the particular application of method used in this experiment. Let us first briefly recapitulate the nature of the problem, for it is upon this that any specific applications of method will be based.

According to the theory from which this study derives (Arnold, Vol. II, 1960), bilateral transection of the hippocampal rudiment behind the motor cortex would block the performance of appropriate action in tasks that depend upon motor recall. In this formulation the hippocampal rudiment receives impulses from the prefrontal, premotor, and motor cortex, as well as from the anterior cingulate gyrus. The hippocampal rudiment relays the impulses via the fornix to the midbrain, then to the thalamic association nuclei, and finally to the association cortex, for recall; and to the midbrain, cerebellum, ventral thalamic nuclei, and frontal, premotor, and motor cortex, for action. According to this formulation, then, bilateral transection of the hippocampal rudiment at the central sulcus (i.e., at the point where the motor and somatosensory cortex meet on the medial surface) would disrupt a link that is needed if motor memory is to affect behavior. After such damage, according to this theory, impulses from the prefrontal cortex (mediating recall), from the

premotor cortex (serving impulses for action), and from the anterior cingulate gyrus (serving appraisal of action and action impulses) could no longer be relayed through the fornix to midbrain and cerebellum, and then to the motor cortex. Consequently, such impulses would no longer affect action. Action would still be possible, on the basis of visual, tactual, or auditory memory and the revived experiences of satisfaction previously achieved. But actions that depend upon motor memory would no longer be successful.

Determination of whether the hippocampal rudiment is part of a recall circuit needed for motor memory presupposes that certain conditions be fulfilled. First, it presupposes that registration has occurred, i.e., of motor patterns that can be remembered. Second, it presupposes that there be some relevant performance to recall which absolutely depends upon motor memory. Without registration, there would be nothing to recall. Without some motor task that needs previous experience, what is registered will not be recalled.

In order to discover whether the hippocampal system is needed for motor memory, it is vitally necessary, of course, to devise an appropriate experimental situation. Preliminary consideration of what motor memory requires, followed by a pilot study, led to choice of an appropriate experimental situation.

#### Considerations Leading to Choice of Task

Recall must be tested through performance. How recall is organized until the desired motor pattern is complete constitutes a learning test. How long and how perfectly this organized recall persists constitutes a retention test. The Present experiment aims to test both learning and retention of a motor habit.

Although all laboratory learning tasks require the registration of motor performance and its recall, only some such tasks are appropriate for testing motor recall unambiguously. In some tasks visual cues are given; these promote visual recall. In other cases auditory, olfactory, or somesthetic cues are provided. A task designed for testing motor recall must be such that sensory cues are not sufficient to lead to appropriate performance. This condition would be met whenever the available sensory cues remain the same or change randomly, when the animal must recall what he has done previously before he can select the action that leads to reward. This is the case, for instance, in single alternation tasks or in go/no go alternation problems. Given the proper control measures, such tasks may test motor recall in an unambiguous way. For the present study a T-maze alternation situation was chosen after careful reflection as a task that could test motor recall unambiguously (Fig. 2)

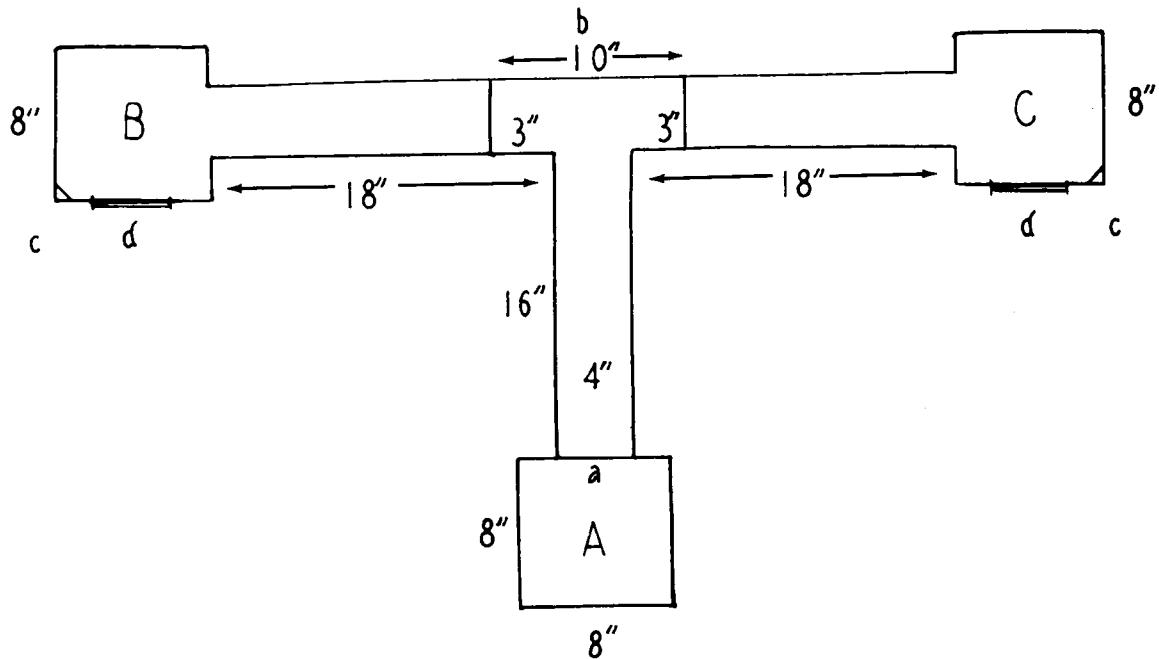
#### Pilot Study

A number of features (such as dimensions of the maze, positioning of sliding doors) incorporated into the final apparatus resulted from the pilot study. Choice of method of lesion-making was also determined on the basis of the pilot study. In the course of coming to a decision about task and techniques, control measures of various sorts were also found necessary, and ways were devised to put them into effect. These will be discussed when relevant under the section on Method.

A stereotactic technique was chosen for making lesions because: (1) it can induce small lesions in the desired location; (2) it has numerous experimental precedents; and (3) the open-brain technique used at first in the pilot study resulted in too many deaths. Although some research workers have

Fig. 2. Floor plan of T-maze used for study of single alternation in the albino rat.

A. starting box; B. and C. goal boxes; a. entryway for maze; b. plastic-covered transparent area, bounded on either side by wall grooves into which sliding doors may be placed; c. dipper region; d. perforated steel panel that extends the height of the maze.



FLOOR PLAN OF T - MAZE

effectively used the open-brain technique of lesion-making (e.g., Moore, 1962, with cats; Thomas & Slotnick, 1962, with rats), this procedure did not work well in the pilot study and so was abandoned. Probably because in open-brain surgery the lesion site approached from the dorsal surface of the brain lies near the midline, the longitudinal sinus was often damaged. This resulted in excessive bleeding and sometimes in the animal's death. Since vascular changes from sinus damage could lead to ambiguity in interpreting the findings, and because a tremendous loss of time results when a trained animal dies before being retested, the open-brain technique was found inadequate for the present purpose.

Albino rats were chosen as subjects. These animals commend themselves on a number of counts: (1) easy availability; (2) docility; and (3) possession of all the brain structures that are found in higher mammals.

Before considering the procedure it seems well to indicate a few of the salient features of the rat brain relevant to the present study. Krieg (1955, p. 169) notes that the degree of correspondence between the rat brain and the human brain is remarkable for two species as widely separated phylogenetically. In the rat, of course, the cerebral hemispheres are not as well developed as they are in man and are not convoluted. The same structures appear in the rat brain, however, as are found in man, though their relative size differs. In the rat, for example, the olfactory bulb is relatively large when compared with that of man. The hippocampus in the rat is likewise very large, compared to the size of that structure in the human being.

The structure intended for damage in the present experiment, the hippocampal rudiment, is made up of a layer of gray matter on the dorsal



surface of the corpus callosum. Within this layer two strands of longitudinal fibers, close together on each side of the median plane, form the median longitudinal striae. On both sides, at the point where the supracallosal gyrus bounds the sulcus in the neighborhood of the corpus callosum, lies the lateral longitudinal striae. According to Ranson and Clark (1959, p. 335) the hippocampal rudiment can be traced from the medial stria toward the rostrum of the corpus callosum, then to the splenium, behind which it becomes continuous with the hippocampus proper. These traced connections are consistent with Arnold's formulation, which holds that impulses from the hippocampal rudiment follow the same course, meeting the fornix as it emerges from the hippocampus.

According to Krieg's atlas, the point of the contemplated lesions for the present study corresponds to 55.5 in antero-posterior direction and at or near the midline, where the hippocampal rudiment is situated. Figure 3 is a cross-section of the rat brain at the intended level. The cross-section reveals two vertical electrode tracks in one of the uninjured subjects.

#### Method

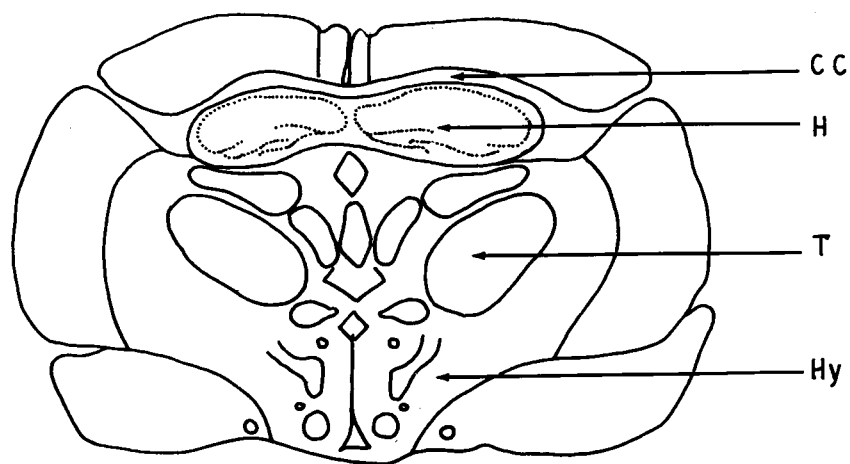
##### Subjects

Thirty-two male albino rats of the same age served as subjects. At the time of arrival in the laboratory, the animals were approximately 100 days old. With a few exceptions (e.g., #65 weighing slightly more than 400 grams) the majority of animals ranged in weight at time of arrival in the laboratory from approximately 260 to 330 grams. Nearly all animals gained weight under the laboratory regimen.

On arrival in the laboratory, the animals were randomly assigned to the following groups (with five animals in each): Group 1, experimental animals

Fig. 3. Schematic brain section at motor level of the brain in rat #66, an unlesioned electrode track control animal.

Vertical lines indicate electrode path. CC. corpus callosum; H. Hippocampus; T. thalamic region; Hy. hypothalamic region.



RAT No. 66

for the study of learning (bilateral rudiment transection intended in these animals before learning); Group 2, experimental animals for the study of retention (bilateral rudiment transection intended in these animals after learning); Group 3, control animals for the study of retention over time (no operation); Group 4, animals to control for the effect of the operation (electrode track controls)<sup>1</sup>; Group 5, control animals with lesions in neighboring structures (lesioned controls).

### Apparatus

A temporal T-maze made of wood, painted a flat gray, with flooring of Con-Tack, and offering no discriminative cues from sensory modalities was used for training (Fig. 2). Except for a ten inch by four inch area ("b" in Fig. 2) at the place where cross alleys and stem alley met, the entire maze had a wooden hinged covering. This mid-maze area, covered with clear plastic, enabled the experimenter to know exactly when the subject started to go down one of the two identical cross alleys. In each cross alley a manually-operated wooden sliding door, at the point where the transparent area met the wooden covered area, could be slid into place as soon as the animal (exclusive of tail) progressed beyond that part of the maze. The doors in the cross alleys prevented what retracing might otherwise have occurred in cases in which the animal arrived in the goal box on the unrewarded side. To prevent injury to the subject's tail, each of the sliding doors had a cutaway portion at its base (approximately three-eighths of an inch in height) which extended the width of the alley. A sliding door similar to those placed near the choice

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<sup>1</sup>In these animals the electrode was introduced into the hippocampal rudiment and withdrawn without turning on the current.

points opened into the stem alley of the maze. This door differed from the other sliding doors only in its not having a cutaway portion at the base. Goal boxes and their corresponding cross alleys were identical in appearance. In each goal box a Foringer dipper capable of holding one-tenth of a cubic centimeter of water jutted out diagonally from the far corner of the side facing the experimenter ("c" in Fig. 2). The side nearest the experimenter in each goal box had a perforated steel panel, which made it possible for the experimenter to see when the animal entered the goal box ("d" in Fig. 2).

The maze rested on a table in an experimental room having a single overhanging fluorescent light, a small table on which the animal's cage was placed during training, and along the wall furthest from the door, a set of control panels used for another experiment. The maze was rotated 45 degrees daily, to control for visual cues from outside the maze. Animals were housed in a room adjoining the experimental room.

#### Procedure

1. Pretraining. In order to get the animals accustomed to their new environment (living in individual cages) and to the manner of feeding, the first two days after arrival in the laboratory were spent free of training. Throughout training food (Rockland Rat Ration) was always available to the animal. After the second day in the laboratory, however, water was available only at a regular fifteen-minute period daily. This measure was necessary since thirst was to be a condition under which the animals would be trained.

Since gentled rats tend to be better maze learners than animals not given this treatment (Barry, 1957), each animal used in the present experiment was handled in the same manner for three minutes daily prior to training. This

procedure continued for a week. It insured that the animals would be accustomed to being handled by the time the regular training began.

The day after the gentling procedure ended, pretraining in the maze began. The first part of the pretraining consisted in giving the animals experience in finding water in the goal boxes. For two successive days each rat was placed in each goal box five times. He remained there until he found the dipper of water. During the period of time between trials the animal was placed in the starting box while the dipper was refilled. The purposes of this training were: (1) to get the animal accustomed to finding water; and (2) to familiarize the animal with the goal boxes and starting box. Thus, this procedure insured that initial speed or slowness in finding water, because of lack of familiarity with the maze itself, would not complicate interpretation of results on motor learning.

For two days after water-finding training ended, each subject received ten forced trials daily, so-called because in one of the cross alleys on any given trial, the sliding door was in place, thus leaving only one alley open. The rat was rewarded alternately, first in one goal box, then in the other. The reasons for giving forced trials were: (1) to familiarize the subjects with the entire maze; (2) to give the animals a start in the performance they would be required to learn. This procedure also led (3) to saving time in maze training, owing to the fact that all animals received this common series of relevant experiences.

2. Training in single alternation. After being water-deprived for twenty-four hours, each subject received ten counted trials daily for the first three successive days of regular training. The first trial on each day (for

the entire period of training) was a "free trial," so-called, because it was not counted as correct or incorrect, but always led to reward since both goal boxes were baited. Whether the pattern of alternation would begin with the right or the left side was determined by which goal box the animal went to on the free trial. On all trials after the free trial of any given day, the animal was rewarded only if he went to the goal box opposite the one in which he had last found water. Thus, if the rat went to the right-hand goal box on each of the first five trials, he would be rewarded only on the first trial.

On the fourth and fifth regular training days all animals (except #65, #55, #79, #81-#84) received two days of twenty trials each. After this, until the end of training, the daily number of counted trials was fifteen. The animals listed earlier in this paragraph as exceptions received fifteen trials a day, after the first three days of ten trials, until the end of training. The reason for reducing the number of trials to fifteen (it had originally been decided to have twenty per day, after the orientation days) was that when twenty trials were given daily, the animals seemed to be less and less motivated during the last trials. Decreasing the trials to fifteen seemed to resolve this problem.

On each trial the animal was allowed between ten and fifteen seconds in the goal box. During this period he had ample time to drink the water in the dipper, and the experimenter had time to fill the dipper in the opposite goal box. Time allowed in the goal box remained constant whether on rewarded or unrewarded trials. Alternation training continued until the subject achieved ninety per cent correct trials or higher on at least forty-five successive trials. Within three days after reaching the criterion the animals to be

operated upon had surgery. Unoperated subjects were treated in every other respect like the operated animals.

Except for four late-operated animals, used for the purpose of increasing the number of rudiment-transected subjects, a waiting period of twelve days elapsed between operation and retesting. A week after operation the animals were again started on the water-deprivation regimen. In the case of learning animals, pretraining in the maze began nine days after operation (except for rat #55, a late-operated animal given pretraining on the days immediately preceding operation). The late-operated rats were placed on water-deprivation training two days after operation as they were trained sooner after operation than was true of the other animals.

3. Operations. For four hours before being operated upon the subjects were deprived of both food and water. All animals to be operated were subjected to ether anesthesia before the operation. To induce anesthesia the animal was placed in an ether cabinet, a cylindrical chamber of five-liter capacity. An electric motor provided power for pumping a measured quantity of an ether-air mixture into the ether chamber. This mixture was allowed to escape through a small hold drilled at the side of the chamber opposite the place the pump tube entered. When needed during the operation, ether was administered on a wad of cotton.

After the anesthetic had taken effect, the dorsal surface of the rat's head was shaved. Then the animal was fastened into the stereotaxic apparatus (Krieg Stereotaxic Instrument, Model #51200, Stoelting). From a point midway between the ears to the back of the neck, a long scalpel cut was made. Then the top skin was clamped back, and the skull cleared of galea and periosteum.



Now the bregma became visible. According to Krieg's Atlas, the point to be penetrated, for inducing damage to the rudiment at the place where the motor and somatosensory areas meet, lies approximately a millimeter and a half behind the bregma. (See Fig. 3)

After finding the 55.5 coordinate, and using the bregma as a guide, trephine holes were drilled slightly to the side of the midline, so as to avoid damaging the median longitudinal sinus. A small pick gently penetrated through the rest of the skull, when the partially drilled skull became paper-thin. At this point the two-pronged electrode was inserted to the desired depth and the current turned on. In experimental animals, bilateral destruction of the hippocampal rudiment was intended. Damage in lesioned control animals (Group 5) was intended to be somewhat more superficial than the hippocampal rudiment. Control animals of Group 4 (electrode track controls) had the electrode inserted to the level of the rudiment, but no current was turned on. In the lesioned animals (Groups 1, 2, 5) a pulsating direct current of 90 volts, 100 pulses per second was applied for 15 seconds. After this ended, the electrode was gently removed, the open area of the skull liberally swabbed with alcohol, the blood flow stopped with Gelfoam when necessary, and the skull sealed with bone wax. At this point, time for the suturing of the wound, some of the subjects already showed signs of reviving. With suturing completed, the animals were returned to their cages, where ample food, water, and warmth awaited them.

4. Histology. After the animals had been tested postoperatively, they were killed with an overdose of ethyl ether. The brains were removed in toto and placed in formalin. Serial sections of the brain were cut between fifteen

and twenty microns in thickness, immediately in front of and behind the gross defect. The Klüver and Barrera staining process was used in preparing the slides.<sup>1</sup> When the histological results became available, it was possible to compare how the animal performed in the alternation task with the site and amount of injury. Reporting the behavioral results in the light of histological findings will be the work of the following chapter.

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<sup>1</sup>Histological work was done by a trained technician at the Loyola University School of Dentistry, under the direction of Dr. Patrick Toto, M.S., D.D.S. Brain slides were read by Dr. David Jones, Ph.D., M.D., Professor of Anatomy at Stritch School of Medicine, and by Dr. Patrick Toto. For one subject (#69) slides were also assessed by Dr. Stanley Jacobsen, Ph.D., of the National Institute of Health.

## Chapter V

### Results

This study of motor recall as affected by rudiment damage in the albino rat yielded one unequivocal and consistent finding: animals that sustained complete bilateral transection of the hippocampal rudiment neither learned nor retained the alternation habit. In contrast, subjects with injuries that spared at least part of the hippocampal rudiment were able to learn and relearn the alternation problem.

Study of the training scores reported in Table 1 indicates the completeness with which animals having complete rudiment transections failed. Even after 500 postoperative trials, many more than control rats required for learning, animals with complete rudiment transections showed no signs of learning or relearning the alternation habit.

#### Determination of Brain Injury

Determination of what led to defective performance in some animals but not in others is only possible through careful analysis and comparison of the histological and behavioral findings. This procedure will reveal the relationships that exist between injury and alternation performance, if and when such relationships occur.

Comparison of behavioral results with brain damage must start, not with assumed or intended damage, but with precise determination of where the lesions were actually made and of what structures other than the intended ones

were involved in the injury. A technique proposed by Krieg (1946) was used to map the site and extent of lesions in this experiment. Krieg's Atlas of Standard Coordinates for the Rat Brain (1946) makes it possible to locate lesions at any level in the brain. These coordinates are expressed in millimetric intervals, in three dimensions. The dorsoventral dimension measures from 0 to 10; the postero-anterior dimension ranges from 44 to 66; the left to right dimension extends from 78 to 90. Using the coordinate system of Krieg, lesions of rats in the present study will be reported as they would be in the fresh rat brain, on which dimensions this system is based. In using this coordinate system, of course, one assumes that brain tissue shrinks proportionately during preparation of slides.

Since in the present experiment the behavioral results for both learning and retention closely mesh with site of injury, results will be ordered in terms of site of the primary lesion. Table 1 and Table 2, which report the behavioral and histological results in operated subjects, respectively, and Table 3, which reports the performance of unoperated control animals, will be the basis for the discussion of results. The discussion will follow the same order as is used in the tables.

#### Comparison of Behavioral and Histological Results

##### Complete transection of the hippocampal rudiment

Three animals (#71, #86, #87) sustained complete transection of the hippocampal rudiment at a point corresponding to Krieg coordinates 2.5, 55.5, 84-82.5. In rat #71 the lesion extended posteriorly to a point between Krieg coordinates 53.5-54. The brains of these rats, then sustained lesions at the depth of the corpus callosum (2.5); at the place where motor and somatosensory

Table 1

## Learning and Retention Records of Operated Rats

Group	Animal Number	Trials Before Operation		Criterion Achieved	Trials After Operation		Criterion Achieved
		<u>Learning</u>	<u>Retention</u>		<u>Learning</u>	<u>Retention</u>	
Complete	86	---	---	---	500+	---	No
Transection	87	---	---	---	500+	---	No
Hippocampal	69 <sup>b</sup>	235	---	Yes	---	500+	No
Rudiment	71	190	---	Yes	---	500+	No
		Ttl.=	<u>425</u>		Tt.=	<u>1000+</u>	<u>1000+</u>
		Mean=	<u>212</u>		Mean=	<u>500+</u>	<u>500+</u>
Partial	88	---	---	---	220	---	Yes
Transection	89	---	---	---	375	---	Yes
Hippocampal	70	175	---	Yes	---	105	Yes
Rudiment	72	205	---	Yes	---	135	Yes
	73	130	---	Yes	---	90	Yes
	77	190	---	Yes	---	75	Yes
	81 <sup>a</sup>	255	60	Yes	---	240	Yes
	83 <sup>a</sup>	180	105	Yes	---	465	Yes
		Ttl.=	<u>1135</u>		Ttl.=	<u>595</u>	<u>1110</u>
		Mean=	<u>189</u>		Mean=	<u>298</u>	<u>185</u>

Note.--Learning mean for all animals reaching criterion, including unoperated control animals (N = 24) is 176 trials; median is 190-205 trials. Retention mean for all animals reaching criterion (N = 20) is 110 trials; median is 75 trials. Striking statistical significance was not achieved though behavioral differences following operation in one group (#86, 87, 69, 71) were marked and long -standing.

<sup>a</sup>This animal also found in Table 3; first used as unoperated control animal for retention over time.

<sup>b</sup>The rudiment is partially intact in this animal. Because of extensive danger to fornix, subject is classified here. See text.

Table 1 (Continued)

## Learning and Retention Records of Operated Rats

Group	Animal Number	Trials Before Operation		Criterion Achieved	Trials After Operation		Criterion Achieved
		<u>Learning</u>	<u>Retention</u>		<u>Learning</u>	<u>Retention</u>	
Lesions in Neighboring Structures	67 <sup>c</sup>	115	---	Yes	---	210	Yes
	74	265	---	Yes	---	75	Yes
	75	385	---	Yes	---	45	Yes
	76	235	---	Yes	---	45	Yes
	78	175	---	Yes	---	45	Yes
		Ttl.= 1175	---		Ttl.= 420		
	Mean= 235	---		Mean= 84			
Electrode Track Controls	61	190	---	Yes	---	45	Yes
	62 <sup>d</sup>	235	---	Yes	---	180	Yes
	65	---	---	---	300	---	Yes
	66	145	---	Yes	---	45	Yes
	68	175	---	Yes	---	90	Yes
	55 <sup>e</sup>	---	---	---	330	---	Yes
	Ttl.= 745	---		Ttl.= 630	360		
	Mean= 186	---		Mean= 315	90		

<sup>c</sup>In this animal, lesion primarily in ventral anterior thalamic nucleus, a structure not damaged in any other animal.

<sup>d</sup>No histological report available for this animal.

<sup>e</sup>Late-operated animal.

Table 2

## Structures Damaged in Operated Animals

Group	Animal Number	Structures Damaged										
		Hippocampal Rudiment		Cingulate Cortex		Cingulum		Cerebral Cortex		Corpus Callosum		Other Structures
		L	R	L	R	L	R	L	R	L	R	
Complete Transection Hippocampal	86 87 69 <sup>a</sup>	X	X	X	X	P	X	X	X	X	X	
		X	X	X	X		X		X	X		
		X		X		X			X			<sup>b</sup> (See below)
Rudiment	71	X	X	X		X			X			
Partial Transection	70 72	X		X		X		X	X		X	P.Des.Rt.
		P	X	X		X		X	X			hippo.
Hippocampal Rudiment	73 77	P	X		X	P	X			X		P.Des.Rt.
		X		X		X		X		X		hippo.
	81	P	X		X		X	X		X	X	L. internal cap.
	83	X		X		X		X		X		
	88	X		X	X	X		X	X	X		
	89	X		X		X		X	X	X		

Note.--"X" indicates moderate to great damage to a structure; "p" indicates partial and slight damage to a structure.

<sup>a</sup>Though the rudiment is partially intact in this animal this subject is classified here because of extensive damage to a link in the proposed memory circuit, i.e., to the fornix.

<sup>b</sup>Left hippocampus destroyed; fornix interrupted.

Table 2 (Continued)

## Structures Damaged in Operated Animals

Group	Animal Number	Hippocampal Rudiment		Cingulate Cortex		Cingulum		Cerebral Cortex		Corpus Callosum		Other Structures	
		L	R	L	R	L	R	L	R	L	R	L	R
Lesions in Neighboring Structures	67 <sup>a</sup>	P		P		P		X		X			(See below)
	74			X	X	X		X	X	X			
	75			X	P	X	P	X	X				
	76			X				X		X			
	78				X		X	X	X		X		
Electrode Track	61			No evidence of injury									
Controls	62			No report									
	65			No evidence of injury									
	66			No evidence of injury									
	68			No evidence of injury									
	55			No evidence of injury									

<sup>a</sup>Extensive damage in right ventral anterior thalamic nucleus and right stria medullaris; changed appearance of tissue in hypothalamic area



areas meet (55.5); and at or near the midline of the brain (84-82.5). Thus, the hippocampal rudiment, which lies above the corpus callosum, immediately to the left and right of the midline, suffered bilateral and complete obliteration at this level.

In another animal (#69) the lesion transected the left-hand side of the hippocampal rudiment, with damage extending below the corpus callosum, injuring the left hippocampus very severely. For a distance of at least a millimeter the fornix fibers below the corpus callosum were obliterated. (See Table 2) Since fornix fibers in lower mammals like the rat course immediately below the corpus callosum and the lesion in this region was extensive, it is very likely that a total interruption of rudiment connections was effected by this lesion (Personal Communication, S. Jacobson, 1963). With rudiment connections apparently cut off completely, this animal (#69), then, seems rightfully to belong with the complete rudiment transected rats. This subject might be more precisely termed an animal with complete hippocampal circuit interruption, rather than rudiment transection, however, since connections from the rudiment and hippocampus in the proposed circuit (not the rudiment itself) were cut off.

Because the hippocampal rudiment is small, other structures (see Table 2) were also damaged when rudiment lesions were inflicted. The commonly-damaged incidental structures include cingulate cortex in the neighborhood of the hippocampal rudiment, cingulum, cerebral cortex, and corpus callosum. In the column to the far right in Table 2, structures lesioned less frequently are listed. Extent of the unintended lesions was inconsistent from animal to animal. Possible behavioral significance of the incidental damage will be discussed in detail in Chapter VI.

Lesions in animals with complete rudiment transections ranged approximately from slightly larger than one millimeter to two millimeters in their greatest extent. This lesion size was normal for animals in the present experiment, whether they were rudiment transected or not. Only in rat #69 (with rudiment connections interrupted) was the lesion somewhat larger than normal (between two and two and a half millimeters).

Behaviorally, animals with complete transection of the rudiment or complete interruption of the hippocampal circuit exhibited complete and long-standing failure in gaining (in the case of #86 and #87) or in recovering (in the case of #69 and #71) the alternation habit. Only animals having complete transection of the hippocampal rudiment or its connections failed to learn or relearn within the limits of the experiment.

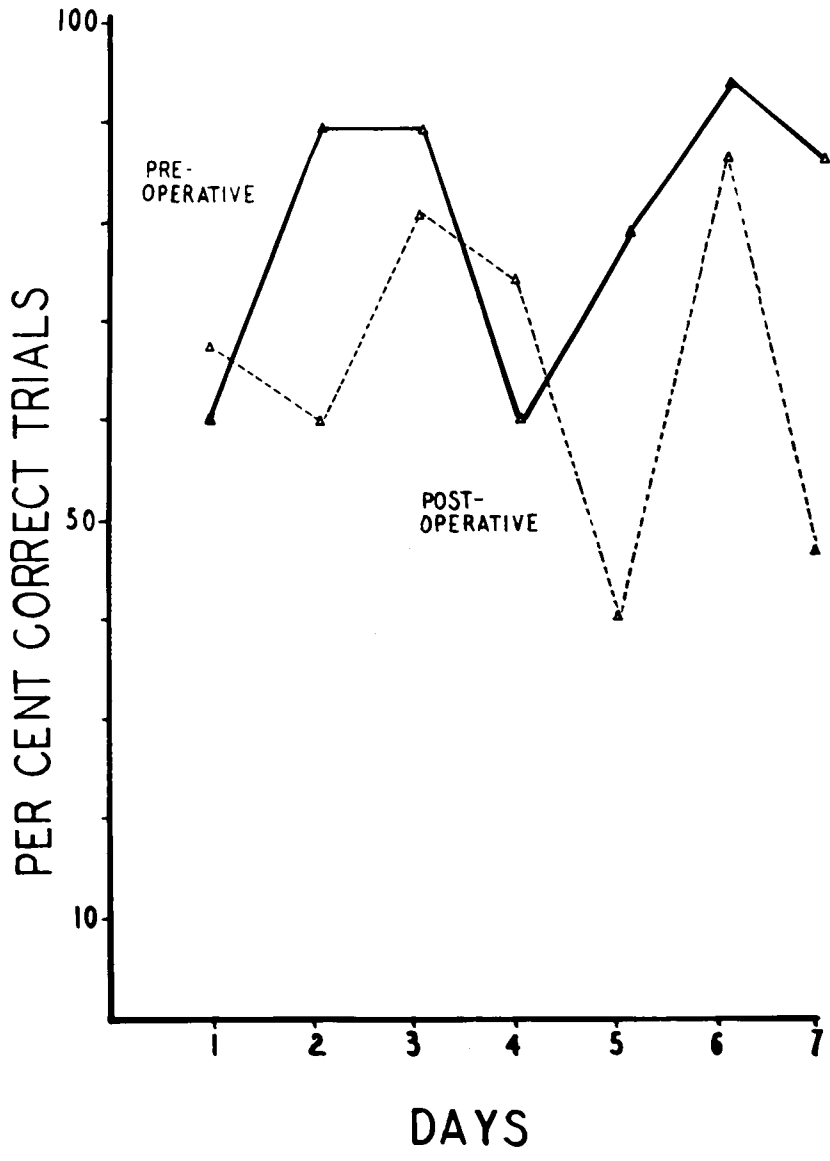
The all-or-none character of the ability to alternate approximately is clearly indicated by a comparison of performance in complete rudiment animals at the very end of training, with performance at the beginning. After complete transection of the hippocampal rudiment, rats for the study of learning (#86, #87) performed no better on the last three days of training (from postoperative trial 466 to 510) than rats normally do on the first three days of alternation training. On the last three postoperative training days rat #86 made correct turns in the maze 53, 47, and 47 per cent of the time while rat #87 made correct turns 73, 53, and 67 per cent of the time. Neither record shows evidence of learning, even though by this time the animals had received post-operative trials that exceeded the number needed by any animal without complete rudiment transection for reaching the learning criterion.

In retention also, rats with complete rudiment transection (#71) or

complete interruption of rudiment connections via the fornix (#69) showed consistently defective alternation performance. Before operation, these animals had reached the high learning criterion of at least forty-five successive trials, with ninety per cent correct trials or higher. Figure 4, which compares the performance of rat #71 on the last seven postoperative training days with the animal's own performance on the first seven counted training days before operation shows the striking character of the loss. Rat #69 exhibited a similar pattern, indicating complete loss. In interpreting Fig. 4 it should be kept in mind that the number of trials per day is not comparable on both graph lines. This is so, since the first three preoperative training days consisted of ten trials daily, followed by two days of twenty trials, then fifteen trials daily until the end of training. The postoperative graph depicts fifteen trials daily. It is noteworthy that by the time the final postoperative week began, rat #71 had already had many more retraining trials (the postoperative graph begins with trial 406) than he had needed to reach the high learning criterion before operation. The results indicate clearly that within the limits of the experiment, there was a total and long-standing loss of a once well-learned alternation habit. At the time training ended, no signs of improvement were evident.

Despite their poor performance in alternating approximately, rats having complete transections of the hippocampal rudiment were indistinguishable in other respects from normal animals that had the same number of alternation trials at the time of comparison. Like normal animals, rats with complete rudiment transections tugged and strained their entire bodies toward the maze when brought into the experimental room for a training session. Once they were

Fig. 4. Comparison of first days of training before operation with last days of re-training after operation, in experimental rat #71, with complete bilateral transection of the hippocampal rudiment.



placed in the maze, and the door to the starting box opened, rats with complete rudiment transections typically darted down the alley, sometimes stopping momentarily at the choice point.

In rat #69, with rudiment connections interrupted (because of interruption of the hippocampal circuit), the tugging and straining toward the maze were absent; but the animal showed normal interest once he was in the maze. For example, like normal animals he frequently scratched the door leading into the stem alley of the "T" between trials. In terms of number of correct trials, however, performance of this animal was defective; he showed no evidence of being able to relearn the maze task. In terms of the theory being tested, it is evident that rat #69 had lost memory of what he had done before, or motor memory.

Summarily, the only consistent way in which rats with complete rudiment transections or interruption of rudiment connection differed from other animals used in this experiment was that rats with those lesions failed to learn or relearn the alternation habit.

#### Partial transection of the hippocampal rudiment

In slight left-right misplacement of the electrode, resulting in partial or subtotal rudiment transection, the rudiment damage was sometimes bilateral (#72, #73, #81), but never completely severed the rudiment connections. Lesions in these animals showed inconsistent damage to neighboring structures, as was also true in animals with complete rudiment transections. Size of lesions was not notably distinguishable from that found in animals with complete rudiment transections.

It is noteworthy that animals in this partial transection group were able

without exception to gain proficiency in the alternation problem. (See Table 1) In fact, the all-or-none difference between the partial and complete transection groups is so marked that it prompted operating upon another group of animals (the late-operated animals) in the hope of accounting for the discrepancy. When the histological results came back, it was discovered that the only way in which animals that learned or relearned differed from those that did not learn or relearn was in completeness of the rudiment lesion. Those with complete transection failed without exception; those with incomplete transection succeeded without exception.

Performance of animals comprising the partial transection group did not differ significantly from that of normal unoperated animals. However, there was some tendency for partial rudiment operates to take longer than unoperated rats to learn or relearn, as Table 1 indicates. Two rats in particular (#81, #83), studied for retention, showed decidedly deficient performance, which requires some interpretation. When used originally as retention over time animals, these rats had showed no loss whatever of the alternation habit. But after operation they did very poorly in comparison with other animals having partial transection of the hippocampal rudiment. How account for the discrepancy?

At least two possibilities could be advanced. The animals with partial rudiment transection showing defective alternation performance differed from other subjects with partial rudiment injuries in: (1) being operated upon approximately six weeks later than the original animals and about one week after being retested for retention over time; and (2) being retrained, beginning on the fourth day after operation, instead of on the thirteenth

postoperative day, as had been the case for other operated animals. That the first possibility, age contributed to the defect seems unlikely. The animals were not much younger when tested for retention over time; yet then they performed well. The reason for impairment after operation, then, appears to lie with the second group of possibilities---with the subjects' being retrained soon after operation. While we cannot be sure that the effect of the operation is the reason for the relative slowness of #81 and #83, it is a distinct possibility.

#### Lesions in neighboring structures

All animals in this group learned or were able to relearn the alternation habit. Lesions were placed at the same antero-posterior level in the brain as was true for rats with rudiment transections. The electrode in this group of animals was angled in laterally, so that the hippocampal rudiment at the level of the motor cortex would be spared.

In most cases the lesions of animals in this group were more superficial than was true for rudiment operates. This was especially notable in rat #75 and rat #76. In one instance, however, the primary lesion went deeper in the brain (#67) than was the case with the rudiment operates, damaging the right ventral anterior thalamus. Tissue in the region of the hypothalamus in this animal appeared to be abnormal. Performance of this animal was irregular and inconsistent following operation though he eventually regained mastery of the alternation habit. Although the abnormal condition of the hypothalamic region complicates interpretation of the behavioral results, one thing is certain and unequivocal: the damage did not prevent relearning. In terms of the theory under test the relative slowness could be explained as owing to partial



interruption of neural impulses leading from the ventral anterior nucleus to the premotor and motor areas. With many of these impulses cut off, executing the specific movements made in the past would be rendered difficult.

#### Electrode track controls

The control animals who had electrodes introduced into the same area as the experimental animals but without having the current turned on revealed "no evidence of injury." In some cases slim lines left by electrodes as they course through the brain tissue were evident, as was illustrated in Fig. 3 (Ch. IV). All animals in the electrode control group learned or relearned the alternation habit. In two electrode track animals tested for learning mastery of the alternation habit took somewhat longer than was true of the typical learning animal before operation. A possible interpretation could be that any brain trauma hinders the recall of a task to be learned. However, the fact that one animal even before operation (#75) needed more trials to learn than either of the electrode track learning animals required suggests that performance of these rats lies within the normal range. The clear-cut and consistent finding in all these subjects is that without question, within the limits set by this experiment, they were able to learn or relearn the alternation habit.

#### Unoperated controls

Table 3 reports the behavioral results for animals tested for retention. The animals were retested twelve days after they had originally reached the learning criterion, a period comparable to the time elapsing between operation and retesting under some of the other treatments. The results indicate no impairment in the alternation habit. Many of the animals re-reached the

criterion in the least number of possible trials (i.e., 45 trials). Passage of time of the order studied here has no effect upon performance in the alternation task.

One clear and consistent result has issued from study of the records of animals used in this experiment. Animals in which the hippocampal rudiment was completely transected completely failed to learn or relearn the alternation habit. In contrast, damage to no other structure consistently led to failure in the alternation habit. How to account for the consistent defect in alternation found in animals having complete rudiment transections will be discussed systematically in the following chapter.

Table 3

Number of Learning and Retention Trials of Intact  
Animals Serving as Unoperated Controls

Animal Number	Number of Trials		Achieved Criterion
	Learning	Retention	
79	165	60	Yes
81 <sup>a</sup>	255	60	Yes
82 <sup>b</sup>	105	45	Yes
83 <sup>a</sup>	180	105	Yes
84	240	45	Yes
	<hr/>		
Ttl.	945	315	
Mean	189	63	Yes

<sup>a</sup>Also found in Table 2; received partial transection of hippocampal rudiment one week after being tested for retention of single alternation habit.

<sup>b</sup>Lesioned after tested for retention of single alternation habit but died two days postoperatively; hence not included in Table 2.

## Chapter VI

### Discussion, Implications, and Summary

The data of the present experiment supply evidence for the hypotheses tested. Complete bilateral transection of the hippocampal rudiment was consistently accompanied by complete loss of an alternation habit once learned and by complete failure to gain the habit for the first time. Consideration of alternative ways to account for the same experimental findings leads to the conclusion that the hippocampal rudiment is critically involved in the psychological function of motor recall. How study of the alternatives led to this conclusion will be the work of this chapter to explain.

#### Extent of damage

Some animals that showed no impairment in the alternation problem (#73, #74, #77, #78) sustained lesions as large as those of animals with complete rudiment transections. Since, however, all animals with complete rudiment transections manifested long-standing loss of the alternation habit while the other animals did not, extent of lesions, at least of the order found in the present experiment, cannot adequately account for poor performance in the alternation task.

#### Motivational and emotional anomalies

It might be objected that brain damage sustained in the experimental animals could have led to: (a) lack of desire to attain the known reward; (b) inability to coordinate activity to reach the goal because of disordered

emotion; or (c) both of the foregoing conditions. If this alternative view is correct, motor memory would not be affected directly; it would be affected, seemingly, because of lassitude or loss of other functions, leading to doing nothing in the maze.

How animals with complete rudiment transections acted in the maze situation, as compared with operated animals without such injuries, is sufficient to indicate that this argument lacks force. For one thing, rats with complete rudiment transections, all of which failed in the alternation task, gave every indication of wanting to reach the water reward. The fact that such animals ran directly to the goal testifies that the animals wanted water (and remembered it). Moreover, like normal animals, rudiment-transected rats gave evidence of wanting to hasten their entry into the maze at the beginning of a day (evidenced by straining toward the maze, behavior common among normal animals familiar with the situation and with what it leads to, the water reward). As soon as the starting gate of the maze opened, rats with complete rudiment transections characterically ran speedily (as is true of normal animals) to the dipper in the goal box. This behavior testifies that animals with complete rudiment transections (1) were able to coordinate their activity toward reaching the goal; and (2) showed normal interest in the situation.

The activity of animals with complete transection of the hippocampal rudiment was in fact indistinguishable from that of normal rats, except that the rudiment-transected animals failed to learn or relearn the action pattern set for obtaining the water reward. Therefore, in the absence of emotional and motivational anomalies, an explanation for poor alternation performance in

rats with complete rudiment transections must come from some other source.

### Physical condition

In weight, eating habits, and vitality exhibited during maze training and at feeding times, animals with complete bilateral transection of the hippocampal rudiment were like other operated rats. One might wonder, however, whether loss of sensory functions could have prevented the rudiment-transected animals from learning and retaining the alternation habit. Once again, both on behavioral grounds and in terms of effects of the operation, this possibility can be excluded.

The sensory modalities most relevant to solving the present T-maze alternation problem are vision and taste. Hearing is not directly relevant, since no systematic auditory cues are provided that could aid solution of the problem. The same is true of touch. Control measures (for preventing olfactory tracking) make the sense of smell irrelevant to the problem. Although, because of the controls employed, vision and taste could not lead to solution of the T-maze alternation, these sensory powers are, surely, useful in learning the T-maze problem. Might it be, then, that defective vision or taste could account for the poor performance of rudiment-transected subjects?

The behavior of the rudiment-transected rats easily leads to the inference that the rats were able to see, an ability rats undoubtedly make use of in learning an alternation habit when vision is available for use. The fact that when brought to the maze at the beginning of a day's training the animals frequently strained their bodies toward the open starting box also suggests that vision was intact. On logical grounds one might say that even if vision were lost, with other functions intact the subjects could be expected to show

some evidence of learning after 500 training trials. Although vision is useful for maze learning, it is not needed for learning an ordinary maze that does not directly test visual discrimination ability (Honzik, 1936).

None of the animals with complete rudiment transection gave evidence of loss of the sense of taste. The animals ran to the goal boxes as before, an indication that the sense of taste was not impaired.

It could be objected, perhaps, that the effect of brain damage of any kind could affect the animals adversely and lead to impaired performance. But if this were true, the animals with lesions in structures other than those of the hippocampal system should be affected equally. In fact, however, lesioned control rats gave no evidence of the all-or-none loss found in rats with complete bilateral rudiment transections, though in some cases the amount of tissue destruction was the same.

Thus, on the basis of the evidence, physical condition, including loss of sensory functions such as vision and taste, used in acquiring the alternation habit, seems inadequate to account for defective alternation performance.

#### Damage to structures other than the hippocampal rudiment

If lesions themselves cannot account for functional loss of alternation ability, perhaps site of lesions can. Since rats with complete transections of the hippocampal rudiment sustained damage to several other structures, too, it is reasonable to consider the possibility that incidental damage to unintended structures accounted for the alternation defect. Animals with complete transection of the hippocampal rudiment sustained damage to several other structures, though the damage was inconsistent from animal to animal: (a) cingulum; (b) cingulate region and cerebral cortex; (c) corpus callosum. In

the case of one subject with disturbed connections in the rudiment (on one side) and obliteration of the hippocampus (on one side) the fornix also suffered serious damage (rat #69).

Study of the lesions made in the inadvertently damaged structures in terms of their correspondence with performance in the alternation task will help to determine whether it is reasonable to maintain that such damage could account for the alternation defect.

(a) cingulum. Animals with complete transection of the hippocampal rudiment sustained slight to moderate damage to the cingulum at the level where the rudiment was transected. Such damage was subtotal and inconsistent from animal to animal (e.g., the cingulum was damaged primarily in one hemisphere in rat #71 and rat #87, while partial damage in both hemispheres was evident in rat #86). Animals showing no defect in alternation, however (#74, #77), also sustained damage to the cingulum at least as extensive and at the same level as was evidenced in animals with complete rudiment transections. Since damage to the cingulum at the same level of the brain was common both to animals with complete rudiment transections and to other operated animals (which showed no behavioral defect), cingulum injury of the kind and extent manifest in the present investigation cannot account for the behavioral defect.

(b) cingulate and cerebral cortex. In its greatest extent damage to cingulate cortex in animals with complete rudiment transections involved areas 23 and 29b moderately, and area 29c to a slight degree. Damage to the cerebral cortex at this level was evident in rat #86. These same areas, however, were damaged at least as extensively and at approximately the same level in two other subjects (#77, primarily unilaterally, and #74, bilaterally). Yet these



animals showed normal performance. Thus, injury to these brain regions, at least to the extent and of the type manifest in the present subjects, was not critical for the alternation habit.

(c) corpus callosum. At least two animals (#88, #78) sustained injury to the corpus callosum at approximately the same anterior-posterior level and at least as great in extent as animals with complete rudiment transection sustained. In rat #78 the lesion in the corpus callosum was made about a millimeter to one side of the midline, with superficial cerebral cortex damage on both sides where the electrode was angled in. In rat #88 the lesion in the corpus callosum extended to the region of the midline and involved part of the hippocampal rudiment. Since these animals showed no defect in alternation performance, corpus callosum injuries at this level do not appear critical for alternation performance that requires motor recall.

(d) hippocampus. In one animal (#69) placed in the complete interruption group because of disruption of the part of the proposed circuit carrying fibers from the fornix back to the cortical association areas, the hippocampus suffered very heavy damage on one side. In two other animals (#72, #73) that lacked the very severe fornix injury but sustained substantial damage to one side of the hippocampus, relearning occurred within normal limits. Thus, damage to the hippocampus on one side appears incapable of accounting for the complete functional loss. The interpretation, that the loss could be attributed to interruption of the fornix fibers (hence part of the memory circuit carrying fibers serving all modalities) stands as more plausible.

On the strength of the evidence found in the present investigation, it can be concluded that damage to incidentally involved structures, of the kind and

extent found in this study, cannot account for defective performance in animals with complete rudiment transections.

#### Complete transection of the hippocampal rudiment

Since the alternative proposals just considered cannot satisfactorily account for the consistent defect in alternation performance found in animals with complete transections of the hippocampal rudiment, the process of elimination leads to the hippocampal rudiment itself. Here the distinction between subtotal and total rudiment transection is seen as extremely important, for it is the only evident lesion difference that demarcates animals that learned or relearned the alternation habit from those that did not. That animals with unilateral or subtotal damage to the hippocampal rudiment were able to learn or relearn the alternation problem seems to indicate that motor recall can function as long as some connecting fibers are intact, even if their total is reduced.

It is true, of course, that in the present experiment only a few subjects sustained total bilateral transections of the hippocampal rudiment, while more rudiment-damaged animals sustained subtotal injuries to the rudiment. The results, though, seem to indicate an all-or-none quality to the rudiment lesions. Either the connection is completely interrupted---in which case no evidence of recall or retention of the alternation habit is shown; or the connection persists with a reduced number of fibers---then recall is possible. With a clear and consistent behavioral difference corresponding to complete or incomplete rudiment transection, even a few cases tellingly depict a clear-but relationship.

Thus, in the presence of confirming evidence (marked alternation defect

in animals having complete transection of the hippocampal rudiment) and in the absence of counter evidence (based on the other alternative explanations considered in this chapter), the hypotheses expressed in Chapter II merit unequivocal support from the data of the present experiment. The hippocampal rudiment does seem to be a critical structure for both learning and retention of an alternation habit. Additional studies would be needed, of course, to determine more precisely the import of the interrupted circuit.

To what do the results of this investigation lead? At the time of writing several investigators are testing other aspects of the general hypothesis, that the hippocampal system is needed for memory in the various modalities. Whether transection of the hippocampal rudiment at the level of the somatosensory cortex will prevent learning and retention of a tactual discrimination; whether visual and auditory learning will be eliminated by lesions that transect the fornix---these questions, arising from the formulation tested in the present study, are being asked today.

A specific line of further investigations could follow directly from the present study of motor recall. Would the alternation habit be lost if lesions were placed at the level of the rudiment further forward in the brain? If the hippocampal rudiment were completely transected at the level of the motor cortex, would the memory loss extend to other modalities? With appropriate tasks for testing memory functions in the different modalities, after placing lesions at strategic points, it should be possible to gain unequivocal answers to such questions.

Starting from the same formulation, other studies could be suggested to investigate the related function of imagination. Whether damage to the

structures proposed for imagination leads to loss of that function could be determined. Would motor memory also be prevented by damage that results in loss of motor imagination? Would motor memory be impaired? Starting from the present formulation the possibilities for extending research are as numerous as they are intriguing.

In discovering an answer to the question, whether rats lose or do not lose ability to perform an alternation task after complete transection of the hippocampal rudiment, the present study has fulfilled its aim. By clarifying what part the hippocampal rudiment at one level of the brain plays in the psychological function of motor recall, this investigation has contributed to a worthwhile quest: to the search for structural requisites for the psychological function of motor recall.

It may well be that further investigations of the kinds suggested here will lead to the sort of positive results, that happy harbor research workers hope for. But whether all the answers be affirmative or not, the persistent following of promising leads cannot but end in promise fulfilled. That the answer to a question put to nature be "yes" or "no" is far less important than is discovery of where the truth in the matter lies.

#### Summary

The present investigation tested one phase of an hypothesis proposed by Arnold (1960), that the hippocampal system (including hippocampus and hippocampal rudiment) serves memory in the various modalities. Specifically, this experiment was designed to discover whether or not lesions in the hippocampal rudiment at the motor level in the brain interfere with learning and retention of single alternation habit, which needs for mastery the function

of motor recall.

To test this hypothesis, twenty-five albino rats were divided into groups, some for the study of learning and others for the study of retention of a single alternation problem. Learning animals had their first training in the alternation problem after surgery. Rats studied for retention acquired the alternation habit before operation. All animals were trained in the same fashion but were subjected to different treatments, so that both learning and retention of the alternation habit could be tested unambiguously.

Separate groups of animals were subjected to the following treatments:

(1) complete bilateral transection of the hippocampal rudiment; (2) partial or subtotal transection of the hippocampal rudiment; (3) lesions in neighboring structures; (4) electrode track controls (in which the electrode was introduced but current not turned on); and (5) no injury (studied for retention over time). Four animals were operated upon six weeks after the original groups of subjects, with the aim of accounting for discrepant behavioral findings already discovered in rats presumed to have complete rudiment transections.

The results indicate that animals with complete bilateral transections of the hippocampal rudiment did not learn or relearn the alternation problem, even after having many more trials than normal rats require. An animal with probable complete interruption of connections of the proposed memory circuit (rat #69) also showed complete and longstanding loss, which in terms of the theory under test would be predicted on the basis of the damage. In contrast, all other animals, whether with partial rudiment transections, lesions in other structures, or no lesions, were able to learn or relearn the alternation problem within the number of trials allowed. Reasons for temporary loss of

alternation ability in the cases of several animals with incomplete rudiment transections were advanced.

Several solutions were proposed to explain the experimental evidence which could not satisfactorily account for the alternation defect following complete transection of the hippocampal rudiment. The tested hypothesis, however, could adequately account for the experimental results. Thus, the tested hypothesis receives confirmation from the present experimental results: complete transection of the hippocampal rudiment at the level of the motor cortex does seem to prevent learning and retention of a single alternation habit. It will remain for further experimentation to determine in detail the import of the interrupted circuit.

The present study could lead to further related investigations that show promise for increasing our knowledge and understanding of the structures needed for learning and retention, as well as of the related function of imagination.

## Chapter VII

### References

Addison, W. H. F. On the rhinencephalon of Delphinus delphis. J. comp. Neurol., 1915, 25, 497-522.

Ades, H. W., & Raab, D. H. Effect of preoccipital and temporal decortication on learned visual discrimination in monkeys. J. Neurophysiol., 1959, 12, 101-108.

Adey, W. R. Studies of hippocampal electrical activity during approach learning. In J. F. Delafresnaye (Ed.), Brain mechanisms and learning. Oxford: Blackwell, 1961, Pp. 577-588.

Adey, W. R., Sunderland, S., & Dunlop, C. W. The entorhinal area: electrophysiological studies of its interrelations with rhinencephalic structures and the brain stem. EEG clin. Neurophysiol., 1957, 9, 309-324.

Akert, K., & Andy, O. J. Experimental studies on Ammon's formation in cats and monkeys. Trans. Amer. Neurol. Assn., 1953, 78, 194-195.

Allen, W. F. Effect of ablating the frontal lobes, hippocampi, and occipito-parieto-temporal (excepting pyriform areas) lobes on positive and negative olfactory conditioned reflexes. Amer. J. Physiol., 1940, 128, 754-771.

Allen, W. F. Effect of ablating the pyriform-amygdaloid area and hippocampi on positive and negative olfactory conditioned reflexes and on conditioned olfactory differentiation. Amer. J. Physiol., 1941, 132, 81-92.

Allen, W. F. Effect of prefrontal brain lesions on correct conditioned differential responses in dogs. Amer. J. Physiol., 1949, 159, 525-532.

Arnold, Magda B. Emotion and personality. New York: Columbia Univ. Press, 2 vols., 1960.

Barris, R. W., & Schuman, H. R. Bilateral anterior cingulate gyrus lesions: syndrome of the anterior cingulate gyri. Neurology, 1953, 3, 44-52.

Barry, H. Habituation to handling as a factor in retention of maze performance in rats. J. comp. physiol. Psychol., 1957, 50, 366-367.

Battig, K., Rosvold, H., & Mishkin, M. Comparison of the effects of frontal and caudate lesions on delayed response and alternation in monkeys. J. comp. physiol. Psychol., 1960, 53, 400-404.

Battig, K., Rosvold, H., & Mishkin, M. Comparison of the effects of frontal and caudate lesions on discrimination learning in monkeys. J. comp. physiol. Psychol., 1962, 55, 458-463.

Boring, E. G. History of experimental psychology. (2nd ed.) New York: Appleton-Century-Crofts, 1950.

Boyle, J., & Nielsen, J. M. Visual agnosia and loss of recall. Bull. Los Angeles Neurol. Soc., 1954, 19, 39-42.

Brady, J. V. Emotional behavior. In J. Field, H. W. Magoun, & V. E. Hall (Eds.), Handbook of physiology. Baltimore: American Physiological Society, Waverly Press, 1960. Sect. I, 3, Pp. 1529-1552.

Brady, J. V., & Hunt, H. F. An experimental approach to the analysis of emotional behavior. J. Psychol., 1955, 40, 313-324.

Brady, J. V., & Nauta, W. J. H. Subcortical mechanisms in emotional behavior: affective changes following septal forebrain lesions in the albino rat. J. comp. physiol. Psychol., 1953, 46, 339-346.

Brady, J. V., Schreiner, L., Geller, I., & Kling, A. Subcortical mechanisms in emotional behavior: the effect of rhinencephalic injury upon the acquisition and retention of a conditioned avoidance response in cats. J. comp. physiol. Psychol., 1954, 47, 179-186.

Brazier, M. A. B. (Ed.) Brain and behavior. Proc. 1st Conf. brain and behavior, 1, Washington: American Institute of Biological Sciences, 1961.

Brodal, A. The hippocampus and the sense of smell: a review. Brain, 1947, 70, 179-222.



Brown, S., & Schäfer, E. A. An investigation into the functions of the occipital and temporal lobes of the monkey's brain. Philos. Trans. Roy. Soc. (London), 1888, 179, 303-327.

Brush, E. S., Mishkin, M., & Rosvold, H. E. Effects of object preferences and aversions on discrimination learning in monkeys with frontal lesions. J. comp. physiol. Psychol., 1961, 54, 319-325.

Bures, J., & Buresova, O. The use of Leao's spreading depression in the study of interhemispheric transfer of memory traces. J. comp. physiol. Psychol., 1960, 53, 558-563.

Buser, P., & Rougeul, A. Somatic functions of the nervous system. In Annu. Rev. Physiol., 1961, 23, 387-418.

Campbell, R. J., & Harlow, H. F. Problem solution by monkeys following removal of the prefrontal areas. V. Spatial delayed reactions. J. exp. Psychol., 1945, 35, 110-126.

Chow, K. L. Lack of behavioral effects following destruction of some thalamic association nuclei in monkey. A. M. A. Arch. Neurol. Psychiat., 1954, 71, 762-771.

Chow, K. L. Brain functions. In Annu. Rev. Psychol., 1961, 12, 281-310.

Chow, K. L., & Hutt, P. J. The "association cortex" of *Macaca mulatta*: a review of recent contributions to its anatomy and functions. Brain, 1953, 76, 625-677.

Clark, W. E. L. The structure and connections of the thalamus. Brain, 1932, 55, 406-470.

Clark, W. E. L. Sensory experience and brain structure. J. ment. Sci., 1958, 104, 1-13.

Correll, R. E. The effect of bilateral hippocampal stimulation on the acquisition and extinction of an instrumental response. J. comp. physiol. Psychol., 1957, 50, 624-629.

Correll, R. E., & Scoville, W. B. Effects of medial temporal lesions on delayed reactions. Amer. Psychologist, 1960, 15, 485. (Abstract)

Craigie, E. H. An introduction to the finer anatomy of the central nervous system based upon that of the albino rat. Philadelphia: Blakiston's Son, 1925.

Crosby, E. C., Humphrey, T., & Lauer, E. W. Correlative anatomy of the nervous system. New York: Macmillan, 1962.

Dell, P., & Bonvallet, M. Somatic functions of the nervous system. In Annu. Rev. Physiol., 1956, 18, 309-338.

Deutsch, J. A. Higher nervous function: the physiological bases of memory. In Annu. Rev. Physiol., 1962, 24, 259-286.

Doty, R. W., Beck, E. C., & Kooi, K. A. Effect of brainstem lesions on conditioned responses in cats. Exp. Neurol., 1959, 1, 360-385.

Ebner, F. F., & Myers, R. E. Inter- and intra-hemispheric transmission of tactile gnosis in normal and corpus callosum-sectioned monkeys. Federation Proc., 19, 1960, 292. (Abstract)

Ellen, P., & Powell, E. W. Temporal discrimination in rats with rhinencephalic lesions. Exp. Neurol., 1962, 6, 538-547.

Elliot Smith, G. Some problems relating to the evolution of the brain. Lancet, 1910, 178, 1-6; 147-153; 221-227.

Ettlinger, G., & Wegener, J. Somaesthetic alternation, discrimination and orientation after frontal and parietal lesions in monkeys. Quart. J. exp. Psychol., 1958, 10, 177-186.

Fagot, H. J. Effects of lesions in the hippocampal rudiment on conditioned olfactory discrimination in the albino rat. Unpublished doctoral dissertation, Loyola Univer., 1962.

Flynn, J. P., & Wasman, M. Learning and cortically evoked movements during propagated hippocampal discharges. Science, 1960, 131, 1607-1608.

Forgays, D. G. Reversible disturbances of function in rats following cortical insult. J. comp. physiol. Psychol., 1952, 45, 216-225.

Franz, S. I. On the functions of the cerebrum: I.-- The frontal lobes in relation to the production and retention of simple sensory-motor habits. Amer. J. Physiol., 1902, 8, 1-22.

Franz, S. I. On the function of the cerebrum: the frontal lobes. Arch. Psychol., 1907, No. 2, 1-64.

Fuller, J. L., Rosvold, H. E., & Pribram, K. H. The effect on affective and cognitive behavior in the dog of lesions in the pyriform-amygdala-hippocampal complex. J. comp. physiol. Psychol., 1957, 50, 89-96.

Fulton, J. F., & Ingraham, F. D. Emotional disturbances following experimental lesions of the base of the brain. J. Physiol., 1929, 67, 27-28.

Galambos, R., & Morgan, C. T. The neural basis of learning. In J. Field, H. W. Magoun, & V. E. Hall (Eds.), Handbook of physiology. Baltimore: American Physiological Society, Waverly Press, 1960. Sect. I, 3, 1471-1499.

Garcia-Bengochea, F., Corrigan, R., Morgane, P., Russell, D., & Heath, R. G. Studies on the function of the temporal lobes. I. The section of the fornix. Trans. Amer. Neurol. Assn., 1951, 76, 238-239.

Garcia-Bengochea, F., De La Torre, O., Esquivel, O., Vieta, R., & Fernandez, C. The section of the fornix in the surgical treatment of certain epilepsies. Trans. Amer. Neurol. Assn., 1954, 79, 176-178.

Gastaut, H. Corrélations entre le système nerveux végétatif et le système de la vie de relation dans le rhinencéphale. J. Physiol. (Paris), 1952, 44, 431-470.

Ghent, L., Mishkin, M., & Teuber, H. L. Short-term memory after frontal lobe injury in man. J. comp. physiol. Psychol., 1962, 55, 705-709.

Glees, P., Cole, J., Whitty, M., & Cairns, H. The effects of lesions in the cingular gyrus and adjacent areas in monkeys. J. Neurol. Neurosurg. Psychiat., 1950, 13, 178-190.

Glees, P., & Griffith, H. B. Bilateral destruction of the hippocampus (cornu ammonis) in a case of dementia. Mschr. Psychiat. Neurol., 1952, 123, 193-204.

Green, J. D. The hippocampus. In J. Field, H. W. Magoun, & V. E. Hall (Eds.), Handbook of physiology. Baltimore: American Physiological Society, Waverly Press, 1960. Sect. I, 2, 1373-1389.

Green, J. D., & Adey, W. R. Afferent projections to the hippocampus of the cat and monkey. Amer. J. Physiol., 179, 640-641.

Green, J. D., & Arduini, A. Changes induced in hippocampal activity induced by afferent stimulation. EEG clin. Neurophysiol., 1953, 5, 473. (Abstract)

Grünthal, E. Über das klinische Bild nach umschriebenem beiderseitigen Ausfall der Ammonshornrinde. Mschr. Psychiat. Neurol., 1947, 113, 1-16.

Harrison, J. M., & Lyon, M. The role of the septal nuclei and components of the fornix in the behavior of the rat. J. comp. Neurol., 1957, 108, 121-137.

Herrick, C. J. The functions of the olfactory parts of the cerebral cortex. Proc. Nat. Acad. Sci., 1933, 19, 7-14.

Honzik, C. H. The sensory basis of maze learning in rats. Comp. Psychol. Monogr., 1936, 13, No. 4 (Whole No. 44).

Isaacson, R. I., Douglas, R. J., & Moore, R. Y. The effect of radical hippocampal ablation on acquisition of avoidance response. J. comp. physiol. Psychol., 1961, 54, 625-628.

Jacobsen, C. F. The functions of the frontal association areas in monkeys. Comp. Psychol. Monogr., 1936, 13, No. 3, 3-60.

Jacobsen, C. F., & Nissen, H. W. Studies of cerebral function in primates. IV. The effect of frontal lobe lesions on the delayed alternation habit in monkeys. J. comp. Psychol., 1937, 23, 101-112.

Jasper, H., Gloor, P., & Milner, B. Higher functions of the nervous system. In Annu. Rev. Physiol., 1956, 18, 359-386.

Jasper, H. H., Procter, L. D., Knighton, R. S., Noshay, W. C., Costello, R. T. (Eds.) Reticular formation of the brain. Boston: Little, Browne, 1958.

Kaada, B. R. Somato-motor, autonomic and electrocorticographic responses to electrical stimulation of "rhinencephalic" and other structures in primate, cat and dog. Acta Physiol. Scand., 1951, 24, Supp. 83.

Kaada, B. R. Cingulate, posterior orbital, anterior insular and temporal pole cortex. In J. Field, H. W. Magoun, & V. E. Hall (Eds.), Handbook of physiology. Baltimore: American Physiological Society, Waverly Press, 1960. Sect. I, 2, 1345-1372.

Kaada, B. R., Rasmussen, E. W., & Kveim, O. Effects of hippocampal lesions on maze learning and retention in rats. Exp. Neurol., 1961, 3, 333-355.

Kaada, B. R., Rasmussen, E. W., & Kveim, O. Impaired acquisition of passive avoidance behavior by subcallosal, septal, hypothalamic, and insular lesions in rats. J. comp. physiol. Psychol., 1962, 55, 661-670.

Kennard, M. A. Effect of bilateral ablation of cingulate area on behavior of cats. J. Neurophysiol., 1955, 18, 159-169.

Kim, C., & Kim, C. U. Effect of hippocampal ablation on audiogenic seizure in rats. J. comp. physiol. Psychol., 1962, 55, 288-292.

Kimble, D. P., & Pribram, K. H. Hippocampectomy and behavior sequences. Science, 1963, 139, 824-825.

Kimura, D. Effects of selective hippocampal damage and avoidance behavior in the rat. Canad. J. Psychol., 1958, 12, 213-218.

- Kling, A., Orbach, J., Schwartz, N. B., & Towne, J. C. Injury to the limbic system and associated structures in cats. A. M. A. Arch. gen. Psychiat., 1960, 3, 391-420.
- Klüver, H. Brain mechanisms and behavior with special reference to the rhinencephalon. J. Lancet, 1952, 72, 567-574.
- Klüver, H., & Bucy, P. S. An analysis of certain effects of bilateral temporal lobectomy in the Rhesus monkey with special reference to "psychic blindness". J. Psychol., 1938, 5, 33-54.
- Klüver, H., & Bucy, P. C. Preliminary analysis of functions of the temporal lobes in monkeys. Arch. Neurol. Psychiat., 1939, 42, 979-1000.
- Konorski, J. Changing concepts concerning physiological mechanisms of animal motor behaviour. Brain, 1962, 85, Part II, 277-294.
- Krieg, W. J. S. Accurate placement of minute lesions in the brain of the albino rat. Quart. Bull. Northwestern Univer. Med. School, 1946, 20, 199-208.
- Krieg, W. J. S. Connections of the cerebral cortex. I. The albino rat. A. Topography of the cortical areas. J. comp. Neurol., 1946, 84, 221-275.
- Krieg, W. J. S. Connections of the cerebral cortex I. The albino rat. C. Extrinsic connections. J. comp. Neurol., 1947, 86, 267-394.
- Krieg, W. J. S. Functional neuroanatomy. (2nd ed.) New York: Blakiston, 1950.
- Krieg, W. J. S. Brain mechanisms in diachrome. (2nd ed.) Evanston, Ill.: Brain Books, 1957.
- Lashley, K. S. Brain mechanisms and intelligence. Chicago: Univer. Chicago Press, 1929.
- Lashley, K. S. Mass action in cerebral function. Science, 1931, 73, 245-254.
- Lashley, K. S. Studies of cerebral function in learning. XII. Loss of the maze habit after occipital lesions in blind rats. J. comp. Neurol., 1943, 79, 431-462.

- Lashley, K. S. Studies of cerebral function in learning. XIII. Apparent absence of transcortical association in maze learning. J. comp. Neurol., 1944, 80, 257-281.
- Lashley, K. S., McDonald, W. T., & Peters, H. N. Studies of cerebral function in learning. X. The effects of dilatation of the ventricles upon maze learning. Amer. J. Physiol., 1933, 104, 51-61.
- Lindsley, D. B. Higher functions of the central nervous system. In Annu. Rev. Physiol., 1955, 17, 311-338.
- Lindsley, D. B., Schreiner, L. H., Knowles, W. B., & Magoun, H. W. Behavioral and EEG changes following chronic brain stem lesions in the cat. EEG clin. Neurophysiol., 1950, 2, 483-498.
- McCleary, R. A. Response specificity in the behavioral effects of limbic system lesions in the cat. J. comp. physiol. Psychol., 1961, 54, 605-613.
- McLardy, T. The hippocampal formation of the brain as detector-coder of temporal patterns of information. Perspectives Biol. Med., 1959, 2, 443-452.
- MacLean, P. D. Psychosomatic disease and the "visceral brain": recent developments bearing on Papez theory of emotion. Psychosom. Med., 1949, 11, 338-353.
- MacLean, P. D. Neurophysiologic symposium: limbic system and its hippocampal formation in animals and their possible application to man. J. Neurosurg., 1954, 11, 29-44.
- MacLean, P. D. Hippocampal studies: their possible bearing on neuropsychiatry. A. M. A. Arch. Neurol. Psychiat., 1955, 74, 219-220. (Abstract and discussion)
- MacLean, P. D. The limbic system ("visceral brain") and emotional behavior. A. M. A. Arch. Neurol. Psychiat., 1955, 73, 130-134.
- MacLean, P. D. The limbic system ("visceral brain") in relation to central gray and reticulum of the brain stem; evidence of interdependence in emotional processes. Psychosom. Med., 1955, 17, 355-366.

- MacLean, P. D. Contrasting functions of limbic and neocortical systems of the brain and their relevance to psychophysiological aspects of medicine. Amer. J. Med., 1958, 25, 611-626.
- MacLean, P. D. The limbic system and two life principles. In The central nervous system and behavior. Transactions of the 2nd conference Josiah Macy, Jr., Foundation, 1959, Pp. 31-118.
- MacLean, P. D., Flanigan, S., Flynn, J. P., Kim, C., & Stevens, J. R. Hippocampal function: tentative correlations of conditioning, EEG, drug, and radioautographic studies. Yale J. Biol. Med., 1955-56, 28, 380-395.
- McNemar, Q. Psychological statistics. (2nd ed.) New York: Wiley, 1955.
- Maher, B. A., Elder, S. T., & Noblin, C. D. A differential investigation of avoidance reduction versus hypermotility following frontal ablation. J. comp. physiol. Psychol., 1962, 55, 449-454.
- Maier, N. R. F. Cortical destruction in the posterior part of the brain and its effect on reasoning in rats. J. comp. Neurol., 1932, 56, 179-214.
- Maier, N. R. F. The effect of cerebral destruction on reasoning and learning in rats. J. comp. Neurol., 1932, 54, 45-75.
- Malmo, R. B. Activation: a neuropsychological dimension. Psychol. Rev., 1959, 66, 367-386.
- Manter, J. T. Essentials of clinical neuroanatomy and neurophysiology. Philadelphia: F. A. Davis, 1959.
- Meyer, V., & Yates, A. J. Intellectual changes following temporal lobectomy for psychomotor epilepsy. J. Neurol. Neurosurg. Psychiat., 1955, 18, 44-52.
- Milner, B. Intellectual function of the temporal lobes. Psychol. Bull., 1954, 54, 42-62.
- Mishkin, M. Visual discrimination performance following partial ablations of the temporal lobe. II. Ventral surface vs. hippocampus. J. comp. physiol. Psychol., 1954, 47, 187-193.



Mishkin, M., & Pribram, K. H. Visual discrimination performance following partial ablations of the temporal lobe. I. Ventral vs. lateral. J. comp. physiol. Psychol., 1954, 47, 14-20.

Mishkin, M., & Pribram, K. H. Analysis of the effects of frontal lesions in monkeys. I. variations of delayed alternations. J. comp. physiol. Psychol., 1955, 48, 492-495.

Mishkin, M., & Pribram, K. H. Analysis of the effects of frontal lesions in monkeys. II. Variations of delayed response. J. comp. physiol. Psychol., 1956, 49, 36-40.

Mishkin, M., & Weiskrantz, L. Effects of delayed reward on visual-discrimination performance in monkeys with frontal lesions. J. comp. physiol. Psychol., 1958, 51, 276-281.

Morgan, C. T., & Stellar, E. Physiological psychology. New York: McGraw-Hill, 1950.

Moore, R. Y. Effects of some rhinencephalic lesions on retention of conditioned behavior in the cat. Unpublished doctoral dissertation, Chicago Univer., 1962.

Mowrer, O. H. Learning theory and behavior. New York: Wiley, 1960.

Munn, N. L. An introduction to animal psychology: the behavior of the rat. Boston: Houghton Mifflin, 1933.

Munn, N. L. Handbook of psychological research on the rat. Boston: Houghton Mifflin, 1950.

Myers, R. E. Localization of function in the corpus callosum. A. M. A. Arch. Neurol., 1959, 1, 74-86.

Nathan, P. W., & Smith, M. C. Normal mentality associated with maldeveloped "rhinencephalon". J. Neurol. Neurosurg. Psychiat., 1950, 13, 191-197.

Nauta, W. J. H. An experimental study of the fornix system in the rat. J. comp. Neurol., 1956, 104, 247-270.

Nielsen, J. M. The ideational motor plan. J. Nerv. Ment. Dis., 1948, 108, 361.

- Nielsen, J. M. Anterior cingulate gyrus and corpus callosum. Trans. Amer. Neurol. Assn., 1951, 76, 183-187.
- Nielsen, J. M. Memory and amnesia. Los Angeles: San Lucas Press, 1958.
- Orbach, J. Immediate and chronic disturbances on the delayed response following transections of the frontal granular cortex in the monkey. J. comp. physiol. Psychol., 1956, 49, 46-51.
- Orbach, J., & Fantz, R. L. Differential effects of temporal neo-cortical resections on overtrained and non-overtrained visual habits in monkeys. J. comp. physiol. Psychol., 1958, 51, 126-129.
- Orbach, J., Milner, B., & Rasmussen, T. Learning and retention in monkeys after amygdala-hippocampal resection. Arch. Neurol., 1960, 3, 230-251.
- Papez, J. W. A proposed mechanism of emotion. Arch. Neurol. Psychiat., 1937, 38, 725-743.
- Papez, J. W. Visceral brain, its component parts and their connections. J. nerv. ment. Dis., 1958, 126, 40-56.
- Pasik, P., Pasik, T., Battersby, W. S., & Bender, M. B. Visual and tactual discriminations by Macaques with serial temporal and parietal lesions. J. comp. physiol. Psychol., 1958, 51, 427-436.
- Peele, T. L. The neuroanatomical basis for clinical neurology. (2nd ed.) New York: Blakiston, 1961.
- Penfield, W. Memory mechanisms. Arch. Neurol. Psychiat., 1952, 67, 178-191.
- Penfield, W. The interpretive cortex. Science, 1959, 129, 1719-1725.
- Penfield, W., & Milner, B. Memory deficit produced by bilateral lesions in the hippocampal zone. A. M. A. Arch. Neurol. Psychiat., 1958, 79, 475-497.
- Feretz, E. The effects of lesions of the anterior cingulate cortex on the behavior of the rat. J. comp. physiol. Psychol., 1960, 53, 540-548.

- Peters, R. H., Rosvold, H. E., & Missky, A. F. The effect of thalamic lesions upon delayed response-type tests in the rhesus monkey. J. comp. physiol. Psychol., 1956, 49, 111-116.
- Petrinovich, L., & Bolles, R. Delayed alternation: evidence for symbolic processes in the rat. J. comp. physiol. Psychol., 1957, 50, 363-365.
- Pribram, K. H. Concerning three rhinencephalic systems. Electroencephalog. clin. Neurophysiol., 1954, 6, 708-709.
- Pribram, K. H. A review of theory in physiological psychology. In Annu. Rev. Psychol., 1960, 11, 1-40.
- Pribram, K. H. A further experimental analysis of the behavioral deficit that follows injury to the primate frontal cortex. Exp. Neurol., 1961, 3, 432-466.
- Pribram, K. H., & Bagshaw, M. Further analysis of the temporal lobe syndrome utilizing fronto-temporal ablations. J. comp. Neurol., 1953, 92, 347-375.
- Pribram, K. H., & Fulton, J. F. An experimental critique of the effects of anterior cingulate ablations in monkey. Brain, 1954, 77, 34-44.
- Pribram, K. H., & Kruger, L. Functions of the "olfactory brain". Ann. N. Y. Acad. Sci., 1954, 58, 109-138.
- Pribram, K. H., & Mishkin, M. Analysis of the effects of frontal lesions in monkey. III. Object alternation. J. comp. physiol. Psychol., 1956, 49, 41-45.
- Pribram, K. H., Mishkin, M., Rosvold, H. E., & Kaplan, S. J. Effects on delayed-response performance of lesions of dorsolateral and ventromedial frontal cortex of baboons. J. comp. physiol. Psychol., 1952, 45, 565-575.
- Pribram, K. H., & Weiskrantz, L. A comparison of the effects of medial and lateral cerebral resections on conditioned avoidance behavior of monkeys. J. comp. physiol. Psychol., 1957, 50, 74-80.
- Pribram, K. H., Wilson, W. A., & Connors, J. Effects of lesions of the medial forebrain on alternation behavior of rhesus monkeys. Exp. Neurol., 1962, 6, 36-47.

Roberts, W. W., Dember, W. N., & Brodwick, M. Alternation and exploration in rats with hippocampal lesions. J. comp. physiol. Psychol., 1962, 55, 695-700.

Rollin, A. R. Mass action effects in learning. J. Psychol., 1955, 39, 301-319.

Rosner, B. S. Effects of cortical lesions on maze retention by the rat. J. comp. physiol. Psychol., 1953, 46, 56-60.

Rosvold, H. E. Physiological psychology. In Annu. Rev. Psychol., 1959, 10, 415-454.

Rosvold, H. E., & Delgado, J. M. R. The effect on delayed-alternation test performance of stimulating or destroying electrically structures within the frontal lobes of the monkey's brain. J. comp. physiol. Psychol., 1956, 49, 365-372.

Rosvold, H. E., & Mishkin, M. Non-sensory effects of frontal lesions on discrimination learning and performance. In J. F. Delafresnaye (Ed.), Brain mechanisms and learning. Oxford: Blackwell, 1961, Pp. 555-576.

Rosvold, H. E., Mishkin, M., & Szwarcbart, M. K. Effects of subcortical lesions in monkeys on visual-discrimination and single-alternation performance. J. comp. physiol. Psychol., 1958, 51, 437-444.

Rosvold, H. E., Szwarcbart, M. K., Mirsky, A. F., & Mishkin, M. The effect of frontal-lobe damage on delayed-response performance in chimpanzees. J. comp. physiol. Psychol., 1961, 54, 368-374.

Rusinov, V. S., & Rabinovich, M. Y. Electroencephalographic researches in the laboratories and clinics of the Soviet Union. Electroencephalog. clin. Neurophysiol., 1958, Suppl. 8.

Russell, R. Brain, Memory, Learning. Oxford: Clarendon Press, 1959.

Schreiner, L., & Kling, A. Behavioral changes following rhinencephalic injury in cat. J. Neurophysiol., 1953, 16, 643-659.

- Schwab, R. S. The rhinencephalon: a symposium of the American EEG Society. EEG clin. Neurophysiol., 1954, 6, 708-716.
- Schwartzbaum, J. S., & Pribram, K. H. The effects of amygdectomy in monkeys on transposition along a brightness continuum. J. comp. physiol. Psychol., 1960, 53, 396-399.
- Schwartzbaum, J. S., Wilson, W. A., & Morrisette, J. R. The effects of amygdectomy on locomotor activity in monkeys. J. comp. physiol. Psychol., 1961, 54, 334-336.
- Scoville, W. B. The limbic lobe in man. J. Neurosurg., 1954, 11, 64-66.
- Scoville, W. B., & Milner, B. Loss of recent memory after bilateral hippocampal lesions. J. Neurol. Neurosurg. Psychiat., 1957, 20, 11-21.
- Small, W. S. An experimental study of the mental processes of the rat. I. Amer. J. Psychol., 1900, 11, 133-165.
- Small, W. S. An experimental study of the mental processes of the rat. II. Amer. J. Psychol., 1901, 12, 206-239.
- Smith, C. J. Mass action and early environment in the rat. J. comp. physiol. Psychol., 1959, 52, 154-156.
- Smith, K. U. Experimental analysis of the associative mechanisms of the human brain in learning functions. J. comp. physiol. Psychol., 1952, 45, 66-72.
- Smith, W. K. The results of ablation of the cingular region of the cerebral cortex. Federation Proc., 1944, 3, 42-43.
- Smith, W. K. The results of stimulation of the uncus and adjacent portions of the hippocampal gyrus. Federation Proc., 1944, 3, 43.
- Spiegel, E. A., Wycis, H. T., Orchinik, C. W., & Freed, H. The thalamus and temporal orientation. Science, 1955, 121, 771-772.
- Stamm, J. S. The function of the median cerebral cortex in maternal behavior of rats. J. comp. physiol. Psychol., 1955, 48, 347-356.

Stamm, J. S., & Pribram, K. H. Effects of epileptogenic lesions in frontal cortex on learning and retention in monkeys. J. Neurophysiol., 1960, 23, 552-563.

Stamm, J. M., & Pribram, K. H. Effects of epileptogenic lesions of inferotemporal cortex on learning and retention in monkeys. J. comp. physiol. Psychol., 1961, 54, 614-618.

Stepien, L. S., Cordeau, J. P., & Rasmussen, T. The effect of temporal lobe and hippocampal lesions on auditory and visual recent memory in monkeys. Brain, 1960, 83, 470-489.

Stern, K. Severe dementia associated with symmetrical degeneration of the thalamus. Brain, 1939, 62, 157-171.

Stewart, J. W., & Ades, H. W. The time factor in reintegration of a learned habit lost after temporal lobe lesions in the monkey (*Macaca mulatta*). J. comp. physiol. Psychol., 1951, 44, 479-486.

Sweet, W. H., Talland, G. A., & Ervin, F. R. Loss of recent memory following section of fornix. Trans. Amer. Neurol. Assn., 1959, 84, 76-82.

Terzian, H., & Ore, G. D. Syndrome of Klüver and Bucy, reproduced in man by bilateral removal of the temporal lobes. Neurology, 1955, 5, 374-380.

Thomas, G. J. Neurophysiology of learning. In Annu. Rev. Psychol., 1962, 13, 71-106.

Thomas, C. J., Moore, R. Y., Harvey, J. A., & Hunt, H. F. Relations between the behavioral syndrome produced by lesions in the septal region of the forebrain and maze learning of the rat. J. comp. physiol. Psychol., 1959, 52, 527-532.

Thomas, G. J., & Otis, L. S. Effects of rhinencephalic lesions on conditioning of avoidance responses in the rat. J. comp. physiol. Psychol., 1958, 51, 130-134.

Thomas, G. J., & Otis, L. S. Effects of rhinencephalic lesions on maze learning in rats. J. comp. physiol. Psychol., 1958, 51, 161-166.

Thomas, G. J., & Slotnick, B. Effects of lesions in the cingulum on maze learning and avoidance conditioning in the rat. J. comp. physiol. Psychol., 1962, 55, 1085-1091.

- Thompson, R. The comparative effects of anterior and posterior cortical lesions on maze retention. J. comp. physiol. Psychol., 1959, 52, 506-508.
- Thompson, R. L. Effects of lesions in the caudate nuclei and dorsofrontal cortex on conditioned avoidance behavior in cats. J. comp. physiol. Psychol., 1959, 52, 650-659.
- Thompson, R. Learning and retentiveness in brain-damaged rats. J. comp. physiol. Psychol., 1959, 52, 501-505.
- Thompson, R., & Hawkins, W. F. Memory unaffected by mammillary body lesions in the rat. Exp. Neurol., 1961, 3, 189-196.
- Thomson, A. F., & Walker, A. E. Behavioral alterations following lesions of the medial surface of the temporal lobe. Folia psychiol. neurol. neurochir. Neerl., 1950, 53, 444-452.
- Valenstein, E. S., & Nauta, W. J. H. A comparison of the distribution of the fornix system in the rat, guinea pig, cat and monkey. J. comp. Neurol., 1959, 113, 337-363.
- Victor, M., Angevine, J. B., Mancall, E. L., & Fisher, C. M. Memory loss with lesions of hippocampal formation. Arch. Neurol., 1961, 5, 244-263.
- Walker, A. E. Recent memory impairment in unilateral temporal lesions. A. M. A. Arch. Neurol. Psychiat., 1957, 78, 543-552.
- Ward, A. A. The cingular gyrus: area 24. J. Neurophysiol., 1948, 11, 13-23.
- Warren, J. M., & Akert, K. Impaired problem solving by cats with thalamic lesions. J. comp. physiol. Psychol., 1960, 53, 207-211.
- Warren, H. B., & Warren, J. M. Reversal learning by cats with prestriate and orbitofrontal lesions. Amer. Psychologist, 1960, 15, 485. (Abstract)
- Warren, H. B., Warren, H. B., & Akert, K. Orbitofrontal cortical lesions and learning in cats. J. comp. Neurol., 1962, 118, 17-42.

Watson, J. B. Kinaesthetic and organic sensations: their role in the reactions of the white rat. Psychol. Rev. Monogr. Suppl., 1907, 8 (Whole No. 33).

Weiskrantz, L., Mihailović, L., & Gross, C. G. Effects of stimulation of frontal cortex and hippocampus on behaviour in the monkey. Brain, 1962, 85, 487-504.

Weiskrantz, L., & Mishkin, M. Effects of temporal and frontal lesions on auditory discrimination in monkeys. Brain, 1958, 81, 406-414.

Weiskrantz, L., & Wilson, W. A. The effect of ventral rhinencephalic lesions on avoidance thresholds in monkeys. J. comp. physiol. Psychol., 1958, 51, 161-171.

White, R. W. Motivation reconsidered: the concept of competence. Psychol. Rev., 1959, 66, 297-333.

Whitty, C. W. M., & Lewin, W. A Korsakoff syndrome in the post-cingulectomy confusional state. Brain, 1960, 83, 648-653.

Williams, M., & Pennybacker, J. Memory disturbances in third ventricle tumours. J. Neurol. Neurosurg. Psychiat., 1954, 17, 115-123.

Wilson, M. Effects of circumscribed cortical lesions upon somesthetic and visual discrimination in monkeys. J. comp. physiol. Psychol., 1957, 50, 630-635.

Wilson, W. A., & Mishkin, M. Comparison of the effects of inferotemporal and lateral occipital lesions on visually guided behavior in monkeys. J. comp. physiol. Psychol., 1959, 52, 10-17.

Wolstenholme, G. E. W., & O'Connor, C. M. (Eds.) Ciba symposium on the neurological basis of behavior, London: Churchill, 1958.



APPROVAL SHEET

The dissertation submitted by Sister Helen Gavin 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.

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Date

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