Chapter 15

The Effects of Thiazesim, LSD-25, and Bilateral Lesions of the Amygdalae on the Release of a Suppressed Response

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INTRODUCTION

A variety of experimental conditions can cause an animal to suppress or "control" a specific somatic response for varying time periods. The Sidman avoidance phenomenon [28], behavior on drl schedules [13], the conditioned-suppression phenomenon [12], and passive-avoidance behavior [26] are examples of temporal control of behavior. The control or suppression of specific somatic responses has been related to a number of variables including intensity of electric shock [18], temporal discriminations [3,], hippocampal EEG changes [9], septal lesions [17], intracranial self-stimulation vs. food reward [8], and subcallosal and cingulate gyrus lesions [23].

The hypothesis tested in the two current experiments is that the amygdalae are involved in the release of a suppressed response on a schedule with a positive reward (food) and response-contingent shock. It was predicted that random pairing of shock with bar pulling for food would inhibit the act of bar pulling; if the functioning of the amygdalae were then suppressed, the bar-pulling response would be disinhibited. These experiments are part of a larger study of "impulse control," which has as one general goal the search for CNS correlates of the control and release of specific somatic responses.

There have been many recent speculations about the CNS mechanisms related to "stop" and "go" phases of behavior. Miller [25] theorized "that there are one or more 'go' or 'activating' mechanisms in the brain which act to intensify ongoing responses to cues and the traces of immediately preceding activities, producing a stronger intensification the more strongly the 'go mechanism' is activated." Also, on a general level, Gerbrandt, in a summary of neural systems of response control and release [14], suggested that "discrete brain systems, mutually inhibiting in their effects, function on the one hand to release highly stabilized responses and on the other hand to allow increases in the elicitation of a response by control over competing responses of higher stability."

Several authors [20, 22, 24] have specifically suggested that the amygdalae may be part of a brain system similar to that described by

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Gerbrandt. Goddard [16] has hypothesized that "the amygdala seems essential for the establishment of avoidance and the modification of approach behavior." Douglas and Pribram [11] postulated that the amygdala "heightens awareness of experiences as a function of previous reinforcement." Pribam [27] more explicitly implicated the amygdala's role in orienting behavior when he observed that "the amygdala is necessary to a focusing function which allows registration of the events oriented to." Barratt [5, 6] has suggested that the amygdalae are related to intraindividual variability of both ANS and somatic responses and are necessary for maintaining suppression or "control" of a specific somatic response; thus, suppressing the functioning of the amygdalae would result in the disinhibition of a "controlled" response.

The experiments reported herein relate amygdalae functioning to the release of a suppressed bar-pulling response in a complex operant schedule designed to allow observation of: (1) the learning of the bar-pulling response; (2) the suppression of the bar-pulling response; and (3) the effects on the bar-pulling response caused by interference with the activity of the amygdalae.

METHODS

Behavioral Procedures

Experiment 1. Twelve naive squirrel monkeys were tested on a series of schedules (Table I) that started with continuous reinforcement for food (crf) and was followed by three fixed-ratio (FR) schedules, a tandem schedule (crf for 5 min alternated with 5 min of FR-6), and a conflict schedule. Part A of the conflict schedule was a 5-min period of random shocks (2.5 mA/sec) randomly paired 50% of

Table I. Sequence and Duration of Sessions

Experiment 1 (12 monkeys)		Experiment 2 (8 monkeys)	
Schedule	Daily sessions	Schedule	Criterion (no. of pulls or no. of sessions)
crf	14	crf	100/4 days
FR-2	14	FR-3	300/4 days
FR-4	14	FR-6	450/4 days
FR-6	14	Tandem	7 days
Tandem (crf vs. FR-6)	28	Conflict Parts A and B same as experiment 1	7 days without bar pulling before experimental test procedures introduced
Conflict Part A (crf + 50% random shock Part B (FR-6)	28 before experimental test procedures introduced		

the time with bar pulls on crf. Part B of the conflict schedules was a 5-min period of FR-6. The monkeys were tested for a fixed number of daily sessions on each schedule (Table I) before the experimental test procedures were introduced. It was predicted that the monkeys would bar pull only on part B of the conflict schedule.

Animals were tested at between 75 and 85% of their normal weight. They were tested in a specially designed chair [9] and were rewarded with a small, banana-flavored pellet. They were placed in the test cubicle at the same time each day, and the tests were run for 40 min; later experimental test sessions were longer when testing the effects of some drugs.

During the crf portion of the tandem schedule and also during part A of the conflict schedule, a light on the left side of the monkey chair was lighted; during the FR-6 portions of these schedules, a light on the right corner was lighted.

At the end of each daily session, the animals were returned to their home cages and given enough monkey chow and fruit to keep them at 75 to 85% of their normal weight.

The monkeys had to pull a 6-in. bar through a ½-in. arc at its tip to receive a reward. If the bar was pulled more often than once per second, it did not activate the reward mechanism. There was a 15-sec period between the different parts of the tandem and conflict schedules during which bar pulling did not result in rewards or shocks.

Experiment 2. This experiment was identical to the first experiment with the following exceptions (Table I): eight animals were run on crf and then proceeded to FR-3 and FR-6 before starting the tandem schedule; animals were changed to new schedules on the basis of behavioral criteria (Table I), e.g., the schedule was changed from crf to FR-3 after at least 100 bar pulls during four consecutive sessions; and the experimental test procedures were introduced during the conflict schedule after at least seven consecutive days during which the animals made no effective bar pulls.

Experimental Test Procedures

Intracranial electrical stimulation (ICS), drugs, and lesions of the amygdalae were used in exploring for the release of the suppressed bar-pulling response. Because of the lack of consistent behavior patterns during the first experiment, it was not possible to have experimental control sessions for all experimental procedures without confounding the results. The second experiment was designed to partially clarify the results obtained in the first, more exploratory, experiment.

ICS. ICS was kept below current levels which produced any observable overt responses to the onset of the current (range 0.4—0.9 mA). ICS parameters were 100 cps and 1 msec duration with a Grass Model S-4 stimulator and stimulus-isolation unit. Voltage and current were monitored during ICS. The amygdala and mesencephalic reticular formation (MRF) were never stimulated on the same day in any one monkey. ICS was used during part A of the conflict schedule.

Drugs. Thiazesim* (SQ 10496, 10 mg/kg), normal saline, and LSD-25 (25 μg/kg and 100 μg/kg) were used in both experiments; d-amphetamine (1–5 mg/kg) was used in the second experiment only. It was predicted that thiazesim would have the same effect on the release of a suppressed bar-pulling response as bilateral lesions of the amygdalae. This prediction was based on the demonstrated suppression of amygdaloid activity accompanying use of the drug [19].

Lesioning. Bilateral lesions were made with a Grass LM-3 radio-frequency stimulator in the basolateral portion of the amygdalae of six monkeys in the first experiment and four monkeys in the second experiment. The size of the lesion varied from 1.5 to 2.0 mm (Fig. 1).

Operative Procedures

Bilateral EEG placements in all animals included: (1) bipolar, side-by-side electrodes in the basolateral amygdalae and MRF and (2) stainless-steel screw electrodes in the skull over the frontal and occipital areas. An indifferent electrode was placed far anterior in dental cement above the frontal sinuses. All subcortical electrode placements and lesions were verified by a modification of the Wade Marshall procedure for frozen sections (Fig. 1). Serial sections 25 μ thick were stained with Thionine blue.

*Thiazesim was formerly known as thiazenone.

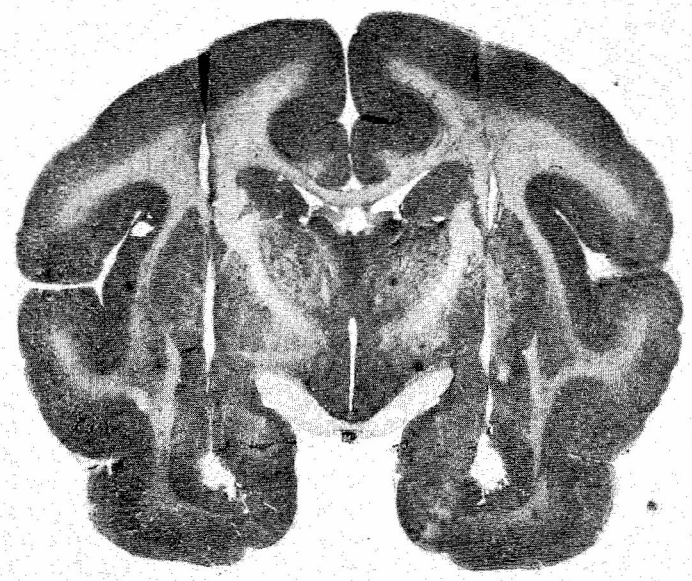

Fig. 1. Bilateral lesions of amygdalae in monkey RT-16.

Table II. Behavior Patterns on Conflict Schedule: Experiment 1

Group 1	(6 monkeys)	Pulled on part B only at high rate and did not pull on part A (suppressed bar pulling on part A).
Group 2	(2 monkeys)	Pulled on parts A and B for five sessions, then ceased pulling on either part. Developed "psychotic-like" behavior both in restraining chair and in home cage.
Group 3	(4 monkeys)	Pulled about equally on both parts A and B. Rate of pulling was lower than on tandem schedule but pattern of response was similar. Did not develop "psychotic-like" behavior.

Recording Apparatus

EEG's were recorded graphically (Grass Model III EEG) and on magnetic tape (seven-channel Ampex SP 300). A code-and-search system was interfaced with an analog-to-digital converter which was interfaced with an IBM 1410 computer. This permitted automatic digitizing of the EEG records for subsequent computer analyses. Behavioral responses, rewards, and shocks were recorded on a Davis cumulative recorder.

RESULTS

Experiment 1

Behavior Patterns. There were no significant differences in the patterns of bar pulling among the 12 monkeys through the tandem schedule although all animals did not perform at the same rate. Their rates of bar pulling steadily increased from the crf schedule through the FR-6 schedule and then declined during the tandem schedule, probably because they received more pellets for fewer pulls on the crf portion of the tandem schedule.

The monkeys did not behave as predicted on the conflict schedule. Instead of bar pulling only on part B of the conflict schedule, three patterns of responses were observed (Table II): six monkeys (Group 1) did as predicted and pulled only on part B; two monkeys (Group 2) continued pulling on both parts A and B for five sessions, and then did not pull on either part; four monkeys (Group 3) pulled about equally on parts A and B. The Group-2 monkeys developed "psychotic-like" behavior. There were no obvious differences in the behavior pattern of the monkeys through the tandem schedule that related to the three different patterns of behavior on the conflict schedule.

The psychotic behavior of the Group-2 monkeys was rather dramatic. They would not eat spontaneously and had to be force-fed; they would sit

Table III. Summary of Behavior Changes with Experimental Test Procedures in Group-1 Monkeys: Experiment 1

Experimental test	Behavioral changes
ICS-unilateral amygdala	Pulled on both parts A and B until received shock on A; continued pulling on B but avoided pulling on A
ICS-unilateral MRF	More active, continued pressing on B, didn't press on A
LSD-25 (100 μg/kg)	After 4 min, alternated between alert and drowsy behavior for 2 hr; shock on part A had no deterrent effect; displayed much catatonic-like behavior
SQ 10,496 (10 mg/kg) 40 min before being placed in cubicle	Pulled on both parts A and B
SQ 10,496 (10 mg/kg) 30 min before LSD-25 (25 μg/kg)	Blocked LSD-25 behavior effects—pulled on both parts A and B
Bilateral lesions of amygdalae	Pulled indiscriminately on both parts A and B

Table IV: Summary of Behavior Changes with Experimental Test Procedures in Group-2 Monkeys: Experiment 1

Experimental test	Behavioral changes
ICS-unilateral amygdala	No effect
ICS-unilateral MRF	No effect
LSD-25 (100 μg/kg)	Changed from "psychotic-like" behavior to more "normal" behavior; pulled on both A and B for 3 days after receiving LSD-25; cage behavior changed to more normal behavior for 3 days
Bilateral lesions of amygdalae	Pulled indiscriminately on both A and B

for hours in sawdust in which they had urinated and defecated. They were easy to handle, in contrast to the normally very active behavior of these monkeys.

Experimental Test Procedures. The responses of the three groups to different experimental test procedures are summarized in Tables III, IV, and V. Among the more relevant results were the following: (1) Among the Group-1 monkeys, thiazesim had the same effect as bilateral lesions of the amygdalae with regard to release of the suppressed bar-pulling response. (2) LSD-25 altered the behavior

of Group-1 and Group-3 monkeys only on the day that they received it; they became "drowsy" within 4 min after the LSD-25 injection (100 μg/kg) and alternated between alert and "drowsy" states for about 2 hr. Some catatonic-like behavior was displayed under LSD-25—the monkeys would extend their arms halfway to the manipulandum and visually fixate on some part of the cubicle for long periods. (3) LSD-25 changed the behavior of the monkeys in Group 2 for about 3 days. After receiving LSD-25, they began to pull the bar in the test cubicle, and their behavior in their home cages was more normal—they would eat spontaneously, were more difficult to handle, and did not crouch in the corner of their cage nor sit in urine or feces. Three days after receiving LSD-25, these monkeys began to revert to their previous psychotic-like behavior. Within 2 weeks, their behavior was as abnormal as it had been before LSD-25. One monkey again given LSD-25 passed through the same behavioral changes as before. (4) Thiazesim given approximately 40 min before LSD-25 blocked or attenuated the LSD-25 effect, depending on the dosage level.

Experiment 2

Behavior Patterns. As predicted, changing the sequence of schedules and the criteria for changing from one schedule to the next resulted in a consistent behavioral response on the conflict schedule (Fig. 2). After 5 days on the conflict schedule, all monkeys ceased bar pulling completely. They would occasionally make abortive pulls, but would not pull the bar to get a reward.

Experimental Test Procedures. No experimental test procedures were tried with these monkeys until they had not pulled the bar for at least 7 consecutive days. The results obtained with the various experimental test procedures are summarized in Table VI. In every instance, thiazesim and bilateral lesions of the amygdalae were both effective in releasing the suppressed bar-pulling response. An acute

Table V. Summary of Behavioral Changes with Experimental Test Procedures in Group-3 Monkeys: Experiment 1

Experimental test	Behavioral changes
ICS-unilateral amygdala	No effect
ICS-unilateral MRF	No effect
LSD-25 (100 μg/kg)	Alternated between alert and drowsy periods for 2 hr; continued pulling on A and B
SQ 10,496 (10 mg/kg)	Continued pulling on A and B
SQ 10,496 (10 mg/kg) 30 min before LSD-25 (25 μg/kg)	Behavioral effects of LSD-25 blocked – animals pulled primarily on B, but next day pulled on both A and B

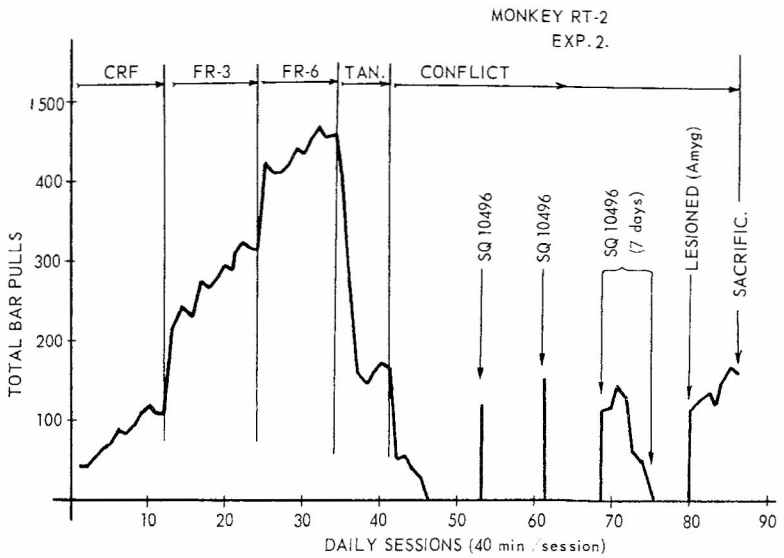

Fig. 2. Total bar pulls on various schedules for monkey RT-2, Experiment 2. The pattern of behavior before the use of experimental test procedures is typical of all monkeys in Experiment 2.

Table VI. Summary of Behavioral Changes Resulting from Experimental Test Procedures: Experiment 2

Experimental test	Behavioral changes
LSD-25	No release of suppressed bar pulling
SQ 10,496	Release of suppressed bar-pulling response on day administered; acute tolerance developed after 4–7 days
ICS-amygdala	No consistent release of suppressed bar-pulling response
ICS-MRF	No release of suppressed bar-pulling response; animals became more active
d-Amphetamine	No release of suppressed bar-pulling response; animals became more active
Normal saline	No change in behavior
Lesions of amygdalae	Evidently permanent release of suppressed bar-pulling response

tolerance to thiazesim developed within 4–7 days if the drug was administered on consecutive days. However, if it was administered every other day, no acute tolerance appeared. Amygdala ICS did not result in a consistent release of the suppressed bar-pulling response. d-Amphetamine, LSD-25, MRF ICS, free food rewards, and normal saline were also ineffective in releasing the suppressed response.

As in the first experiment, when thiazesim was administered approximately 40 min before LSD-25, the behavioral effects of LSD-25 were blocked or attenuated. Also, thiazesim, administered after LSD-25 had produced behavioral changes, appeared to potentiate the behavioral changes produced by LSD-25.

EEG Results. The frontal and amygdala leads were characterized by low-voltage, fast activity in the range 25–40 cps within 40–60 min after the administration of thiazesim. This appearance of low-voltage, fast activity coincided with an increase in activity level and the release of the suppressed bar-pulling response. Approximately 80 min after thiazesim administration, the 25- to 40-cps activity was more predominant in the frontal leads than in the amygdala leads.

Five minutes after the administration of LSD-25 (100 μg/kg), the EEG was characterized by bursts of 14- to 18-cps activity which appeared in the frontal and amygdala leads. Approximately 20 min after the administration of LSD-25, the 14- to 18-cps activity accounted for 60% of the total energy in the frontal EEG; this lasted for about 2 hr. The administration of thiazesim (10 mg/kg) approximately 40 min before LSD-25 (100 μg/kg) blocked or attenuated the usual EEG changes seen with LSD-25 alone.

Although the above analyses were made with the use of a computer, the EEG changes following the administration of thiazesim and LSD-25 were clearly evident in the ink tracings.

DISCUSSION

The results substantiate the hypothesis that the amygdalae are involved in the release of a specific suppressed response. The following two conditions consistently caused release of the suppressed bar-pulling response: (1) the administration of thiazesim and (2) bilateral lesions of the amygdalae. Although the animals became more active after administration of thiazesim, release of the suppressed response cannot be attributed to an increase in activation level alone; MRF ICS and the administration of d-amphetamine both resulted in increased levels of behavioral activity but did not result in the release of the suppressed bar-pulling response.

Unilateral amygdala ICS did not result in a persistent release of the suppressed response, but in a few animals it did produce a momentary release of the response. Two facts must be considered here: first, the stimulation was unilateral and bilateral stimulation might possibly have had a more persistent effect; second, the ICS stimulation was at a very low level—lower, for example, than the level at which Ursin and Kaada

[29] reported "attention" responses in cats after stimulation of the same area of the amygdala.

The differences in response patterns between the first and second experiment indicate the importance of the schedule used in testing hypotheses involving independent variables like drugs. One goal of Experiment 2 was to get a more consistent pattern of behavior on the conflict schedule. It was hypothesized that the three patterns of behavior observed in the first experiment were related to incidental learning or a behavioral set which resulted from keeping the animals on all of the schedules too long. As predicted, telescoping the overall length of the experiment resulted in a consistent response to the conflict schedule in the second experiment. It is also important to note that some monkeys continued pulling the bar after they had been shocked. Appel [4] had observed that in squirrel monkeys electric shock could suppress a response for as long as 50 days. The behavioral results, especially in the first experiment, emphasize the importance of carefully considering the type of schedule and the learning history of an animal when generalizing about the aversive effects of electric shock.

Why did suppressing the activity of the amygdalae lead to the release of the suppressed bar-pulling response? Since the behavioral effects of thiazesim were reversible, it would appear that biochemical changes temporarily bring about the same functional neurophysiological results as lesions of the amygdalae. The 25- to 40-cps EEG activity started to increase in the amygdala about 20 min after the administration of thiazesim. Within 40 to 60 min, this fast activity was evident in the frontal leads and after 80 min was more prominent in the frontal than in the amygdala leads. No changes were observed in the occipital or MRF leads at this time. The EEG changes in the frontal leads coincided with an increase in the activity levels of the monkeys and also with release of the bar-pulling response. This suggests that amygdala–frontal-lobe interaction was associated with the release of the suppressed response. This suggestion is even more plausible if one considers that thiazesim blocks the behavioral effects of LSD-25 if it is administered before the LSD-25 but appears to potentiate the effect of LSD-25 if administered while the animal is already under the influence of LSD. LSD-25 also produced EEG changes which were first observed in the amygdala and frontal leads, the main leads in which thiazesim produced changes.

LSD has been shown to increase the serotonin level in the brain. It has also been shown that the amygdalae and contiguous limbic-system structures have a relatively high serotonin concentration. The EEG changes in the frontal–amygdala leads after administration of LSD-25 could relate to biochemical changes involving a competition between serotonin and LSD-25, which has a structural similarity to serotonin. It is difficult to relate thiazesim to this biochemical picture, however, since it is not structurally similar to either LSD-25 or serotonin.

From a neuroanatomical viewpoint, it is possible that the amygdala is acting as a "thermostat" that has both downstream and upstream effects. Upstream it is involved in the activity of the frontal lobes, possibly by a change in frequency modulation of impulses from temporal

cortex to frontal cortex; this could produce a change in behavioral inhibition if one accepts the popular conception that certain frontal areas are related to impulse control and that the temporal lobes are more involved with storage of information codes. The change in frequency modulation in neuronal pools through which impulses pass from the temporal areas to the frontal areas could markedly change the ability of an animal to respond to signals that ordinarily would result in the suppression of a response. This hypothetical change in frequency modulation and signal transmission could be a function of impedance changes in the cells [2]. If one completely destroyed (e.g., by lesions) a neuronal pool that was acting as a frequency-modulation interface, this would interfere with the transmission between temporal lobe and frontal lobe. However, one could also achieve the same result biochemically, by changing the impedance characteristics of the neuronal pool.

The downstream influence of the amygdalae could be made manifest at varous basal-ganglia nuclei as Gloor [15] suggests. This downstream influence could result in a general change in motor output. Barratt [5, 6] has hypothesized that certain limbic-system structures (especially the amygdalae) are related to intraindividual variability of behavior. Lesions of the amygdalae rendered the performance of animals more variable. It is possible that this increase in intraindividual variability of somatic responses is related to a modulating influence of the amygdalae on basal-ganglia nuclei.

SUMMARY

The purpose of these experiments was to test the hypothesis that the amygdalae are involved in the release of a suppressed response on a schedule involving a positive reward (food) and response-contingent shock. The following two experimental test conditions consistently caused release of a suppressed bar-pulling response: (1) the administration of thiazesim and (2) bilateral lesions of the amygdalae. Possible mechanisms underlying these changes are discussed.

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