AMYGDALOID LESIONS AND BEHAVIORAL INHIBITION IN THE RAT

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by

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A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Doctor of Philosophy

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Montreal

March, 1967
Acknowledgements

I would like to gratefully acknowledge the assistance of the following people: Mr. W. Mundl for his technical assistance in the design and construction of the apparatus; Mrs. A. Szikita for typing the manuscript; and my fellow graduate students, M. Baum, H. Buchtel and R. Musty for their helpful suggestions during preparation of the manuscript.

I am most indebted to my wife, Ann, for her encouragement, patience and assistance throughout this study.
# TABLE OF CONTENTS

**INTRODUCTION** ................................................................. 1

The Concept of Response Inhibition ......................................... 1
Septal Area ............................................................................. 2
Hippocampus .......................................................................... 4
Amygdala ................................................................................ 7
The Present Investigation .......................................................... 14

**GENERAL SURGICAL AND HISTOLOGICAL PROCEDURES** .......... 15

**EXPERIMENT I.**
Effects of Amygdaloid Lesions on Passive Avoidance Behavior .......... 18

**EXPERIMENT II.**
Effects of Amygdaloid Lesions on DRL Performance ....................... 23

**EXPERIMENT III.**
Effects of Amygdaloid Lesions on Discrimination Learning and Reversal . . 27

**EXPERIMENT IV.**
Effects of Amygdaloid Lesions on Alternation Behavior .................... 30

**EXPERIMENT V.**
Effects of Amygdaloid Lesions on Fixed Ratio and Runway Performance . . . 33

**DISCUSSION** ................................................................. 37

**SUMMARY** ........................................................................ 50

**REFERENCES** ............................................................... 51

**FIGURES** ......................................................................... 63
The present investigation is concerned primarily with the effects of amygdaloid lesions on behavioral inhibition. Since much of the research which will be reported in this thesis stems from similar studies on the septal area and the hippocampus, these data will first be summarized briefly. Then the relevant anatomical and behavioral data on the amygdala will be discussed more fully. Since the recent literature contains two comprehensive reviews (Gloor, 1960; Goddard, 1964b) of the extensive research that has been done on amygdaloid function, it seems unnecessary to duplicate these efforts. This review, therefore, will deal only with those studies that are directly relevant to the experiments to be reported.

The Concept of Response Inhibition

In the past fifteen years there has been a growing interest in the response modulating functions of the structures in the limbic system. This interest began with the extensive electrophysiological investigations of Kaada (1951) with the cat, dog and monkey. Among other findings, Kaada reported that stimulation of the area surrounding and below the genu of the corpus callosum (septal area) produced inhibition of on-going autonomic and somatomotor responses, while stimulation of the anterior and medial cingulate cortex
produced facilitation of these same motor responses. He also reported somatomotor inhibition following stimulation of the medial portions of the amygdala and facilitation following stimulation of the more lateral aspects of the amygdala.

From these electrophysiological observations, McCleary (1961) hypothesized that lesions in Kaada's inhibitory area should disrupt an animal's ability to inhibit responding, thus producing perseverative behavior. Conversely, lesions in Kaada's motor facilitatory areas should disrupt the performance of an active response. McCleary predicted that septal lesions would disrupt passive avoidance learning but not the learning of an active avoidance response, and conversely, that cingulate lesions would disrupt active avoidance behavior but not passive avoidance. Indeed, McCleary (1961) found just such a double dissociation and concluded that the septal area was part of a circuit mediating response inhibition. In the six years since McCleary's report a considerable amount of evidence has collected in the literature concerning this hypothesis of limbic system function. In order to simplify matters, the relationship of three structures: septal area, hippocampus and amygdala to the response inhibition hypothesis will be dealt with separately.

**Septal Area**

The evidence implicating the septal area in response
inhibition comes both from studies of fear motivated behavior (active and passive avoidance and conditioned emotional behavior) and from studies where food or water motivation is employed.

It has generally been found that electrolytic lesions of the septal area produce a deficit in passive avoidance behavior (Kaada, Rasmussen & Kveim, 1962; McCleary, 1961; Schwartzbaum & Spieth, 1964; Zucker & McCleary, 1964) but either facilitate or have no effect on two-way shuttlebox avoidance (Fox, Kimble & Lickey, 1964; Kenyon, 1962; Kenyon & Krieckhaus, 1965; Krieckhaus et al., 1964; McCleary, 1961). Rewarding levels of septal stimulation interfere with the acquisition of a conditioned emotional response (Brady & Conrad, 1960; Goldstein, 1962) and low level septal stimulation disrupts the acquisition of a passive avoidance response (Kasper, 1964; Schwartzbaum & Donovick, 1965).

Evidence of perseverative behavior following septal lesions has also been found in situations where there is no fear motivation present. Zucker and McCleary (1964) found that although septal lesions had no effect on the acquisition of a food rewarded position habit, the lesions did produce a deficit in the reversal of this habit. Schwartzbaum et al. (1964a) trained rats preoperatively to discriminate between two tones in a bar-pressing situation. After placing septal
lesions in these rats, they noted that the animals showed sustained increases in bar-pressing during nonreinforced conditions (during $S^A$). These authors also reported that a high number of perseverative errors were made by rats with septal lesions during the acquisition of a brightness discrimination. Disruption of both fixed interval responding (Ellen & Powell, 1962) and DRL performance (Ellen, Wilson & Powell, 1964) have also been reported in rats with septal lesions.

Some of these results have been replicated with the use of septal stimulation. Olds & Olds (1961) found that non-contingent rewarding septal stimulation produced an impairment in the ability of rats to learn daily response reversals. Deficits in DRL performance (Kaplan, 1965) and position habit reversal (Kasper, 1965) have also been reported when nonrewarding septal stimulation was used.

The septal data that have been briefly reviewed above support the response inhibition hypothesis of septal function proposed by McCleary (1961). Several alternative hypotheses have been offered by other investigators but a discussion of these is beyond the scope of this review (see McCleary, in press).

Hippocampus

One pattern of behavior which follows hippocampal lesions or ablations has been described by Kimble (1963) as
an increased degree of perseverative behavior. The hippocampal animal generally shows a tendency to persist in previously learned responses when these responses are no longer appropriate in the situation. This pattern of behavior is so similar to the perseverative behavior of the septal animal that, according to McCleary (in press) they cannot as yet be convincingly differentiated from one another.

Several investigators (Isaacson & Wickelgren, 1962; Kimble, 1963; Kimble et al., 1966; Kimura, 1958; Snyder & Isaacson, 1965; Teitelbaum & Milner, 1963) have reported that the hippocampal animal is unable to inhibit a previously learned approach response in a passive avoidance situation. It appears that large posterior dorsal lesions (Teitelbaum & Milner, 1963) are more effective than anterior dorsal hippocampal lesions (Kaada et al., 1962; Kveim et al., 1964) in producing a deficit in passive avoidance. In addition to lesion locus, the nature of the passive avoidance task used (Snyder & Isaacson, 1965) and the amount of pretraining (Isaacson et al., 1966; Kimble et al., 1966) appear to be important variables in the degree of passive avoidance impairment that is found after hippocampectomy. Hippocampal lesions have also been shown to facilitate the acquisition of an active avoidance response (Isaacson et al., 1961).

Hippocampal animals have been found to persist in loco-
motor activity when it is measured in large chambers (Douglas & Isaacson, 1964; Teitelbaum & Milner, 1963), mazes (Roberts et al., 1962), and exploratory boxes (Kaplan, 1966) but not when measured in small chambers (Kim, 1960) or running wheels (Kaada et al., 1961; Leaton, 1963). Kaplan (1966) has suggested that conditions which enhance "approach" or exploratory behavior appear to be more likely to produce prolonged activity in hippocampectomized animals.

Perseverative behavior has also been observed when hippocampal animals are shifted from a continuous to an intermittent schedule of reinforcement. Hippocampal animals are unable to learn to withhold or delay their responses on a DRL schedule (Clark & Isaacson, 1965). This DRL deficit, like the passive avoidance deficit, has been shown to depend upon the amount of pretraining the subjects receive on a continuous reinforcement schedule (Schmaltz & Isaacson, 1966). It appears that the longer the pretraining period, the greater the DRL deficit as measured by rate of response. High rates of response have also been observed from hippocampal animals on a variable interval schedule of reinforcement (Jarrard, 1965).

Kimble (1966) has interpreted the slower extinction of hippocampectomized animals in a runway (Jarrard et al., 1964), in operant conditioning situations (Niki, 1965; Peretz, 1965; Teitelbaum, 1961), and avoidance tasks (Isaacson, Douglas &
Moore, 1961) as evidence of a perseverative tendency. Additional evidence for perseveration in hippocampal animals comes from deficits in alternation behavior (Lash, 1964; Pribram et al., 1962; Mahut & Cordeau, 1963). However, Kaplan's (1966) recent findings that rats with hippocampal damage do not perseverate a conditioned freezing response and habituate as fast as controls in certain situations indicate that there are limitations to the concept of perseveration.

Amygdala

The amygdala is a relatively small but complex mass of gray matter in the depth of the mammalian temporal lobes. It is usually divided into two closely related groups of nuclei: the phylogenetically older corticomedial complex and the more recent basolateral group (Johnston, 1923; Humphrey, 1936; Gloor, 1960).

The basolateral group consists of the lateral nucleus, the accessory basal nucleus, and the large-celled lateral portion of the basal nucleus. The corticomedial group can be subdivided into the cortical, central, and medial nuclei and the small-celled medial portion of the basal nucleus. The nucleus of the lateral olfactory tract is also part of the corticomedial group (Gurdjian, 1928; Gloor, 1960).

Afferent Connections. Of all the afferent connections
to the amygdala, only the olfactory fibers are anatomically well documented. Projections from the olfactory bulb, traversing via the lateral olfactory tract, terminate in all of the corticomedial nuclei except the central nucleus (Cowan, Powell & Raisman, 1965; Gloor, 1960).

Other afferent input to the amygdala has been demonstrated from the piriform cortex (Gloor, 1960; Cowan et al., 1965), the reticular formation (Machne & Segundo, 1956), the hippocampus (Gloor, 1960), the thalamus (Wendt & Albe-Fessard, 1962), and the rostral hypothalamus (Cowan et al., 1965).

Efferent Connections. There are two main efferent pathways from the amygdala: the stria terminalis and the ventral amygdalofugal pathway. The stria terminalis appears to originate mainly in the corticomedial region but may also receive some fibers from the basolateral group via intramygdaloid connections (Gloor, 1960). The stria terminalis projects to the basal septal region, head of the caudate nucleus, preoptic area and anterior and ventromedial hypothalamic nuclei (Gloor, 1960). The ventral amygdalofugal pathway originates in the piriform cortex, receives additional fibers from the basolateral nuclei and projects to the same region of the rostral hypothalamus as the stria terminalis (Cowan et al., 1965; Gloor, 1960).
There is now substantial evidence (Fox, 1949; Nauta, 1961; Valverde, 1963) for a third efferent pathway which projects to the dorsomedial nucleus of the thalamus and eventually connects with the orbitofrontal cortex.

Gloor (1960) has divided the amygdaloid efferent connections into two classes. The primary amygdaloid projection field, which characteristically shows short latency responses to amygdaloid stimulation, consists of the basal septal region, head of the caudate nucleus, preoptic area, anterior and ventromedial hypothalamic nuclei, anterior temporal and insular cortex. The secondary projection field, which shows longer latency responses to amygdaloid stimulation, consists of the remaining hypothalamic nuclei, subthalamus, entopeduncular nucleus, mesencephalic tegmentum and hippocampus.

From the anatomical evidence reviewed above, it becomes apparent that the amygdala is directly connected to the septal area and indirectly to the hippocampus. Since the latter two areas appear to be involved in behavioral inhibition, it seems quite possible that the amygdala plays a similar role in behavior. However, while a considerable amount of research effort has been devoted to studying the inhibitory functions of the hippocampus and septal area, it is unfortunate that the amygdala has received relatively little attention in the
attack on this general problem.

It was mentioned earlier that Kaada (1951) found a trend toward somatomotor facilitation in the lateral areas of the amygdala and somatomotor inhibition in the medial region of the amygdala. However, it should be emphasized that points producing facilitation and those eliciting inhibition are not clearly separated but overlap extensively (Gloor, 1960). Studies on autonomic, somatomotor and behavioral effects of amygdaloid stimulation are numerous, confusing and often contradictory in the effects reported. After a careful review of these experiments, Gloor (1960) concluded that a topographical organization of function appeared to be absent in the amygdala.

Suggestions of a possible inhibitory role of the amygdala come from several sources. Brutkowski, Fonberg and Mempel (1960) trained dogs to place a leg on a platform for food reinforcement when a tone was presented ("excitatory conditioned response") and to inhibit this response when the tone was paired with a rattle ("inhibitory conditioned response"). Following bilateral removal of the amygdala by aspiration, the performance of the "inhibitory conditioned responses" was severely impaired while "excitatory conditioned responses" remained unaffected.

Amygdaloid lesions have been shown to impair the
acquisition of a conditioned emotional response (CER) (Kellicutt & Schwartzbaum, 1963). None of the lesioned animals in this study showed any clear-cut evidence of suppression of a bar-pressing response for food reinforcement. A similar deficit in CER acquisition has been reported (Goddard, 1964a) when low level, continuous stimulation of the amygdala was used instead of lesions. Goddard suggested that the stimulation was acting as a "functional lesion" by scrambling the otherwise orderly traffic of impulses through the amygdala.

Using a passive avoidance paradigm, Ursin (1965) found that lesions of the stria terminalis and/or medial nucleus of the amygdala in cats interfered with the inhibition of a previously learned approach response to a food cup. Lesions of the lateral nucleus disrupted the acquisition of an active avoidance response, but did not affect passive avoidance behavior. These behavioral data fit nicely with Kaada's (1951) electrophysiological findings. However, Horvath (1963) found a small but statistically significant passive avoidance deficit from basolateral lesions in cats. The small size of the deficit may reflect the fact that the cats had extensive experience with electric shock in an active avoidance task before they were tested in the more sensitive passive avoidance task. Pellegrino (1965) found that low-level continuous stim-
ulation of the amygdala produced a passive avoidance deficit in rats. Thirsty rats receiving stimulation of the amygdala, particularly the basolateral region, were unable to inhibit an approach response to an electrified water spout from which they had previously been taught to drink. Thus it is not yet clear which of the two subdivisions of the amygdala is critical for passive avoidance behavior. It may well be, as Gloor (1960) has suggested for other behavioral functions of the amygdala, that there is no topographical localization of this task within the amygdala.

Fonberg & Delgado (1961) found that if they stimulated the basolateral region of the amygdala while a cat was spontaneously eating, the eating would immediately cease. The inhibition of food intake usually continued for several minutes after the stimulation was turned off. A similar inhibitory effect occurred if the cats were stimulated while bar-pressing for food. If the animal had raised one of its paws and touched the bar when the stimulation was turned on, the response would not be completed and the cat would put down its paw. The effect apparently could not be attributed to a deficit in motor coordination.

It has often been observed that many laboratory cats will not spontaneously attack rats. Electrical stimulation of the lateral hypothalamus of these cats will produce an
effective, well-directed attack on a rat placed in the test
cage with the cat (Egger & Flynn, 1962; 1963; Wasman &
Flynn, 1962). Simultaneous stimulation of this hypothalamic
attack area and the basolateral area of the amygdala results
in a complete suppression of hypothalamically elicited attack
responses (Egger & Flynn, 1962; 1963). Hypothalamic stimula-
tion has also been shown to elicit fear reactions (Fonberg,
1963a). These fear reactions can also be inhibited by simul-
taneous stimulation of the amygdala (Fonberg, 1963b).

Finally, Schwartzbaum et al. (1964b) have found that
rats trained in a "go-no-go" bar-pressing situation to dis-
 criminate between two tones and tone and no-tone displayed
severe impairment on retention performance after large bi-
lateral lesions in the amygdala. The amygdaloid lesioned rats
actually increased responding under nonreinforced conditions
(during S^). The authors concluded that the amygdala was
implicated in some forms of behavioral inhibition that are
normally associated with nonreinforced events. Amygdaloid
damage results in the perseveration of responses that are no
longer adaptive, that is, nonreinforced events fail to exert
adequate control over behavior.

After a careful review of the evidence cited above
Goddard (1964b) concluded that "the amygdala is primarily
involved in the active suppression of motivated approach
behavior. Once an amygdalectomized animal has overcome the initial postoperative depression and lethargy, it overeats, responds sexually to all stimuli whether dangerous or not with curiosity and is insensitive to variations in deprivation and food reward. In other words, it does not know when to stop."

The Present Investigation

The suggestion has been made in the above review that the amygdala may have an inhibitory role similar to that clearly demonstrated for the septal area and the hippocampus. The experiments in the present investigation were designed with several purposes in mind. First, by using tasks such as passive avoidance, go-no-go discrimination learning and reversal, DRL performance, and alternation behavior, an attempt was made to compare the behavioral effects of amygdaloid damage with those associated with septal and hippocampal damage. A second purpose was to test the hypothesis suggested by Goddard (1964b) that one function of the amygdala was the suppression of approach motivated behavior. Finally, throughout all of the experiments an attempt was made to determine whether the anatomical division of the amygdala into two distinct groups of nuclei, basolateral and corticomedial, has a functional correlate.
General Surgical and Histological Procedures

The subjects (Ss) in all experiments were male hooded rats obtained from the Quebec Breeding Farm, weighing 290-350 grams at the time of surgery. The Ss were individually housed in stainless steel wire mesh cages measuring 8.5 X 10 X 8 in.

Surgery was performed under Nembutal anesthesia (60 mg./Kg.) with the S's head held stationary in a Stoelting stereotaxic instrument. Bilateral amygdaloid lesions were produced by passing a two milliampere anodal current (d.c.) for 15 to 20 sec. through a formvar-insulated stainless steel electrode. The nose bar of the stereotaxic instrument was used as the indifferent electrode. The coordinates for basolateral lesions were: 0.75 to 1.0 mm. posterior to the bregma, 5.0 mm. lateral to the midline, and 6.5 to 7.0 mm. below the dura. The coordinates for corticomedial lesions were: 0.75 to 1.0 mm. posterior to the bregma, 3.5 to 4.0 mm. lateral to the midline, and 8.0 to 8.25 mm. below the dura.

Two types of sham operations were performed. One group of shams had two small holes drilled in their skulls and then were sutured and removed from the stereotaxic instrument. In the second group of shams the electrode was lowered down through the caudate/putamen to the edge of the amygdala, but not into the amygdala, and then removed without passing
any current.

Following surgery each S was given 200,000 units of penicillin IM, 0.5 cc. of Megimide (5 mg./ml.) IP, and Achromycin surgical powder was placed on the wound. All operated Ss were given a 10 to 14 day recovery period before behavioral testing began.

At the completion of testing the operated Ss were sacrificed with ether and perfused intracardially with 10% formol-saline. Following storage under refrigeration for 24 hours, the brains were removed from the skulls and kept in 10% formalin for 48 hours. All brains with lesions were sectioned at 40 μ on a freeze-microtome. Every fifth section through the lesion was mounted on a slide coated with a 1% gelatin solution and stained with luxol fast blue for myelinated fibers and neutral red for cell groups. The lesions were reconstructed by projecting the stained sections onto bilateral drawings made from the deGroot (1959) atlas. The lesions were assessed using a "single blind" procedure. Only those lesions which bilaterally damaged either the basolateral or the corticomedial groups of nuclei were considered acceptable. The data from Ss with damage overlapping these two areas or with bilateral damage to any surrounding structure were discarded and therefore are not included in the analyses reported here. The locus and extent of the bilateral
damage in these two types of lesions can be seen in the serial reconstructions in Fig. 1.
Experiment I. Effects of Amygdaloid Lesions on Passive Avoidance Behavior

It has previously been demonstrated that amygdaloid lesions in cats (Horvath, 1963; Ursin, 1965) and low level amygdaloid stimulation in rats (Pellegrino, 1965) produce deficits in passive avoidance. Since there is some question (Pellegrino, 1965; Ursin, 1965) about which of the two subdivisions of the amygdala is critical in this task, this experiment was designed to shed further light on this problem by investigating the effects of selective amygdaloid lesions in rats on passive avoidance.

Method

Subjects

The Ss were 49 experimentally naive male hooded rats. The groups consisted of 16 Ss with basolateral lesions, 11 Ss with corticomedial lesions, 11 sham-operated controls, and 11 normal controls.

Apparatus

Testing was carried out in a round metal chamber (Fig. 2) measuring 12 in. high X 11 in. in diameter. The chamber contained a metal water spout which was recessed in a 2 in. square opening in the wall. A sensitive acceleration trans-
ducer was built into the floor of the chamber for measuring motor activity in terms of dynamic energy output. This information was amplified, integrated and transformed into a numerical score which was recorded on an electromechanical counter (Mundl, 1966). A photocell beam passing 0.5 in. in front of the spout was used to measure approaches to the spout. Each time S came within 0.5 in. of the spout with his nose, the beam was broken and the event was electronically recorded as an approach response on an electromechanical counter. The metal water spout could be electrified by E so that each time the S touched the spout he would complete a circuit between the spout and the grid floor and receive a 0.1 milliampere shock on the mouth. This event was also recorded on an electromechanical counter. Since the opening in which the spout was recessed was just large enough for the S to get his head through, he was prevented from accidentally breaking the photocell beam by backing into it and also prevented from "testing" the spout with his paw to determine whether it was electrified or not.

Procedure

When the Ss arrived from the breeding farm, they were placed in individual cages to which graduated drinking tubes had been attached. Their 24-hour water intakes were measured for 5 days and will be referred to as "preoperative water
intakes." On day 6 surgery was performed as previously described. During the first 7 days of the postoperative recovery period, 24-hour water intakes were measured to determine whether the lesions had affected water intakes. On the 10th postoperative day the Ss were placed on a 23-hour water deprivation schedule and were maintained on that schedule for the next 9 days. For the first three days of this period each S was placed in the chamber for 20 min. and allowed free access to the water spout. During the subsequent 6 days of the experiment, each S was allowed to drink freely from the spout for the first 5 min. or until he drank 2 cc. of water whereupon E electrified the spout for the remainder of the session. The number of approaches made, number of shocks received and the activity score were recorded for each session. After each session the S was returned to his home cage and 20 min. later given free access to water for another 20 min. The quantity of water each S drank during this period was measured and will be called "home cage water intake." After the last test session all Ss were put back on ad lib. water. Their 24-hour water intakes were measured for 9 days and will be referred to as "post-deprivation water intakes."

**Results**

**Measures of water intake**
There were no significant differences between any of the groups in the preoperative water intake measure ($F=2.00$, $df=3/33$). During the postoperative recovery period, the water intakes of the corticomedial group were significantly lower than those of the control group ($F=4.54$, $df=1/27$, $p<.05$) but the basolateral group did not differ from the controls ($F=0.10$, $df=1/24$). The surgical trauma, as indicated by the water intake data, appears to have been greater for the corticomedial group than for the basolateral group. Additional analyses revealed no significant differences between the groups in either the home cage water intakes ($F=0.20$, $df=3/45$) or in the post-deprivation water intake measure ($F=1.38$, $df=3/44$).

**Behavioral measures**

Figure 3, shows that the Ss with basolateral lesions received more mouth shocks than either the corticomedial Ss ($F=11.61$, $df=1/25$, $p<.01$) or the controls ($F=55.55$, $df=1/36$, $p<.001$). There was a much smaller but significant difference ($F=4.87$, $df=1/31$, $p<.05$) between the corticomedial group and the control group. Observation of the Ss by E during testing revealed that all groups responded to the shock in a similar manner by jumping back and occasionally vocalizing.

The basolateral group approached the spout more often
(Fig. 4) than either the corticomedial group (F=11.69, df=1/25, p<.01) or the control group (F=26.40, df=1/36, p<.001). The corticomedial group did not differ from the controls (F=0.95, df=1/31) on this measure.

Although there was no difference between the groups with lesions (F=0.01, df=1/25) in the activity measure (Fig. 5), both the basolateral group (F=11.90, df=1/36, p<.01) and the corticomedial group (F=10.10, df=1/31, p<.01) were more active than the control Ss.
Experiment II. Effects of Amygdaloid Lesions on DRL Performance

The previous experiment demonstrated that rats with basolateral amygdaloid lesions are severely impaired in learning to inhibit a previously acquired approach response to an electrified water spout. The following experiment was designed to investigate whether the deficit found in Experiment I was specific to fear motivated tasks. The behavioral task, differential reinforcement of low rates of response (DRL), was selected for two reasons. First, in order to perform well on this schedule, an animal must learn to withhold a bar-pressing response. Second, data are available for comparative purposes on the effects of septal (Ellen, Wilson & Powell, 1964) and hippocampal (Clark & Isaacson, 1965; Schmaltz & Isaacson, 1966) lesions on DRL performance.

Method

Subjects

The Ss were 45 experimentally naive male hooded rats. The groups consisted of 14 Ss with basolateral lesions, 11 Ss with corticomedial lesions, 10 sham-operated controls, and 10 normal controls.

Apparatus

The apparatus was a standard operant conditioning
chamber measuring 9.5 X 8.5 X 11 in. One wall of the chamber was constructed from clear Plexiglas to permit observation of the S during testing. The bar and pellet cup were 0.5 in. apart on the left wall of the chamber. A calibrated drinking tube was attached to the right wall of the chamber. The chamber was housed in a ventilated, sound-insulated enclosure. The ventilation system also provided a source of masking noise. A 12 volt lamp (bulb # 67) in the roof of the enclosure provided illumination. Reinforcement consisted of 45 mg. Noyes pellets. The programming equipment provided the following raw data on electromechanical counters: total bar-presses, number of reinforcements, and burst responses. Burst responses consisted of bar-presses which occurred within one second of each other. Each S's performance was also recorded on a Gerbrands cumulative recorder.

Procedure

Following the postoperative recovery period, the Ss were placed on a 23-hour food deprivation schedule for 5 days. After the Ss had adapted to this regimen, they were trained to bar-press on a continuous reinforcement schedule for 7 days. On the 8th day they were put on a DRL-20 sec. schedule of reinforcement, 45 min. per day for 20 consecutive days. On this reinforcement schedule, the S received a pellet only if he refrained from responding for at least 20 sec. Responses
which occurred during this 20 sec. delay period were not
rewarded, and they reset the timers back to the beginning of
the delay period. Thus the animal had to learn to respond
at a very low rate and space his responses at least 20 sec.
apart. After each daily test session, the S was returned
to his home cage and given 13-15 grams of standard laboratory
chow. The Ss usually ate this ration in 2 to 3 hours, and
thus can be considered to have been deprived for approximately
20 hours at the beginning of each daily test session.

Results

There were no differences between any of the groups
in rates of response during the pretraining period while the
Ss were on a continuous reinforcement schedule (F=0.60,
df=3/41).

The performance of the basolateral group on the DRL
schedule as measured by per cent reinforced or correctly
timed responses (Fig. 6) was clearly impaired when compared
with the performance of either the corticomedial group (F=13.37,
df=1/23, p<.01) or the control group (F=18.55, df=1/32, p<.001).
Although there appears to be a small difference between the
corticomedial group and the control group, particularly dur-
ing the last 10 days of testing, this difference is not sta-
tistically reliable (F=1.17, df=1/29).
Data from the burst response measure are only available for the basolateral and control groups. Figure 7, shows that the basolateral group made many more burst responses than the control group (F=10.79, df=1/25, p<.01). The analysis of variance revealed that the effect over days was significant (F=2.00, df=19/475, p<.01). The group X day interaction was also significant (F=3.72, df=19/475, p<.001) indicating opposite trends in this measure.

Figure 8, shows that this schedule of reinforcement generated gradually decreasing rates of response in both the corticomedial group and the control group. This trend over days was found to be highly significant (F=26.50, df=19/551, p<.001). After an initial drop the rate of response of the basolateral group leveled off (Fig. 8). Statistical analysis revealed that the response rates of both the basolateral group (F=27.42, df=1/32, p<.001) and the corticomedial group (F=11.30, df=1/29, p<.01) were significantly higher than that of the control group. The difference between the two groups with lesions was not significant (F=3.29, df=1/23).
Experiment III. Effects of Amygdaloid Lesions on Discrimination Learning and Reversal

The purpose of the following experiment was to determine the effects of amygdaloid lesions on the acquisition of a “go-no-go” visual discrimination and upon a reversal of this discrimination. If one of the effects of amygdaloid lesions is to produce perseveration of previously learned responses, it might be expected that such animals would have difficulty learning this problem.

Method

Subjects

The Ss were 51 experimentally naive male hooded rats. The groups consisted of 17 Ss with basolateral lesions, 13 Ss with corticomedial lesions, 9 sham-operated controls, and 12 normal controls.

Procedure

The Ss were given 6 days pretraining on a continuous reinforcement schedule in the same operant conditioning box used in Experiment II. During discrimination training, the house light was used to signal the “go” period in the first part of the experiment. When the S pressed the bar after the house light came on, a 45 mg. pellet was delivered and the
house light went off simultaneously, signalling the "no go" period, during which bar-presses were not reinforced. The light was programmed to come on according to a VI-15 sec. schedule. Responses which were made during the "no go" blackout period, with the exception of those which were made within 0.5 sec. after the pellet was delivered, were counted as errors.

Each period of illumination was counted as a trial. The Ss were given 5 warm-up trials and then 65 test trials daily. On each trial there was a 20 sec. limited hold, that is, if the animal did not respond within 20 sec. after the light came on, the light was automatically turned off and the trial was terminated. After 7 days training on this schedule, the reinforcement contingencies were reversed; that is, the S would not receive a pellet for a bar-press when the light went off and would not receive pellets for bar-presses while the light was on. All other procedures remained the same as in the first half of the experiment. The first part of the experiment will be referred to as "original learning" and the second part will be called "reversal learning."

Following each session the S was returned to his home cage and given supplementary food. The amount of food given each S in his home cage depended upon how much
food he had received during testing, and was adjusted to ensure that each S received 13-15 grams of food daily.

Results

Although there was a slight tendency for the basolateral group to make more errors (Fig. 9), that is, respond more during the "no-go" period, than either the corticomedial or the control groups, there were no statistically significant group differences in either the original learning (F=0.61, df=2/48) or in the reversal learning (F=0.73, df=2/48). Although, the analyses of variance revealed highly significant day effects in both original learning (F=40.04, df=6/12, p < .001) and the reversal learning (F=35.49, df=6/12, p < .001), there were no significant interaction effects in either the original learning (F=0.41, df=12/288) or in the reversal learning (F=1.29, df=12/288). An additional confirmatory nonparametric trend test (Ferguson, 1965) revealed a highly significant (p < .001) decreasing monotonic trend in the error scores of the basolateral group during reversal learning.
Experiment IV. Effects of Amygdaloid Lesions on Alternation Behavior

In the previous experiment no reliable indications of perseverative behavior were observed in Ss with amygdaloid lesions in a go-no-go visual discrimination and reversal problem. Since perseverative behavior was readily observable in tasks in which there were no visual cues (Experiments I & II) but not in a task in which behavior is guided by visual cues (Experiment III), it seemed useful to investigate this parameter further. The purpose of this experiment was to determine the effects of amygdaloid lesions on alternation behavior with and without a visual cue.

Method

Subjects

The Ss in Part A of this experiment (no visual cue) were 52 experimentally naive male hooded rats. The groups consisted of: 17 Ss with basolateral lesions, 13 Ss with corticomedial lesions, 9 sham-operated controls, and 13 normal controls. In Part B of the experiment (cued alternation) the Ss were 25 experimentally naive male hooded rats. The groups consisted of 12 Ss with basolateral lesions, 6 sham-operated controls and 7 normal controls. No Ss with corticomedial lesions were tested in Part B of the experi-
ment because no deficit was found in the corticomedial group in Part A.

Apparatus

The test chamber was the same operant conditioning box used in Experiments II & III with two modifications. A second bar was placed on the left side of the pellet cup. Both bars were similarly located an equal distance (0.5 in.) from the pellet cup. In Part B of the experiment, a small pilot light (Dialco socket # 81410-111, bulb # 1819) was placed 1.75 in. over each bar. The lights were programmed to indicate which bar would deliver the next pellet. There was no blackout period, that is, as soon as the S pressed the correct bar, the light immediately shifted from that bar to the other one.

Procedure

All Ss were first given 6 days pretraining during which bar-presses on either bar were rewarded on a continuous reinforcement schedule. In Part B of the experiment this pretraining was done with the cue lights off. On the 7th day the apparatus was programmed to reinforce simple left-right alternation between the two bars. The first reinforcement for each session was always delivered from the right bar. Responses made on the same bar after delivery of a pellet
were counted as errors. Each test session was 30 min. long. Following each session the Ss were returned to their home cages and were given supplementary food as described previously (Experiment III).

Results

In Part A of the experiment (Fig. 10, left side) the basolateral group made more perseverative errors, that is, continued to press the same bar after delivery of a pellet, than either the corticomedial group (F=4.71, df=1/28, p<.05) or the control group (F=10.37, df=1/37, p<.01). The performance of the corticomedial group did not differ from that of the control group (F=1.53, df=1/33).

In Part B of the experiment, when the cue was added to the task, the Ss with basolateral lesions learned to alternate between the two bars as quickly as the controls (Fig. 10, right side). There was no difference (F=1.72, df=1/23) between the two groups. Additional analysis revealed that the Ss with basolateral lesions in the cued condition made significantly fewer perseverative errors than the basolateral Ss in the noncued condition (F=16.46, df=1/27, p<.001).
Experiment V. Effects of Amygdaloid Lesions on Fixed Ratio and Runway Performance

Many investigators have reported changes in food intake following amygdaloid lesions, but there seems to be little agreement among these reports about the direction of the changes. Some studies report postoperative increases in food intake while others report changes in the opposite direction (see Goddard, 1964b). According to Schwartzbaum (1961) the hyperphagia which follows amygdalectomy does not seem to be the result of an increase in hunger drive, but rather a defect in some form of satiety mechanism. Amygdalectomized monkeys will become hyperphagic under ad lib. feeding conditions but they are less responsive than normals to changes in food deprivation or amount of reward (Schwartzbaum, 1960; 1961).

The purpose of the following experiment was to determine whether the DRL and alternation deficits (Experiments II & IVA) could be attributed to changes in food motivation.

Method

Subjects

The Ss in this experiment had previously been used in Experiment II. All Ss, with the exceptions mentioned
below, were tested for both fixed ratio performance and runway running speeds. In Part A of the experiment (fixed ratio performance) the groups consisted of: 13 Ss with basolateral lesions, 11 Ss with corticomedial lesions, 10 sham-operated controls, and 9 normal controls. In the interval between Part A and Part B of this experiment, one basolateral S, one corticomedial S and two normal controls became ill and for this reason were not used in Part B.

Apparatus

In Part A of the experiment the Ss were tested in the same operant conditioning chamber that was used in Experiment II. In Part B the Ss were tested in a straight runway measuring 42 X 5.5 X 14 in. with a start box measuring 8 X 5.5 X 14 in. The entire runway was painted flat black. At the end of the runway the S had to put his head through an opening measuring 2 X 5.5 in. to obtain two 45 mg. Noyes pellets. A photocell system connected to a Hewlett Packard Electronic Counter measured the time it took the S to run the length of the runway. This elapsed time score was converted into a running speed score by dividing the length of the runway by the running time.

Procedure

In Part A of the experiment the apparatus was pro-
grammed for the following fixed ratio schedules: days 1 and 2, 1 to 1; days 3 and 4, 10 to 1; days 5 and 6, 20 to 1; days 7 and 8, 30 to 1; days 9 and 10, 40 to 1; days 11 and 12, 50 to 1. On days 13 and 14 an extinction procedure was used and no bar-presses were reinforced. Each daily session was 30 min. long and the measure of performance was the total number of responses made in this period. Following each session the S was returned to his home cage and given supplementary food as described previously (Experiment III).

Following fixed ratio testing all Ss were returned to an ad lib. feeding schedule for three weeks before they were tested in the runway. During runway testing the Ss were maintained on the same deprivation schedule as during fixed ratio testing.

On day 1 of runway testing each S was given 5 pre-training trials followed by 10 test trials. On days 2 and 3 each S was given 10 trials in the runway. On all test days the intertrial interval was 30 sec. during which the Ss were placed in a cardboard waiting box. After each S had been tested, the runway was washed with a damp sponge.

Results

Fixed ratio performance

Although there appears to be a trend toward a higher
rate of response in the basolateral group (Fig. 11) at the higher ratios, an analysis of variance indicated that there were no significant differences between the groups (F=2.56, df=3/39). There also were no differences between the groups during extinction (F=1.95, df=3/39).

Runway performance

The basolateral group had significantly slower running speeds (Fig. 12) than either the corticomedial group (F=6.71, df=1/20, p < .05) or the control group (F=15.95, df=1/27, p < .001). The difference between the corticomedial group and the controls was not significant (F=0.25, df=1/25).
Discussion

The present experiments have demonstrated that the behavior of rats with amygdaloid lesions is often similar to the behavior of rats with septal and hippocampal lesions; that is, these animals perseverate in emitting previously learned responses when these responses are no longer appropriate. The present results also indicate, however, that perseverative behavior is not observed in all situations in rats with amygdaloid lesions. Thus, any general statement about the behavior of these animals needs careful qualification with respect to the nature of the particular task.

In Experiment I a clear-cut passive avoidance deficit was observed in Ss with basolateral amygdaloid lesions and a marginally significant deficit was found in Ss with corticomedial amygdaloid lesions. All of the water intake measures that were recorded indicate that the deficit probably cannot be attributed to an increase in water intake caused by the lesions. Although changes in water intake following septal lesions have been reported (Harvey & Hunt, 1965), similar effects were not observed in Experiment I following amygdaloid lesions.

It also seems unlikely that the deficit can be attributed to an increase in general activity level. Although
both groups of animals with lesions were more active than controls (Fig. 5), only the basolateral group showed a striking passive avoidance deficit as measured by the large number of mouth shocks and approaches.

Similar passive avoidance deficits have been reported following septal lesions (Kaada et al., 1962; McCleary, 1961; Schwartzbaum & Spieth, 1964; Zucker & McCleary, 1964) and hippocampal lesions (Isaacson & Wickelgren, 1962; Kimble, 1963; Kimble et al., 1966; Kimura, 1958; Snyder & Isaacson, 1965; Teitelbaum & Milner, 1963). Passive avoidance deficits can also be produced by continuous low-level stimulation of the amygdala (Pellegrino, 1965), septal area (Kasper, 1964; Schwartzbaum & Donovick, 1965), or the hippocampus (Musty, personal communication), supporting Goddard's (1964a) suggestion that this type of stimulation produces a "functional lesion" of the area stimulated. These passive avoidance deficits have generally been interpreted in terms of a loss in response inhibition.

The data from Experiment I appear to conflict with Ursin's (1965) study on cats with respect to the particular amygdaloid region which is important in passive avoidance performance. Ursin found that lesions in the corticomedial region produced passive avoidance deficits, while lesions in the basolateral region did not. Almost directly opposite
results were found in Experiment I with rats. Gerbrandt (1964) has suggested that there might be a species difference in the functional organization of the amygdala. According to him, the corticomedial group in the cat is passive avoidance specific and in the rat the basolateral group is passive avoidance specific. In light of the fact that a small but significant deficit was observed in the corticomedial group in Experiment I and also that Horvath (1963) reported a small but significant deficit from basolateral lesions in cats, it seems more likely that this may be another example of an imperfect topographical organization of function within the amygdala (Gloor, 1960). Certainly, the data from the passive avoidance task do not constitute clear-cut support for a species difference, with complete reversal of function in the two amygdaloid regions, for the cat and the rat.

The deficit in DRL-20 acquisition (Experiment II) indicates that perseveration of previously learned responses following amygdaloid lesions can occur in tasks where no shock motivation is employed. As in Experiment I, basolateral lesions seem to be far more effective than corticomedial lesions in producing perseverative behavior. Indeed, the only suggestion of a deficit from the corticomedial group was in terms of a higher rate of response (Fig. 8).
when compared with controls. However, when the performance of the corticomedial group is compared with the controls in terms of correctly timed responses (Fig. 6), it becomes apparent that there is no deficit.

A similar deficit in DRL-20 performance in the rat has been reported with septal lesions (Ellen, Wilson & Powell, 1964) and continuous low level septal stimulation (Kaplan, 1965). The authors of both experiments attribute the DRL deficits to impairments in response inhibition rather than impaired temporal discrimination. In support of this interpretation, Ellen et al. (1964) point out that the interresponse time (IRT) distributions of Ss with septal lesions show that the DRL impairment occurred only when intervals of 10 sec. or less had elapsed since the preceding response. If, however, the animal withheld his bar-press response beyond 10 sec., there was evidence in the IRT distributions of good temporal discrimination.

The effects of hippocampal lesions on this task (DRL) have been investigated in greater detail than have the effects of amygdaloid lesions (Experiment II). In both cases, the locus as well as the size of the lesion appears to be important. When small anterior dorsal hippocampal lesions are made, no DRL deficit is observed (Ellen, Wilson & Powell, 1964), but when larger lesions are placed in the more poster-
ior portions of the hippocampus, DRL performance is severely disrupted (Clark & Isaacson, 1965; Schmaltz & Isaacson, 1966). In addition to lesion size and locus, the amount of pretraining on a continuous reinforcement schedule is an important variable. When animals with hippocampal damage were given extensive pretraining they were severely impaired in their ability to withhold the bar-press response on the DRL schedule, but hippocampal Ss who received no pretraining on a continuous reinforcement schedule did not differ from controls in their DRL performance (Schmaltz & Isaacson, 1966). Unfortunately, similar data on the effects of pretraining are not available for animals with septal and amygdaloid lesions, to the best of my knowledge. Research on this important aspect of the problem is needed.

The failure to find any clear-cut evidence of perseverative behavior in the successive go-no-go visual discrimination and reversal tasks (Experiment III) was somewhat unexpected and certainly would not have been predicted from the results of Experiments I and II. The passive avoidance and DRL deficits confirmed Goddard's (1964b) hypothesis that animals with amygdaloid damage would be unable to suppress established responses; but the results of Experiment III are inconsistent with this hypothesis.

These results (Experiment III) also appear to be in-
consistent with the findings of Schwartzbaum et al. (1964b) and Thompson and Schwartzbaum (1964). Using an auditory frequency discrimination task, these authors reported an increase in responding under nonreinforced conditions (during $S^A$) following amygdaloid lesions in rats. In Experiment III no such increase in responding during the no-go period ($S^A$) was found. In addition, Thompson and Schwartzbaum (1964) reported that lesions placed in the corticomedial region of the amygdala produced greater increases in responding during $S^A$ than basolateral lesions. The greater effect from corticomedial lesions is contrary to what would be predicted from the results of Experiments I and II. However, it should be kept in mind that these inconsistencies could easily be attributed to the different experimental procedures employed. Specifically, Schwartzbaum et al. (1964b) and Thompson & Schwartzbaum (1964) investigated postoperative retention of an auditory frequency discrimination whereas Experiment III was designed to investigate postoperative acquisition of a visual discrimination.

In contrast to the lack of deficit in reversal learning following amygdaloid lesions (Experiment III), reversal deficits have been found in several tasks following continuous septal stimulation (Kasper, 1965; Olds & Olds, 1961),
septal lesions (Zucker & McCleary, 1964) and hippocampal lesions (Kimble & Kimble, 1965; Lash, 1964).

The negative findings of Experiment III did suggest, however, that the presence of a specific visual cue might be important in determining whether perseverative behavior would be observed in animals with amygdaloid lesions. In other words, these findings suggested that animals with amygdaloid lesions might be capable of withholding a previously established response provided there was a visual cue available to guide their behavior. This possibility was tested in Experiment IV, and it is clear from the results (see Fig. 10) that the Ss with basolateral lesions had no difficulty learning to alternate from left to right when given a visual cue to guide their behavior. In the absence of this cue, however, the Ss with basolateral lesions were slower than the controls in learning to alternate. There is a suggestion here that rats with basolateral lesions are unable to suppress or inhibit responses in situations where they must use the information provided by some form of internal cue (Experiments I, II & IVA), but are capable of inhibiting responses in situations where their behavior is guided by a visual cue (Experiments III & IVB).

Some additional support for this modification of Goddard's hypothesis comes from a report by Pribram et al.
septal lesions (Zucker & McCleary, 1964) and hippocampal lesions (Kimble & Kimble, 1965; Lash, 1964).

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Some additional support for this modification of Goddard's hypothesis comes from a report by Pribram et al.
(1966) who have studied the effects of limbic lesions which included the amygdala on classical and go-no-go alternation.

In the classical alternation situation, there are two identical covered food wells facing the monkey and he is required to alternate from the left food well to the right one on successive trials. There are no additional cues available in this task. Although the responses are different, the general nature of this task is similar to the uncued alternation task used in Part A of Experiment IV. Like the rats with basolateral lesions in Experiment IVA, the monkeys with limbic lesions were severely impaired on this task.

In the go-no-go alternation task, one centrally placed food well faces the monkey and is baited on alternate trials. The S must learn to withhold the response for at least 5 sec. on the unbaited trials. There are no additional cues available, other than the information the S received on the previous trial, to indicate whether the food well is baited or not. Thus this problem is an uncued analogue of the go-no-go task used in Experiment III. On the basis of the results of Experiment III and the modification of Goddard's hypothesis suggested above, one would predict that monkeys with limbic lesions would learn this task slower than controls. Indeed, Pribram et al. (1966) report just such a deficit. It is difficult however to draw any definite
conclusions from this experiment about the relative contribution of amygdaloid damage to these deficits because the lesions included orbitofrontal, insular and temporal cortex as well as the amygdala. It would be important to know whether the deficits could be replicated with damage restricted solely to the amygdala.

Since at the present time there are insufficient data available to assess adequately the modification of the response inhibition hypothesis suggested above, I have mentioned it here very tentatively and only for its possible heuristic value. I do not, of course, presume to account for all the various behavioral effects that occur following damage to the amygdala, by means of this hypothesis.

Another modification of the response inhibition hypothesis has been suggested by Schwartzbaum and his co-workers (Kellicutt & Schwartzbaum, 1963; Schwartzbaum et al., 1964b). These authors suggest that the failure of nonreinforced events to exert adequate control over the behavior of animals with amygdaloid lesions, as seen in the persistence of response tendencies that are no longer adaptive, may reflect a defect in the development of emotional reactions to such events (Amsel, 1958; 1962). The numerous studies (see Goddard, 1964b) which have demonstrated decreases in emotional reactivity following amygdaloid lesions lend some support
to this approach. This hypothesis, however, would predict a deficit in the cued go-no-go discrimination and reversal tasks (Experiment III); but, as mentioned above, amygdaloid lesions did not produce any deficits in these tasks.

Several other possible alternative explanations for the poor performance of Ss with basolateral amygdaloid damage in the passive avoidance, DRL, and uncued alternation tasks should be considered. First, it may appear that the deficits could be due to the lesions causing an increase in motivation for food or water, thus leading to the observed increase in approach responses in these tasks. This seems unlikely for several reasons. First, as mentioned above, there was no indication in any of the water intake measures in Experiment I of an increase in 24-hour water consumption as has been observed following septal lesions (Harvey & Hunt, 1965). Second, there were no differences between the groups with lesions and the control group in bar-pressing rate on either a continuous reinforcement schedule (Experiment II) or on a steadily increasing fixed ratio schedule with food as reward (Experiment V). Third, the results of the runway test (Experiment V) were directly opposite to what would be predicted from such an interpretation. The Ss with basolateral lesions actually ran slower than the other groups in this test. Finally,
Schwartzbaum's (1960; 1961) findings that amygdalectomized monkeys are less sensitive than controls to changes in food deprivation or amount of reward would also be inconsistent with an "increased drive" interpretation.

A second possible explanation (also unlikely) is that the behavioral deficits observed in Ss with basolateral lesions could be attributed to a general impairment in learning ability. The fact that the rats with basolateral lesions were capable of learning the go-no-go discrimination and reversal problem (Experiment III), and the cued alternation task (Experiment IVB) would be inconsistent with such an interpretation. Also inconsistent with this interpretation is the failure to find any deficit in the acquisition of a delayed response and several types of discrimination problems in monkeys with amygdaloid lesions (Mahut & Cordeau, 1963; Orbach et al., 1960; Schwartzbaum, 1965; Schwartzbaum & Pribram, 1960).

In conclusion, the experiments which make up this study were undertaken with the intention of investigating the effects of amygdaloid lesions on response inhibition. Although several of the present experiments support the response inhibition hypothesis (Goddard, 1964b), others clearly contradict it. These contradictory findings led to the modified hypothesis that animals with amygdaloid lesions are
deficient in inhibiting responses only when the animal must depend upon the information provided by internal cues, but are not deficient when there is a visual cue to guide their behavior.

At a more theoretical level, Gloor (1960) has proposed that the amygdala (and possibly other limbic structures) may be concerned with the reinforcement of behavioral patterns by modulating the hypothalamic integration of basic somatomotor and autonomic functions. Specifically, the basic defect produced by amygdaloid lesions might consist of a "disturbance in those motivational mechanisms which normally allow the selection of behavior appropriate to a given situation" (Gloor, 1960). Since the passive avoidance, DRL, and alternation deficits that were observed in rats with amygdaloid lesions are clear evidence of inappropriate behavior in these situations, the present results are generally in accord with Gloor's hypothesis. However, the results of the cued alternation and go-no-go experiments indicate that rats with amygdaloid lesions are capable of making the appropriate behavioral adjustments in some situations. These latter results suggest that any attempt to ascribe a unitary function to a structure as complex as the amygdala is likely to be an oversimplification. They also suggest that future research in this area should be concern-
ed with the stimulus aspects as well as the response aspects of a behavioral situation.
Summary

Rats with bilateral lesions of the basolateral region of the amygdala were impaired in passive avoidance, DRL performance, and spatial alternation without a cue, but learned a visually cued spatial alternation task and a go-no-go visual discrimination and reversal problem as readily as controls. With the one exception of a small deficit in passive avoidance, rats with lesions in the corticomedial region of the amygdala were not impaired in any of these tasks. Control experiments indicate that the deficits produced by basolateral lesions can not readily be attributed to an increase in motivation for food or water. The results of these experiments suggest that rats with basolateral amygdaloid lesions are unable to inhibit established responses when the animal must depend upon the information provided by some form of internal cue, but not when there is a visual cue available to guide their behavior.
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Fig. 1. Reconstructions of typical bilateral basolateral and corticomedial lesions on sections redrawn from the deGroot (1959) stereotaxic atlas. Numbers in center of figure refer to anterior-posterior coordinates in the deGroot atlas.
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Fig. 2. Apparatus used to test passive avoidance behavior in Experiment I. See text for detailed description.
Fig. 3. Mean number of mouth shocks received by experimental and control Ss during successive test sessions.

(ABL = Ss with basolateral lesions, n=16; CMA = Ss with corticomedial lesions, n=11; CONTROL = normal and sham-operated Ss, n=22).
Fig. 4. Mean number of approach responses to the spout made by experimental and control Ss during successive test sessions. (ABL = Ss with basolateral lesions, n=16; CMA = Ss with corticomedia l lesions, n=11; CONTROL = normal and sham-operated Ss, n=22).
Fig. 5. Activity scores of experimental and control Ss during successive test sessions. (ABL = Ss with basolateral lesions, n=16; CMA = Ss with corticomedial lesions, n=11; CONTROL = normal and sham-operated Ss, n=22).
Fig. 6. The ratio (X 100) of the number of reinforcements received to the number of bar-presses for experimental and control Ss on the DRL-20 schedule. (ABL = Ss with basolateral lesions; CMA = Ss with corticomediai lesions; CON = normal and sham-operated Ss).
Fig. 7. Mean number of bar-presses with an interresponse time of 1 sec. or less. (ABL = Ss with basolateral lesions; CON = normal and sham-operated Ss).
Fig. 8. Mean number of bar-press responses made by experimental and control Ss on the DRL-20 schedule. (ABL = Ss with basolateral lesions; CMA = Ss with corticomedial lesions; CON = normal and sham-operated Ss).
Fig. 9. Learning curves for go-no-go visual discrimination and reversal tasks. (ABL = Ss with basolateral lesions; CMA = Ss with corticomedial lesions; CON = normal and sham-operated Ss).
Fig. 10. Learning curves for cued and noncued spatial alternation tasks. (ABL = Ss with basolateral lesions; CMA = Ss with corticomedial lesions; CON = normal and sham-operated Ss).
Fig. 11. Fixed ratio and extinction performance of experimental and control Ss. (ABL = Ss with basolateral lesions; CMA = Ss with corticomedial lesions; CON = normal and sham-operated Ss).
Fig. 12. Runway running speeds of experimental and control Ss. (ABL = Ss with basolateral lesions; CMA = Ss with corticomedial lesions; CON = normal and sham-operated Ss).