The effects of amygdaloid stimulation on passive avoidance[1]

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Abstract

Noncontingent, continuous stimulation of the amygdala in the rat, particularly the basolateral area, impairs the acquisition of a passive avoidance response. Experimental Ss (N = 23) approached and received more mouth shocks from an electrified water spout than control Ss (N = 23).

Problem

Several investigations suggest that the amygdala plays a significant role in behavioral inhibition. Bilateral lesions of the amygdala have been shown to impair inhibitory conditioned responses (Brutkowski et al, 1960) and the acquisition of a conditioned emotional response (CER) (Kellicutt & Schwartzbaum, 1963). When tested in a bar-pressing discrimination situation, rats with amygdaloid lesions typically persisted in responses that were no longer adaptive; that is, they increased responding under nonreinforced conditions (Schwartzbaum et al, 1964).

Electrical stimulation of the amygdala has been shown to inhibit spontaneous food intake (Fonberg & Delgado, 1961), and attack behavior elicited by hypothalamic stimulation (Egger & Flynn, 1962). In addition, continuous stimulation of the amygdala impairs the acquisition of the CER (Goddard, 1964).

The purpose of this study was to determine the effects of noncontingent, continuous, low-level amygdaloid stimulation on the acquisition of a passive avoidance response.

Method

The Ss were 46 experimentally naive, male hooded rats, weighing about 300 gm. Group E consisted of basolateral (N = 15) and corticomedial (N = 8) placements. Group C consisted of stimulated controls (N = 7); fully operated controls (N = 8); and normal controls (N = 8).

The Ss were tested in an open top, wooden box with a grid floor measuring 13 x 9 x 16 in. The front wall of the chamber was clear plexiglas, permitting observation during testing. A stainless steel water spout projected from the middle of one wall, 2 in from the grid floor. The mouth shock, applied between the water spout and the grid, was supplied by a transformer adjusted to 40-v rms.

Brain stimulation, from a Grass S4 Stimulator, consisted of biphasic square waves at 100 cps., 0.5 msec. pulse duration. The current was kept constant at 20 μa for all stimulated animals. Electrodes consisted of twisted strands of formvar coated stainless steel wire (0.01 in) soldered to No. 27-9 Amphenol plugs.

All surgical implantations were unilateral and were performed under Nembutal anesthesia. Coordinates for electrode placements were : AP:0.0—1.0 mm posterior to bregma; L:5.0 mm; H:8.0—9.0 mm from the skull. All operated Ss were given a seven day postoperative recovery period, during which both they and the normal Ss were handled daily.

During the testing period all Ss were maintained on 23 hr. water deprivation. The experiment consisted of seven daily 20 min. sessions. During the first session the water spout was not electrified and the Ss were permitted to drink freely for the entire 20 min. During each of the six test sessions all Ss were allowed to drink freely for the first 5 min., and then the water spout was electrified for the remaining 15 min. of the session. The number of mouth shocks each S received, the number of approach-withdrawal responses, and the latency of the first daily contact with the spout were recorded for each session.

After the last test session, the implanted Ss were perfused and their brains removed and fixed in 10% formalin. Frozen sections of the electrode track were stained with luxol fast blue and neutral red (see Fig. 2).

Results

Figure 1 shows that the experimental animals (basolateral and corticomedial placements) received more mouth shocks than control Ss ($F = 51.06$; df 1/264; $p < .001$). A further analysis revealed that those animals with basolateral placements received more mouth shocks than those with corticomedial placements ($t = 2.52$; $df = 136$; $p < .02$).

The experimental animals also approached the spout more often than the control Ss ($F = 11.58$; df 1/264; $p < .001$). Animals with basolateral placements approached the spout more often than the corticomedial group ($p < .01$, Mann-Whitney U-test).

The Ss in the control group were slower than the experimental Ss in making their first daily contact with the spout ($F = 4.91$; df 1/264; $p < .05$), and frequently did not even approach the spout during the 20 min. test session.

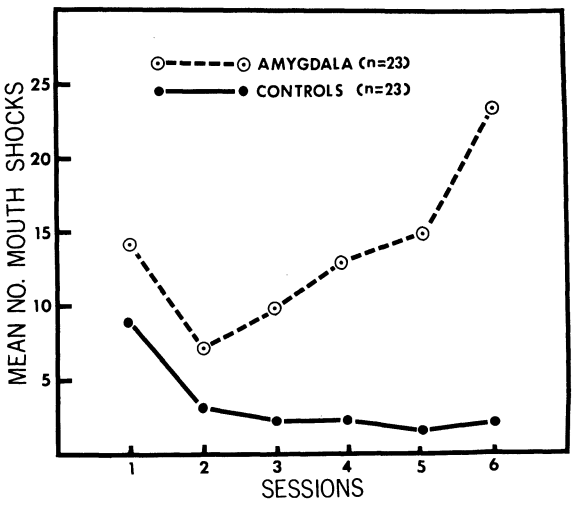

Fig. 1. Mean number of mouth shocks taken during daily sessions.

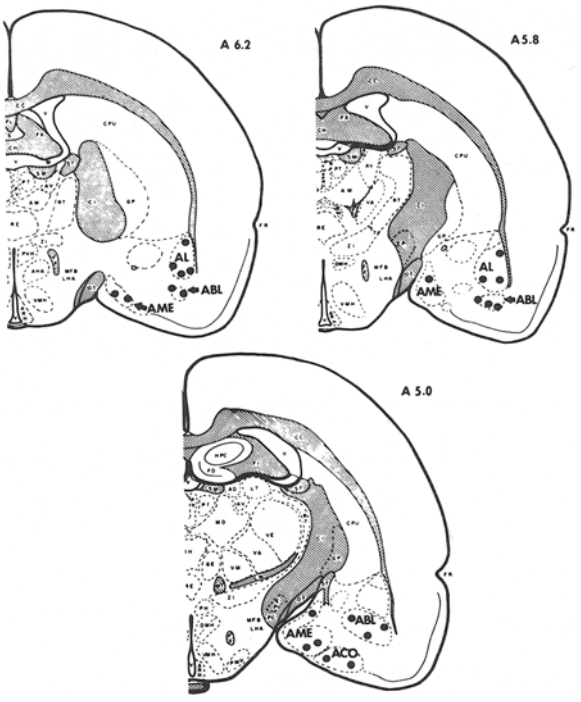

Fig. 2. The distribution of Group E electrode placements after deGroot (1959). Abbreviations: AL, lateral amygdala; ABL, basolateral amygdala; ACO, cortical amygdala; AME, medial amygdala.

A separate control experiment was designed to test whether the passive avoidance deficit could be attributed to increased thirst caused by the amygdaloid stimulation. Six animals with amygdaloid placements were tested for 10 days with stimulation on alternate days. No difference in water intake during the test session was found between days on which the animals were stimulated and those on which no stimulation was administered.

Although the amygdala is sensitive to seizure activity, behavioral seizures were never observed at the current intensity (20μ a) used.

Discussion

The results of this experiment indicate that noncontingent, continuous stimulation of the amygdala, particularly the basolateral area, impairs the acquisition of a passive avoidance response. It is probable that the stimulation produces its behavioral effect by disrupting the normal activity of the amygdala. This disruption is reflected in the persistence of response tendencies that are no longer adaptive.

Several investigators have suggested that deficits in the acquisition of a passive avoidance response caused by septal lesions (McCleary, 1961; Kaada et al, 1962) and by septal stimulation (Kasper, 1964) are due to the interruption of a circuit mediating response inhibition. The anatomical connections that exist between the septal area and the amygdala (Gloor, 1960) and the results of this experiment suggest that the amygdala may also be part of a circuit mediating response inhibition.

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Note

1. This research was supported in part by grants from the U.S. Public Health Service, and from the National and Medical Research Councils, and the Defence Research Board of Canada. Portions of the work presented are included in a thesis submitted to the Graduate Faculty of McGill University in partial fulfillment of the requirements for the M.A. degree in psychology.