Retrograde amnesia produced by localized electrical stimulation of brain through chronically implanted electrodes¹

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Retrograde amnesia produced by electrical stimulation of brain through chronically implanted electrodes in either the anterior or posterior regions of the cerebrum was compared in rats. Postlearning brain stimulation through either site produced retrograde amnesia, but there was a tendency for stimulation through anteriorly placed electrodes not to induce full tonic-clonic seizures. Differences between these findings and those in cats should not be attributed to species differences.

Although retrograde amnesia (RA) induced by electroconvulsive shock has received considerable attention in recent psychological literature, few studies have attempted to identify areas of brain critical for this phenomenon. Generally, RA is produced by an intense electric shock delivered through alligator clips attached to the ears. This technique has two distinct disadvantages. The shock must be painful (aversive) because the current passes through the skin and dura to reach the brain, and the electric current probably affects much or all of the brain. In the present study low intensity electroshocks were delivered through chronically implanted electrodes that extended through the dura into the neocortex in an attempt to reduce the aversiveness of the shock and to restrict the amount of brain activated. This study compares the effectiveness of brain shock to antero-lateral neocortex (presumably motor cortex) and to postero-lateral neocortex which is spatially close to the underlying hippocampus. Evidence supports the notion that the hippocampus plays an essential role in memory (Hostetter, 1968).

METHOD

The Ss were 16 male hooded rats that weighed between 190 and 304 g at operation. The behavioral apparatus was a 12 x 8 x 8 in. Skinner box designed to deliver water rewards on a continuous reinforcement schedule and to record the number of bar presses. Its floor was constructed from stainless-steel rods through which footshocks were delivered. Latency to the first bar press in each session was taken from the time the rat was placed in the box and was measured with a stop watch.

Brain shocks were delivered through thin wires, insulated except for about 0.5 mm at the tips, and attached to a miniature socket (Plastic Products) that was fixed to the skull with screws and Cranioplastic cement. The electrodes were positioned either bilaterally about 1 to 2 mm from the midline in an anterior position (2 to 3 mm anterior to bregma) or 4 to 6 mm from the midline bilaterally in the posterior position (5 to 6 mm caudal to bregma). Twenty-five mA of 60-cycle current (850 V through 34-kilohm resistance) with a duration of 0.2 sec was used for brain shock. The rats were placed in a box with a foam rubber floor for the administration of brain shock to minimize injuries consequent to seizure.

The rats were trained to press the bar for water in sessions lasting 10 min daily. Thirty min after each session the rats were given access to water for 1 h in their home cages. Otherwise no water was available, but food was available ad lib in their home cages. When response rates and latencies to the first bar press in each session had stabilized, that is, did not vary by more than 20% over four consecutive days, the rats were divided into five groups that were matched in terms of rates of bar pressing. Three rats received footshock and brain shock through posteriorly located electrodes (Group P-FS); three rats got footshock plus brain shock through anteriorly located electrodes (Group A-FS); three rats received brain shock through anteriorly placed electrodes but no punishing footshock (Group A); three rats received only brain shock through posterior electrodes (Group P); and four rats got footshock only with no brain shock (Group FS). One rat from Groups A, P, and FS, respectively, was removed before the study

was completed: two rats died in surgery and one was removed because of labyrinthitis. Data are reported for 13 rats.

After recovery from surgery (four weeks) retraining was initiated and continued until performance had again stabilized to the same criterion that was held for preoperative training. The next day the experimental treatments were administered. Immediately following the first bar press, rats in Groups P-FS and A-FS received a footshock of 1 mA (60-cycle ac) for 2 sec followed within 10 sec by a brain shock through the implanted electrodes. Rats in Groups P and A received brain shock within 10 sec after the first bar press, but no footshock; and rats in Group FS received footshock, but no brain shock following their first bar press. After the various treatments, all rats were returned to their home cages. The following day all rats were returned to the Skinner box and tested for retention of the electric shocks.

RESULTS

Latencies to the first bar press (seconds) and characterization of the seizure pattern for each rat are presented in Table 1. Scores for the pretreatment condition are the means of the latencies to first response of the last four postoperative retraining sessions. The posttreatment scores are latencies for the single test day. Response rates reflected the pattern of latency scores, that is, when latencies increased, response rates went down, so these data are not presented.

Footshock was an effective aversive stimulus because all rats that received only this treatment (Group FS) showed greatly increased posttreatment latencies (see Table 1). Brain shock alone, through either anterior or posterior electrodes, did not have an aversive effect (Groups A and P). At least one brain shock did not increase first response latencies at all. All but two rats that received brain shock showed a typical tonic-clonic convulsion followed by coma. The two rats that showed only partial convulsions (both were in Group A-FS) reared up on their back legs and exhibited clonic movements of their front legs, but they did not fall down nor go into coma. The probability that both rats would by chance fall into the group that received anterior electroshock rather than posterior electroshock was not significant by Fisher's exact test (Siegel, 1956). However, it seems likely that the threshold for full convulsions would be different for different electrode placements.

A rat was considered to demonstrate amnesia for footshock if its latency to the first response on the test day did not differ markedly from its mean latency over the last four pretreatment sessions. The differences between pretreatment latencies and posttreatment latencies for each rat (RA values) were evaluated by

Table 1
Latencies to First Bar Press (sec) Before and After Experimental Treatments and Characterization of Seizure Patterns

Group	Rat No.	Pretreatment	Posttreatment	Seizure
P-FS	1	8	9	Full
	9	8	8	Full
	16	10	10	Full
A-FS	4	9	8	Full
	7	9	9	Partial
	13	8	92	Partial
P	6	9	9	Full
	10	5	5	Full
A	2	10	11	Full
	3	10	10	Full
FS	8	9	517	None
	12	9	577	None
	14	8	572	None

means of Mann-Whitney U tests (Siegel, 1956). Groups P-FS and A-FS differed significantly from the footshock control group (Group FS) but did not differ significantly from the other control groups (Groups A and P). Thus RA was produced by brainshock delivered through both anterior and posterior electrodes.

DISCUSSION

Although RA was produced by brain shock delivered through anterior and posterior neocortical electrodes, the seizure patterns for these groups sometimes differed markedly. All rats in Group P-FS, Group P and Group A, and one rat in Group A-FS had full tonic-clonic seizures followed by coma. Two rats in Group A-FS did not exhibit full seizures and coma, but one of them (rat No. 7) clearly showed RA. The other rat (No. 13) that had a partial convulsion did show perhaps some memory for the punishing footshock. Its latency to first bar press increased from 8 to 92 sec. On the other hand, control rats (footshock only) increased their latencies from 8 or 9 sec to over 500 sec. Clearly, the results (Table 1) demonstrate that RA can occur from electrical stimulation of the brain without a full tonic-clonic convulsion.

Kesner & Doty (1968) delivered brain shock through stainless-steel screws anchored in the skulls of cats. Brain shock delivered bilaterally over ectosylvian areas (posterior neocortex) induced both full convulsions and RA, whereas brain shock to the postcruciate area (anterior neocortex) produced convulsions but

did not induce RA. Thus, in cat as well as rat, both RA and convulsion were induced by brain shock delivered through posteriorly positioned electrodes. In the rat effects of brain shock differed from those in cat when the shock was delivered to anterior neocortex. Electrical stimulation through the anterior placements (at the current strength used) did not consistently produce full tonic-clonic convulsions in the rat, but it did produce RA. However, the brains of cat and rat differ in size as well as degree of functional and topographic differentiations which might affect electrical thresholds for convulsions. Therefore, these results regarding difference between cat and rat in brain-shock induced RA do not necessarily indicate a species difference.

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NOTE

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