The Response of American Journals of Opinion to the Rise and Consolidation of National Socialism in Germany

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EXTINCTION AND RE-ACQUISITION OF A FOOD-REINFORCED
MOTOR RESPONSE IN HIPPOCAMPECTOMIZED CATS

by

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CHAPTER I

INTRODUCTION

Studies of hippocampectomy in rats, cats and monkeys have been variously interpreted as indicating the possible involvement of this structure in recent memory mechanisms (Drachman and Ommaya, 1964), mechanisms involving passive avoidance (Isaacson and Wickelgren, 1962), orienting activity (Karmos and Grastyan, 1962), and in many other activities.

Two interpretations of the results observed in hippocampectomized animals have especially attracted this author's attention.

The first may be called the Perseveration theory. This interpretation suggests that animals with hippocampal lesions are unable to inhibit learned responses when the reward is removed or when rewarded and unrewarded stimuli are reversed (Thompson and Langer, 1963; Mahut and Coreau, 1963; Teitelbaum, 1964).

The other interpretation may be referred to as the Positive Reinforcement theory. This theory suggests that lesions in the hippocampus may have the effect of increasing the effectiveness of positive reinforcement (Peretz, 1965). Thus, if a reward previously associated with a stimulus is removed, the animal will continue responding to this stimulus because of its strong
association with positive reinforcement.

The present experiment attempts to clarify the nature of the mechanism responsible for the two interpretations, and discuss the extent to which these theories can account for existing experimental results.
CHAPTER II

THE HIPPOCAMPUS

General Anatomy

The hippocampus is usually included among the structures of the rhinencephalon (literally, "nose-brain"). Other structures included here are the olfactory and the vomeronasal nerves; the olfactory bulb and stalk; the anterior olfactory nucleus; the anterior perforated spaces; the prepyriform cortex and portions of the hippocampal gyrus; the isthmus of the fornicate gyrus, and the cingulate region (Crosby, et al., 1962). Although included among the structures of the rhinencephalon, the role of the hippocampus in olfaction has been questioned for quite some time (Green, 1964). In fact, its connections with the olfactory system are questionable at best (Arnold, 1960; Green, 1964).

Embryology

Both embryologically and phylogenetically, the hippocampus first appears in the dorsomedial hemisphere wall, before the development of the corpus callosum (Crosby, et al.). It is molded into its final form by the rapid proliferation of the thalami, striatum, and neocortex (Ballard, 1964). As the corpus callosum and neopallial cortex develop, the hippocampus is forced to retreat downward into the temoral lobe region, leaving remnants above and below the corpus callosum. Eventually, three major parts
become differentiated: (1) a ventral portion, the forerunner of the dentate gyrus; (2) the intermediate portion, which becomes the major part of the hippocampus, the cornu ammonis; and (3) the dorsal portion differentiating into the various subicular areas (See figure 1).

This course of development, especially that of the dentate gyrus, may have contributed to the misconception that the hippocampus is an important olfactory structure. Although the dentate gyrus develops from the ventral portion of the hippocampal mass, it is often regarded distinct from the hippocampus. Its size is very often dependent on the degree of development of olfaction in some animals. Cateceans, who have poorly developed olfactory systems, have the dentate gyrus greatly reduced, while in insectivores, with large olfactory nerves, it attains great size.

Structure and Fiber Pathways

The hippocampus and associated structures are illustrated in figure 2. Fibers arising from the pyramidal cells of the hippocampus enter the fimbria of the hippocampus, which lies medial to the hippocampus in the floor of the inferior horn of the lateral ventricle of each side (Netter, 1962). There, fibers run posteriorly and curve upward in a close bundle called the crus of fornix, then pass anteriorly, just beneath the corpus callosum. The fibers also course medially, thus meeting those from the other hippocampal structure symmetrically at the midline. In this vicinity they are called the bodies of fornix, which, in turn, continue forward
Fig. 1. Section through the hippocampus showing its various subdivisions. (Adapted from Grosby, et al., 1962, p. 421.)
Fig. 2. Relationship of hippocampus to other structures.
The hypothetical circulation of impulses during emotion are shown in solid arrows. (Adapted from Hanter & Gatz, 1961, p. 101.)
and again turn ventrally, forming the columns of fornix. These terminate in the mammillary nuclei of the hypothalamus. From the mammillary bodies, efferent fibers course directly to the anterior nuclei of the thalamus, constituting the mammillo-thalamic tract. The anterior thalamic nucleus sends these and all other fibers to the cingulate gyrus, (see figure 2), which encircles the corpus callosum. Through its narrow posterior part (the isthmus), the cingulum is continuous with the hippocampal gyrus, the most medial convolution of the temporal lobe (Manter and Gatz, 1961). (The hippocampal gyrus is not to be confused with the hippocampus.) The mammillary bodies are connected with the brainstem tegmentum and the reticular formation via the mammillo-tegmental tract (Arnold, 1960). Interconnection of the hippocampi is formed by fibers passing between the structures at the crura of fornix, forming the hippocampal commissure. It is also possible that these commissural fibers include fibers from the hippocampal gyri, as well as fibers from the septal grey traversing to the hippocampus (Crosby, et al., 1962).

According to a theory suggested by Papez, the hippocampus, fornix, mammillo-thalamic tract, and anterior thalamic nucleus form a circuit by which impulses course between the hypothalamus and cortex (Manter and Gatz, 1961). Thus, hypothalamic stimuli pass through the the anterior nuclei of the thalamus to the cingulate gyrus, and continue to the cortex by means of association fibers. This is said to produce the subjective experience of emotion. Psychic activity can produce emotional displays by the
reverse side of the circuit. The cortex may send stimuli through
the cingulate gyrus to the hippocampal gyrus, which is connected
to the hippocampus by short fibers. The excitation continues
through the fornix to the mammillary nuclei, thus activating the
autonomic nervous system (see figure 2).

Some support of this theory is provided by the effects of
temporal lobotomies, which produce individuals with greatly sub-
dued emotional patterns.

A number of motor reactions have been elicited by the sti-
mulation of the hippocampus. Beginning with the rostral temporal
pawt and proceeding posteriorly and caudally, stimulation brings
about facial movements, vocalization, neck and shoulder movement,
and movement of the upper extremities. Stimulation also produces
changes in autonomic activity. Arousal reactions have been achie-
ed even while the animal was under anesthesia, lapsing back into
unconsciousness upon cessation of the stimulus (Crosby, et al.,
1962).

Connections of the hippocampus with visual, tactile, and
olfactory fibers are evidenced physiologically by evoked poten-
tials after stimulation of the proper sensory receptors. The
pathways by which these potentials occur are obscure. However,
it must be noted that the latencies involved are rather long
(Crosby, et al., 1962), suggesting several synapses.

Arnold (1960) points out that the hippocampus seems to be a
part of an information retrieval circuit. Such retrieval is ini-
tiated via visual areas and the hippocampal gyrus on the basis of visual, auditory, and some tactual cues; recall based on other tactual cues, olfactory cues, and motor memory is mediated by the hippocampal rudiment (indusium griseum).

Evoked Potentials

Some of the potentials evoked in the hippocampus have already been mentioned. Green (1964) points out that the reports of evoked potentials in the hippocampus are very confusing, particularly due to differences in procedure, since evoked potentials are readily modified by anesthetics, hyperstimulation, types of electrodes, amount of injury, and other factors.

Bard (1961) states that all investigations of impulses evoked in this structure by stimulation of the olfactory bulbs have yielded either negative results or potentials so attenuated in size or latency as to suggest a number of synapses. This opinion is thus receiving support from many sources.

Renshaw, et al., (1940) recorded responses from various portions of the hippocampus upon stimulation of the entorhinal area. Derado, et al., (1964) found that stimulation of the visual system by light flashes evoked activity in the hippocampus. Similar responses were obtained by tactile stimulation of the nostrils.

Green (1964) implies that evoked potentials may be due in part to the effect of potential changes on the extraneuronal currents around the hippocampal pyramids. At any rate, even these studies alone have demonstrated the complexity of the structure, its involvement in a variety of sensory processes, and only slight
importance in olfactory processes.

State of Consciousness

As already mentioned, stimulation of the hippocampus can bring about arousal (Crosby, et al., 1962). Green (1964) points out that because early concentration on the midbrain reticular formation, other areas were long neglected in studies of arousal. He emphasizes that the midbrain represents a narrow pathway which in actuality contains opposing centers in close proximity. This sort of criticism is extended to the phenomenon of hippocampal arousal as well.

According to Green, the changes which occur in the EEG of the neocortex are directly opposite of those evidenced in the hippocampus. While arousal results in "desynchronization" of the neocortex, the hippocampus shows rhythmic, "synchronised", theta waves of 4 to 7 per second. The theta response was found to occur in the hippocampus of the rabbit due to olfactory, visual, auditory, and tactual stimulation (Green and Arduini, 1954), and could not be abolished by removal of the mammillary body or decortication.

Conditioning and Learning

Allen (1941) conditioned dogs to elevate a paw when the odor of cloves was presented. Bilateral temporal lobectomy, removal of the hippocampus, or destruction of the pyriform-amygdaloid complex had no effect on the habit. However, discrimination tasks requiring discrimination between two odors, bilateral
destruction of the pyriform-amygdaloid complex abolished the habit. In another study, (1948), he points out that in no case has the removal of Ammon's horns had an effect on a previously acquired olfactory conditioned reflex. Apparently, the question of whether the hippocampus plays an important olfactory role is again decided in the negative.

A number of studies involving EEG analysis have pointed out the importance of the hippocampus in orienting activity.

On the basis of changes of hippocampal electrical activity during the establishment of conditioned reflexes, Grastyan, et al., (1959) suggested that the hippocampus plays an essential role in the formation of the conditioned reflex, perhaps by inhibiting or controlling the orienting reflex. Grastyan supports this assumption in a later study (Karmos and Grastyan, 1962) in which hippocampectomized cats are found to exhibit a hyperactive orientation reflex. Their findings, however, are far from complete. Holmes and Adey (1960) point out that the theory; is not adequate since it cannot explain the decrease in frequency of the hippocampal theta rhythm with increased training, an effect which is opposite of what Grastyan would predict.

Adey, et al., (1963) studied the impedance shifts in the hippocampi of cats during discriminative approach in a T-maze. Choice of the correct visual cue resulted in a food reward. It was found that during early stages of training only irre-
gular deviations around the baseline were seen. With increasing performance levels, the impedance drop observed during the approach phase continually increase, with a maximum drop during maximum performance. This effect persisted with overtraining, but was abolished with extinction. Although these results are discussed in terms of mechanisms underlying these impedance shifts, they underline the importance of the hippocampus in discriminative approach conditions. The confusing aspect is that these events are found to occur without either gross orienting behavior or the imposition of discriminative performance (Radulovacki and Adey, 1965). Apparently, the mere fact of an alerted state is sufficient to produce the effect. Porter, et al., (1964) throw some light on the situation. The data reported by these authors clearly indicates that great regional differences exist within the hippocampus, pointing out that precise anatomical location is of utmost importance in seeking behavioral correlates of its electrical activity (Radulovacki and Adey, 1965). The authors suggest that the establishment of "memory traces" in the brain may depend on such rhythmic activity. The reestablishment of these wave patterns are then involved in the recall of information. This is in agreement with a similar processes proposed by Arnold (1960).

**Seizure Activity**

It is well known that the hippocampus is often involved in
seizure activity (Karmos, 1962; Kim and Kim, 1962; Green, 1964; and Arnold, 1960). Kim and Kim (1962) found that hippocampal ablation resulted in greater susceptibility to audiogenic seizures in rats. Another interesting fact is that seizures initiated in the hippocampus may rapidly spread to adjacent areas. They are often evoked by continued stimulation of this structure (Green, 1965) and are the frequent result of electrically induced lesions (Karmos and Grastyan, 1962). This suggests that lesions in this structure should be performed by other means, such as aspiration, whenever possible.

Learning and Retention

Whether or not hippocampectomized animals will be impaired in learning and retention seems to be dependent on the type of task involved.

In simultaneous visual discriminations ablation of the hippocampus does not seem to result in a deficit. This effect was found in rats (Kimble, 1963; Grossman, 1963) and monkeys (Stepien, et al., 1960; Kimble and Pribram, 1963). Neither was impairment found in the acquisition of a tactile discrimination in cats (Teit (Teitelbaum, 1964; Webster and Voneida, 1964). Similarly, Ellen and Wilson (1963) concluded that hippocampal lesions do not produce a deficit in the acquisition of a bar pressing response in rats. Finally, Arnold has found learning and retention to be normal in simultaneous tactual and successive olfactory discrimination in rats (Arnold, 1966).
In the learning and retention of successive visual discriminations however, hippocampectomized rats have been found to be deficient (Kimble, 1963; Grossman and Mountford, 1964). Lesioned monkeys have also been found to be deficient in a sequential bar-pressing task (Kimble and Pribram, 1963; Stepien, et al., 1960).

Similar deficits were found by Drachman and Ommaya (1964) in retention of a visual matching-from-sample task and delayed matching-from-sample. They also found their lesioned monkeys impaired in post-operative acquisition of a visual pattern discrimination.

Apparently, the crucial point is whether the discrimination involves simultaneous presentation or successive presentation of stimuli. The data cited supports such a generalization with the exception of Arnold's (1966) work with successive olfactory discrimination and Drachman and Ommaya's (1964) finding of impairment in post-operative acquisition of a matching-from-sample visual discrimination. It may be that the complexity of the problem is the important factor. A successive discrimination is certainly more difficult than a simultaneous one. Matching-from-sample is even more difficult.

Rats with hippocampal lesions show defective maze learning as compared with either operated or non-operated controls (Kaada, et al., 1960; Madsen and Kimble, 1965; Kimble, 1963; Thomas and Otis, 1958). None of these studies can be gauged as to complexity of task, since there is no basis of comparison with discrimi-
nation studies. Furthermore, different mazes differ in complexity. Kimble (1963) attempted to investigate just this point by having his rats learn two hebb-williams mazes of differing complexity. He found that hippocampectomized animals made more errors on the simple maze than on the complex one. Paradoxically, the "simple" maze also provided more opportunity for error (7 error lines) than the "complex" maze (4 error lines). Small wonder that the number of errors was approximately double for the "simple" maze.

Orientation

In the section on the electrical activity of the hippocampus, it was mentioned that electrophysiological evidence points to some form of involvement in orientation. Similar conclusions have been made from behavioral observations, such as Isaacson (1963, who found that these lesions impaired the ability of rats to respond to normal or irrelevant stimuli placed in a straight-alley runway which they were accustomed to traverse for food. A conflicting result was found by Karmos and Grastyan (1962) who observed a hyperactive orienting reaction in hippocampectomized cats. If, at the time of delivery of the auditory cue which signalled the cat to approach a food tray, the animal happened to sit with its back to the tray, "long lasting, searching, sniffing orienting movements ensued". Similar hyperactive orientation reactions were observed during a delayed auditory conditioning task. The authors conclude that the
hippocampus inhibits or in some other fashion controls the orientation reflex. In terms of this explanation, Wickelgren and Isaacson's (1963) rats should have exhibited increased, rather than decreased, orienting reactions to the novel stimulus introduced into the runway. The apparent discrepancy is probably due to a rather subjective interpretation of what orienting reactions consist of. Searching and sniffing behavior does not guarantee a deficit necessarily connected with orientation—a memory defect, for example, could be involved as well. The Wickelgren and Isaacson study, however, does point out the possibility that lesioned animals might be less distractible. Their results can be interpreted simply in terms of an increase in the effect of positive reinforcement (Peretz, 1965).

Kim (1960b) reported that some of his rats showed "poor orientation after the brain lesion and would lose the female while chasing after her". This can support either Wickelgren and Isaacson or Karmos and Grastyan. In the first case the explanation would be that the rats were not sufficiently attracted by the stimulus of the female, while in the second case it could be said that the orienting reaction was not rapid enough to locate the female. In any event, it is sure that the structure is involved in orientation. Exactly how, remains undecided.

Behavioral Manifestations

A number of behavioral manifestations are associated with hippocampal lesions. These can be divided into sexual activity,
general activity, and aggressiveness (or timidity).

The data on sexual activity is somewhat conflicting. In a systematically conducted study, Kim (1960b) found an increase in frequency of mounting in rats with hippocampal ablations when compared with a control period. He also found that neocortical ablation tended to reduce the frequency, thus the effect was not due to neocortical damage.

In monkeys, however, no such clear-cut results can be found. Stepien et al., (1960) found only meager evidence of hypersexuality in one of two lesioned monkeys. This manifested itself in immediate erection when the experimenter approached the cage, but no attempt was made to mount another male or female in the cage. A result entirely opposite to Kim's was found by Gel, et al., (1963) who reported that sexual activity was never observed between monkeys in communal cages. In fact, one large rhesus monkey, who was a persistent masturbator before hippocampal resection, ceased the habit and did not resume it while under observation for over a year. These differences between rats and monkeys are probable due to species differences.

At this point increases in other pleasurable reactions may also be mentioned. Karmos and Grastyen (1962), for example, reported a tendency of lesioned cats to rub themselves against all objects. Green (1964) reports that electrical or chemical stimulation of the hippocampus is often associated with signs of pleasure, such as increased willingness of the animals to bar-press for stimulation.
A number of authors found an increased level of activity in lesioned rats (Kimble, 1963; Teitelbaum and Milner, 1963), cats (Karmos and Grastyan, 1962), and monkeys (Stepien, et al., 1960). Other authors, however, reported just the opposite results in rats (Kim, 1961; Kaada, et al., 1961) and monkeys (Gol, et al., 1963). Most of these were not systematic studies, but incidental observations. Teitelbaum and Milner obtained their activity increases on the basis of the number of light beam interruptions in an activity cage, and in increased running-speed in an unbaited T-maze. Kimble (1963) found an increase on the basis of number of squares of an open field entered by the rats. The significance of increased activity levels in these animals is ambiguous, but some authors (Kimble, 1963; Teitelbaum and Milner, 1963) believe that it might account for their inability to perform passive avoidance problems.

The most outstanding behavioral change following hippocampectomy is the docility reported by some investigators. Gol, et al., (1963) describes how cats, monkeys, and baboons, wild or aggressive before the operation, become tame and difficult to arouse. A lesioned monkey would even permit a snake to crawl between its legs, whereas its mere visual presence had previously elicited violent withdrawal reactions. Previously wild cats appeared quite tame after surgery, and were unresponsive to dogs placed in their cages. Only extreme provocation would elicit rage or aggressive responses of a transitory nature. Milder, but
similar results are reported in monkeys by Stepien, et al., (1960).

The present author has also observed docility in a previously wild cat after a hippocampal lesion. This effect however, could also be due to the post-operative care of the animal. It was handled daily, and received its first food by hand. Since handling of the animal continued, the animal could have learned to trust the experimenter.

Memory

Hippocampal lesions have often been associated with defects in memory, generally referred to as "recent memory". Green (1964) suggests that as far as clinical cases are concerned, the term "post-distractional amnesia" would be more precise and descriptive. The concept of "short-term memory", or "recent memory" as applied to either human or animal studies has also been questioned by Drachman and Emmaya (1964).

Clinical Cases of Memory Loss

Several cases of memory loss after temporal lobectomy have been reported by Milner (1959). Two of them are briefly reviewed here. Both cases involved unilateral temporal lobectomy of the dominant hemisphere for the correction of focal epilepsy. In these cases, only slight recovery of a recent memory loss occurred. Neither patient exhibited a change in I.Q., and were able to continue their daily work—one a glove cutter, the other a civil engineer.
The engineer, although able to recite 9 digits forward and 7 backwards, cannot recall small test items if his attention has been diverted for a short time. (Hence Green's suggestion for "post-distractor amnesia"). Both patients exhibit anterograde amnesia for ordinary daily events due to such distractions. In addition, both patients show a retrograde amnesia, going back four years for the glove cutter, three months for the engineer. The memory loss in these patients was explained in terms of a pathological condition in the opposite temporal lobe, which resulted in a bilateral, rather than a unilateral lesion.

A third case of bilateral temporal lobectomy is related in which the patient also forgot everything of the previous moment the instant he was distracted. In another instance, a patient with a similar operation was unable to recognize the picture of a dog he had drawn a half hour earlier, and even called it a deer. The author concluded that the hippocampus must be essential in the consolidation of memory traces. Those traces which had gained independence of the hippocampus were available to the patient, others were lost.

Although the evidence for a recent memory loss in these and similar patients seems obvious at a glance, Drachman and Ommaya criticise the loose application of the term. They point out that some of these patients have normal digit spans, yet are unable to recall a short paragraph read to them. Apparently, the impairment depends more on the nature of the memorandum, rather
than on the time elapsed since the sensory information is received. The label seems to be an oversimplification of the situation.

**Experimental Studies of Memory**

Generally, segments of studies dealing with delayed response have resulted in conflicting and inconclusive results. Authors who performed simple delayed response experiments found no impairment in retention (Mahut and Cordeau, 1963; Orbach, et al., 1960). Stepien, et al., (1960) employed a method for the investigation of recent memory suggested by Konorski (1959) and found an impairment in monkeys. This "compound stimulus test" involves the reinforcement of a "compound" consisting of two successive stimuli when the stimuli are the same. If the second stimulus differs from the first, the "compound" is not reinforced. The authors conclude that a recent memory loss must have been responsible for the deficient performance, since the first stimulus had to be retained for comparison with the second in order for the animal to perform successfully.

Orbach, et al., (1960) and Mahut and Cordeau (1960) and Pribram, et al., (1962) discovered an impaired performance in delayed alternation of monkeys with medial temporal lesions, but the authors do not interpret their findings in terms of recent memory loss. This may in part be due to the fact that in two of these studies (Orbach et al., and Mahut and Cordeau) longer intertrial delays did not produce a deficit, while shorter delays did.
Mahut and Cordeau conclude that delayed alternation deficits must be related to something other than the difficulty of the problem as such.

In delayed matching-from-sample experiments, Correll and Scoville (1965) and Drachman and Ommaya (1964) obtained similar results. It was quite evident that bilateral lesions of medial temporal lobe structures result in a marked deficit. Surprisingly, the latter authors found that animals with extensive lesions reached criterion at the long delay of 12 seconds in fewer trials than the subjects with small lesions! They conclude that not the ability to delay is impaired, nor short-term memory, but that loss of pre-operative retention and impairment of acquisition are the chief contributors to the results.

Drachman and Ommaya present an interesting speculation as to the reason that complex memoranda cannot be repeated by subjects with medial temporal lobe lesions. They suggest that "simple memoranda can be 'held' in the transient mechanism of short-term memory", but that lengthier, more complex memoranda "must be 'stored' using relatively permanent mechanisms of memory". Thus, he considers the impairment to be one dealing with storage, not recall.
CHAPTER III

REVIEW OF RELATED LITERATURE

Although this study concerned itself primarily with the perseverance theory and the positiverinforcement theory, there is one other manifestation which is of interest here: the effect of hippocampectomy on problems of passive avoidance. The interest derives from the fact that some of the results explained in terms of inability to perform passive avoidance lend themselves to interpretation under the other two theories. Some of the literature under each of these three attempts to explain the results of hippocampectomy are reviewed here in the following order (a brief definition of the terms and theories is also provided):

1. Passive avoidance. The process of avoiding punishment by inhibiting a learned response. Hippocampectomized animals are thought to be deficient in this function.

2. Perseveration. The tendency to continue performing a learned task; inability to alter the mode of performance of a learned task.

3. Positive Reinforcement. Increased effectiveness of positive reinforcement as a motivator.
Passive Avoidance

There have been a number of reports that lesions of the hippocampus produce deficits in passive avoidance tasks in rats (Kimble, 1963; Clark and Isaacson, 1965; Isaacson and Wickelgren, 1962, and Snyder and Isaacson, 1965), but not in active avoidance (Goldstein, 1965; Isaacson, et al., 1962). The latter is usually referred to as the conditioned avoidance response, CAR. In the typical CAR experiment, the rat is placed in a shuttle box and is given a shock paired with a sensory cue. By crossing to the other compartment it can avoid the shock. Thus, this is an active response.

In the case of passive avoidance, the typical experiment consists of teaching an animal to enter a certain goal box of a maze or runway, where it is rewarded with food. When the response is established, a shock is introduced along with the food in order to induce the animal to withhold the learned response. While normal animals refuse to enter the goal box for many trials even after a single shock, lesioned rats show little change in their behavior.

Since the animals are able to perform one type of avoidance task but not another, these studies can be interpreted as an inability to perform a specific type of avoidance. It may be even simpler to interpret these results in terms of an inability to withhold a learned task, as Clark and Isaacson do. Such an interpretation would be equal to stating that hippocampal lesions
produce response perseveration tendencies.

Furthermore, the results of passive avoidance experiments can also be interpreted in terms of the positive reinforcement theory. This theory contends that the food reward (positive reinforcement) becomes so strong in hippocampectomized animals as to counteract the effects of a punishing shock. In any case, to simply state that the lesioned animals are deficient in performing passive avoidance is inadequate.

**Perseveration**

A variety of experiments have indicated that lesions of the hippocampus interfere with ability to perform certain types of reversals. This effect is sometimes interpreted in terms of perseveration tendencies. The simplest example of this is the reversal of a position habit. Thompson and Langer (1963) demonstrated the inability of hippocampectomized rats to choose a certain arm of a T-maze if the other arm had previously been the rewarded one. The hippocampus, however, is not the only structure that produces this result. Bilateral lesions destroying either the mammillary bodies, mammillo-thalamic tract, anterior thalamus, precallosal limbic area, septal area, proptic hypothalamus, or substantia nigra result in a significant impairment of this task. All of them are either anatomically or electrophysiologically related to the hippocampus. On the other hand, not all structures related to the hippocampus produce this deficit. Lesions in the amygdala, for example, do not. Neither do lesions
of the nucleus medialis dorsalis. Evidently the effect is a complicated matter.

Thompson and Langer do not subscribe to any one explanation of the results. They do consider that the most probable explanation has to do with either the disruption of a memory process or an inability to inhibit a learned response.

Mahut and Cordeau (1963) found a severe deficit in a spatial reversal problem with lesioned monkeys, and considered the possibility that this deficit and the deficit in delayed alternation may be related. Both results are interpreted in terms of response perseveration tendencies.

Perseveration was observed in another type of task, tactile discrimination, by Teitelbaum (1964) and Webster and Veneida (1964). Teitelbaum's technique consisted of teaching cats to depress one of two pedals, each with different tactile qualities, to receive a food reward. Reversal training consisted of rewarding the previously negative pedal, and omitting the reward from the previously positive stimulus. He showed that although cats with hippocampal lesions were comparable to normal animals in discrimination learning per se, and were able to learn new tactile discrimination problems, mastering a reversal took more than twice as many trials as for normals.

Webster and Veneida also found similar results in commissurectomized cats. Animals with unilateral hippocampal lesions were found to take up to six times as long to reverse learning
involving the limb contralateral to the lesion, as compared with the ipsilateral limb. Again, the evidence seems to point to response perseveration.

If response perseveration is to explain the preceding results, then it certainly can explain inability of animals to extinguish a response after the reinforcement has been removed. Webster and Voneida found the same results in simple extinction as in tactile discrimination reversal. Resistance to extinction has also been reported by other authors (Jarrard and Isaacson, 1965; Jarrard, et al., 1964).

Furthermore, the perseveration interpretation can be applied to the lack of change in the response of rats traversing a straight-alley runway when a novel stimulus is introduced (Wickelgren and Isaacson, 1963). While normal rats stopped to investigate the novelty, hippocampectomized rats did not. Apparently, the strongly dominant running response could not be inhibited by these rats.

Two more studies dealing with this problem in a different way may be cited. Ellen and Wilson (1963) trained lesioned rats to bar-hold to avoid an electric shock, then reversed the task to bar-pressing. As expected, these animals could not learn the new response. Since the animals could learn a new task of similar nature, the authors conclude that the deficit involves the giving up of old responses, rather than learning of new ones.

Similarly, Clark and Isaacson found that rats with hippo-
cortical ablations could not switch from a CRF (continuous reinforcement for each bar-press) to a DRL (differential reward of low rates of responding) schedule of bar-pressing. When switched to the DRL schedule, they were rewarded with water only if there was a 20 second interval since the previous bar-press. Lesioned rats tended to continue bar-pressing rapidly, and consequently received very little water under the DRL schedule.

All these studies in some way implicate the hippocampus as necessary for the inhibition of a learned response, giving up of old response patterns, or modifying them.

**Positive Reinforcement**

Recently, Peretz (1965) suggested that another interpretation of some of the results may be possible. The finding by Isaacs and Wickelgren (1962) for example, that lesioned rats respond to food more rapidly after previously being shocked in the food compartment can be interpreted in terms of an increased effectiveness of the reinforcing qualities of the food reward, thus overcoming the adverse effects of shock.

In general, Peretz believes that "hippocampectomy may have the general effect of increasing the effectiveness of positive reinforcement". Other studies cited by Peretz as lending themselves to interpretation in similar fashion are as follows: Kim (1960a) found an increase in water consumption, as well as an increase in sexual mounting (1960b); increases in exploratory behavior found by Roberts, Dembr, and Brodwick, (1962).
Jarrard, Isaacson, and Wickelgren (1964) showed that hippocampal ablation can interfere with the extinction of a food-reinforced response.

The large number of errors made by monkeys in the Study by Stepien et al., (1960) regarding recent memory also support this hypothesis. Here, a delay was involved during which the response was to be withheld. The inability to do so might have been due to the great attractive force of the reward. Finally, Kimura (1958) found that the increase in the latency of a response to food after small electric shocks was smaller for hippocampectomized animals than for normal rats. Thus, there certainly seems to be the possibility that positive reinforcement increases motivation level and accounts for a number of experimental results.

To test his hypothesis that positive reinforcement acquires a greater significance for animals with hippocampal lesions, Peretz devised the following method. Cats were taught to open a window in the wall of a wooden box by pushing on it with their nose or paw. The response was rewarded with a piece of beef. Response latencies were measured on each trial. When the animal performed the response for ten consecutive trials with a latency under 15 seconds, twenty extinction trials were given in which the reward was omitted. Peretz found that the extinction latencies were far shorter for the hippocampectomized animals than for the controls; i.e., the lesioned animals seemed to ha-
ve acquired a stronger habit. This result supported his hypothesis. However, the result can be explained by the perseveration theory as follows.

Peretz taught his cats a motor task which was then extinguished. In terms of the perseveration theory, the dominant tendency at the beginning of extinction is to continue responding. In the lesioned animals this tendency is stronger than in the control animals, therefore it is more difficult for them to give up this response: the "perseverate".

It is the purpose of this study to replicate Peretz's experiment, and to introduce another task which might put his theory to a more rigorous test, and indicate if his conclusions are justified. The question at the moment is this: what would the two theories predict if we were to try to re-establish the response after extinction? In the light of the perseveration theory, the dominant tendency of the animal after extinction is to inhibit its response, i.e., simply to "not respond". If it were to perseverate in this tendency, it would be more difficult to re-establish the response in a lesioned animal than in a control.

The positive reinforcement theory, on the other hand, would predict just the opposite result. Since the reinforcement should have increased effectiveness for the lesioned cats, reinstatement of the reward would result in more rapid return of the response in the control animals.

Peretz's positive reinforcement theory does not actually
contradict the perseveration theory, but merely suggests an alternative. Since the present experiment is a partial replication of Peretz's study, it may be hypothesized that similar results will be obtained. Thus:

1) extinction speed will be greater for control animals than for hippocampectomized animals; this will manifest itself in an increased response latency during the extinction trials, as well as in a smaller number of trials needed by control animals to achieve extinction;

2) re-acquisition will be more rapid for hippocampectomized animals than for control animals; this will manifest itself in longer re-acquisition response latencies for the control group than for the hippocampectomized group.
Subjects consisted of 8 adult cats, 4 males and 4 females.

Grouping. Cats were randomly assigned to one of three groups: Bilateral hippocampectomy (4 cats), operated controls (2 cats), and normal controls (2 cats).

Surgery. Bilateral hippocampectomies were conducted by aspiration through an incision in the ectosylvian gyrus, which penetrated into the lateral ventricles. The hippocampus was thus visualized, and removed as completely as possible by applying suction.

Operated controls were prepared by exposing the ectosylvian gyrus in the same manner as for hippocampectomized animals. An incision was made in the gyrus, penetrating to the lateral ventricles, but all other tissue was left intact.

Operations were conducted under Nembutal anesthesia, and penicillin shots (200,000 units) were given before and after the operation to combat infection. Cilimiacycin was sprinkled into the wound before sewing up the incision. Animals were permitted two weeks for recovery after the operation.

Histology. Since the animals will be retained for further testing, no histological analysis of the lesions will be made.
at this time.

Apparatus consisted of a Wisconsin General Test Apparatus with an automatic timing device which measured intervals as short as 0.10 seconds.

The Wisconsin Apparatus consists of a wooden box divided into two compartments by a guillotine door. One compartment (20" x 20" x 18") houses the animal during the testing session. The other compartment (20" x @20" x 15") contains a tray, 2" high, with three food wells seperated by vertical sheets of plexiglass (see fig.3). Each food well is 2" in diameter and 3/8" deep. Only one food well was utilized in the experiment. A grey wooden block, $2\frac{1}{2}" \times 2\frac{1}{2}" \times 2\frac{1}{2}"$ served to cover the well.

The timing device was turned off automatically by means of a microswitch operated by the sliding door as it began to open at the beginning of a trial. It was turned off automatically as the animal uncovered the well by pushing the block, at the same time interrupting a light beam which activated a photoelectric system and stopped the timer. The time interval measured, between the moment the door began to open and the moment the block uncovered the food well, constituted the response latency.

Method. Each of the animals followed an identical procedure as described below.

**Phase 1. Response to a positive stimulus. (Acquisition.)**

The cats were taught to push the block to uncover the food in five stages. First they were permitted to eat the food from
an open well. The well was then \( \frac{1}{2} \) closed, necessitating a slight push on the block to attain access to the meat. In the last three stages the well was 1/2 closed, 3/4/ closed, and finally fully closed. The criterion for advancement to the succeeding stage was similar to that used by peretz: the cat advanced when the response latencies at any particular stage were 15 seconds or less for five consecutive trials. Cats received 20 trials per day. When the training criterion was achieved with the well fully closed, training was terminated for that day even if 20 trials were not used.

Testing, or measured acquisition of the response, began with the first trial on the following day, also at 20 trials per day, and continued for 7 days (140 trials). On day 8, the first 10 trials were rewarded, bringing the acquisition set to 150 trials. Immediately (trial 11), 20 non-rewarded trials followed, initiating the extinction series, Phase 2. (If the animal failed to respond after 2 minutes, a latency of 120 seconds was recorded. No such trials occurred in Phase 1.)

**Phase 2. Extinction.**

Extinction began on day 8, and continued at a maximum of 2 20 trials per day until the extinction criterion was reached. This criterion consisted of absence of response by the animal on five successive trials for three successive days. (Absence of response was defined as failure to push the block within two minutes after the trial was initiated.) When five such
Fig. 3. The food-tray and food well as the cat sees it when the guillotine door to its compartment is raised. The cat approaches it from the direction of the arrow. The block is shown covering the middle food well and the reward. The plexiglass permits approach from only one side, yet does not obstruct vision.
successive trials were recorded, testing was terminated for that day.

**Phase 3. Re-establishment of the response.**

During the first trial on the day after the extinction criterion was reached, meat was again placed in the food well. This constituted an "information trial"—the animal was informed that the reward was being reinstated. Response latencies were measured for this trial as well as for the following 20 trials. The cats therefore received 21 trials on this day. Testing continued until the animals performed at latencies comparable to the mean latencies of the last three days of the acquisition series.
CHAPTER V

RESULTS

Mean response latencies were computed for each day, dropping the fastest trial and the slowest trial for that day. Previous experience with cats has indicated that these animals will occasionally be distracted from their task. In order to prevent a single extremely long trial from giving an inaccurate estimate of the performance, the longest trial was discarded. The shortest trial was discarded to prevent prejudicing the data in one direction.

Since the speed with which an animal responds may be expected to be dependent on its motivation, the animals in the different groups were matched on their response latencies during acquisition, thus controlling for the motivational level of the animals during testing. It was found that two normal control cats (9N and 11N) had mean response latencies of 1.00 and 1.20 seconds during the last 10 acquisition trials. Mean scores of these two animals were used as the basis of comparison for the lesioned cat (6L) with a similar mean response latency of 1.6 seconds. One operated control animal (6C) was found to have a mean response latency of 2.88 seconds. Its record was matched with the lesioned animal (7L) with similar speed (2.50
Finally one animal in the control group (5C) was found to have achieved a mean response latency of 4.73 seconds. Its records were used as the basis of comparison for two operated animals (4L and 3L) with latencies of 4.49 sec. and 4.58 sec., respectively. In this way, the motivational level of the animals was taken into consideration during data analysis.

**Rate of Extinction**

The mean latencies of the first 20 extinction trials were divided into 4 blocks of 5 trials each (trials 1-5, 6-10, 11-15, and 16-20). The percent increase in latency for each animal during these extinction trials was computed and is found in table 1. On basis of the matching as described above, these increases were compared between the lesioned group and the control group by a t-ratio for paired measures. It was found that the means of the percent increases were significantly different between the two groups (p = .04).

A comparison of the mean number of trials to extinction yielded similar results. The t-ratio for paired measures was significant with p<.01 (Table 2).

Thus, extinction speed was greater for control animals than for hippocampectomized animals, and hypothesis 1 was supported.

Figure 4 represents the graph of the response latencies during the last ten acquisition trials and the first twenty extinction trials, averaged in blocks of five.
Rate of Re-acquisition

The response latencies of each group during re-acquisition were compared with their own acquisition latencies during the last 10 trials of acquisition. Therefore, in this case each group served as its own control (Table 3).

The response latencies of the lesioned group during re-acquisition did not differ significantly from their latencies at the end of the acquisition series. Thus, re-acquisition for this group was nearly instantaneous. The latencies between end of acquisition and re-acquisition did differ \( (p=.04) \) for the control animals. Since the control animals needed a longer time to re-acquire previous response speed, hypothesis 2 was supported.

Figure 5 is a graph representing the mean response latencies of the two groups during the last 10 acquisition and first 20 re-acquisition trials.
### Table 1*

<table>
<thead>
<tr>
<th>Cat #</th>
<th>Acquisition latencies, last 10 trials (sec)</th>
<th>Extinction latencies, first 20 trials (sec)</th>
<th>% Increase (%x.1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>8L</td>
<td>1.60</td>
<td>7.17</td>
<td>34.8</td>
</tr>
<tr>
<td>7L</td>
<td>2.50</td>
<td>36.56</td>
<td>136.4</td>
</tr>
<tr>
<td>4L</td>
<td>4.49</td>
<td>55.85</td>
<td>114.3</td>
</tr>
<tr>
<td>3L</td>
<td>4.58</td>
<td>13.50</td>
<td>19.5</td>
</tr>
<tr>
<td>9N</td>
<td>1.00</td>
<td>3.34</td>
<td>23.4</td>
</tr>
<tr>
<td>11M</td>
<td>1.20</td>
<td>53.69</td>
<td>437.5</td>
</tr>
<tr>
<td>6C</td>
<td>2.88</td>
<td>68.82</td>
<td>229.0</td>
</tr>
<tr>
<td>5C</td>
<td>4.73</td>
<td>82.83</td>
<td>165.1</td>
</tr>
</tbody>
</table>

Mean: 

| Cat # | | | |
|-------| | | |
| 9N    | | | |
| 11M   | | | |
| 6C    | | | |
| 5C    | | | |

Difference between means: 117.3

t: 2.36

df: 3

p = .04

*Animals designated by L have hippocampal lesions; those designated by N are normal controls; those designated by C are operated controls.
Fig. 4. Mean response latencies of the last ten acquisition trials (ACQ), and the first twenty extinction trials (EXT), averaged in blocks of five trials each.
### TABLE 2*

**EXTINCTION TRIALS TO CRITERION**

<table>
<thead>
<tr>
<th>Cat</th>
<th>Trials to criterion</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8L</td>
<td>52</td>
<td></td>
</tr>
<tr>
<td>7L</td>
<td>27</td>
<td></td>
</tr>
<tr>
<td>4L</td>
<td>17</td>
<td>Mean: 32.25</td>
</tr>
<tr>
<td>3L</td>
<td>33</td>
<td></td>
</tr>
<tr>
<td>5C</td>
<td>13</td>
<td></td>
</tr>
<tr>
<td>6C</td>
<td>13</td>
<td>Mean: 20.50</td>
</tr>
<tr>
<td>9N</td>
<td>37</td>
<td></td>
</tr>
<tr>
<td>11N</td>
<td>19</td>
<td></td>
</tr>
</tbody>
</table>

Difference between means: 11.75
\[ t; 4.79 \]
\[ df: 3 \]
\[ p < .01 \]

*See note below table 1.*
<table>
<thead>
<tr>
<th>Cat #</th>
<th>Last 10 acquisition latencies (sec.)</th>
<th>First 20 re-acquis. latencies (sec.)</th>
<th>Diff. betw. means: 0.55</th>
</tr>
</thead>
<tbody>
<tr>
<td>8L</td>
<td>1.60</td>
<td>3.73</td>
<td>t: 0.687</td>
</tr>
<tr>
<td>7L</td>
<td>2.50</td>
<td>3.42</td>
<td>df: 3</td>
</tr>
<tr>
<td>4L</td>
<td>4.49</td>
<td>2.81</td>
<td>p: ns</td>
</tr>
<tr>
<td>3L</td>
<td>4.58</td>
<td>5.41</td>
<td></td>
</tr>
<tr>
<td>Mean:</td>
<td>3.29</td>
<td>3.84</td>
<td></td>
</tr>
<tr>
<td>9N</td>
<td>1.00</td>
<td>1.17</td>
<td>Diff. betw. means: 2.72</td>
</tr>
<tr>
<td>11N</td>
<td>1.20</td>
<td>4.39</td>
<td>t: 2.49</td>
</tr>
<tr>
<td>6C</td>
<td>2.88</td>
<td>8.31</td>
<td>df: 3</td>
</tr>
<tr>
<td>5C</td>
<td>4.73</td>
<td>6.81</td>
<td>p: .04</td>
</tr>
<tr>
<td>Mean:</td>
<td>2.45</td>
<td>5.17</td>
<td></td>
</tr>
</tbody>
</table>

*See note below table 1.
Fig. 5. Mean response latencies of control and lesioned animals during the last ten acquisition trials (ACQ) and the first twenty re-acquisition trials (RE).
CHAPTER VI

DISCUSSION AND SUMMARY

Apparently the results support Peretz's hypothesis that one of the effects of hippocampectomy is the increased effectiveness of positive reinforcement. However, some studies report results that are contrary to what this theory would predict. Other results, although not contradicting Peretz's theory, can be explained in simpler ways.

The study by Roberts, Dember, and Brodwick, for example, reported increased exploratory behavior in rats with hippocampal lesions. Although exploration can be regarded as a positive reinforcement, we do not need to go to this dfactor to explain the results. An increase in the general activity level could explain the results just as well.

Thomas and Otis reported in their experiment that lesioned rats ran faster in a maze than a control group. Yet their maze performance was inferior to that of the controls. The positive reinforcement theory can therefore account for only a part of the results. Strong evidence countering the interpretation proposed by Peretz is provided by Grossman and Mountford, who induced hippocampal dysfunction by means of KCl injections into the

45.
structure. Contrary to expectations, these authors found a more rapid extinction in KCl injected rats than in controls. Furthermore, injections into the hippocampi of both sated and 23-hr food deprived animals did not result in increased consumption of food. They report that the animals' behavior was similar to that of animals working under low motivational conditions.

As already mentioned in Chapter II, lesions in the hippocampus produce impairment in learning and retention of successive visual, tactual, and auditory discriminations, as well as in maze learning. Yet in all these studies, positive reinforcement was present. According to Peretz's reinforcement theory, all post-operative learning should be accelerated due to the increased effect of the reinforcement. According to the perseveration theory, postoperative retention of a preoperatively learned task should be improved, provided no reversal is required. Since the results mentioned above are clearly opposed to such a conclusion, neither theory adequately accounts for the existing findings.

Arnold (1960) has presented a comprehensive theory of hippocampal function which might take into account the available data. Although not all aspects of the theory have been investigated, its general characteristics are briefly presented here.

According to the theory, the hippocampal system is composed of the hippocampus, the hippocampal rudiment (indusium griseum) and the fornix. The system is responsible for the retrieval of modality-specific impressions by sending impulses to the sensory
nuclei of the thalamus and the cortical association areas, where these impressions are reactivated. Different regions of the hippocampus serve different sense modalities, depending on the portions of the limbic system that are nearest the hippocampus at that point, and which primary sensory areas that portion of the limbic system serves. Thus, in the case of motor memory, the modality is served by the subcallosal and cingulate gyri, which are connected to the indusium griseum. Transection of the rudiment at the proper location can be expected to impair motor memory.

The hippocampus itself is important in the retrieval of visual, auditory and some tactual cues. Experimental results involving lesions of the hippocampus are therefore dependent to a large extent on the location of the lesion.

Affective memory, on the other hand, is mediated by the limbic system, including the hippocampal gyrus, fornix, mammillary bodies, anterior thalamic nucleus, and cingulate gyrus. This involves the appraisal of a percept as "good and desireable for me" or "bad for me".

Since the hippocampus is involved in sensory memory, all tasks involving retrieval of sensory information are expected to be difficult for lesioned animals. This is why such animals are impaired in successive discrimination tasks. Simultaneous discrimination involves no retrieval, and the animals are not impaired. The theory does have difficulty explaining the results of Orbach, et al., and Mahut and Cordeau (discussed in Chapter II), who
found that monkeys with hippocampal lesions were not impaired in a delayed alternation task with long delays.

In Peretz's and the present study, no discrimination was involved—only an appraisal of the food well and block as "good for me". Since the hippocampus is not involved in this function, no impairment in acquisition of the task occurred in lesioned animals.

Summary

A motor-response for a food reward was established in four hippocampectomized, two normal, and two operated control cats. The response was extinguished by omitting the reward, and was then re-established. Extinction rate was found to be greater for control animals than for animals with hippocampal ablations. Re-acquisition was found to be more rapid for hippocampectomized animals than for controls.

It was shown that the positive reinforcement theory can account for the results observed by Peretz and those of the present experiment, and that the perseveration theory can account for other experimental findings. Neither theory can account for all the results obtained by different investigators, and in many instances both theories would predict the opposite results. They are clearly inadequate. It was suggested that Arnold's theory of hippocampal function can account for many of the results not adequately covered by other theorists, and that her theory predicts the present findings also.


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Allen, W.F. Fiber degeneration in Ammon's Horn resulting from the extirpation of the piriform and other cortical areas and from transection of the horn at various levels. J. Comp. Neurol., 1948, 88, 425-438.


Arnold, Magda B. 1966. Personal communication.


The thesis submitted by Peter G. Kaufmann has been read and approved by three members of the Department of Psychology.

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

The thesis is therefore accepted in partial fulfillment of the requirements for the Degree of Master of Arts.

Date

Signature of Adviser

May 26, 1966