The effect of insular neocortical lesions on passive and active avoidance behavior in the rat¹

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Abstract

Rats with insular lesions were deficient in adopting a passive-avoidance response. However, Ss with insular lesions did not differ from operated and normal control Ss in the acquisition of an active-avoidance response.

Problem

Rats with hippocampal lesions do not readily inhibit a previously dominant response (Isaacson et al., 1962; Jarrard et al., 1964). This has implicated the hippocampus as a response inhibitory structure. Kaada (1960) has presented evidence showing that the stimulation of the orbito-insulo-temporal cortex exerts a strong inhibition on various somatomotor activities. He further suggests that hypothalamic nuclei which subserve limbic inhibition may also receive the inhibition from the orbito-insulo-temporal cortex. It therefore seems possible that animals with lesions in the insular neocortex may manifest the same deficit which hippocampectomized rats exhibit on a passive-avoidance task. The purpose of this study was to investigate the effect of ablations, restricted to the insular neocortex, on a passive-avoidance response. The study was also extended to investigate the effect of insular lesions on the acquisition of an active-avoidance response.

Method

Ss were 31 male Long-Evans rats, 96 days old at the beginning of the experiment. Ss were randomly assigned to three groups: a neocortical group (N=11), an operated control group (N=10), and a normal control group (N=10). Half of all Ss in each group were assigned to the passive-avoidance task; the other half were assigned to the active-avoidance task.

The passive-avoidance apparatus consisted of a runway with internal dimensions of 54 x 4 in. and 9 1/2 in high. A guillotine door was situated 11 in from the starting end and formed the start box. Another guillotine door separated the goal box from the runway. A food cup was situated in the far wall of the goal box. The runway had a wood floor except the goal-box floor which consisted of brass rods spaced 1/2 in apart.

The active-avoidance apparatus consisted of two identical boxes placed end to end. These were both 12 x 7 in, and 8 in high. Both boxes were mounted on a common track. Adjacent doors connected the two compartments. On a particular trial, the connecting doors were opened, and the S advanced into the forward compartment. The doors were subsequently closed and the fore and aft compartments were interchanged. The same procedure occurred on the next trial. The S always responded by entering into the forward compartment. In this sense, the response was unidirectional. The compartment floors consisted of brass rods spaced 1/2 in apart. A 7.5 w bulb was located in the ceiling of both compartments and a speaker was installed beyond the far wall.

Surgery was performed under nembutal anesthesia (40 mg/kg) and ablations were made while Ss were held in a stereotaxic instrument. Animals in the operated control group received the same surgical treatment minus the aspiration of cortical tissue. All Ss had re-

turned to their pre-operative weight levels during the 14-day post-operative period.

Passive-avoidance Ss were placed on a deprivation schedule and maintained at 80% body weight. For five days, all Ss received 10 daily acquisition trials in the runway. A trial consisted of placing S in the start box and opening the guillotine door. After S had entered the goal box and consumed the three 45-mg food pellets in the food cup, S was returned to the start box. Running time was recorded for each trial. On the fourth trial of the sixth day, a 3-sec. 0.5 ma shock was administered, via the grid floor, when the S touched the food cup. Subsequent trials, on that day, and the next two days were identical to those administered during the first five days.

Ss on the active-avoidance task received 10 acquisition trials daily in the one-way avoidance apparatus. S was placed in the rear compartment. A trial commenced with the simultaneous onset of tone and light in the rear compartment and opening of the doors connecting the two compartments. A timer was simultaneously activated. Continuous grid shock was presented 5 sec. after tone-light onset. Entry into the fore compartment by the S terminated the tone and stopped the timer. The forward compartment was then placed in the rear position and another trial was administered. Training continued for six days.

Results

Histological evaluations revealed that bilateral ablations in experimental Ss were located along the rhinal fissure and involved areas 13 and 20 according to Krieg's (1946) delineations. Typical lesions of the insular neocortex are illustrated in Fig. 1. Animals with insular neocortical lesions manifested a significant deficiency in acquiring a passive-avoidance response (Fig. 2). Analysis of running-time scores for days 6 through 8 revealed that the significant difference between groups ($F=93.58$, $df=2/13$, $p<.001$) was attributable to the performance of neocortical Ss ($p<.01$). Figure 3 summarizes the mean latency scores of Ss on the active-avoidance task. Analysis of these data did not show significant differences between groups.

Fig. 1. Reconstruction of lesions showing representative destruction of insular cortical tissue.

Fig. 2. Mean running time in seconds for groups in the passive-avoidance apparatus.

Fig. 3. Mean latency scores in seconds for groups in the one-way active-avoidance apparatus.

Discussion

The performance of the insular neocortical Ss on the passive-avoidance task resemble the performance of hippocampectomized Ss on the same task (Isaacson et al., 1962). Kaada et al. (1962) reported a passive-avoidance deficit in rats with extensive lesions of the insular and temporal cortex. The present findings are in accord with these results but point out that the passive-avoidance deficit occurs as a result of smaller lesions restricted to the insular cortex. Negative findings on the active-avoidance task may reflect the relative lack of difficulty inherent in this task. Practically all Ss reached a performance plateau early in training. The two tasks also differ in terms of salient motivational components. Whereas shock-avoidance is involved in both tasks, hunger adds to the motivational structure of the passive-avoidance situation. This may be particularly relevant since it is postulated that the cortical areas in question are concerned with food-seeking, licking, and chewing (MacLean, 1958). Further research should delineate the relative influence of

hunger and shock avoidance on the passive avoidance deficit manifested by animals with insular lesions.

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Note

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