EFFECT OF AMYGDALOID NUCLEAR LESIONS ON HYPOTHALAMIC LUTEINIZING HORMONE-RELEASED FACTOR IN THE MALE DEERMOUSE

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Recently, it was found that lesions placed in the basolateral amygdaloid nuclear complex result in the continuous release of luteinizing hormone (LH) from the hypophysial of the male and female deermouse (Eleftheriou & Zolovick, 1967; Eleftheriou, Zolovick & Norman, 1967). These findings, together with previous work on the amygdala, indicate the existence of a separate inhibitory centre, in both sexes, for the regulation of LH secretion.

The mechanism by which lesions in the amygdaloid complex affect changes in the pituitary and plasma levels of LH is not established. The present report deals with the effects of lesions placed in the basolateral amygdaloid complex on the content of hypothalamic luteinizing hormone-releasing factor (LH–RF) in the stalk-median eminence region of the male deermouse (Peromyscus maniculatus bairdii).

Adult male deermice, weighing 15–19 g., were lesioned bilaterally by thermocoagulation in the basolateral nuclear group of the amygdaloid complex according to the stereotaxic atlas for the species, as described previously (Eleftheriou & Zolovick, 1965; Eleftheriou et al. 1967).

Three groups of 15 animals each were killed 1, 2 and 3 weeks (45 animals/period) after the lesions had been produced. Three similar groups of non-lesioned controls were also killed. An additional six groups were sham-operated by inserting the needle into the amygdala without administering current. Three of these groups were killed 1 week and the remaining three 2 weeks after the operation. All experiments were conducted during December, January and February. The position of lesions was confirmed by histological examination. Light and diet were not altered throughout the experiment. The body weight of the operated animals did not show any significant changes.

After the animals had been killed the stalk-median eminence region and adjacent ventral hypothalamus were dissected out (3–10 mg.) and pooled for each group of 15 animals. The pooled tissue was extracted as described by McCann, Taleisnik & Friedman (1960) and McCann (1962). The extract from each group of 15 deermice was divided into three equal parts and assayed according to McCann et al. (1960). There were 3 hypothalamic extracts/treatment. Each of these hypothalamic extracts contained the pooled stalk-median eminences (SME) of 15 deermice. Since each pooling of 15 SME was assayed in 3 female rats, and there were three such pools of tissue/treatment, 9 assay rats were used at each treatment period for a total of 54 assay rats for the 6 treatments. Analysis of variance and 't' tests were used to test the significance of differences.
The lesions were cylindrical with an average height of about 9.8 mm. and a diameter of 0.7 mm. The limits of the lesions in the lateral plane extended from the middle of the basolateral amygdaloid nuclei to the middle of the lateral amygdaloid nuclei. In the anterior-posterior direction, the lesions extended from outside the first one-third of the dorsal and ventral hypothalamic nuclei posteriorly and adjacent to the level of the anterior limits of the posterior hypothalamic nuclei. No lesions exceeded these boundaries and no damage to hypothalamic nuclei was involved.

Table 1. Ovarian ascorbic acid depletion (mg./100 g.) induced by hypothalamic extracts from intact and sham-operated male deer mice, and by extracts from deer mice with lesions in the basolateral amygdaloid nucleus 1, 2, and 3 weeks after the operation

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Percentage ovarian ascorbic acid depletion (± S.D.)</th>
<th>P v. controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intact controls</td>
<td>17.9±3.8</td>
<td>NS</td>
</tr>
<tr>
<td>Sham-operated at 1 week</td>
<td>21.5±4.2</td>
<td>NS</td>
</tr>
<tr>
<td>Sham-operated at 2 weeks</td>
<td>18.6±3.1</td>
<td>NS</td>
</tr>
<tr>
<td>Lesioned, 1 week</td>
<td>1.9±0.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Lesioned, 2 weeks</td>
<td>3.1±2.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Lesioned, 3 weeks</td>
<td>7.3±1.2</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

NS = not significant.

Within 1 week after lesions had been placed in the basolateral amygdaloid complex, the hypothalamic content of LH-RF declined significantly (P < 0.001) as indicated by the very low ability to induce depletion of ovarian ascorbic acid (OAAD) of only 1.9% (Table 1). Although 3 weeks after the operation hypothalamic extracts induced OAAD of 7.3%, it was still significantly (P < 0.01) lower than OAAD induced by control hypothalamic extracts. The latter hypothalamic extracts which induced OAAD of 17.9–21.5% were obtained from intact and from sham-operated male deer mice.

Previous studies in the male deermouse (Eleftheriou et al. 1967) have indicated that after lesions in the basolateral amygdaloid nuclear groups, the plasma and pituitary levels of LH increase significantly. Thus, the fact that the LH–RF content of the hypothalamus of similarly treated deer mice declined significantly was not surprising.

The results of this study and previous work on the deermouse indicate that lesions placed in the basolateral amygdaloid complex result in a significant decrease in hypothalamic LH–RF and a significant increase in plasma LH.

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