1-1-1971

Position and response learning in hippocampal lesioned rats.

Jean Paul E. Laroche
University of Windsor

POSITION AND RESPONSE LEARNING IN HIPPOCAMPAL LESIONED RATS

by

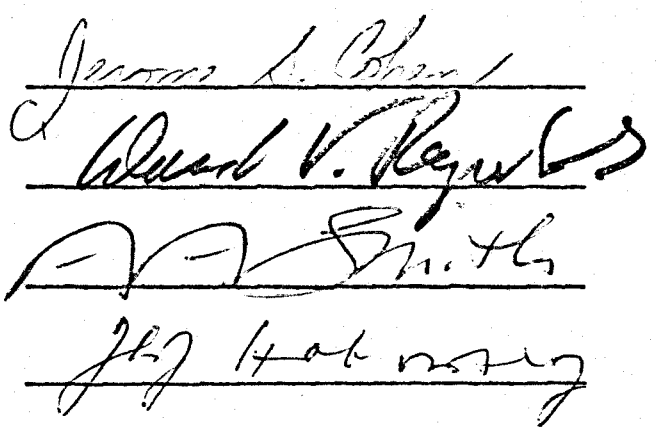
Jean Paul E. Laroche
B.A., Loyola College, 1969

A Thesis
Submitted to the Faculty of Graduate Studies through the
Department of Psychology in Partial Fulfillment
of the Requirements for the Degree of
Master of Arts at the
University of Windsor

Windsor, Ontario, Canada

1971

APPROVED BY:



ABSTRACT

The present study was designed to investigate the response and stimulus hypotheses of perseverative locomotor behavior observed in hippocampal lesioned rats. The response cue hypothesis maintains that hippocampal lesioned animals suffer from an inability to inhibit an ongoing locomotor response (Kimble, 1968). The stimulus cue hypothesis states that these animals suffer from an inability to inhibit attention to previously responded to stimuli (Douglas, 1967).

Forty-eight albino rats were subjected to either bilateral hippocampal lesions or sham operations. An equal number of hippocampal and sham animals were placed into four brightness groups. Subjects were required to learn a left-right discrimination in a T maze supplied with either black versus black, black versus dark grey, black versus light grey, or black versus white doors at the choice point. After learning had occurred, the animals were required to respond to the same maze arm when run from a start box 180 degrees from the original. Based on a previous study by Cohen, Laroche, and Beharry (1971), it was hypothesized that response perseveration usually observed in hippocampal lesioned rats would decrease as the brightness differences become greater at the choice point.

The results did not confirm the hypothesis. No significant differences were observed between the hippocampal and

sham lesioned animals. Failure to obtain differences was discussed in terms of possible external inhibition supplied by the doors to the normally inferiorly inhibited hippocampectomized subjects. A study to investigate this possibility was proposed.

PREFACE

The present author is greatly indebted to Dr. J.S. Cohen for his patience, guidance, and enthusiasm in this study. Dr. A.A. Smith offered invaluable advice and assistance in both statistical and design aspects of this paper. Problems in physiological methodology were greatly reduced by Dr. D. Reynolds.

Technical assistance was received from Mr. Bill Soames, who was responsible for constructing the apparatus. Mrs. Marla Howitt was patient and more than capable in typing the present paper. My wife, Zena, also spent hours typing and a year and a half supplying incentive and moral support to an at times discouraged author.

A word of thanks is also given to Dr. J.E.J. Habowsky of the Biology Department, for his contribution in participating as the outside reader.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

LIST OF TABLES (Continued)

CHAPTER I

INTRODUCTION

Statement of the Problem

Perservative locomotor responding has been shown to be a reliable behavioral deficit in animals with hippocampal lesions. Kimble (1968) maintains that animals with hippocampal lesions suffer from an inability to stop an ongoing motor response regardless of external stimulus change. This is an example of a deficit of internal inhibition. Douglas (1967), however, contends that such animals suffer from a deficit in stimulus habituation. That is, the hippocampal injured animal is unable to stop responding to a previously attended to external stimulus.

It was the purpose of the present study to investigate the response inhibition and stimulus habituation hypotheses of perseveration by using hippocampal lesioned rats. Difference in brightness cues were varied in a maze learning task requiring the reversal of a previously learned turning response into the arm of a T maze.

Background and Related Research

The Hippocampus (Ammon's horn) is a combination of pyramidal and dentate areas making up most of the horn-like structure which lies along the floor of the inferior horn of each lateral ventricle. The hippocampal formation,

according to Green (1960), is the hippocampus and dentate gyrus along with their adjacent and continuous regions of the brain and their main afferent and efferent pathways. These pathways lead largely to the subiculum and entorhinal cortex, the cingulate gyrus and amygdala, the hippocampal commissure, the septum, and the fornix.

Ammon's horn has been found to be related both to the brain stem and the cortex. The main connections of the brain stem and hippocampus enter through the septum lucidum among the fibers of the fornix system. Fibers of the post commissural fornix descend deeply behind the anterior commissure and pass through the hypothalamus to the mammilary bodies. Fibers of the precommissural fornix enter the septum and pass in front of the anterior commissure. The hippocampus-cortex connections are chiefly existent in the temporo-ammonic tract, connecting the entorhinal cortex to the hippocampus. Green (1960) cites studies where stimulation of the hippocampus led to evoked potentials in the entorhinal cortex but only to slight and irregular potentials in the fornix. Large evoked potentials were found in the hippocampus following stimulation of the fornix. The implication is that the main pathways from the brain stem to the hippocampus are afferent to the hippocampus. The pathways connecting the cortex and the hippocampus are efferent from the hippocampus. A further implication is that the hippocampus is involved with a type of information control,

mediating afferent stimulation from the brain stem to the cortex.

Penfield and Milner (1958) found that bilateral damage to the hippocampus in humans led to a deficit in recent memory. Memory of events as far back as two or three years prior to the damage and memory of current experience were seriously impaired. However, work skills and concepts that had been acquired two or three years preoperative were not impaired. Milner (1965) concluded that human subjects with bilateral hippocampal damage could sustain memory of current experience as long as no distracting stimuli were present, implying that the observed deficits might be due to some malfunctioning of the consolidation process. In the animal research, however, results have not completely replicated human short term memory deficits. Kimble (1968) reports that in rats and monkeys, only in successive discrimination tasks for both acquisition and retention, is there a deficit due to hippocampal lesions. Animal deficits have more reliably been found in tasks requiring a change or reversal of an ongoing or previously learned response. Kimble considered that dual deficits in the consolidation process and in action decrement (Walker, 1958) occurred in both human and animal subjects with hippocampal damage. The action decrement hypothesis holds that inhibition of responding to current stimulation is necessary before memory consolidation can occur. Kimble proposed that an inhibitory deficit due to

hippocampal damage could explain both the animal deficits in response change and the human deficits in recent memory. In animals, the inability to inhibit responding to current stimulation would incapacitate the animal to respond to another stimulus. In humans, the inability to inhibit responding to distracting stimuli would prevent consolidation.

To test the short term memory hypothesis, Kimble (1963) ran hippocampal lesioned experimental, and operated and unoperated control group rats in a simultaneous discrimination task with an 8 min. intertrial interval. No differences in acquisition were observed between the three groups. These results were contrary to predictions from the short term memory hypothesis. Jarrard, Isaacson, and Wickelgren (1964) tested hippocampal ablated animals in a runway acquisition task. They found no differences between animals with a 10 sec. intertrial interval and a 10 min. intertrial interval. Jarrard et al. concluded that the short term memory hypothesis of hippocampal functioning is not applicable to infrahuman animals.

As discussed above, hippocampal damage in animals does not result in the same short term memory deficits as found in humans. Other physiological techniques besides lesions, such as electrophysiological recording and stimulation, however, have pointed to a possible attention monitoring process of the hippocampus. It should be noted, however, that these two main approaches are not mutually exclusive.

For example, Grossman and Mountford (1964) used a technique whereby they produced a temporary lesion in the hippocampus by the administration of KCl and recorded electrical activity in this same structure at the same time. Brunner and Rossi (1969) used hippocampal lesioned groups and hippocampal stimulated groups in a passive avoidance task. The authors came to the conclusion that it is possible for hippocampal stimulation to disrupt functioning as does lesioning.

In the electrical studies, a general finding has been that hippocampal slow wave (4-7 cps) activity is correlated with neocortical arousal. Green and Arduini (1954) reported an inverse relationship between hippocampal activity and neocortical activity in rabbits, cats, and monkeys. Basically, when the cortex showed a desynchronized pattern, hippocampal slow waves were observed. When cortical spindle waves were recorded, hippocampal desynchronization was present. This relationship was found to be most easily observed in the rabbit. In the cat, slow waves in the hippocampus were accompanied by fast desynchronized activity in this same structure. The hippocampal slow wave trains were shorter than in the rabbit. In monkeys the slow waves were even more difficult to observe, were of shorter duration, and were interspersed with even more desynchronized activity. In all these animals, the slow wave activity was observed after afferent stimulation which caused an arousal response. Green and Arduini concluded that the slow waves in the

hippocampus were also a type of arousal response. This view that theta (4-7 cps) wave activity in the hippocampus was an arousal reaction was also strengthened by the observation that it appeared as a result of stimulation of the reticular formation. The fact that theta rhythms were found in the hippocampus even after decortication suggested that the arousal reactions of the hippocampus and neocortex, while having a lawful relationship, were not entirely interdependent. It can also be observed that the obtained results concur with the fact that the human data is different from the animal data. Proceeding from rabbit to cat to monkey, it can be found that the relation of theta activity in the hippocampus to desynchronized activity in the neocortex is more obscured. It may be that, advancing to man, the relationship no longer exists and the functions of the hippocampus are of a different nature.

In another experiment relating hippocampal slow wave activity to neocortical arousal, Holmes and Adey (1960) recorded activity from the hippocampus and entorhinal cortex in cats in a delayed response situation. The animals were delayed on a platform until a bridge was lowered which enabled them to approach one of two pans. One pan contained food that was placed in it while the animal was watching. The authors found that hippocampal slow waves were present initially during approach to the goals and gradually disappeared as 100% correct responses were reached. These slow

waves reappeared during extinction. Holmes and Adey concluded that hippocampal theta rhythms, which were not observed in spontaneous locomotor activity, are involved with an aroused state of the organism during goal directed motor activity. Pickenhain and Klingberg (1967) tested rats in a delayed response situation where a series of light flashes was paired with shock. The authors found that after conditioning had occurred, initial presentations of the light were accompanied by hippocampal slow waves. The slow waves then disappeared and returned. They were greatest in frequency just before avoidance behavior was elicited. The experimenters concluded that in rats, slow hippocampal activity is correlated with not yet automated motivational behavior.

Grastyan (1959) and Grastyan, Lissak, Madarasz, and Danhoffer (1959) have stated that the hippocampal theta rhythm corresponds to a state where familiar but still "uncertain" stimuli are present, i.e., stimuli that are familiar but new to the present situation. These investigators implied that the theta rhythm corresponds to a state of orientation to uncertain stimuli and at the same time represents a state of inhibited attention to other stimuli. Theta activity in the hippocampus is considered to occur in the initial states of learning, before the response to the stimuli becomes automated.

Grastyan et al. (1959) found in cats that during

conditioning the CS on initial presentation elicited orienting responses and hippocampal slow waves. After orienting responses disappeared, hippocampal desynchronization was observed with the presentation of the now conditioned stimulus. These findings, along with the finding that hippocampal stimulation inhibits orienting or alaimentary or defensive reflexes (Grastyan, 1959), led the author to view the theta rhythm as an indication of a non functioning state of the hippocampus. According to this hypothesis, ongoing attention cannot be inhibited by the hippocampus when it is in a theta state. Inhibition of attention is a function associated with the hippocampus when it is in a desynchronized condition. According to Grastyan's hypothesis, it follows that a lesioned hippocampus can be considered to be effectively in a theta state, where the attention of the initial stages of learning cannot be inhibited and consequent attention to new stimuli that have become relevant is defective. Thus, according to this model, the prediction is that perseverative behavior will appear in the hippocampal lesioned animals during a reversal task because the necessary changes in attention cannot occur.

In summary, it seems that in general the electrophysiological findings indicate that attentional flexibility is mediated by the hippocampus. Green and Arduini (1954) conclude that hippocampal theta rhythms are related to afferent stimulation which causes arousal. Holmes and Adey

(1960) refer to the theta rhythm as being related to arousal in goal directed motor behavior and Pickenhain and Klingberg (1967) find that the theta rhythm is related to not yet initiated, motivated (goal directed) behavior. Grastyan's (1959) model is perhaps the most directly attentional. He hypothesizes that the theta rhythm is related to behavioral orienting to familiar but not yet conditioned stimuli.

On the behavioral level, one finding has been that animals with hippocampal lesions are deficient in their ability to stop an ongoing motor response or to reverse a previously learned locomotor response. In testing for the effects of visual and auditory distracting stimuli on an ongoing locomotor response, Riddell, Rothblatt, and Wilson (1969) trained hippocampal lesioned, neocortical lesioned, and unoperated rats to run a straight alley runway for food reward. The authors found the hippocampal lesioned animals to be less distractable than the two control groups when running speed was used as a measure of distractability. Cohen and Swenson (1970) observed that hippocampal lesioned rats did not perseverate previous responding to the exploratory (E) arms of a straight runway. The animals continued the response of running down the straight alley. In studying the effect of hippocampal lesions on spontaneous alternation in a T maze and on free locomotor exploration of an unfamiliar environment in rats, Roberts, Dember, and Brodwick (1962) found that hippocampal lesioned rats had significantly lower

alternation scores and higher locomotor exploration scores than did rats in a non lesioned control group. Leaton (1965) established base rates for exploratory behavior in a T maze and then observed exploratory locomotor activity after hippocampectomy in rats. He found that the hippocampal lesioned animals had significantly higher rates of exploratory activity than did cortical lesioned and sham operated control animals. Jackson and Strong (1969) tested hippocampal ablated animals in a Lashley 3 box and found that these animals made significantly more "door errors" (run straight past the door into a cul) then did control animals. In testing for habit reversal in a brightness discrimination task, Silveira and Kimble (1968) found that hippocampal lesioned animals took significantly more trials to learn the reversal because they consistently perseverated responding to one arm. Interpretations of the above findings have generally taken one of two positions.

One interpretation, supported by Douglas (1967), is that hippocampal lesions lead to deficits in habituation to the stimuli controlling the response. This deficit in external (stimulus) cue inhibition is represented in Douglas' model as a deficit in the stimulus gating mechanism, causing hippocampal lesioned animals to continue responding to previously reinforced but presently inappropriate stimuli. Roberts et al. (1962) maintain that since hippocampal damaged and control animals reacted similarly in the initial

phases of both the alternation and exploratory phases of their experiment, the differences that were observed in later trials indicate differences in susceptibility to familiarization. Also, hippocampal lesions may have impaired the inhibitory processes through which memory can reduce the exploration of remembered and thus familiar stimuli. In Leaton's (1965) study, hippocampal lesioned rats were found not to decrease running speeds on forced trials, whereas control animals did. These results indicated to Leaton that hippocampal damaged animals did not habituate to novel stimulation as well as did the control animals. Leaton went on to suggest that the destruction of the hippocampus would produce deficits in learning to the extent that habituation was important to the problem. The author discussed the memory and inhibition hypotheses proposed by Roberts et al. (1962). It was concluded that because the hippocampectomized animals responded differently on forced trials to the exploratory and neutral boxes, the deficit was an inability to habituate rather than an inability to remember. The exploratory end box was defined as the one which contained little springs, blocks, and other novel objects. Jackson and Strong (1969) found that hippocampal lesioned animals made more door errors in a Lashley 3 box. The hippocampal animals did not, however, make more door errors at door 6, from where the goal box was visible. These animals made more door errors at the first five doors, where nothing but

the doors and the walls behind them were visible. The authors interpreted these results to suggest that the hippocampus relates to attentive functioning more than it does to locomotor perseveration. They suggested that door 6 was a more salient stimulus because the goal box was visible from it. Silveira and Kimble (1968) also concluded that hippocampal lesioned animals were deficient in attentional processes. Specifically, even after the animals stopped responding to the previously reinforced brightness cue, attention to new relevant cues was still found to be inhibited.

In a study designed specifically to test the stimulus cue hypothesis of perseverative behavior in hippocampal lesioned rats, Kirkby, Stein, and Kimble (1967) investigated spontaneous alternation in hippocampal lesioned and control animals after varying lengths of confinement (50 sec., 10 min., and 50 min.) in the first choice arm of a T maze. The authors observed that only the hippocampal animals in the 50 sec. condition failed to alternate consistently. These results led Kirkby et al. to conclude that damage to the hippocampus impairs the rate of information acquisition, thus increasing the amount of time needed for satiation to novel stimuli. If this is the case, the present author maintains that it might require more salient stimulus differences to allow for faster acquisition leading to discrimination. Ackil, Melgren, Halgren, and Frommer (1969) used 0 and 30

preexposures in a shuttle box to test for differences in habituation and attention in hippocampal lesioned and control animals. They found that 30 preexposures did not interfere with the acquisition of an avoidance response in the hippocampal damaged animals. These preexposures did, however, interfere with acquisition in the operated and unoperated control groups. Hippocampal lesioned animals were also different in so far as they failed to reduce initial shuttling during the preexposure trials. Hippocampal operates resisted extinction more than did the control groups. It was observed that there were no differences between hippocampal operated animals in the 0 and 30 preexposure groups. Ackil et al. interpreted these results to indicate that there was a deficit in stimulus integration by the hippocampal lesioned animals. Ellen and Deloache (1968) investigated spontaneous alternation in hippocampal, cortical, and sham lesioned rats in a T maze. Brightness cues (black and white inserts) in the choice arms and spatial direction cues (east or west position) of the main alley were varied to study their effects on alternation behavior. The results indicated that the hippocampal and control groups both alternated above the chance level when brightness cues and spatial cues were varied so that alternation to one or both cues led to the same response. When brightness and spatial cues were arranged so that alternation to one set necessitated repetition of response to the other, hippocampal lesioned animals

alternated at chance levels and control animals continued to alternate above chance levels to spatial direction cues. Ellen and Deloache interpreted these results to mean that hippocampal damaged animals responded primarily to external brightness cues. Control animals responded to position cues.

Other studies of perseverative responding in hippocampal lesioned rats have led to another non attentional interpretation. Kimble (1968) proposed an internal Pavlovian inhibition concept to explain response perseveration in hippocampectomized animals. In Kimble's response (internal) cue model, the animal is considered to perseverate as a result of an inability to stop an ongoing or previously learned response. Riddell et al. (1969) interpreted their results as supporting this hypothesis. In their study, hippocampal lesioned animals were found to be distractable to some extent, though not as much as control animals. The fact that the hippocampal operates showed any signs of distractability, however, led the authors to agree with Kimble's (1968) hypothesis. They concluded that the observed deficit was one of an inability to stop the ongoing motor response. In the Cohen and Swenson (1970) study, hippocampal damaged rats ran straight past the previously responded to side arm and continued along the straight alley. Cohen and Swenson agreed with the response cue hypothesis. They concluded that the running response to the straight alley was

perhaps too strong for the hippocampal lesioned animals to inhibit it.

Means (1969) and Dalland (1970) used procedures similar to Ellen and Deloache (1968) to determine whether hippocampal lesioned animals respond to stimulus or response cues. Means found that when rats were tested in cross maze problems designed to separate response from position cue responding, hippocampal damaged rats perseverated response cues in reversal training significantly more than did sham operated animals. Although all groups used response cues after over-training, control animals responded to exterioceptive cues while the hippocampal lesioned animals did not. In a similar experiment, Dalland compared rats with hippocampal lesions, septal lesions, and sham lesions in a T maze alternation test. As a means of determining whether stimulus or response cues were the ones that dominated responding, the main runway was turned 180 degrees after alternation testing. In this new situation, if the response cues were the perseverated ones, the animals would have to alternate arm entries. If the stimulus cues were the perseverated ones, the animals would have to perseverate turning responses. The results showed that the hippocampal lesioned animals perseverated response cues. The above results lead to conclusions that differ from those found in the Kirkby et al. (1967), Ackil et al. (1969), and Ellen and Deloache (1968) studies. Different (stimulus versus response cue perseveration)

interpretations of perseverative responding in hippocampal lesioned animals have been supported by similar types of experiments where this type of behavior has been observed.

Perseverative locomotor responding in hippocampal damaged rats, however, has not always been observed. Jackson and Strong (1969) found that when animals had to learn left-right-left-right sequences in a two bar Skinner box, the hippocampal lesioned animals did so in significantly fewer trials and were also able to learn longer sequences than were control animals. This was also the case in three bar sequences (e.g., A-B-A-B-C). The experimenters concluded that unless the sequence could be considered to be one response, the perseveration hypothesis could not account for the superior performance of the hippocampal operates.

Some studies support an attentional (exterioceptive stimulus) deficit (Roberts, Dember, & Broderick, 1962; Leaton, 1965; Jackson & Strong, 1969; Silveira & Kimble, 1968; Kirkby, Stein, & Kimble, 1967; Ackil, Melgren, Halgren, & Frommer, 1969; Ellen & Deloache, 1968), while others support a response perseverative deficit per se (Riddell, Rothblatt, & Wilson, 1969; Cohen & Swenson, 1970; Means, 1969; Dalland, 1970) in hippocampal lesioned rats. A recent study by Cohen, Laroche, and Beharry (1971) may shed some light on these contradictory results. Cohen et al. observed perseverative behavior in hippocampal lesioned rats in a T maze situation when the stimulus qualities of the maze arms

were the same but not when they were more distinctively different. Rats were taught a left-right discrimination in the T section of a cross maze. Afterwards, they were tested for perseveration by changing the start box to the opposite side. Hippocampal lesioned rats were found to perseverate the turning response in the reversal situation and to take significantly longer than the operated control animals to reach extinction. In this case, both choice arms were the same neutral grey color. In the second experiment the procedure remained the same except that one choice arm was painted flat black. This procedure did not lead to the above stated differences between the hippocampal lesioned and the operated control animals. The results in the minimal cue difference situation support the response inhibition findings of Means (1969) and Dalland (1970) who used similar procedures. The results in the maximal cue difference situation, however, are in opposition to these findings and support a stimulus bound interpretation of hippocampal functioning. An hypothesis to explain the findings of Cohen et al. (1971) is that response perseveration will be evident in hippocampal lesioned animals when cue differences are not great enough to allow the hippocampal operates to integrate these differences. As already mentioned, this ability to integrate stimuli has been observed by many researchers to suffer a deficit after damage to the hippocampus.

The present study was designed to study the effects of

brightness cue differences on response perseveration in hippocampal lesioned rats. The procedure used is essentially the same as in the Cohen et al. (1971) study except that the animals are additionally required to push through a black, dark grey, light grey, or white door which is paired with a black door at the choice point. Specifically, several sham operated control groups and hippocampal operated experimental groups were randomly assigned to one of the black-black, black-dark grey, black-light grey, or black-white door combinations. They were trained to make a left or right turn to enter the choice arm of a T maze. After learning this task, a rat was then tested for perseverative responding by being started 180 degrees from the original start box. The hypothesis derived from Cohen et al.'s (1970) research maintains that only in those groups of rats presented dark grey versus black, and black versus black choices, should a greater response perseveration be found in hippocampal damaged rats than sham operates. Less perseverative behavior should be found in hippocampal lesioned rats as a function of increased brightness differences between choice point doors.

CHAPTER II

METHOD

Subjects

Subjects were 48 male albino rats bred at the colonies of Woodlyn Farms, Guelph, Ontario. Each subject was between 90 and 110 days old at the time of operation and weighed approximately 300 grams. Half were randomly chosen as the experimental group, hippocampal operates (HP). The other half were used as the control group, sham operates (Sh).

Apparatus

A cross maze was used. The main runway was 172 cm. long x 10 cm. wide x 14 cm. high and was equipped at either end with a wood covered start box 22 cm. long. The choice arms were 80 cm. long and a metal drinking tube protruded 4 cm. from the floor at the end of each arm.

The maze was illuminated by a 7.5 watt frosted light, one foot over the choice point. Two more of these bulbs were located 7 cm. above the drinking tubes at the end of each of the choice arms. A stimulus door, either black, dark grey, light grey, or white was placed in the right choice arm at a point 1 cm. inside from the main runway. The left arm was always equipped with a black door. Each door was hinged on the choice arm so that a rat could push it open, thus gaining entry into the arm.

The entire maze, except for the choice point, was covered by $\frac{1}{2}$ x $\frac{1}{2}$ inch wire mesh. Plexiglass covered the choice point. In an attempt to reduce extraneous visual cues, cheese-cloth was placed over the wire mesh and extended vertically along the stand which held the 7.5 watt light bulb in place at the choice point. The board to which the 7.5 watt bulb was secured blocked direct vertical vision at this point.

A 60 second stop-watch was used to record latencies in running from the raising of the start box door to the commencement of drinking.

Procedure

Surgery. After a one week handling period, all animals were subjected to surgery under aseptic conditions. Each subject received a 3 cc. intraperetenail injection of Nembutal solution (1 mg./kg.). The skull was exposed and the animal was placed in a Stoelting sterotaxic instrument. Two small holes were drilled in the skull to allow for passage of the lesioning electrode. A stainless steel insulated electrode was used (Clay-Adams insect pin, size 00). The electrode was insulated except for 3 mm. on one side of the electrode tip. The exposed portion was inserted so that it faced the cortex, leaving the thalamus protected by the insulated side of the electrode. The electrode was entered 2 mm. interior to the inter-aural line, $5\frac{1}{4}$ mm. bilaterally from mid point, and 6 mm. down from and perpendicular to

the skull surface. A current of 30 ma. was administered for 5 seconds. A Grass lesion maker, model LM4, was used to supply the current.

The same procedure was used for the sham operates except that no current was passed through the electrode.

All animals were given a seven to 10 day recovery period before the beginning of pretraining.

Pretraining. Animals were placed on a 23 hour deprivation schedule one day before the start of pretraining. Each subject (S) was run individually. On the first day, the animals were placed in the left then right choice arms for five minutes. A 10% sucrose solution was placed at the end of each arm and the number of runs back and forth was recorded.

On day two, the same procedure was used except the sequence of arm placement was reversed.

The third day Ss were allowed to run from one of two start boxes to either choice arm, whereupon they were allowed to drink for 10 seconds. The animals were then handled for 30 seconds. Half the rats were run from start box A and half were run from start box B. Each S received six massed trials in this manner. The two black stimulus doors were left completely open. Pretraining on days four and five followed the same procedure except that the doors were left half open during the trials on day four. On day five, the doors were left slightly open for the first three

trials and were closed for the last three. This progression of door position was used in order to train Ss to push the doors open.

Running time and arm choice were recorded on each trial. Upon completion of the 18 pretraining trials, the number of left and right choices by each animal was noted and the sides chosen most frequently by each were designated as the preferred arm.

When the preferred arm for each animal had been determined, animals in each surgical group were randomly divided into four stimulus pair combinations. Six animals were chosen for each combination. Depending on which group they were in, animals had to choose arms blocked off by either black doors (B-B group), black versus dark grey doors (B-G^2 group), black versus light grey doors (B-G^1 group), or black versus white doors (B-W group). In the present study the black door was always positioned on the left arm. Shades of B, G^2, G^1, and W corresponded to numbers 2, 4, 6, and 8 on Munsell card series.

The reinforced arm was determined from the last three days of pretraining trials. Half of the Ss showing a preference were reinforced in the non preferred arm and half were reinforced in the preferred arm. For the Ss showing no preference, half were given reinforcement in one arm and the other half were given reinforcement in the other arm. A 10% sucrose solution was made available at the end of the

reinforced arm. An empty drinking tube was positioned at the end of the other. The alleys were wiped with a damp cloth after each trial in an attempt to prevent odour traces.

Training. All Ss received 10 massed, corrected trials per day. They were run from the same start box used in pre-training. A trial consisted of as many runs as it took the animals to enter the correct, reinforcing arm. If S entered the incorrect arm, it was left there for 30 seconds, then replaced in the starting chamber. If the correct arm was chosen, the animal was allowed to drink for 10 seconds and was then removed and handled for 30 seconds. Once S was placed in the start box, immediate access to the rest of the maze was given. If an animal did not reach the stimulus doors within 5 minutes or did not enter a choice arm within 10 minutes, he was replaced in his home cage and run the next day. If failure to enter occurred for three days in a row or for a total of five days, the animal was removed from the experiment.

Testing. After Ss had learned to a criterion of 18 out of 20 correct runs (two consecutive days) they were started from the opposite start box and reinforced the same choice arm as in the training phase. The same criterion of 18 out of 20 correct runs was used in this part of the experiment.

Extinction. Animals were run from the same start box used in testing. However, neither water tube delivered re-

inforcement. Entry into the incorrect arm did not lead to a 30 second confinement period. Confinement was a condition contingent upon entry into the formerly reinforced choice arm. After entry into the formerly correct arm, Ss were confined there for 30 seconds and then handled for 30 seconds before the beginning of the next trial. The criterion of extinction was a latency of 60 seconds to enter the previously correct arm on two consecutive trials. Ten extinction trials per day were carried out. Latencies and number of errors (i.e., entering the originally non reinforced arm) were recorded.

Perfusion. After experimentation, all animals were sacrificed with ether and perfused through the heart with .09% physiological saline and 10% formalin. The head of the perfused animal was removed and the brain was exposed and left to stand in 10% formalin for at least 48 hours. The brains were then removed from the skulls and allowed to stand in 10% formalin for at least another 48 hour period.

Histology. A frozen sections technique for examining 50u coronal sections was carried out according to procedures described by Hutchinson and Renfrew (1967). Visual examination of coronal sections of the brain were made until evidence of a lesion became visible. Every fifth 50u section was then mounted on a slide and an image of it was projected on a screen. The images were recorded by photographs from a 35 mm. camera. If evidence of a bilateral

hippocampal lesion which did not invade the thalamus was not obtained in the experimental animals, their data was not used in the experiment. A visual examination of the sham operated animals was also made to check for irregularities. Photographs of approximately every third slide were taken. These photographs as well as examination of all mounted slides were compared with altas plates from DeGroot (1959) to determine locus and extent of the lesion.

CHAPTER III

RESULTS

Examples of the smallest and largest lesions are given in Figure 1. Lesions invaded the hippocampal arch. The thalamus was not injured but in some cases, damage extended into the neocortex. Six HP animals were discarded because the lesion either invaded the thalamus or did not result in bilateral damage to the hippocampus. Histological examination of the sham operates did not indicate damage in any case.

Behavioral Results

Since a number of animals had to be rejected by histological criterion, there were only five HP subjects in the B-B condition rather than six. Analyses of variance were carried out on the two basic measures, running speed and number of errors. Running speeds were calculated by multiplying the reciprocal latency of time x 1,000.

In order to ascertain the presence and amount of response perseveration, the following comparisons were made: mean speeds in the last training block and the first testing block; mean speeds on the first trial of the last training block and the first trial of the first testing block; and mean speed for training and testing. Comparisons between the last training block or trial and the first testing block

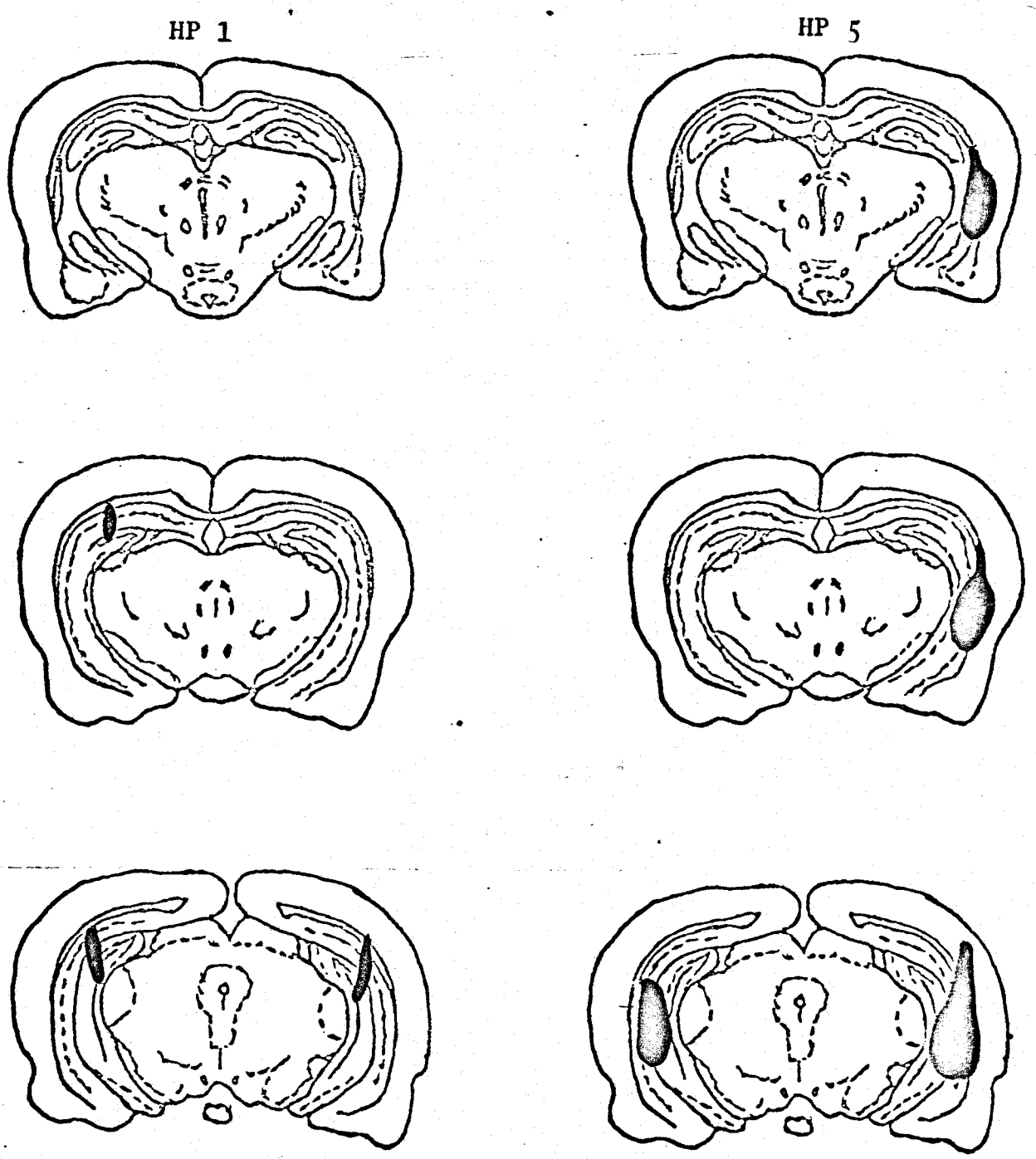


Figure 1. Example of the extent of hippocampal lesions in HP animals.

Table I

Mean Speed, Training and Testing
(Standard Deviations in Parentheses)

	Sham Operates				Hippocampal Operates			
Phase	Black vs. White	Black vs. Grey[1]	Black vs. Grey[2]	Black vs. Black	Black vs. White	Black vs. Grey[1]	Black vs. Grey[2]	Black vs. Black
Training	287.33	233.33	253.00	300.50	239.50	297.83	242.33	311.20
	(65.28)	(75.48)	(87.56)	(147.50)	(24.40)	(34.04)	(174.85)	(119.69)
Testing	272.67	293.17	316.67	308.33	247.83	361.17	275.33	349.20
	(58.01)	(32.65)	(70.56)	(111.03)	(77.59)	(120.24)	(78.26)	(185.38)

Table II

Analysis of Variance on Mean Speed, Training and Testing

Source of Variation	SS	df	MS	F
Between Subjects	860,013.81	45		
Surgical Group	1,763.45	1	1,763.45	.09 (NS)
Brightness Condition	7,307.13	3	2,435.71	.12 (NS)
Interaction	43,580.76	3	14,526.92	.71 (NS)
Error Between	773,698.78	38	20,360.49	
Within Subjects	236,755.00	46		
Training-Testing	26,571.23	1	26,571.23	5.31*
Surg. x Train-Test	93.70	1	93.70	.02 (NS)
Bright. x Train-Test	16,239.70	3	5,413.23	1.08 (NS)
Surg. x Bright. x Train-Test	3,454.06	3	1,151.35	.23 (NS)
Error Within	190,016.45	38	5,000.43	

NS > .05

Table III

Mean Speed on Last Training Block and on First Testing Block
(Standard Deviations in Parentheses)

	Sham Operates				Hippocampal Operates			
Phase	Black vs. White	Black vs. Grey^1	Black vs. Grey^2	Black vs. Black	Black vs. White	Black vs. Grey^1	Black vs. Grey^2	Black vs. Black
Training	377.33	316.50	296.50	339.17	1708.00	409.67	309.67	377.20
	(92.10)	(83.64)	(56.80)	(153.56)	(28.95)	(103.59)	(145.59)	(176.92)
Testing	240.50	265.50	296.00	279.00	208.83	311.33	238.33	288.80
	(62.55)	(54.31)	(53.72)	(102.54)	(79.62)	(78.81)	(90.29)	(105.57)

Table IV

Analysis of Variance on Mean Speed, Last Block Training and First Block Testing

Source of Variation	SS	df	MS	F
Between Subjects	831,911.74	45		
Surgical Group	118.67	1	118.67	.006 (NS)
Brightness Condition	42,068.88	3	14,022.96	.73 (NS)
Interaction	57,085.07	3	19,028.36	.99 (NS)
Error Between	732,175.26	38	19,267.77	
Within Subjects	332,034.00	46		
Training-Testing	124,236.81	1	124,236.81	26.80*
Surg. x Train-Test	2,672.34	1	2,672.34	.58 (NS)
Bright. x Train-Test	14,593.98	3	4,864.66	1.05 (NS)
Surg. x Bright. x Train-Test	14,579.62	3	4,859.87	1.05 (NS)
Error Within	176,137.90	38	4,635.21	

NS > .05

Table V

Mean Speed, First Trial, Last Training Block and First Trial First Testing Block (Standard Deviations in Parentheses)

	Sham Operates				Hippocampal Operates			
Phase	Black vs. White	Black vs. Grey[1]	Black vs. Grey[2]	Black vs. Black	Black vs. White	Black vs. Grey[1]	Black vs. Grey[2]	Black vs. Black
Training	173.00	143.67	176.00	293.00	144.33	278.00	147.67	102.80
	(97.09)	(165.96)	(132.28)	(147.28)	(62.72)	(179.26)	(167.53)	(78.24)
Testing	90.67	100.00	114.83	83.50	76.00	130.17	126.50	72.60
	(43.99)	(70.34)	(45.07)	(37.95)	(36.07)	(58.06)	(69.64)	(66.27)

Table VI

Analysis of Variance on Mean Speed, First Trial, Last Training Block and First Trial, First Testing Block

Source of Variation	SS	df	MS	F
Between Subjects	715,960.21	45		
Surgical Group	795.32	1	795.32	.05 (NS)
Brightness Condition	23,904.58	3	7,968.19	.51 (NS)
Interaction	102,474.58	3	34,158.19	2.19 (NS)
Error Between	592,206.62	38	15,584.38	
Within Subjects	687,371.50	46		
Training-Testing	186,817.39	1	186,817.39	16.85*
Surg. x Train-Test	2,293.90	1	2,293.90	.21 (NS)
Bright. x Train-Test	9,991.71	3	3,330.57	.30 (NS)
Surg. x Bright. x Train-Test	61,131.46	3	20,377.15	1.84 (NS)
Error Within	421,373.36	38	11,088.77	

NS $>$ .05

Table VII

Number of Errors, Training and Testing
(Standard Deviations in Parentheses)

	Sham Operates				Hippocampal Operates			
Phase	Black vs. White	Black vs. Grey[1]	Black vs. Grey[2]	Black vs. Black	Black vs. White	Black vs. Grey[1]	Black vs. Grey[2]	Black vs. Black
Training	7.33	7.83	5.17	11.33	6.67	6.83	10.67	7.40
	(3.09)	(2.54)	(1.34)	(3.68)	(2.49)	(2.41)	(5.09)	(5.43)
Testing	3.67	4.17	4.00	3.17	3.83	4.83	5.17	6.20
	(1.49)	(2.54)	(1.63)	(1.34)	(2.27)	(1.68)	(1.46)	(4.02)

Table VIII

Analysis of Variance on Number of Errors, Training and Testing

Source of Variation	SS	df	MS	F
Between Subjects	387.47	45		
Surgical Group	8.91	1	8.91	1.18 (NS)
Brightness Condition	33.58	3	11.19	1.49 (NS)
Interaction	58.01	3	19.34	2.57 (NS)
Error Between	285.61	38	7.52	
Within Subjects	9.30	46		
Training-Testing	291.07	1	291.07	22.22*
Surg. x Train-Test	9.55	1	9.55	.73 (NS)
Bright. x Train-Test	11.31	3	3.77	.29 (NS)
Surg. x Bright. x Train-Test	93.94	3	31.31	2.39 (NS)
Error Within	497.68	38	13.10	

NS $>$ .05

Table IX

Number of Errors, First Block Training and Testing
(Standard Deviations in Parentheses)

	Sham Operates				Hippocampal Operates			
Phase	Black vs. White	Black vs. Grey[1]	Black vs. Grey[2]	Black vs. Black	Black vs. White	Black vs. Grey[1]	Black vs. Grey[2]	Black vs. Black
Training	3.33	3.17	3.33	3.67	3.50	3.33	5.00	3.80
	(0.94)	(0.90)	(1.11)	(1.11)	(11.52)	(1.60)	(3.79)	(3.82)
Testing	2.33	1.67	2.33	2.00	1.83	2.33	3.83	3.40
	(0.94)	(0.75)	(0.75)	(0.57)	(1.21)	(0.47)	(1.57)	(2.33)

Table X

Analysis of Variance on Number of Errors, First Block Training and Testing

Source of Variation	SS	df	MS	F
Between Subjects	178.83	45		
Surgical Group	9.84	1	9.84	2.59 (NS)
Brightness Condition	14.71	3	4.90	1.29 (NS)
Interaction	9.43	3	3.14	.83 (NS)
Error Between	144.57	38	3.80	
Within Subjects	171.00	46		
Training-Testing	32.41	1	32.41	9.18*
Surg. x Train-Test	0.35	1	0.35	.10 (NS)
Bright. x Train-Test	0.35	3	0.12	.03 (NS)
Surg. x Bright. x Train-Test	3.11	3	1.04	.29 (NS)
Error Within	134.10	38	3.53	

NS > .05

Table XI

Number of Errors on First Trial Training and First Trial Testing
(Standard Deviations in Parentheses)

	Sham Operates				Hippocampal Operates			
Phase	Black vs. White	Black vs. Grey^1	Black vs. Grey^2	Black vs. Black	Black vs. White	Black vs. Grey^1	Black vs. Grey^2	Black vs. Black
Training	1.67	1.67	1.33	2.17	1.37	2.17	2.33	3.40
	(0.47)	(0.75)	(0.75)	(1.34)	(0.47)	(1.78)	(2.56)	(4.32)
Testing	1.50	2.00	1.83	1.83	1.83	1.83	2.33	3.80
	(0.50)	(0.58)	(0.69)	(0.37)	(1.07)	(0.37)	(1.37)	(2.78)

Table XII

Analysis of Variance on Number of Errors, First Trial Training and First Trial Testing

Source of Variation	SS	df	MS	F
Between Subjects	10.63	45		
Surgical Group	.41	1	.41	1.78 (NS)
Brightness Condition	1.17	3	.39	1.70 (NS)
Interaction	.47	3	.16	0.70 (NS)
Error Between	8.97	38	.23	
Within Subjects	8.19	46		
Training-Testing	.18	1	.18	.90 (NS)
Surg. x Train-Test	.00	1	.00	.00 (NS)
Bright. x Train-Test	.06	3	.02	0.10 (NS)
Surg. x Bright. x Train-Test	.23	3	.08	0.40 (NS)
Error Within	7.71	38	.20	

.05

Table XIII

Errors to Extinction
(Standard Deviations in Parentheses)

	Black vs. White	Black vs. Grey[1]	Black vs. Grey[2]	Black vs. Black
Sham Operates	7.17	10.33	6.00	5.83
	(4.26)	(9.27)	(4.73)	(3.67)
Hippocampal Operates	8.83	16.50	7.00	6.60
	(4.88)	(16.03)	(4.04)	(7.45)

Table XIV

Analysis of Variance on Errors to Extinction

Source of Variation	SS	df	MS	F	
Surgical Groups	66.36	1	66.36	.89	(NS)
Brightness Condition	387.36	3	129.12	1.74	(NS)
Interaction	55.81	3	18.60	.25	(NS)
Error Within	2,898.52	39	74.32		

NS > .05

Table XV

Trials to Extinction
(Standard Deviations in Parentheses)

	Black vs. White	Black vs. Grey[1]	Black vs. Grey[2]	Black vs. Black
Sham Operates	13.50	14.67	11.33	10.00
	(6.08)	(8.26)	(5.82)	(4.86)
Hippocampal Operates	14.17	19.33	9.83	10.80
	(6.54)	(14.20)	(4.88)	(8.70)

Table XVI

Analysis of Variance on Trials to Extinction

Source of Variation	SS	df	MS	F	
Surgical Groups	15.44	1	15.44	.20	(NS)
Brightness Condition	337.59	3	112.53	1.48	(NS)
Interaction	56.74	3	18.91	.25	(NS)
Error Within	2,963.95	39	76.00		

NS > .05

Table XVII

Number of Errors, Training and Testing
(Newman-Keuls)

Hippocampal-Training

	B/B	B/W	B/G^1	B/G^2
B/B	-	NS	NS	*
B/W		-	NS	*
B/G^1			-	*
B/G^2				-

Hippocampal-Testing

	B/W	B/G^1	B/G^2	B/B
B/W	-	NS	NS	NS
B/G^1		-	NS	NS
B/G^2			-	NS
B/B				-

		2	3	4
** q.01	$(r,38) \times \sqrt{n_h MS \text{ error bet.}}$	24.18	30.70	31.08
* q.05	$(r,38) \times \sqrt{n_h MS \text{ error bet.}}$	18.96	22.80	25.12

Table XVIII

Number of Errors, Training and Testing
(Newman-Keuls)

Sham-Training

	B/G^2	B/W	B/G^1	B/B
B/G^2	-	NS	NS	**
B/W		NS	NS	*
B/G^1			NS	*
B/B				-

Sham-Testing

	B/B	B/W	B/G^2	B/G^1
B/B	-	NS	NS	NS
B/W		-	NS	NS
B/G^2			-	NS
B/G^1				-

		2	3	4
** q.01	$(r,38) \times \sqrt{\frac{nMS \text{ error bet.}}{}}$	25.63	32.54	32.95
* q.05	$(r,38) \times \sqrt{\frac{nMS \text{ error bet.}}{}}$	19.19	23.08	25.43

Table XIX

Number of Errors, Hippocampal versus Sham
(Newman-Keuls)

Training			
B/W	NS		
B/G^1	NS		
B/G^2	**	HP Sh	
B/B	**	HP Sh	
** q.01	(2,38)	$\times \sqrt{nMS \text{ error between}}$	25.63
* q.05	(2,38)	$\times \sqrt{nMS \text{ error between}}$	19.19
** q.01	(2,38)	$\times \sqrt{n_h MS \text{ error between}}$	25.33
* q.05	(2,38)	$\times \sqrt{n_h MS \text{ error between}}$	18.96
Testing			
B/W	NS		
B/G^1	NS		
B/G^2	NS		
B/B	NS		

Table XX

Number of Errors, Train versus Test at Each Brightness Level (Newman-Keuls)

Hippocampal			
B/W	NS		
B/G^1	NS		
B/G^2	*	Train	Test
B/B	NS		
** q.01	(2,38) x $\sqrt{\text{nMS error within}}$		33.85
* q.05	(2,38) x $\sqrt{\text{nMS error within}}$		25.33
** q.01	(2,38) x $\sqrt{\text{nMS error within}}$		30.90
* q.05	(2,38) x $\sqrt{\text{nMS error within}}$		23.13
Sham			
B/W	NS		
B/G^1	NS		
B/G^2	NS		
B/B	**	Train	Test

Table XXI

Number of Errors, Rate of Change from Train to Test, HP versus Sh (Newman-Keuls)

B/W	NS		
B/G^1	NS		
B/G^2	*	HP Sh	
B/B	**	HP Sh	

** q.01	(2,38)	$\times \sqrt{nMS \text{ error pooled}}$	30.03
* q.05	(2,38)	$\times \sqrt{nMS \text{ error pooled}}$	22.47
** q.01	(2,38)	$\times \sqrt{n_h MS \text{ error pooled}}$	29.64
* q.05	(2,38)	$\times \sqrt{n_h MS \text{ error pooled}}$	22.19

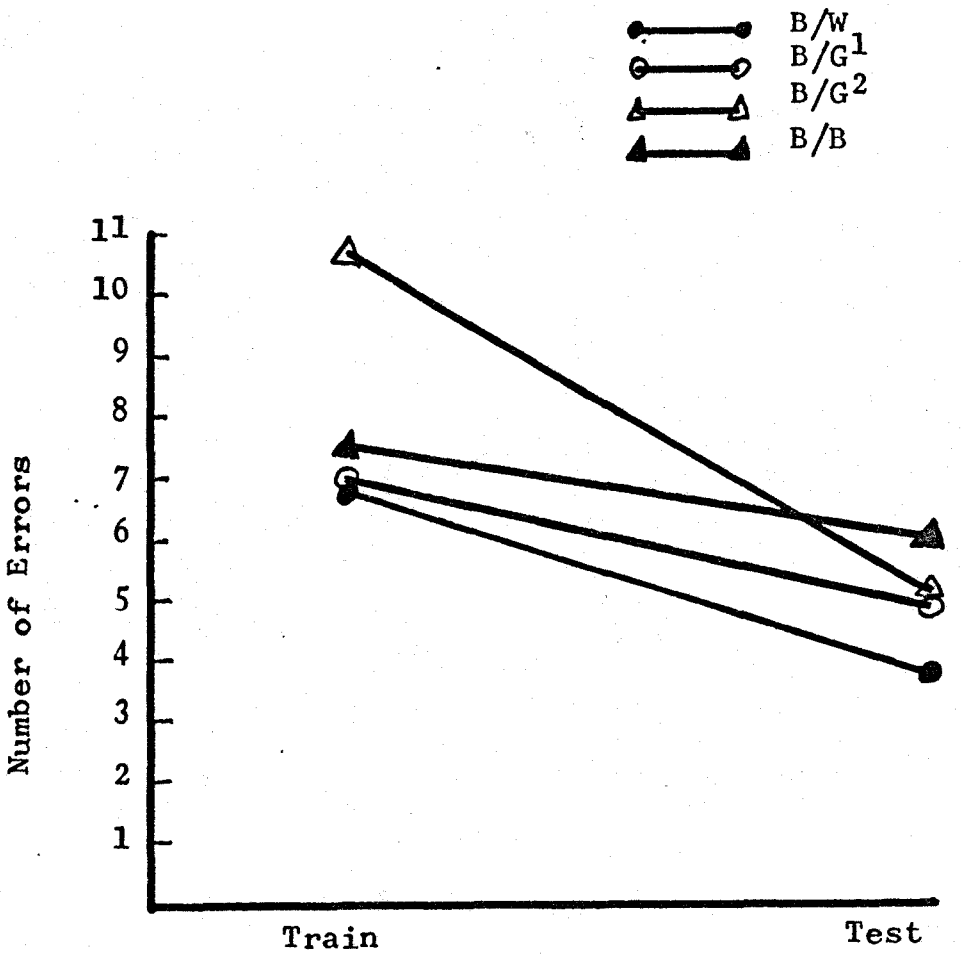


Figure 2. Number of errors, training and testing, for HP.

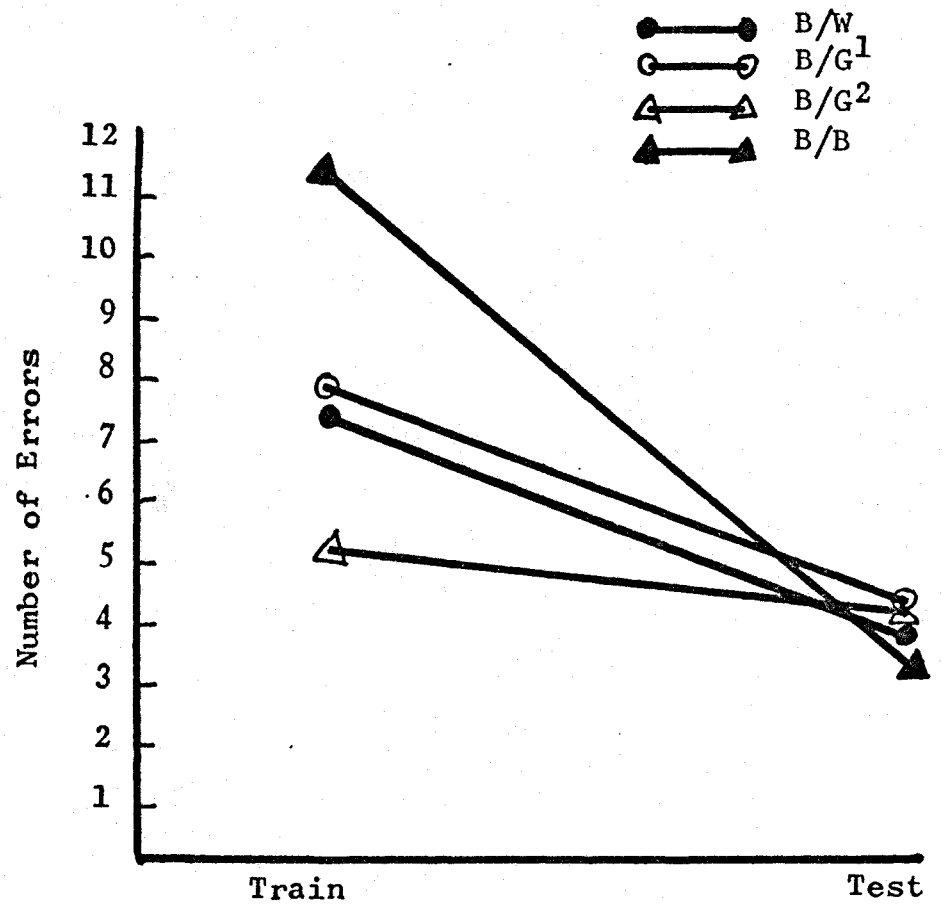


Figure 3. Number of errors, training and testing, for Sh.

or trial were carried out in the event that any response perseveration in HP animals is short lived and habituated after a few runs.

Mean speeds and standard deviations for training and testing are shown in Table I. A summary of the analysis of variance for mean speed during training and testing is given in Table II. No significant main or interaction effects for between subjects comparisons were found. For within subjects analysis, however, significant main effect for phase change ($F = 5.31$, df 1/38, $p < .05$) was found. No significant within subjects interactions were found. Mean speed and standard deviations in the last training block and on the first testing block are presented in Table III. A summary of the analysis of variance for these results is presented in Table IV. Again, only the within subjects training-testing phase main effect was significant ($F = 26.80$, df 1/38, $p < .01$). A significant main phase effect was found ($F = 16.85$, df 1/38, $p < .01$) for the overall mean speed from training to testing.

Since there were only two phases, training and testing, the main effects significance indicates the direction of change. The overall mean speed from the training to the testing phase significantly increased (270.63 to 303.05). However, when the mean speed from the last training block (516.76) or the first trial of that block (188.56) was compared to the mean speed of the first testing block (266.04)

or first trial of that testing block (99.28), a significant decrease was found. These results indicate that the starting position change initially affected both surgical groups similarly but that this effect was soon habituated.

Errors were also an indication of response perseveration. Similar to the running speed measure, error frequency overall training training and testing trials, for the first training and first testing block, and for the first training trial and the first testing trial, were analyzed.

Analysis of variance on the number of errors in the first trial training and the first trial testing for each surgical group under each brightness condition (see Tables IX and X) yielded a significant main effect for phase change ($F = 9.18$, df 1/38, $p < .01$). Errors significantly decreased from training to testing. The overall number of errors and standard deviations, training and testing, are presented in Table VII. The analysis of variance of the number of errors is presented in Table VIII. As in the above case, a significant main effect for phase change was found ($F = 22.22$, df 1/38, $p < .01$). No other differences were significant at the .05 level. However, the surgical group x brightness condition x training-testing interaction reached .10 level of probability. Although not a significant interaction, individual comparisons (Newman-Keuls) were carried out in hopes of discovering some meaningful differences between surgical groups under each brightness condition (see Tables

XVIII through XXI and Figures 2 and 3). Hippocampal lesioned animals in the B-G^2 group made significantly more errors than did any of the other HP groups ($p < .05$) during training. No significant differences in the number of errors were found between any of the brightness condition groups in testing (see Table XVII). For the Sh operates in the training phase, only animals in the B-B condition made significantly more errors than the B-W group ($p < .05$), the B-G^1 group ($p < .05$), or the B-G^2 group ($p < .01$). No other significant differences were found. As was the case for the HP operates, no differences were found in the number of errors between Sh brightness groups in the testing phase (see Table XVIII). In comparing the number of errors in training for the HP versus Sh operates (Table XIX), it was found that the HP animals in the B-G^2 condition made significantly more errors than did the Sh animals in the same condition ($p < .01$). For animals in the B-B condition, the Sh operates made significantly more errors than did the HP operates ($p < .01$). No differences were found in the B-W and B-G^1 conditions. There were no differences in the number of errors (HP versus Sh) in the testing phase. Significant changes in error frequency from the training to the testing phase were found only in the B-G^2 HP animals and the B-B Sh animals. In both cases, significant error reductions were found ($p < .05$). The expected increase in errors for HP groups with minimum brightness differentiation failed to

occur. No other significant differences were found (see Tables XX and Figures 2 and 3).

A further comparison between the rate of change in error frequency between HP and Sh subjects was carried out (see Table XXI and Figures 2 and 3). For the B-G^2 condition, HP subjects reduced their errors more than Sh subjects ($p < .01$). This finding was counter to predictions. However, B-B HP subjects showed a significantly smaller error reduction than Sh operates ($p < .01$). This result was the only finding consonant with the predictions that HP animals would show more response perseveration under minimally distinctive external cues.

Analysis of variance for the number of errors to extinction and trials to extinction yielded no significant differences (see Tables XII through XVI).

CHAPTER IV

DISCUSSION AND CONCLUSIONS

The prediction that the B-B and the B-G^2 hippocampal lesioned groups would show greater response perseveration was not validated. There were no differences between the brightness conditions, either within the hippocampal group or between the hippocampal and sham operates in the testing phase. The only differences were differences observed in the training phase.

As indicated in the results section of this paper, significant main effects in mean speed measures were found for within subjects differences on the phase variable. Overall training and testing speeds increased from the former to the latter. Speeds decreased from training to testing on the measures of mean speed for the last block training and the first block testing and for the first trials on these blocks. These results seemed to indicate that the habituation processes played an important role in decreasing running speeds in the latter measures. The lack of between subjects differences and the lack of within subjects interactions suggests a number of hypotheses. Running speed may not have been a good measure of response perseveration in this type of task. Support for this hypothesis is indicated by Cohen et al. (1971), where differences were not obtained

on speed measures but were obtained on error measures.

In error measurements, the number of errors, first trial training and testing, yielded no significant differences. Differences in training and testing measures were the only significant differences obtained in the number of errors, first block training and testing. In addition to phase differences, the total number of errors showed significant number of differences in the surgical group x brightness condition x training-testing interaction at the .10 level. Individual comparisons failed to validate the predictions of response perseveration in B-B and B-G^2 HP rats. Hippocampal lesioned animals in the B-G^2 condition made more errors than did any other HP group, including the B-B group. These differences only occurred, however, in the training phase. Generally, findings have indicated that initial learning is not impaired by hippocampal lesioning. These results are further complicated by the fact that Sh animals in the B-B group made significantly more errors than the Sh animals in any other brightness condition. Any explanation of the results of the HP, B-G^2 group in terms of lesion effects could not explain the results in the Sh, B-B group or the fact that this result was not obtained in the HP, B-B group. A possible explanation of these differences may be in terms of pretraining preferences. In both HP, B-G^2 and Sh, B-G cases, there were two animals who made many more errors than the other animals in the other brightness

conditions. In each case, these animals started training in the nonpreferred arm. No explanation can be presently offered for the observed perseverative behavior in these two Sh rats.

Also, HP animals in the B-G^2 condition made significantly more errors than the Sh animals in training. Hippocampal animals reduced their errors from training to testing significantly more than did sham animals in this brightness condition. Analysis of the results for the B-B conditions are the same, except in this case the Sh showed significantly greater decrease. No differences were obtained in any of these measures for the B-W and B-G^1 groups.

Discrepancies between the results of the present study and those of Cohen et al. (1971) may be due to differences in procedure. In the present study, the animals were required to push open a door before they could open the choice arm. In the previous study, only the choice arms were changed in cue distinctiveness. No doors were used. The present procedure may have supplied the subjects with an external inhibitor that replaced the inhibition supposedly deficient in hippocampal lesioned rats. Another possibility may be that the stimulus doors provided an extra discriminative cue which compensated for the minimal differences in the B-G^2 and B-B conditions. These doors were hinged on one side of the choice arms. Therefore, when the animals were run from the opposite start box, the new position of the

hinges may have been an extra discriminative cue.

Implication for Further Research

An experiment to test the above hypotheses can be easily designed and is presently underway by Cohen and Laroche. Hippocampal and sham lesioned rats are being run in the same maze as in the present study, under three conditions. In the first condition, animals will be taught a left-right discrimination without doors. They will then be run from the opposite start box and required to run to the same choice arm. The second group will be required to learn a left-right discrimination and will have to push through plexiglass doors to enter the choice arm. They will be tested from the opposite start box and then required to make reversal shift from the same start box. If the doors merely provide an external inhibition for HP rats, then response perseveration should only be seen for such animals who are not trained with the doors. If the doors, however, decreased response perseveration in 180 degrees turn by providing more cue distinctiveness, then response perseveration should also be observed in HP animals required to push open the doors during reversal shift training.

Preliminary data on five hippocampal damaged (HP) and six sham (Sh) operated animals trained and tested with doors and on six HP and five Sh animals tested and trained without doors confirm these hypotheses. That is, only those HP - no door rats typically showed an increase in errors (body

turn perseverations) during the testing phase.

REFERENCES

Ackil, J.E., Mellgren, R.L., Halgren, C., & Frommer, G.P. Effects of CS preexposures on avoidance learning in rats with hippocampal lesions. Journal of Comparative and Physiological Psychology, 1969, 69 (4), 739-747.

Brunner, R.L., & Rossi, R.R. Hippocampal disruption and passive avoidance behavior. Psychonomic Science, 1969, 15 (5), 228-229.

Cohen, J.S. Exploration in the hippocampal-ablated albino rat. Journal of Comparative and Physiological Psychology, 1970, 73 (2), 261-268.

Cohen, J.S., Laroche, J.P., & Beharry, E. Response perseveration in the hippocampal lesioned rat. Psychonomic Science, 1971, 23 (3), 221-223.

Cohen, J.S., & Swenson, L.C. The effect of hippocampectomy and prior experience in exploratory activity in the albino rat. Psychonomic Science, 1970, 18 (5), 275-277.

Dalland, T. Response and stimulus perseveration in rats with septal and dorsal hippocampal lesions. Journal of Comparative and Physiological Psychology, 1970, 71 (1), 114-118.

Douglas, R.J. The hippocampus and behavior. Psychological Bulletin, 1967, 67, 416-442.

Ellen, P., & Deloache, J. Hippocampal lesions and spontaneous alternation behavior in the rat. Physiology and Behavior, 1968, 3, 857-860.

Grastyan, E. The hippocampus and higher nervous activity. In M.A.B. Brazier (Ed.), The central nervous system and behavior. New York: Josiah Macy, Jr. Foundation, 1959. Pp. 119-205.

Grastyan, E., Lissak, K., Madarasz, I., & Danhoffer, H. Hippocampal electrical activity during the development of conditioned reflexes. Electroencephalography and Clinical Neurophysiology, 1959, 11 (3), 409-430.

Green, J.D. The hippocampus. In J. Field, H.W. Magoun, and V.E. Hall (Eds.), Handbook of Physiology, Neurophysiology, II. Washington, D.C.: American Physiological Society, 1960. Pp. 1373-1389.

Green, J.D., & Arduini, A. Hippocampal electrical activity in arousal. Journal of Neurophysiology, 1954, 17, 532-557.

Grossman, S.P., & Mountford, H. Learning and extinction during chemically induced disturbance of hippocampal function. American Journal of Physiology, 1964, 207 (6), 1387-1393.

Holmes, J.E., & Adey, W.R. Electrical activity of the entorhinal cortex during conditioned behavior. American Journal of Physiology, 1960, 199 (5), 741-744.

Hutchinson, R.R., & Renfrew, J.W. A simple hiatological technique for localizing electrode tracks and lesions within the brain. Journal of the Experimental Analysis of Behavior, 1967, 10, 277-280.

Jackson, W.J., & Strong, P.N. Differential effects of hippocampal lesions upon sequential tasks and maze learning by the rat. Journal of Comparative and Physiological Psychology, 1969, 68 (3), 442-450.

Jarrard, L.E., Isaacson, R.I., & Wickelgren, W.O. Effects of hippocampal ablation and intertrial interval on runway acquisition and extinction. Journal of Comparative and Physiological Psychology, 1964, 57 (3), 442-444.

Kimble, D.P. The effects of bilateral hippocampal lesions in rats. Journal of Comparative and Physiological Psychology, 1963, 56 (2), 273-283.

Kimble, D.P. Hippocampus and internal inhibition. Psychological Bulletin, 1968, 70, 285-295.

Kirkby, R.J., Stein, D.G., Kimble, R.J., & Kimble, D.P. Effects of hippocampal lesions and duration of sensory input on spontaneous alternation. Journal of Comparative and Physiological Psychology, 1967, 64 (2), 342-345.

Leaton, R.N. Exploratory behavior in rats with hippocampal lesions. Journal of Comparative and Physiological Psychology, 1965, 59 (3), 325-330.

Means, L.W. Cue utilization and perseveration in the hippocampectomized rat. Dissertation Abstracts, 1969, 29 (12-B), 4870-4871.

Milner, B. Memory disturbance after bilateral hippocampal lesions. In P. Milner and S. Glickman (Eds.), Cognitive processes and the brain. New York: Van Nostrand, 1965. Pp. 97-111.

Penfield, W., & Milner, B. Memory deficit produced by bilateral lesions in the hippocampal zone. A.M.A. Archives of Neurological Psychiatry, 1958, 79, 475-497.

Pickenhain, L., & Klingberg, F. Hippocampal slow wave activity as a correlate of basic behavioral mechanisms in the rat. In W.R. Adey and T. Tokizane (Eds.), Structure and function of the limbic system. New York: Elsevier, 1967. Pp. 218-227.

Riddell, W.I., Rothblatt, L.A., & Wilson, W.A., Jr. Auditory and visual distraction in hippocampectomized rats. Journal of Comparative and Physiological Psychology, 1969, 67 (2), 216-219.

Roberts, W.W., Dember, W.M., & Brodwick, M. Alternation and exploration in rats with hippocampal lesions. Journal of Comparative and Physiological Psychology, 1962, 55 (5), 696-700.

Silveira, J.M., & Kimble, D.P. Brightness discrimination and reversal in hippocampally-lesioned rats. Physiology and Behavior, 1968, 3, 625-630.

Walker, E.J. Action decrement and its relation to learning. Psychological Review, 1958, 65 (3), 129-142.

APPENDIX A

Mean Speed Scores for Each Subject

Hipp. No.	Group	Train		Test	Last block Training		First block Testing	First trial Last block Training		First trial First block Testing
1	B-W	209	-	178	276	-	229	250	-	125
2	B-W	277	-	390	336	-	378	071	-	050
3	B-W	248	-	154	254	-	156	111	-	125
4	B-W	224	-	281	293	-	162	143	-	067
5	B-W	261	-	265	298	-	162	200	-	056
6	B-W	218	-	219	251	-	166	091	-	033
7	$B\text{-}G^1$	250	-	352	448	-	358	333	-	100
8	$B\text{-}G^1$	303	-	163	323	-	169	125	-	022
9	$B\text{-}G^1$	257	-	260	335	-	297	168	-	200
10	$B\text{-}G^1$	312	-	511	327	-	388	500	-	167
11	$B\text{-}G^1$	346	-	473	408	-	392	042	-	167
12	$B\text{-}G^1$	319	-	408	617	-	264	500	-	125
13	$B\text{-}G^2$	218	-	300	370	-	317	250	-	167
14	$B\text{-}G^2$	093	-	296	192	-	170	016	-	143
15	$B\text{-}G^2$	234	-	295	312	-	139	166	-	045
16	$B\text{-}G^2$	062	-	113	091	-	140	004	-	063
17	$B\text{-}G^2$	600	-	370	557	-	354	500	-	250
18	$B\text{-}G^2$	247	-	278	336	-	310	250	-	091
19	B-B	196	-	336	225	-	242	091	-	071
20	B-B	173	-	143	172	-	126	035	-	023
21	B-B	290	-	252	371	-	322	038	-	048
22	B-B	472	-	321	447	-	305	100	-	021
23	B-B	425	-	694	671	-	449	250	-	200
24	B-B	-		-	-		-	-		-

APPENDIX A (Continued)

Sham No.	Group	Train		Test	Last block Training		First block Testing	First trial Last block Training		First trial First block Testing
25	B-W	226	-	236	239	-	199	111	-	063
26	B-W	235	-	238	357	-	218	200	-	056
27	B-W	391	-	338	369	-	378	077	-	167
28	B-W	191	-	185	317	-	220	250	-	111
29	B-W	312	-	345	461	-	230	067	-	111
30	B-W	329	-	294	529	-	198	333	-	036
31	B-G^1	342	-	358	392	-	222	024	-	100
32	B-G^1	215	-	277	259	-	223	167	-	077
33	B-G^1	144	-	283	309	-	199	035	-	029
34	B-G^1	199	-	263	242	-	250	059	-	077
35	B-G^1	301	-	310	461	-	353	500	-	067
36	B-G^1	139	-	268	236	-	246	077	-	250
37	B-G^2	119	-	177	218	-	186	048	-	083
38	B-G^2	295	-	316	337	-	342	333	-	063
39	B-G^2	267	-	305	325	-	285	250	-	100
40	B-G^2	405	-	342	382	-	310	333	-	200
41	B-G^2	205	-	352	242	-	348	050	-	143
42	B-G^2	227	-	408	275	-	305	042	-	100
43	B-B	426	-	422	340	-	338	500	-	077
44	B-B	303	-	274	243	-	204	333	-	125
45	B-B	036	-	122	072	-	082	009	-	006
46	B-B	201	-	225	354	-	353	250	-	111
47	B-B	478	-	410	523	-	353	333	-	091
48	B-B	359	-	397	503	-	344	333	-	091

APPENDIX B

Number of Errors for Each Subject

Hipp. No.	Group	Train		Test	Last block Training		First block Testing	First trial Last block Training		First trial First block Testing
1	B-W	06	-	01	02	-	00	00	-	00
2	B-W	08	-	03	03	-	02	00	-	00
3	B-W	05	-	02	03	-	01	01	-	01
4	B-W	03	-	05	02	-	04	00	-	03
5	B-W	11	-	08	06	-	02	00	-	00
6	B-W	07	-	04	05	-	02	01	-	01
7	B-G^1	10	-	06	04	-	02	00	-	01
8	B-G^1	07	-	04	01	-	03	00	-	01
9	B-G^1	10	-	02	06	-	02	05	-	01
10	B-G^1	05	-	06	04	-	02	01	-	00
11	B-G^1	05	-	04	03	-	03	00	-	01
12	B-G^1	07	-	04	02	-	02	01	-	01
13	B-G^2	12	-	05	03	-	03	00	-	01
14	B-G^2	17	-	05	09	-	04	00	-	00
15	B-G^2	08	-	05	04	-	03	01	-	00
16	B-G^2	16	-	08	11	-	07	07	-	04
17	B-G^2	02	-	05	00	-	04	00	-	02
18	B-G^2	09	-	03	03	-	02	00	-	01
19	B-B	04	-	03	02	-	02	00	-	02
20	B-B	15	-	06	02	-	02	00	-	00
21	B-B	00	-	14	00	-	08	00	-	08
22	B-B	12	-	04	11	-	02	11	-	01
23	B-B	06	-	04	04	-	03	01	-	03
24	B-B	-		-	-		-	-		-

APPENDIX B (Continued)

Sham No.	Group	Train		Test	Last block Training		First block Testing	First trial Last block Training		First trial First block Testing
25	B-W	05	-	02	03	-	02	01	-	00
26	B-W	04	-	02	03	-	01	01	-	00
27	B-W	11	-	03	04	-	02	01	-	01
28	B-W	11	-	05	05	-	03	01	-	01
29	B-W	04	-	04	02	-	02	00	-	01
30	B-W	09	-	06	03	-	04	00	-	00
31	$B-G^1$	05	-	02	04	-	02	01	-	02
32	$B-G^1$	13	-	09	04	-	02	01	-	01
33	$B-G^1$	07	-	05	02	-	01	00	-	00
34	$B-G^1$	06	-	05	04	-	03	00	-	01
35	$B-G^1$	08	-	02	03	-	01	02	-	01
36	$B-G^1$	08	-	02	02	-	01	00	-	01
37	$B-G^2$	06	-	01	04	-	01	00	-	00
38	$B-G^2$	07	-	04	03	-	02	00	-	01
39	$B-G^2$	03	-	05	02	-	03	00	-	00
40	$B-G^2$	04	-	06	02	-	03	00	-	01
41	$B-G^2$	05	-	03	04	-	02	02	-	01
42	$B-G^2$	06	-	05	05	-	03	00	-	02
43	B-B	11	-	04	03	-	02	01	-	01
44	B-B	11	-	04	02	-	02	00	-	01
45	B-B	04	-	01	03	-	01	01	-	00
46	B-B	13	-	05	05	-	03	00	-	01
47	B-B	13	-	02	05	-	02	04	-	01
48	B-B	16	-	03	04	-	02	01	-	01

APPENDIX C

Number of Trials to Extinction and Number of Incorrect Entries to Extinction for Each Subject

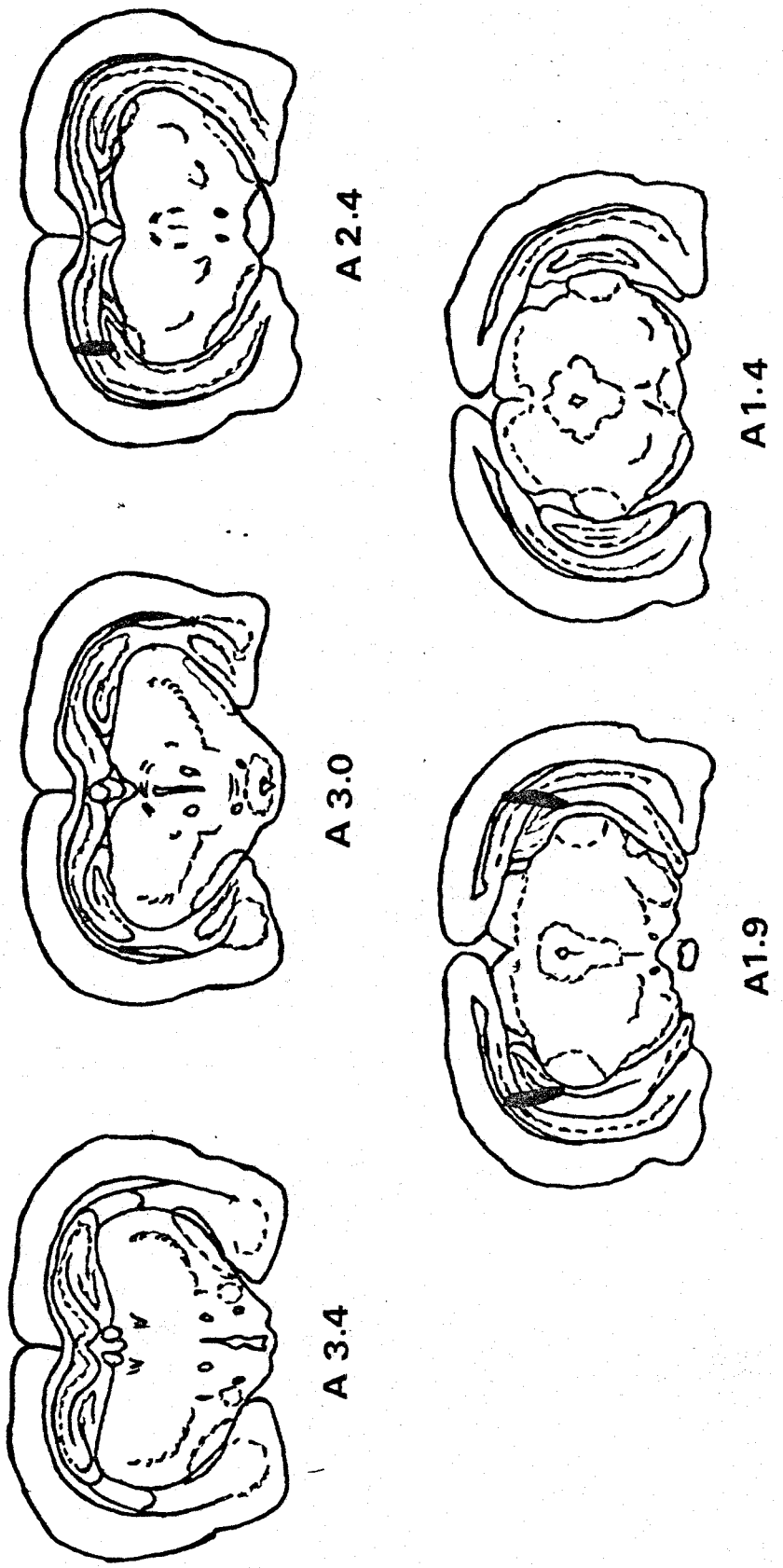
Hipp. No.	Group	Number of trials	Number of Incorrect Entries
1	B-W	08	06
2	B-W	24	15
3	B-W	16	10
4	B-W	18	15
5	B-W	04	03
6	B-W	15	04
7	$B\text{-}G^1$	19	12
8	$B\text{-}G^1$	09	04
9	$B\text{-}G^1$	09	07
10	$B\text{-}G^1$	24	21
11	$B\text{-}G^1$	48	50
12	$B\text{-}G^1$	07	05
13	$B\text{-}G^2$	13	13
14	$B\text{-}G^2$	05	05
15	$B\text{-}G^2$	16	07
16	$B\text{-}G^2$	02	00
17	$B\text{-}G^2$	10	10
18	$B\text{-}G^2$	13	07
19	B-B	04	01
20	B-B	08	01
21	B-B	07	04
22	B-B	28	21
23	B-B	07	06
24	B-B	-	-

APPENDIX C (Continued)

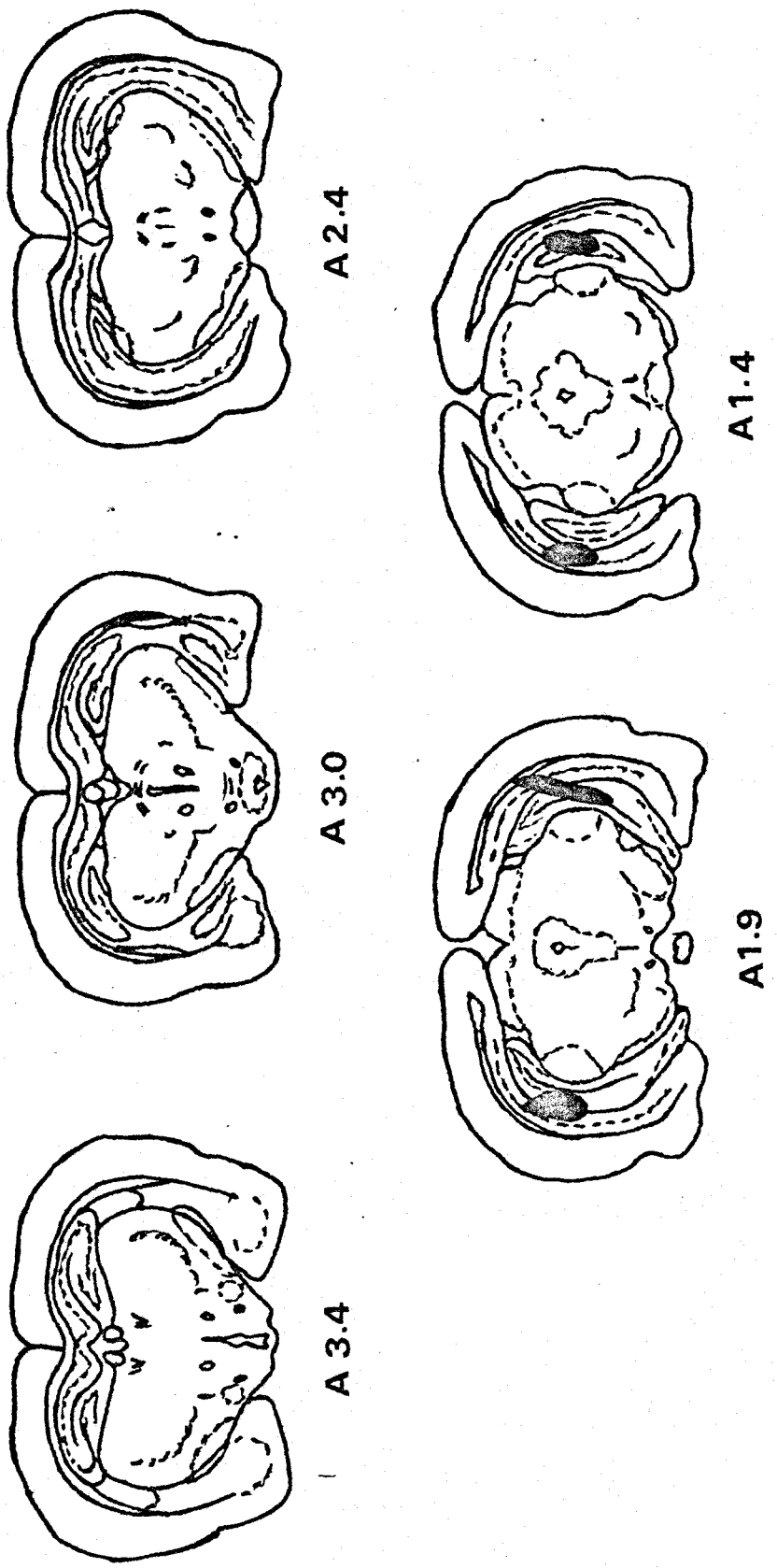
Sham No.	Group	Number of trials	Number of Incorrect Entries
25	B-W	17	10
26	B-W	08	04
27	B-W	09	06
28	B-W	06	02
29	B-W	19	15
30	B-W	22	06
31	$B-G^1$	30	30
32	$B-G^1$	19	09
33	$B-G^1$	07	05
34	$B-G^1$	13	05
35	$B-G^1$	14	11
36	$B-G^1$	05	02
37	$B-G^2$	06	02
38	$B-G^2$	04	04
39	$B-G^2$	13	03
40	$B-G^2$	08	02
41	$B-G^2$	17	11
42	$B-G^2$	20	14
43	B-B	16	09
44	B-B	08	05
45	B-B	06	01
46	B-B	04	03
47	B-B	17	12
48	B-B	09	05

APPENDIX D

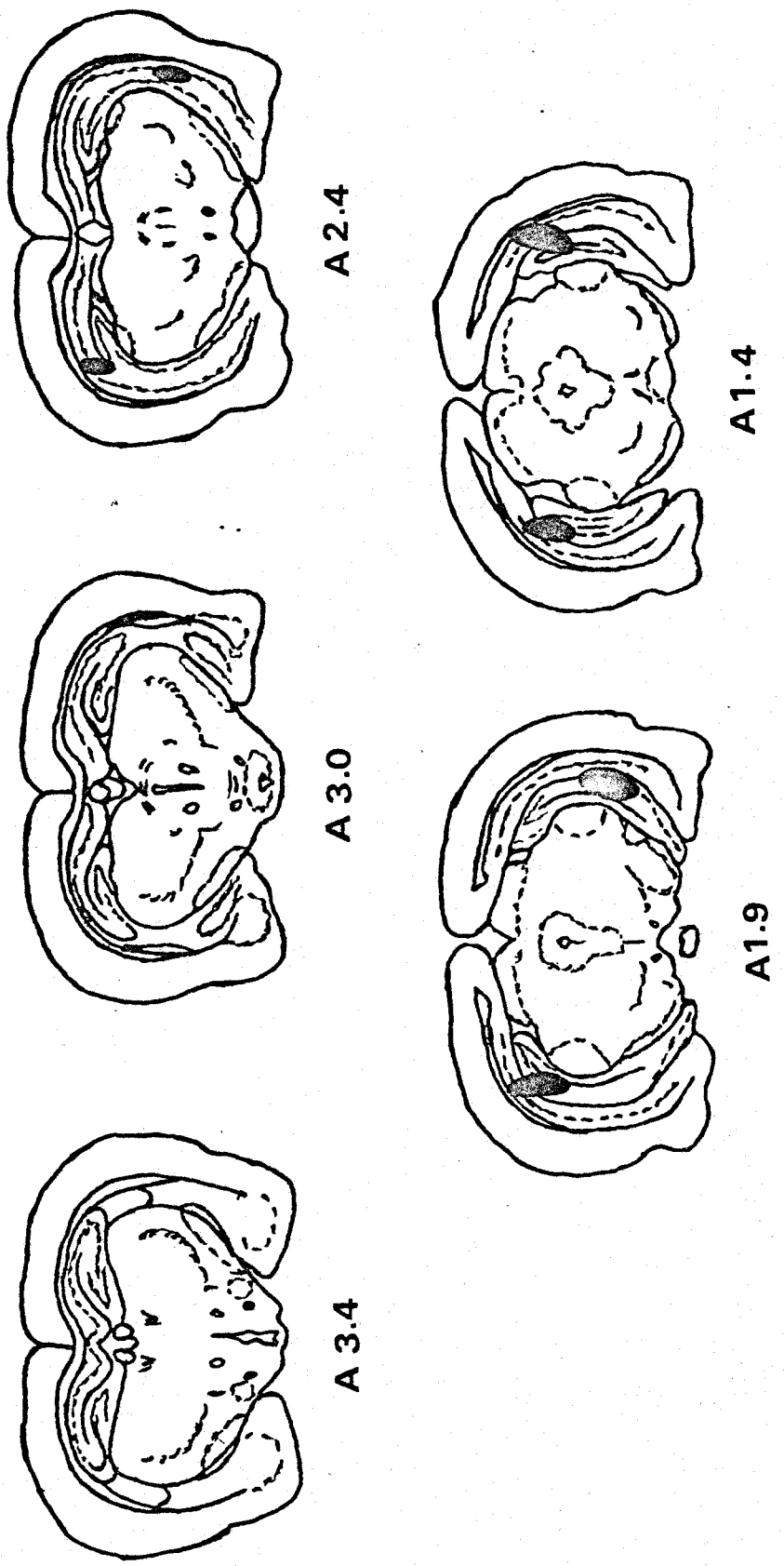
Extent of Lesion for Animals in HP Group



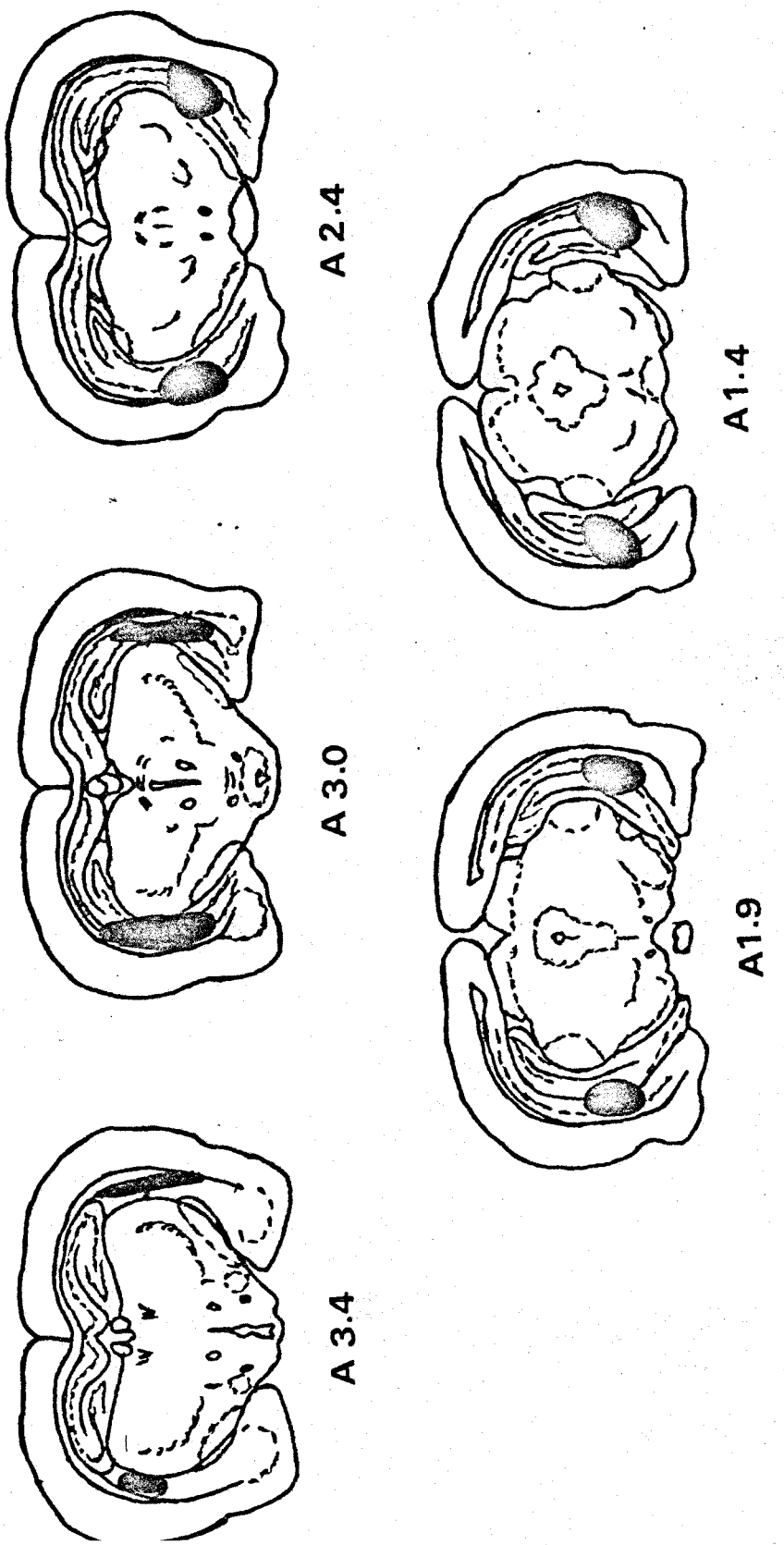
Hipp. 1



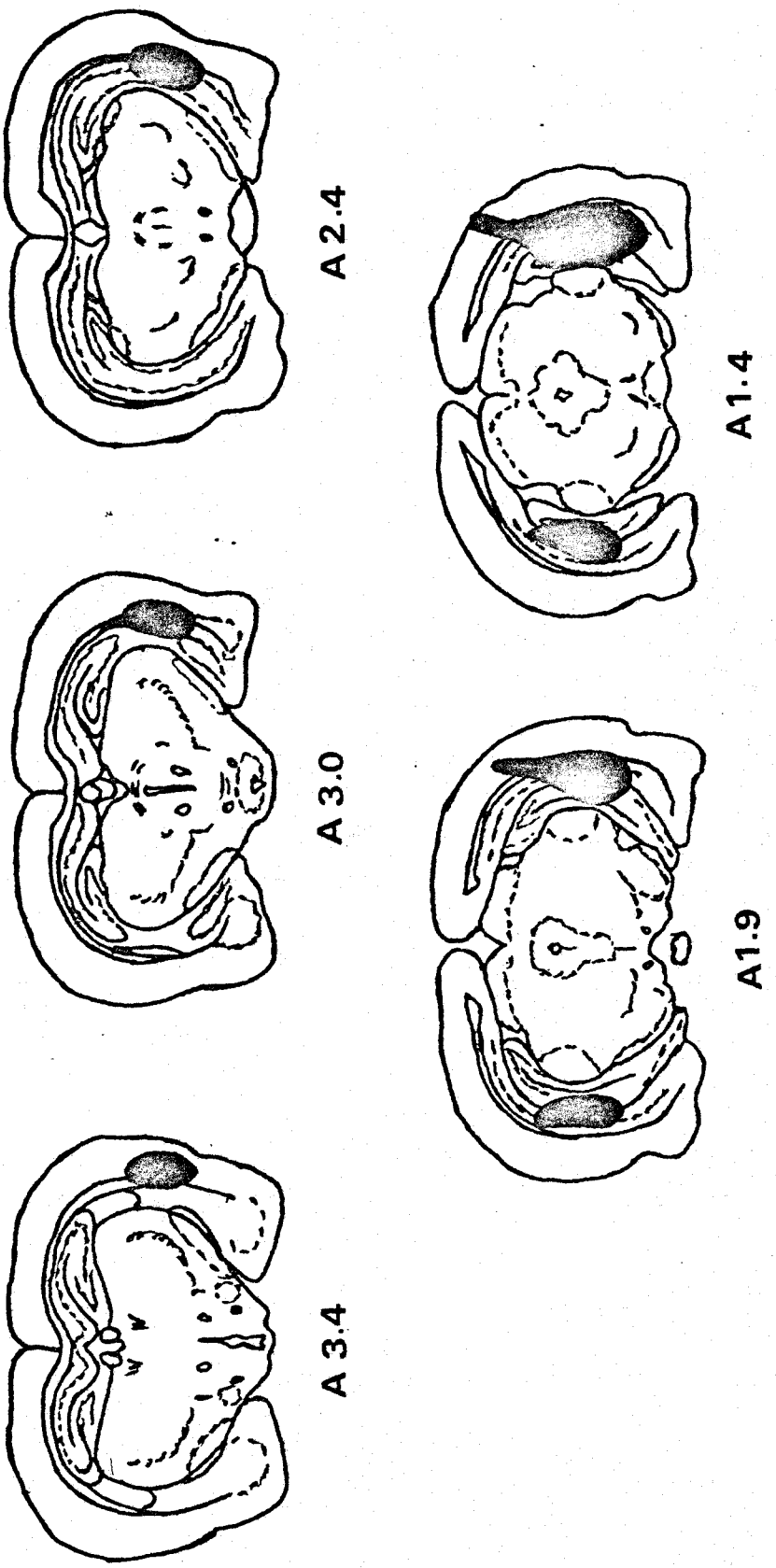
Hipp. 2



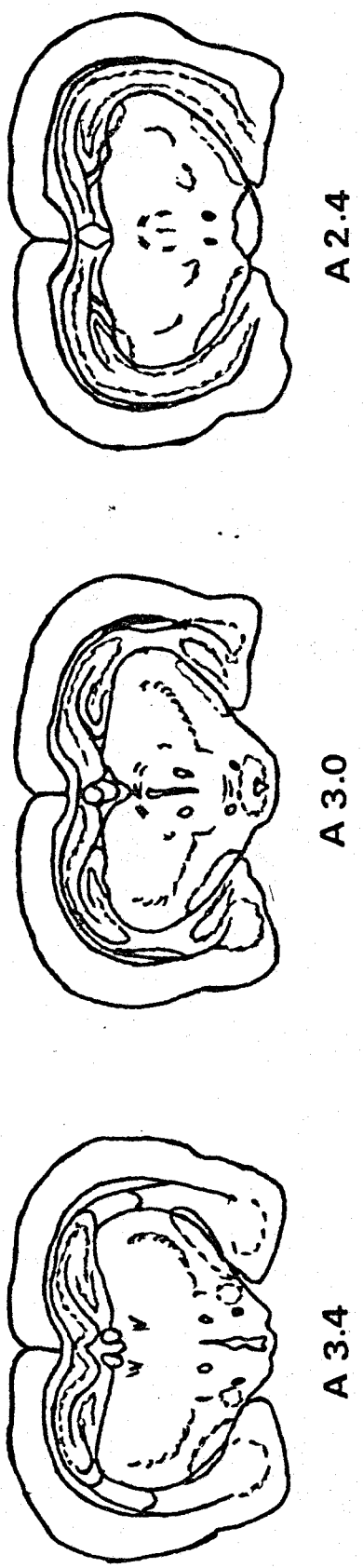
Hipp. 3

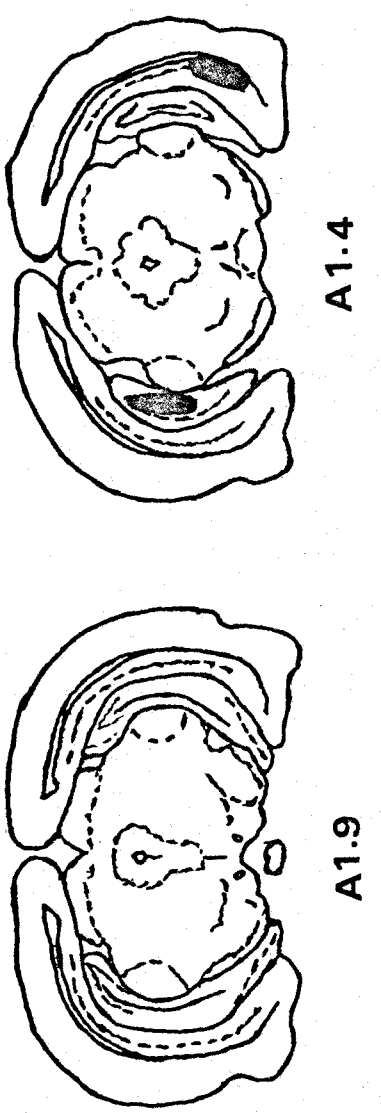


Hipp. 4

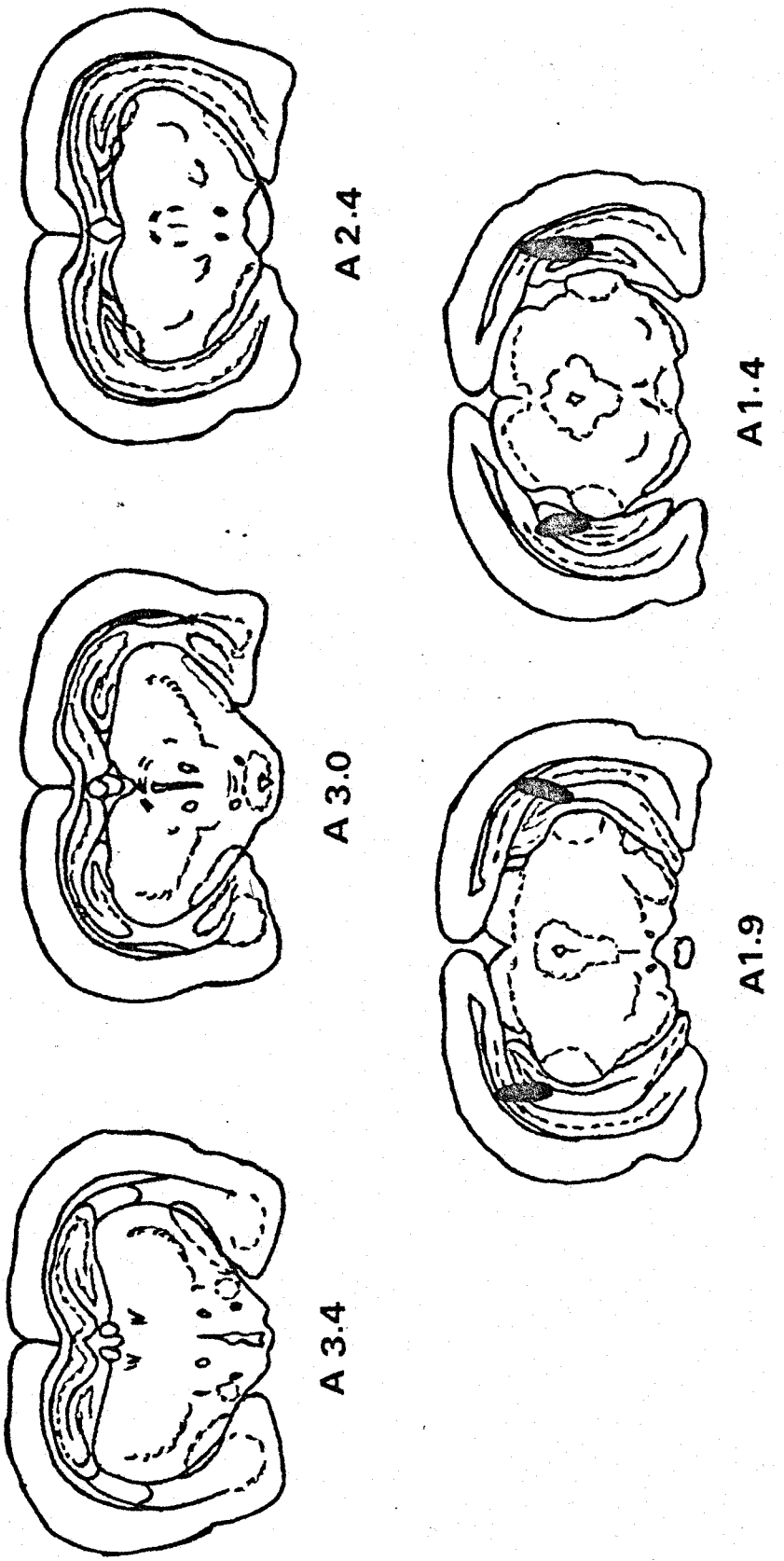


Hipp. 5

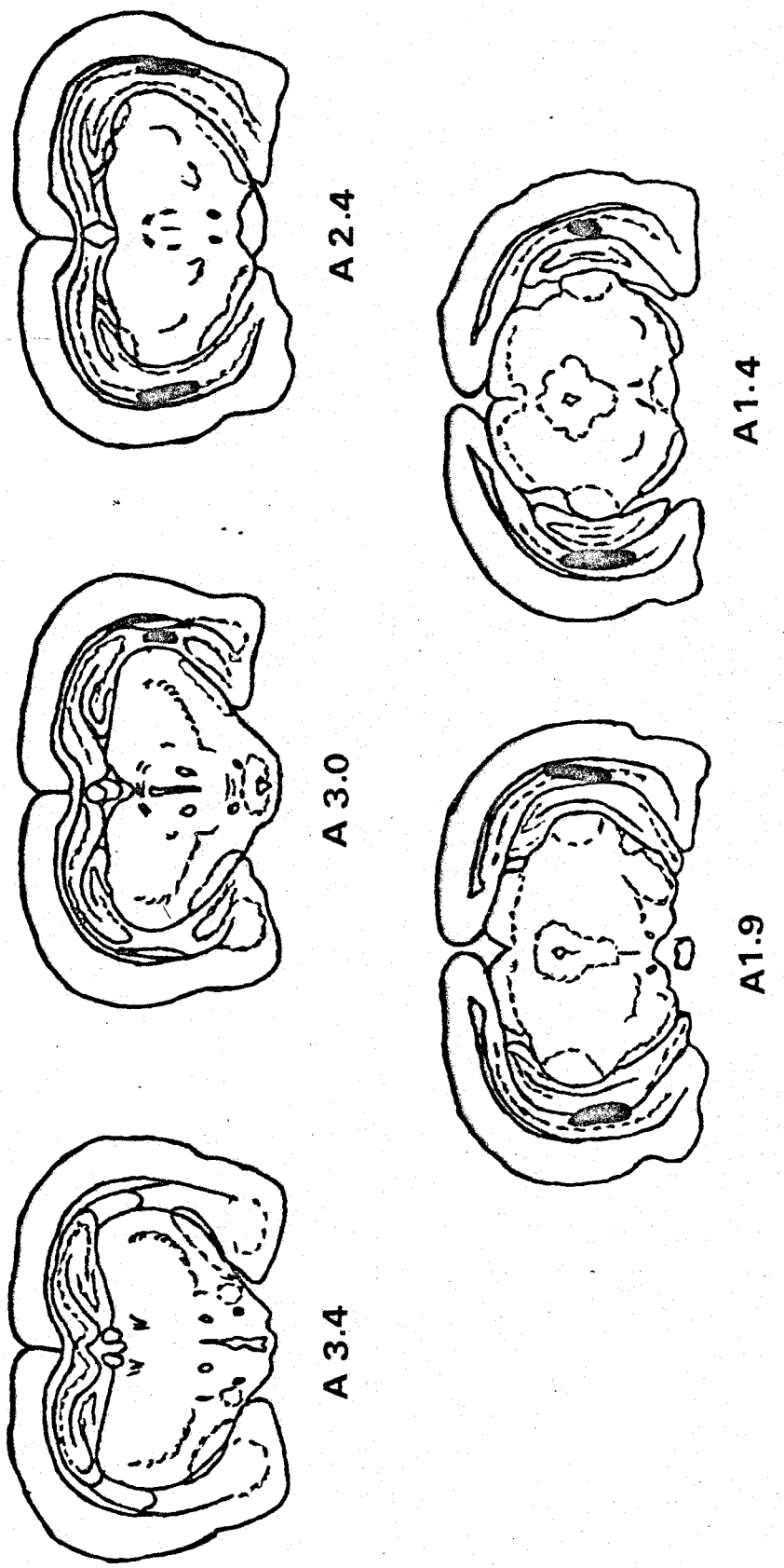




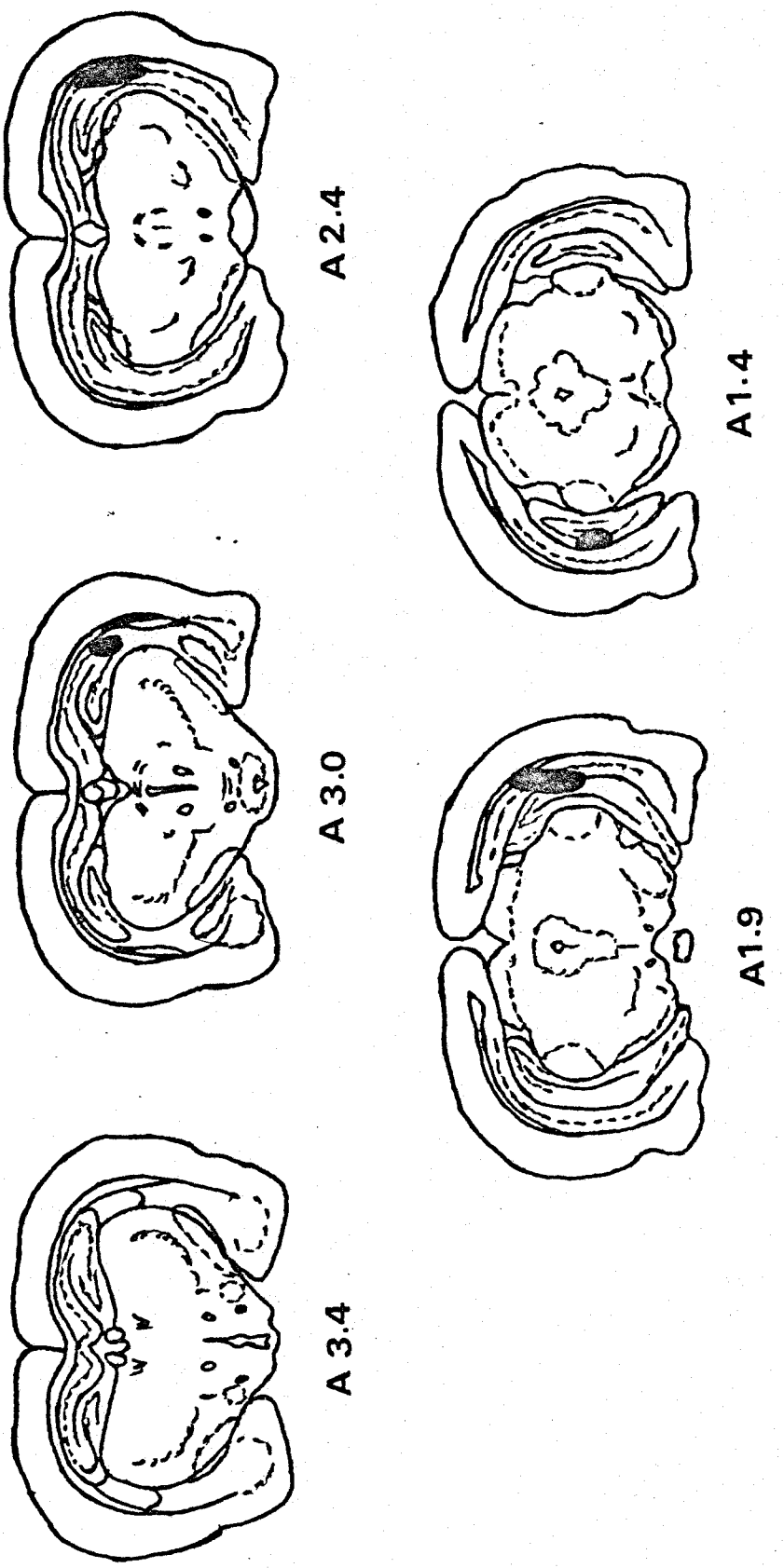
Hipp. 6



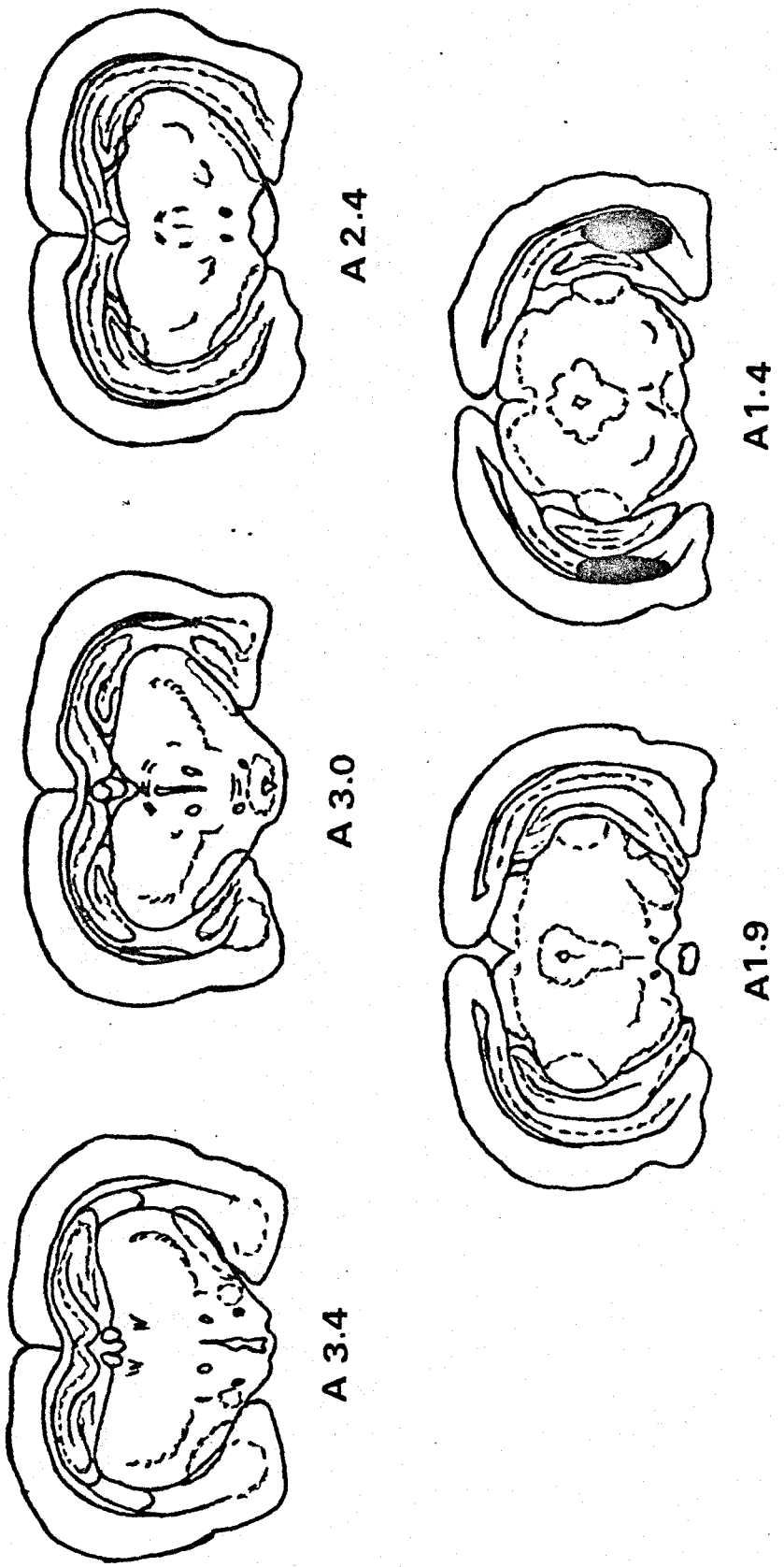
Hipp. 7



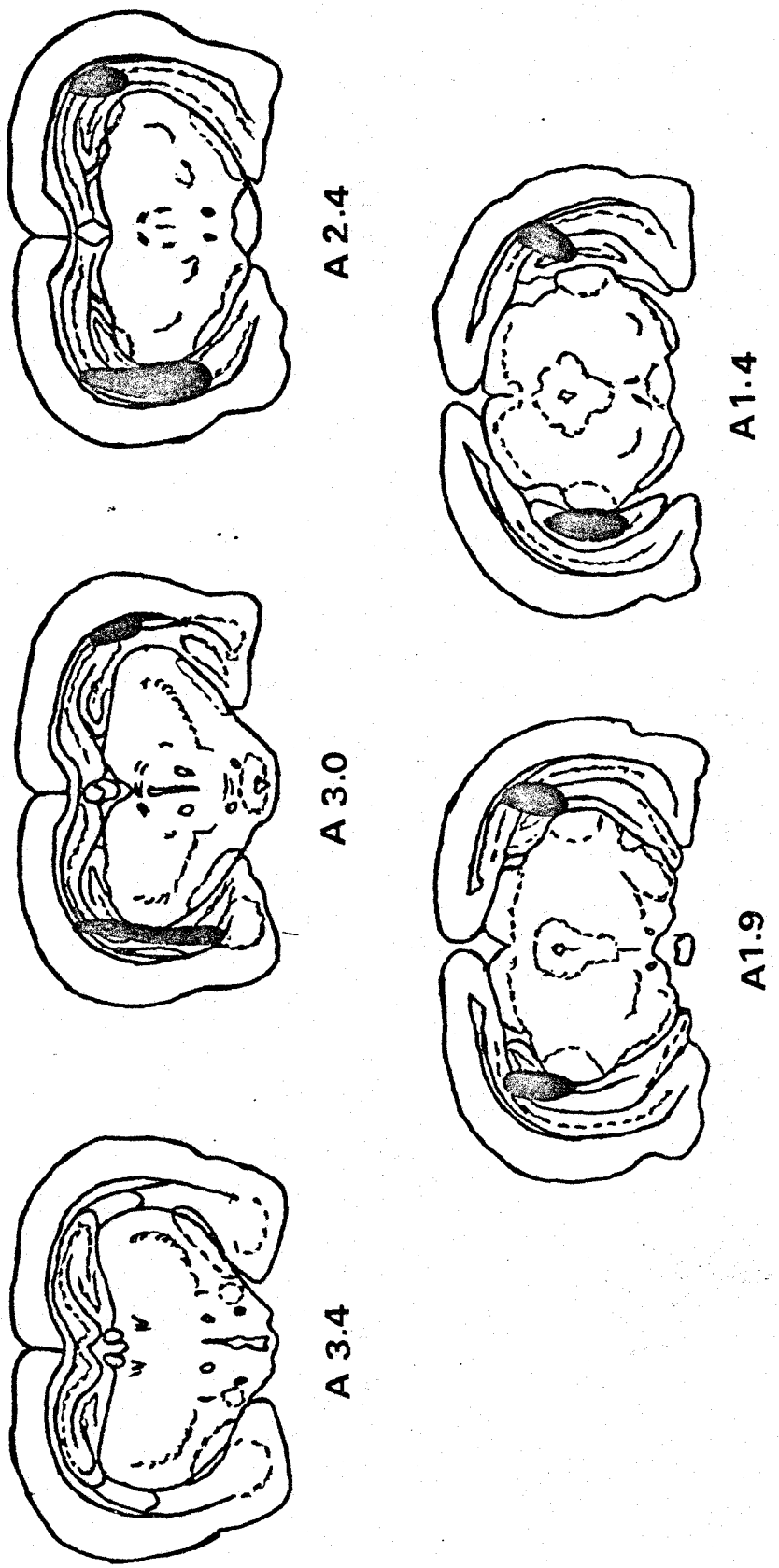
Hipp. 8



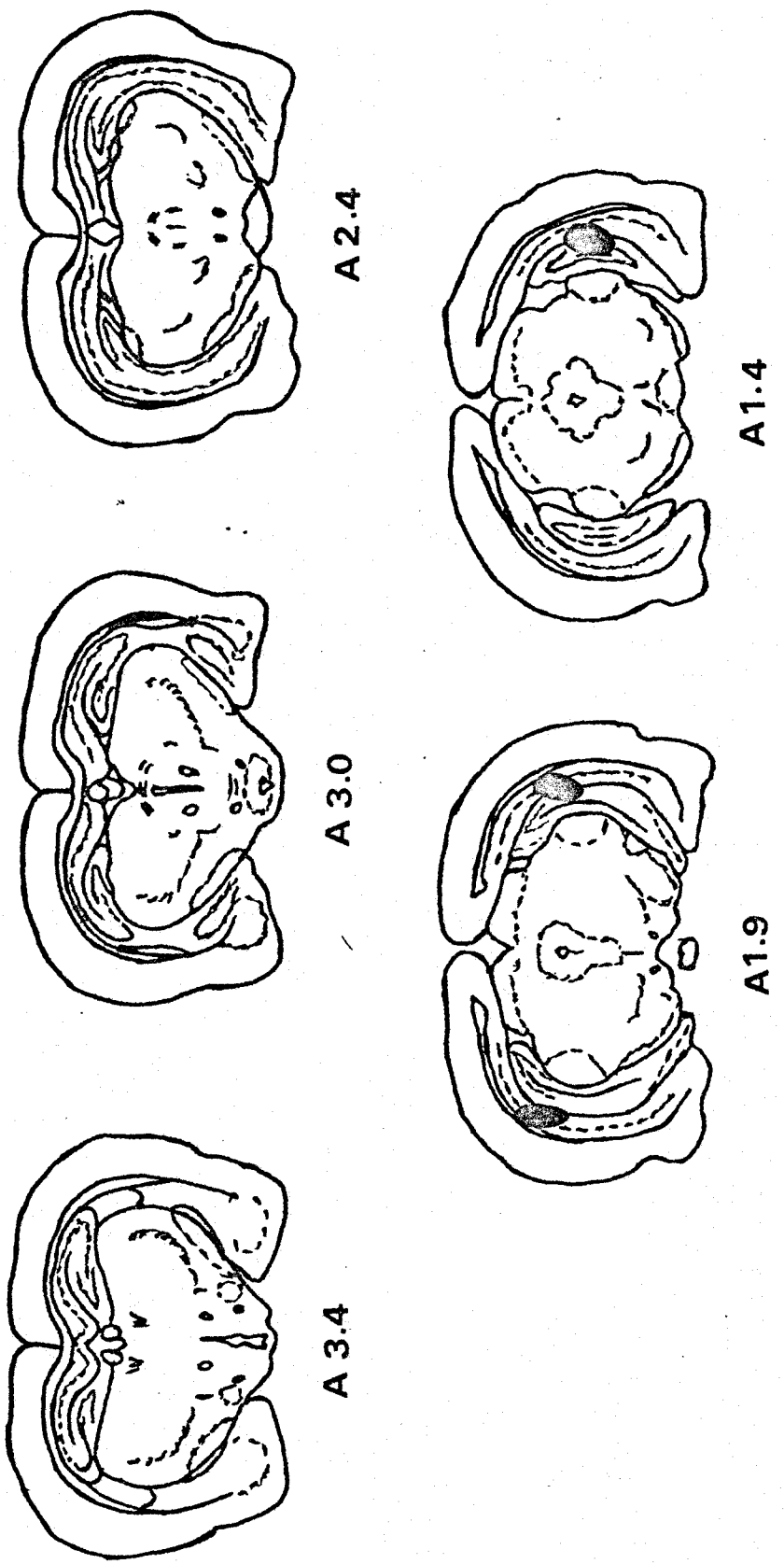
Hipp. 9



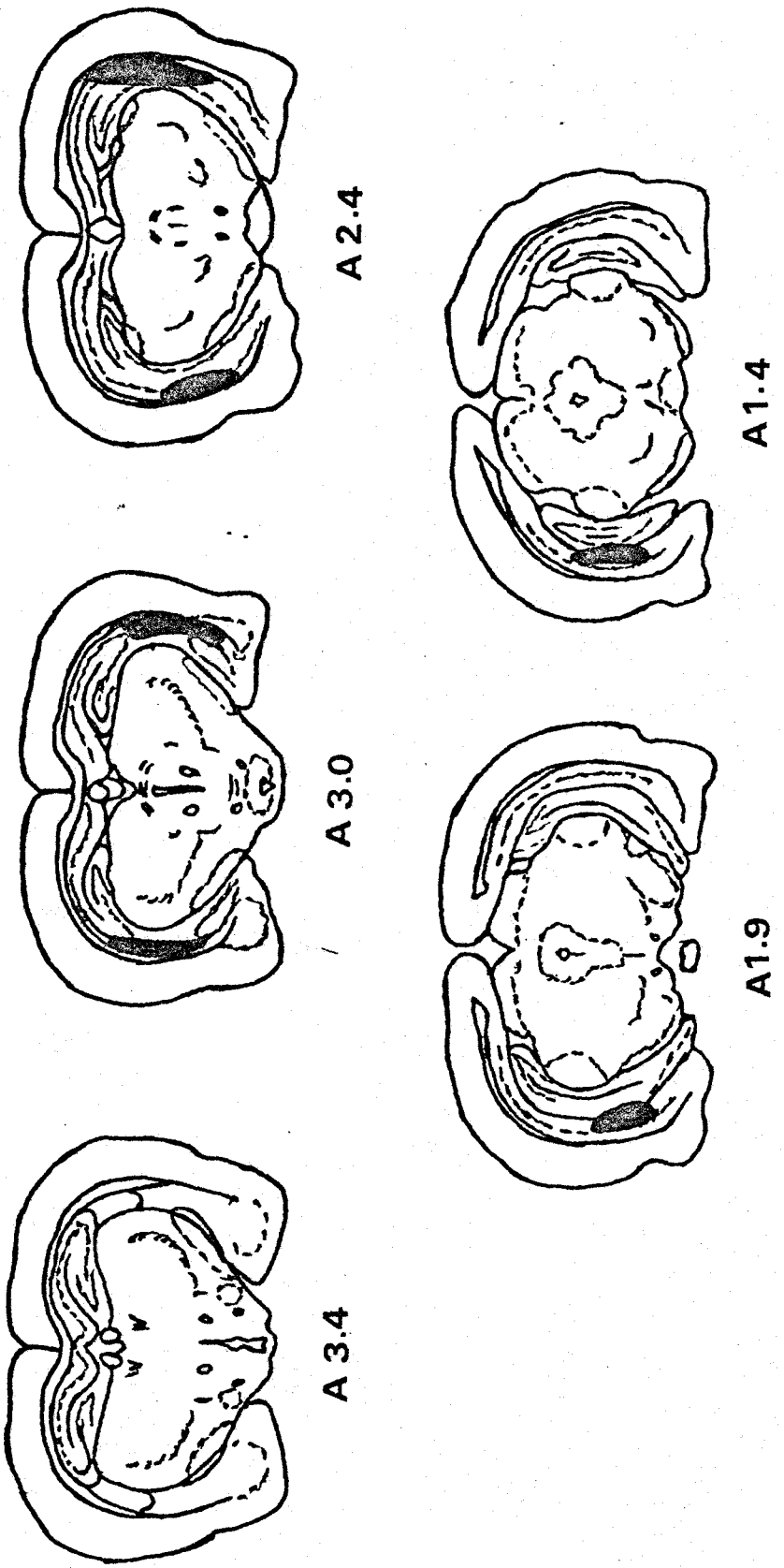
Hipp. 10



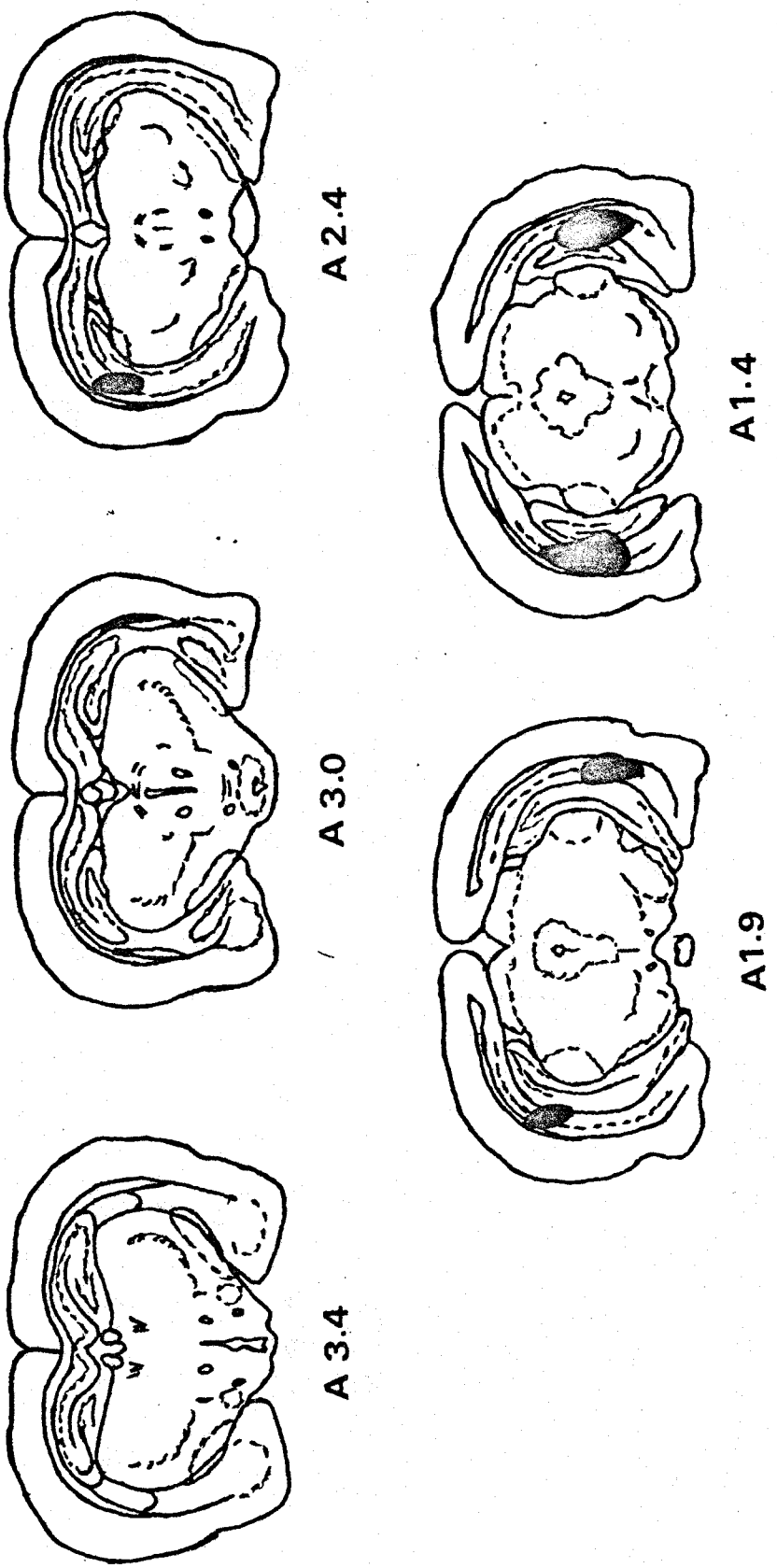
Hipp. 11



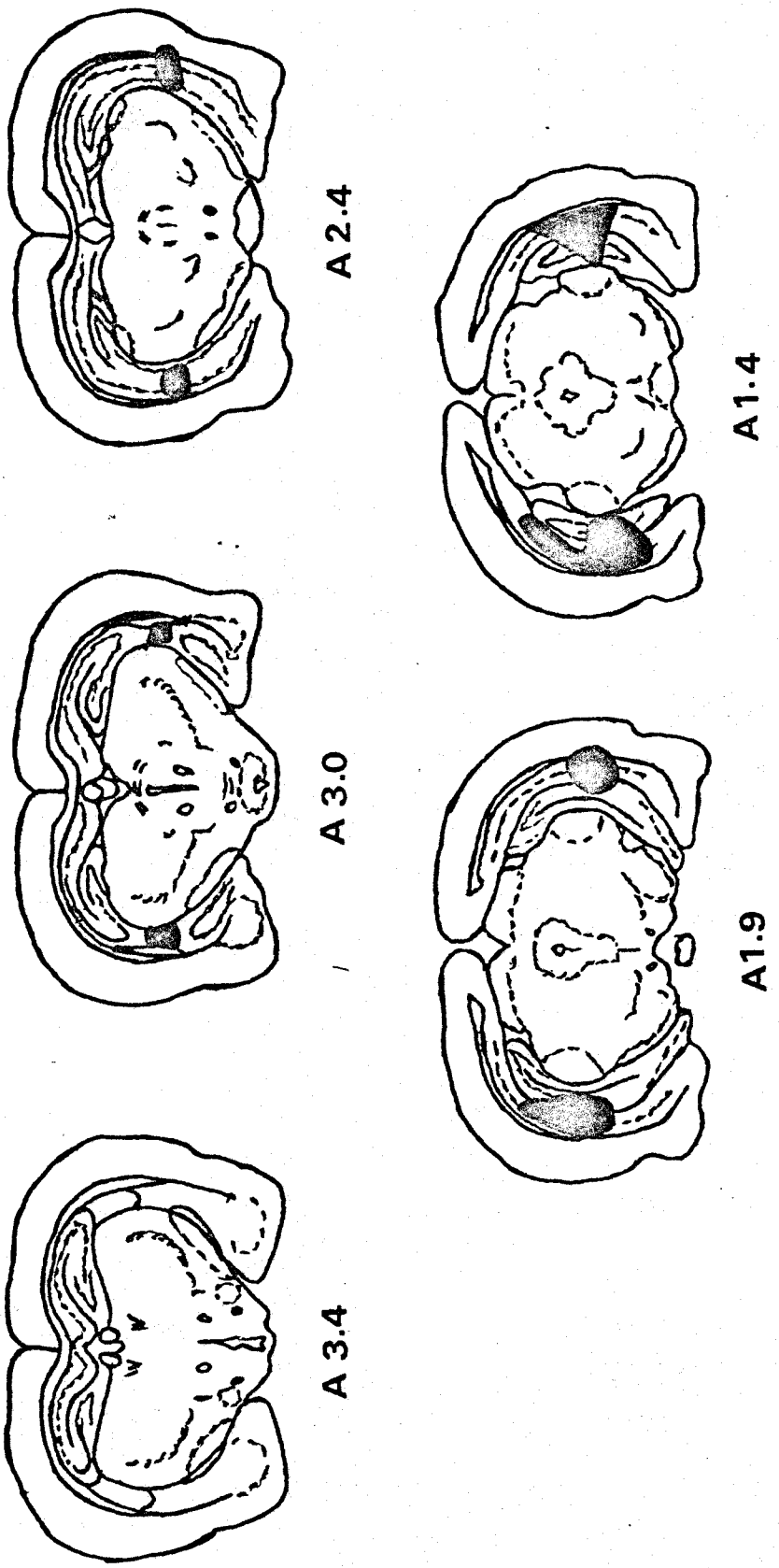
Hipp. 12



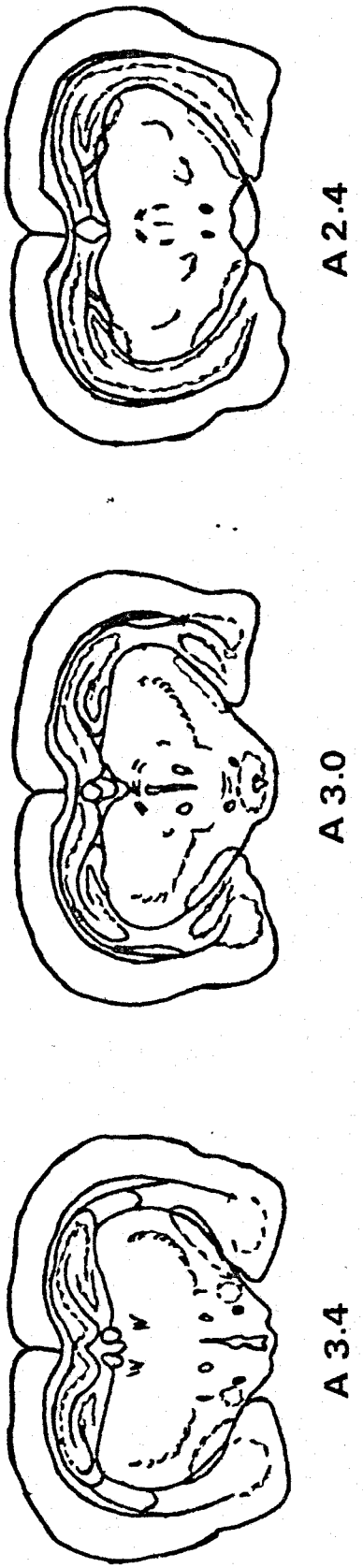
Hipp. 13

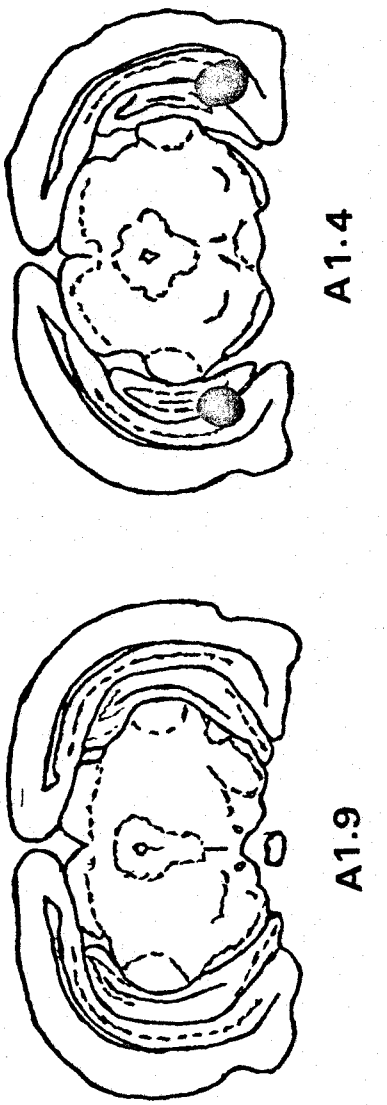


Hipp. 14

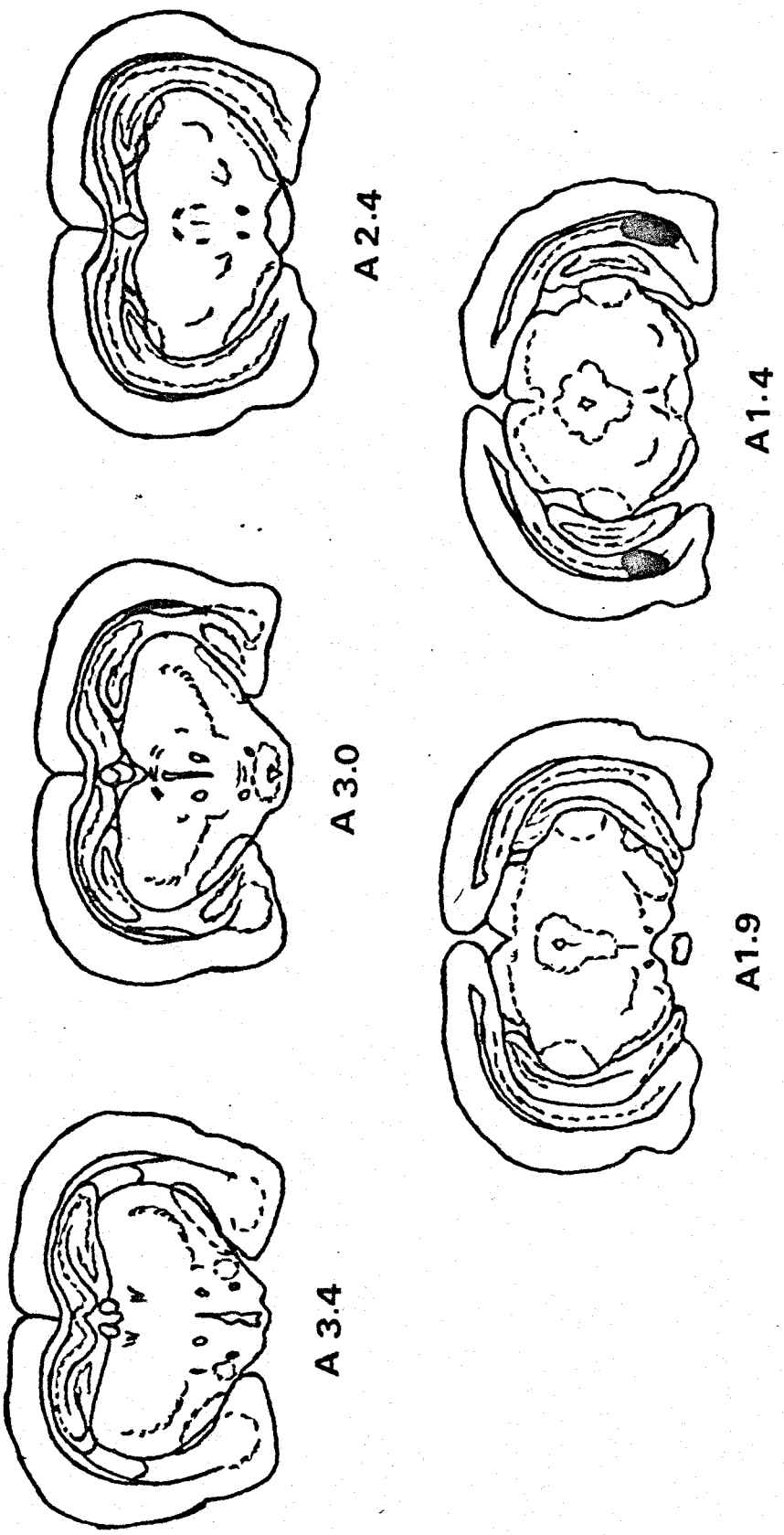


Hipp. 15

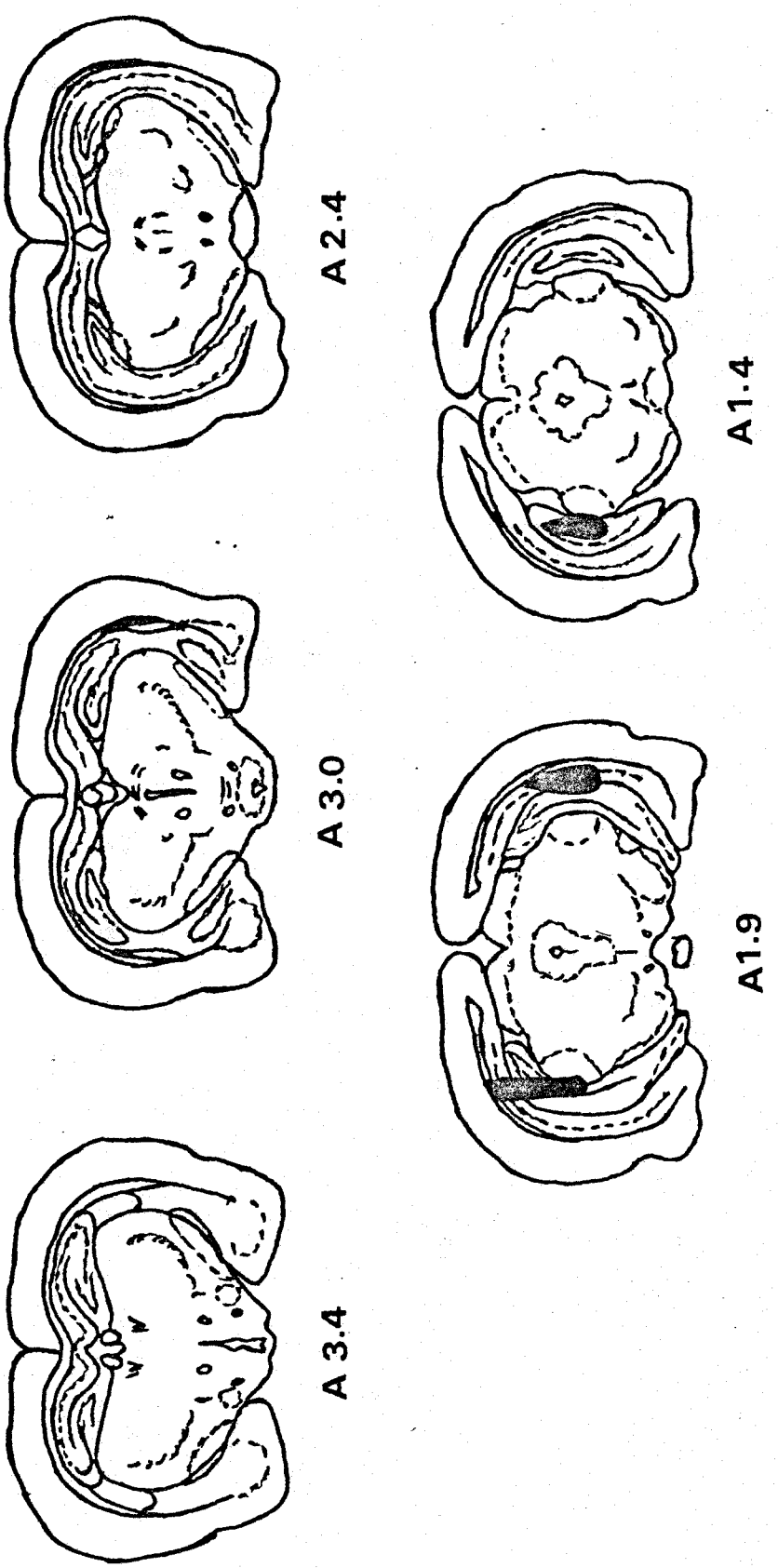


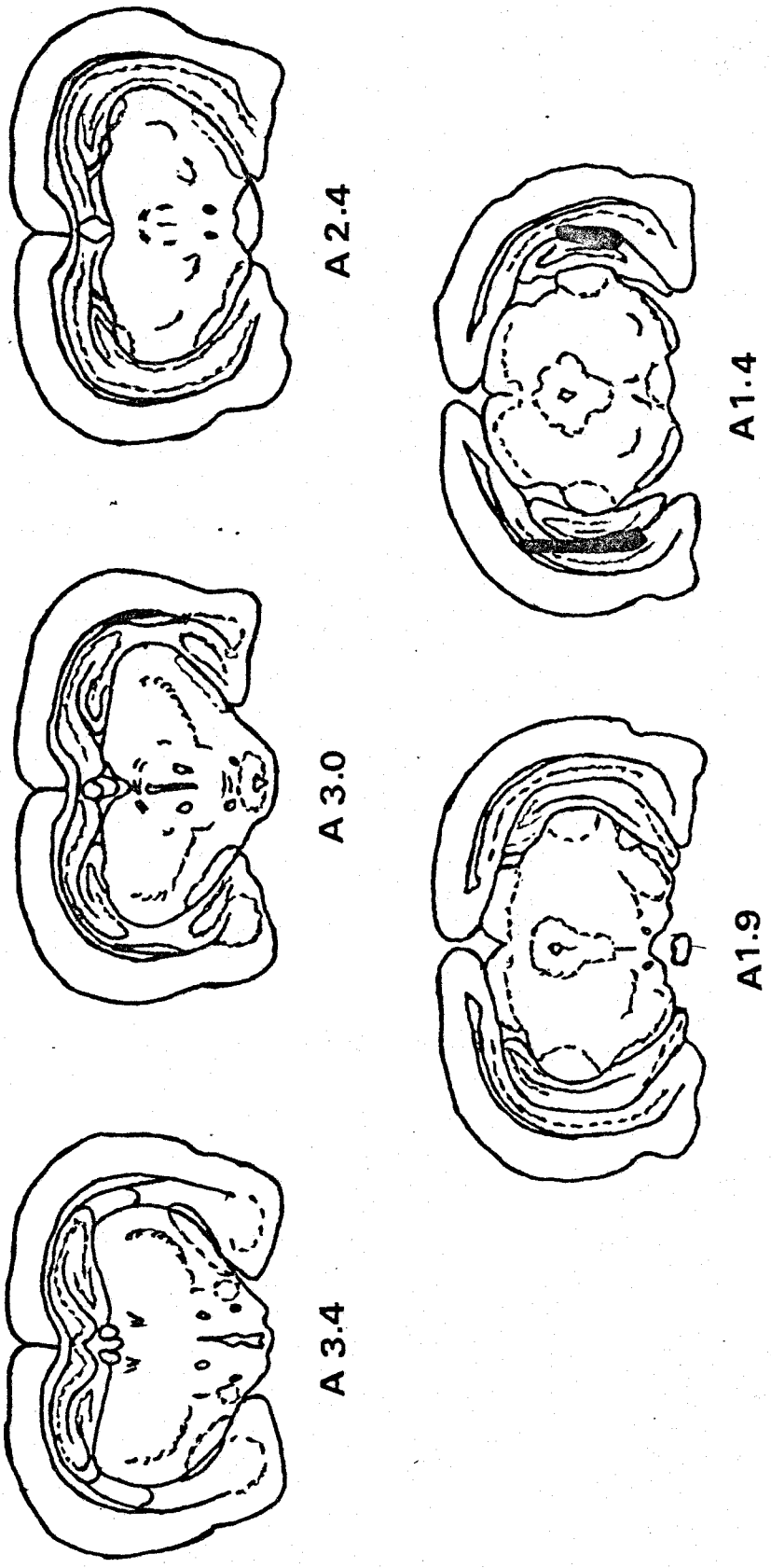


Hipp. 16

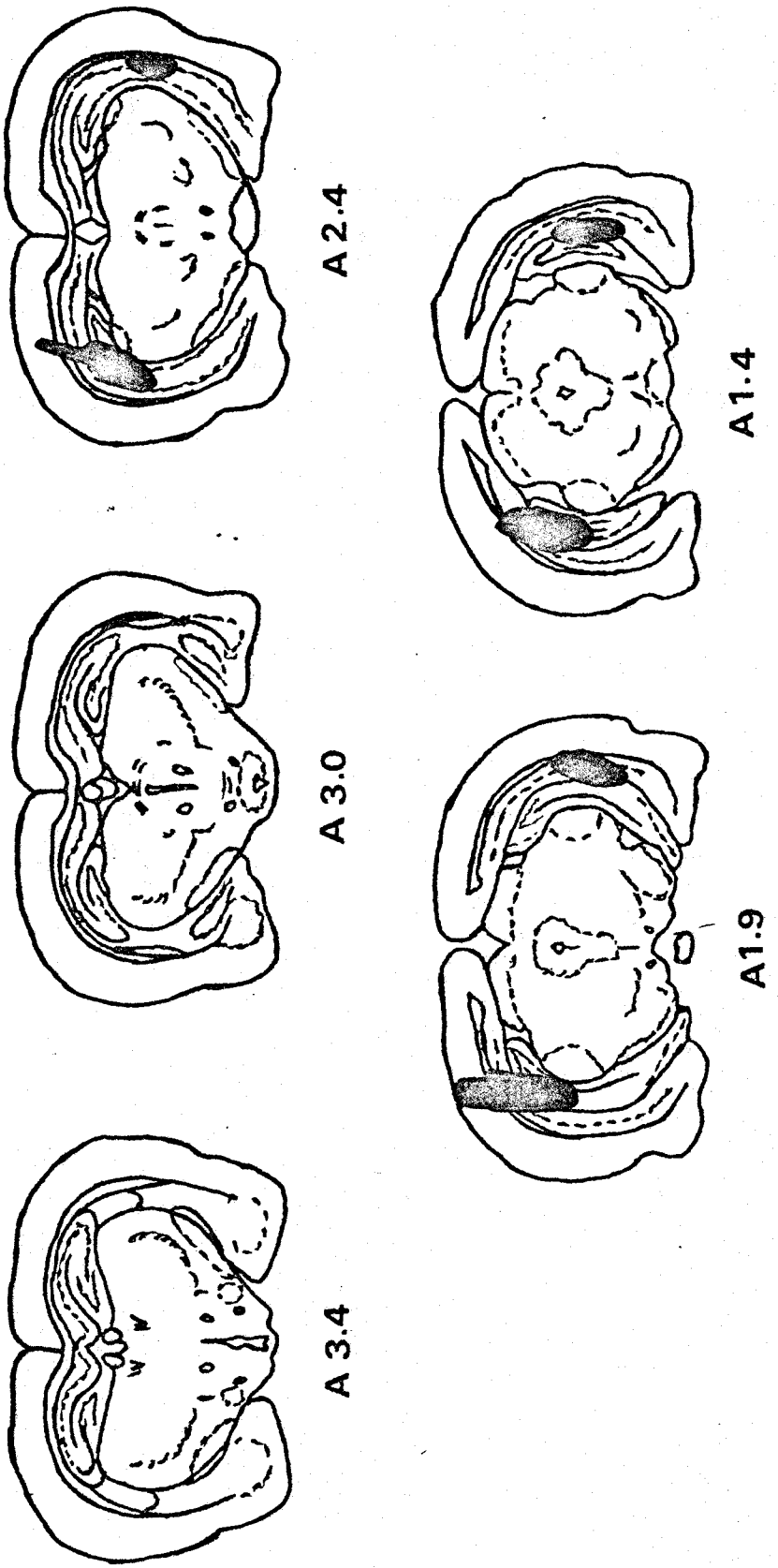


Hipp. 17

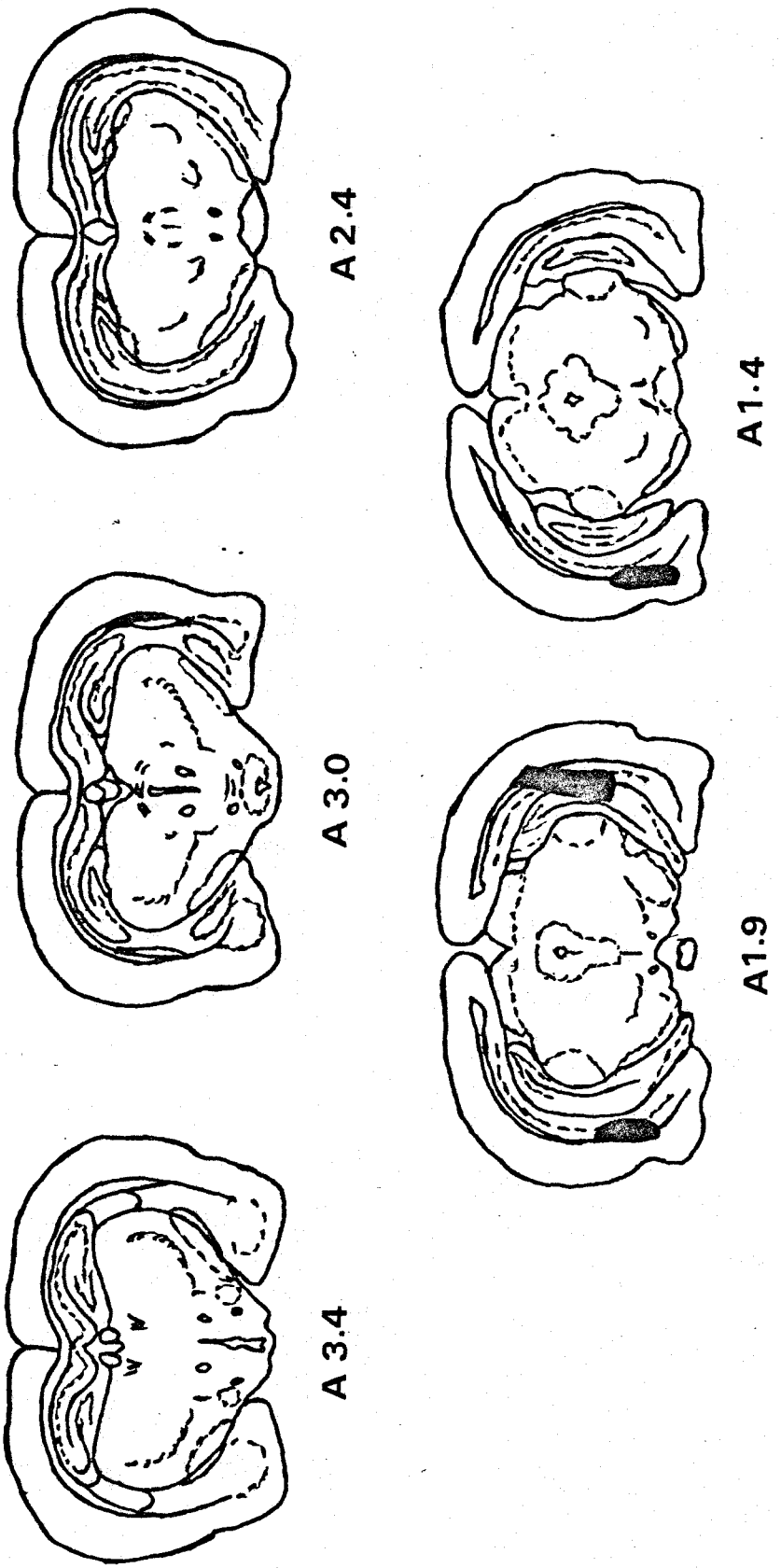


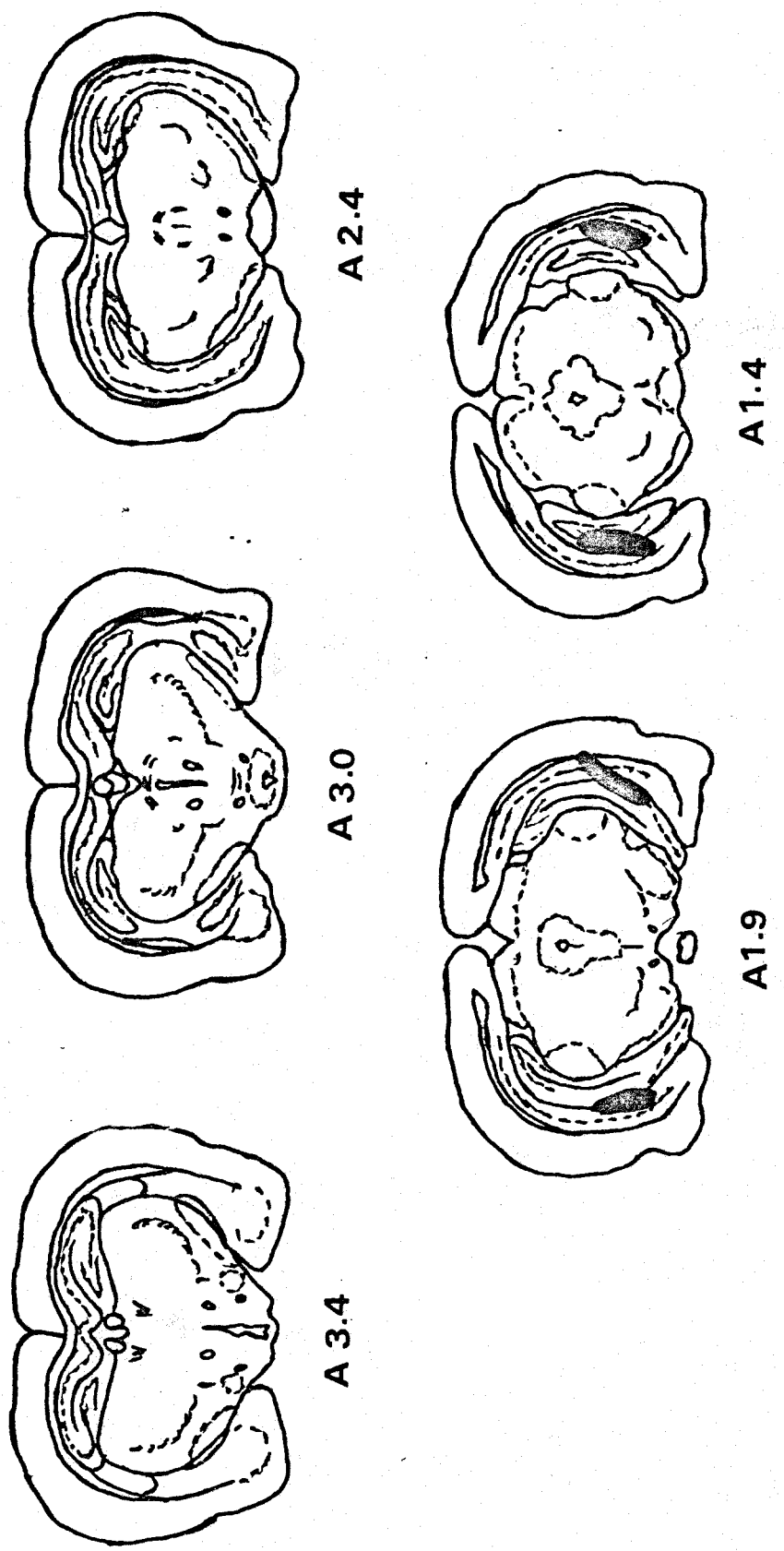


Hipp. 19

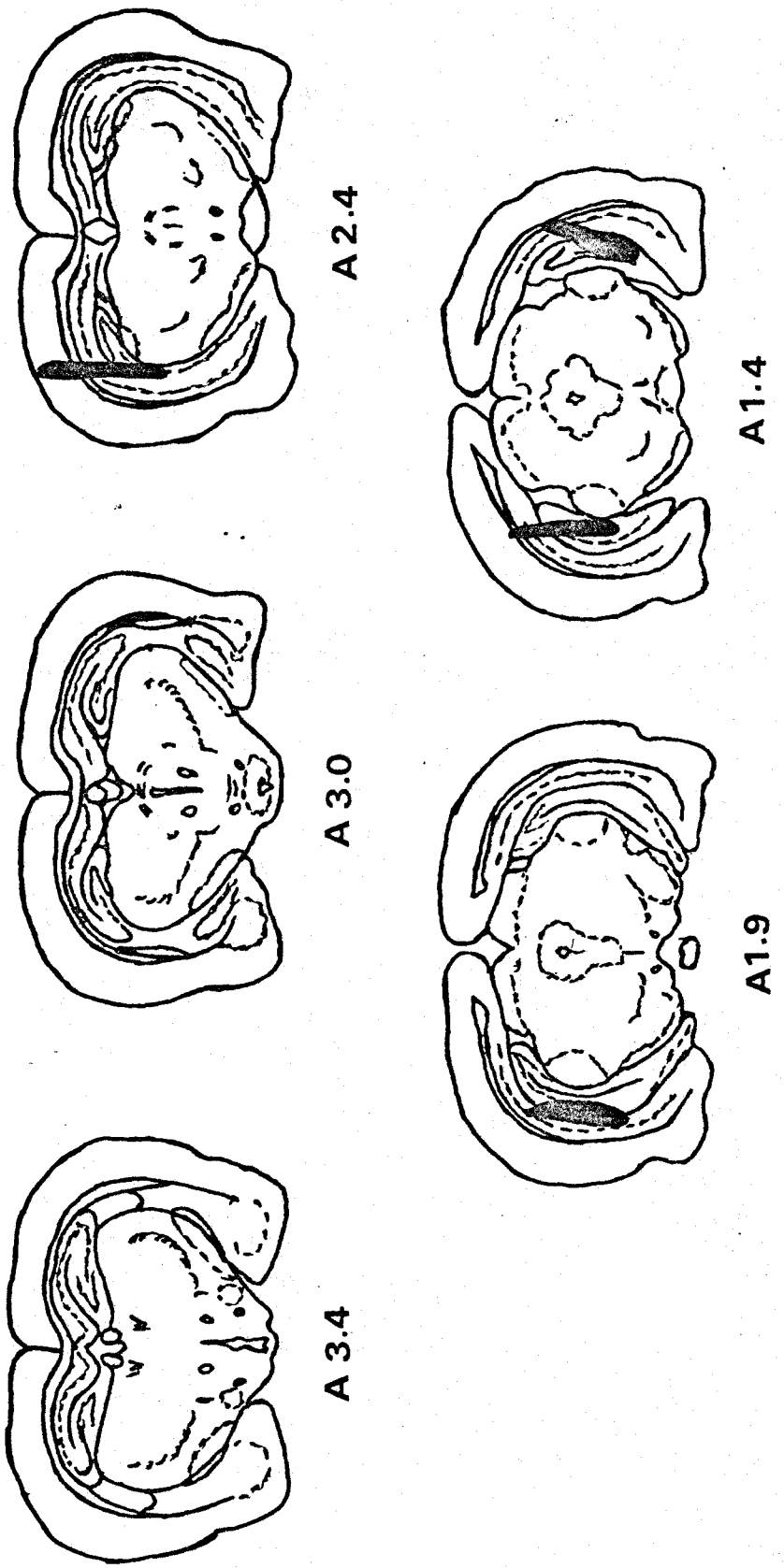


Hipp. 20

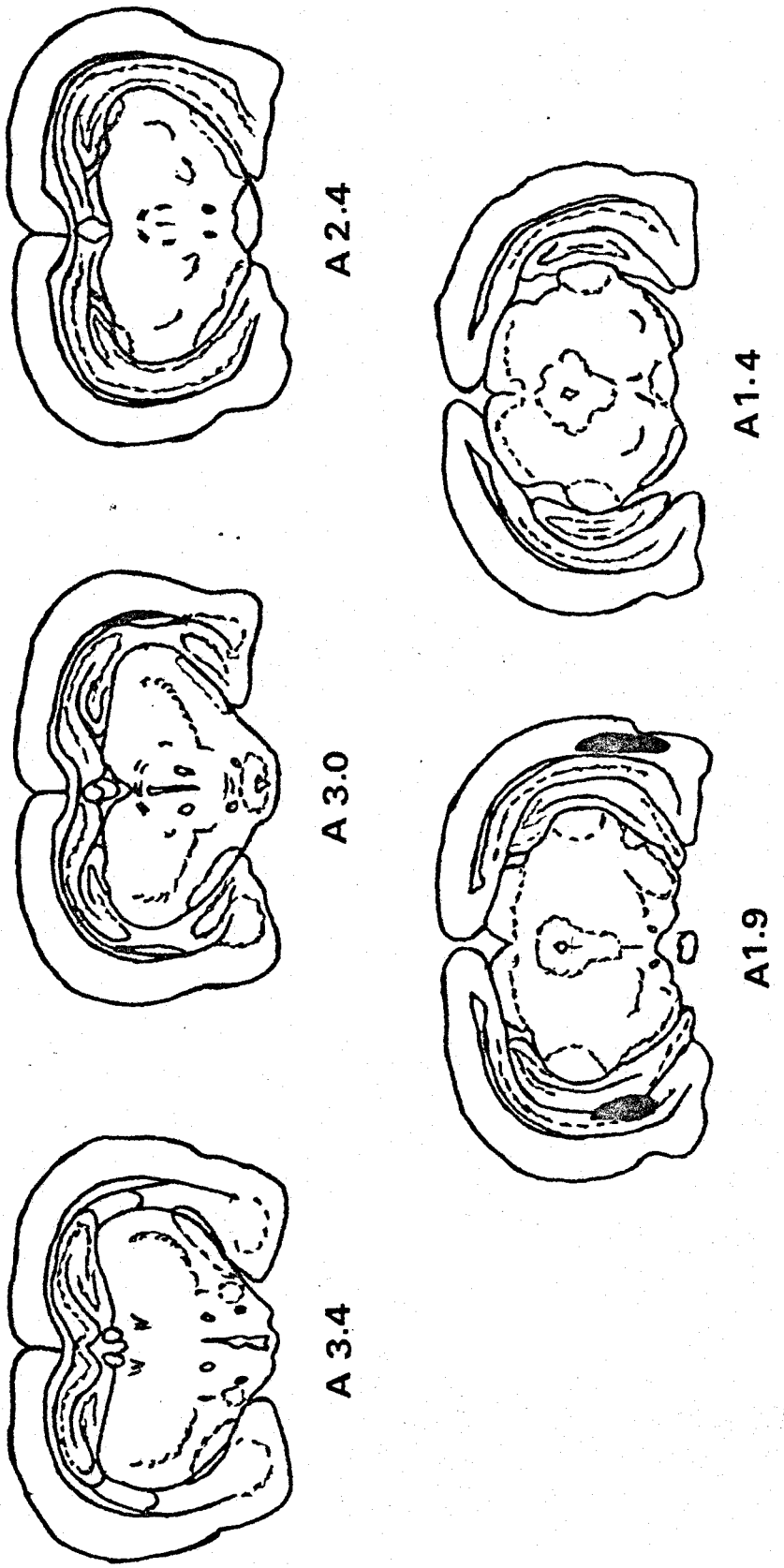




Hipp. 22



Hipp. 23



Hipp. 24

VITA AUCTORIS

NAME:	Jean Paul Emile Laroche
PLACE AND YEAR OF BIRTH:	Montreal, Quebec, 1948
EDUCATION:	St. Alice School, Montreal, Quebec 1954-1961
	St. Pius X High School, Montreal, Quebec 1961-1965
	Loyola College, Montreal Quebec Graduated with B.A. degree. 1965-1969
	University of Windsor, Windsor, Ontario Registered as a full-time graduate student. 1969
EXPERIENCE:	Loyola College, Experimental Psychology Lab Instructor 1968-1969
	University of Windsor, Psychology 15 (Introductory) Teaching Assistant 1969-1970
	University of Windsor, Experimental Psychology Teaching Assistant 1970-1971
AWARDS:	Province of Ontario Graduate Fellowship May, 1970 - September, 1971
PUBLICATIONS:	Cohen, J.S., Laroche, J.P., & Beharry, E. Response perseveration in the hippocampal lesioned rat. Psychonomic Science, 1971, 23 (3), 221-223.