A rhinencephalic feeding center in the cat

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Lesions of the rhinencephalon, primarily restricted to the pyriform lobe and amygdaloid complex, modifies the eating behavior of cats so that their food intakes and body weights are significantly increased. Neodecortication has no significant effect on either food intake or body weight whether done before or after the amygdalecotomy. The amygdaloid cats cat significantly more than their controls and gain weight at a significantly more rapid rate when on an ad libitum feeding regimen. It is concluded that the amygdaloid complex constitutes some type of feeding center in the cat probably exerting its effects on more primitive lower brain stem feeding mechanisms.

In previous papers (1, 2) we have reported hyperphagia to be the predominant finding following bilateral amygdalecotomy in the cat. The present report will present histological control on all the animals in the earlier papers and will give additional data concerning these changes in appetitive behavior in two new groups of animals.

Several recent studies (3–5) have noted hyperphagia to result from amygdalar lesions in both dogs and cats, thus confirming our original observations. In addition, Sawa et al. (6), Terzian and Ore (7) and Alajouanine et al. (8) have reported obesity in human cases following rhinencephalic removals. However, little or no quantitative data are given in these reports.

Our original experiment was primarily designed to test Bard and Mountcastle’s hypothesis (9) that the amygdaloid complex acts as a rage suppressor center in the cat. This paper presents the results of these tests in addition to giving quantitative data on food intake and body weight following amygdalecotomy.

METHODS

Seventeen adult male and female cats were used as experimental animals. After initial adjustment to caged laboratory conditions the behavior of each animal was regularly observed. The observations included reactivity to routine care, general cage activity, response to petting and handling, reactions to the presence of other animals, degree and type of aggressive behavior induced by application of moderately noxious stimuli, and various characteristics of the feeding patterns such as approach to edible and nonedible objects, rapidity of eating, amount eaten at one sitting, etc. Body weights and food intakes on a horse-meat diet in the ad libitum feeding situation were duly recorded on all animals at regular intervals.

The experiment was conducted in two parts. In the first part seven cats (group 1) were bilaterally amygdalecotomized in two stages by the open suction technique previously described by Schreiner and Kling (10). These animals were subsequently neodecorticated after their body weights had reached a stable plateau for several weeks. This period of stable body weight following the rising weight phase we refer to as the static phase of amygdalar obesity. In the second part of the experiment 10 cats were divided into two groups of 5 animals each (groups 2 and 3). Animals of group 2 were neodecorticated in a manner previously described by Bard and Mountcastle (9) and after being studied for several months were subsequently bilaterally amygdalecotomized. Animals of group 3 were bilaterally amygdalecotomized and then subsequently neodecorticated during the rising phase of body weight, i.e. during the dynamic phase of amygdalar obesity.

Following the postoperative studies the brains were perfused through the carotid artery with 10% formalin, and were removed, sectioned and stained with nigrosin. Histological reconstruction of the extent of the lesions was obtained in all of the operated animals.

RESULTS

The results of these experiments are shown in tables 1 and 2.

Group 1. This group of seven animals was shown in our original reports to gain weight at a significantly more rapid rate than their controls when on an ad libitum feeding regimen and that this gain was at least in part due to an increased food intake. From table 1 it can be seen that the difference in weight between this group and their controls was highly significant at the end of the dynamic phase of amygdalar obesity, whereas the preoperative weights of the group did not differ significantly from their controls. The total gain and percentage.
TABLE 1. Mean Body Weight (kg) of Amygdaloeutomized and Control Animals

<table>
<thead>
<tr>
<th>Group</th>
<th>No.</th>
<th>Preop. Wt. of Cats</th>
<th>Wt. End Dynamic Phase</th>
<th>Total Gain</th>
<th>Percentage Gain</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7</td>
<td>2.90±0.19</td>
<td>4.31±0.17</td>
<td>1.40±0.09</td>
<td>49±5.8</td>
</tr>
<tr>
<td>Controls*</td>
<td>7</td>
<td>3.96±0.13</td>
<td>4.44±0.13</td>
<td>0.18±0.09</td>
<td>5.5±0.7</td>
</tr>
<tr>
<td>P values, group 1 vs. controls</td>
<td>&gt; .1</td>
<td>&lt; .001</td>
<td>&lt; .001</td>
<td>&lt; .001</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>5</td>
<td>3.59±0.03</td>
<td>4.41±0.05</td>
<td>0.91±0.03</td>
<td>19±2±0.09</td>
</tr>
<tr>
<td>Controls†</td>
<td>18</td>
<td>4.00±0.12</td>
<td>4.10±0.12</td>
<td>0.10±0.01</td>
<td>3.5±0.2</td>
</tr>
<tr>
<td>P values, group 2 vs. controls</td>
<td>&gt; .1</td>
<td>&gt; .1</td>
<td>&lt; .001</td>
<td>&lt; .001</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td>3.75±0.08</td>
<td>4.72±0.14</td>
<td>0.97±0.09</td>
<td>26±0.2</td>
</tr>
<tr>
<td>Controls†</td>
<td>18</td>
<td>4.02±0.12</td>
<td>4.10±0.12</td>
<td>0.14±0.01</td>
<td>3.5±0.2</td>
</tr>
<tr>
<td>P values, group 3 vs. controls</td>
<td>&gt; .1</td>
<td>&gt; .02</td>
<td>&lt; .001</td>
<td>&lt; .001</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± S.E. *4 Normals, 3 decorcticates. †9 Normals, 2 sham operates, 2 fasted, 5 decorcticates.

Gain of this group as compared to their controls is also highly significant by group comparison testing. Food intakes during the dynamic and static phases of amygdalar obesity are shown in table 2. From this table it can be seen that the food intakes of group 1 as compared to their controls is highly significant by group comparison during the dynamic phase of amygdalar obesity, but that during the static phase of amygdalar obesity the difference between this group and their controls was not significant. These animals were studied for over a year and were subsequently neodecorticated while in the static phase of amygdalar obesity. This procedure during this particular phase was found to have no appreciable effect on the food intake or rate of weight gain in the operates as compared to their controls. Reconstructions of the lesions of the animals of group 1 are shown in figures 1 and 2A.

During the entire course of study of this group of animals, both pre- and postoperatively, there were no other significant changes in behavior. No rage behavior, 'placidity,' hypersexuality, or any form of oral behavior, was observed at any time although repeated tests were made for these behavioral characteristics at regular intervals in all animals of the experimental group.

Group 2. This group was studied in a manner similar to group 1, except that more extensive preoperative food intake and weight studies were made, these latter being duly recorded at regular intervals. Tables 1 and 2 show that in the preoperative period this group did not differ significantly from their controls as regards food intakes.

TABLE 2. Food Intake (gm/Day, Dry Weight, in Ad Libitum Feeding Situation) for Amygdaloeutomized and Control Animals

<table>
<thead>
<tr>
<th>Group</th>
<th>No.</th>
<th>Food Intake of Cats During Dynamic Phase</th>
<th>Food Intake of Cats During Static Phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7</td>
<td>149±6.0±1.08</td>
<td>76±4.0±1.20</td>
</tr>
<tr>
<td>Controls*</td>
<td>7</td>
<td>67±4.0±1.20</td>
<td>74±9.0±1.18</td>
</tr>
<tr>
<td>P values, group 1 vs. controls</td>
<td>&lt; .001</td>
<td>&gt; .1</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>5</td>
<td>69±4.0±1.40</td>
<td>64±4.0±1.20</td>
</tr>
<tr>
<td>Controls†</td>
<td>18</td>
<td>66±4.0±1.38</td>
<td>62±3.0±1.26</td>
</tr>
<tr>
<td>P values, group 2 vs. controls</td>
<td>&gt; .1</td>
<td>&lt; .001</td>
<td>&gt; .1</td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td>69±4.0±1.40</td>
<td>64±4.0±1.20</td>
</tr>
<tr>
<td>Controls†</td>
<td>18</td>
<td>62±3.0±1.26</td>
<td>61±3.0±1.26</td>
</tr>
<tr>
<td>P values, group 3 vs. controls</td>
<td>&gt; .1</td>
<td>&lt; .001</td>
<td>&gt; .1</td>
</tr>
</tbody>
</table>

Values are means ± S.E. *4 Normals, 3 decorcticates. †9 Normals, 2 sham operates, 2 fasted, 5 decorcticates.

FIG. 1. A: Extent of lesions at base of the brain in group 1. Solid line shows the minimal extent of brain tissue removed in all 7 animals while dotted line shows the maximal lesion for all the animals. Nomenclature from Papez (11). B, C: Frontal sections through the amygdaloid complex at Horsley-Clarke anterior plane 13.5. Solid line indicates the maximal extent of lesion through the different nuclei of the amygdaloid complex of group 1, while dotted line shows the minimal removal of amygdaloid tissue in this group.
Group 3. This group was studied in a manner similar to group 2. Tables 1 and 2 show that in the preoperative period this group did not differ significantly from their controls as regards food intakes and body weights. In this group amygdalectomy was done first and all animals proceeded to become hyperphagic, their food intakes becoming significantly different from their control group. Their rate of weight gain was also significantly different from their controls so that their total gain and percentage gain during the dynamic phase of amygdalar obesity were both significantly different from their controls. Subsequent neodecortication was done in the dynamic phase of hyperphagia in this group and was not found to alter the upward trend of the food intakes or body weights, these latter proceeding to stay above the control levels until the group finally entered the static phase of amygdalar obesity. During this period when the weights were in a stable elevated plateau their food intakes again became not significantly different from their control group. Lesions in this group are reconstructed in figures 2C and 4. As with the previous two groups, no other changes in behavior became apparent during the entire course of the observation period.

DISCUSSION

That the structures on the medio-basal aspect of the temporal lobe are in some way concerned with the organization of feeding behavior seems to be well documented. Various types of 'oral behavior' and alterations in the eating patterns have been prominently mentioned in the literature as resulting from bilateral damage to the deep temporal lobe structures. In 1888 Brown and Schäfer (12) described two rhesus monkeys which among other alterations in behavior, showed marked hyperphagia. In the first instance the lesion was a total temporal lobectomy while the second case was a bilateral removal of the superior temporal convolution with extensive deep vascular damage. Then in 1939 Klüver and Bucy (13) noted 'oral behavior' and altered eating habits as part of a syndrome following bilateral temporal lobectomy in the rhesus monkey. This behavior, although not described by the authors as hyperphagia, did consist of great alterations in the general feeding patterns. For example, monkeys which are normally frugivorous became rabid meat eaters for a limited period of time, a situation Klüver maintains is never seen in normal monkeys. In addition, the animals mouthed and sniffed indiscriminately all objects with which they came into contact, discarding the inedible objects and devouring the edible ones. No distinct quantitative measurements were made regarding food intake but Klüver (personal communication) has stated that he believes there was an increased food consumption in all of his bilateral temporal

mained stabilized in an elevated plateau despite normal food intake. The extent of the lesions of this group are shown in figures 2B and 3A, B. As with group 1, no other changes in behavior were observed during the entire period of observation.
monkeys. This was associated in at least some of the monkeys with a weight increase, although Klüver maintains that despite tremendous food consumption some animals remained lean for periods of up to 10 years. Although there were no systematic weight curves obtained in the animals, Bucy and Klüver in a later paper (14) report on one monkey that showed marked 'greediness' and a 'tremendous appetite' with an increased food consumption and marked obesity. The weight in this animal increased from 5.93 to 7.67 kg in a period of 2 years even though it was not on an ad libitum feeding program. Thus it appears that at least some of Klüver and Bucy's monkeys were hyperphagic and did show significant weight gains. Anatomical control has subsequently shown that all of these monkeys sustained bilateral removal of the amygdaloid complex.

In addition, several recent papers have made references to hyperphagic behavior after temporal lobe lesions, but little in the way of quantitative data is given. Green, Clemente and de Groot (3) observed hyperphagia in some of their cats with stereotaxically placed lesions in the amygdala, but made no special attempt to study this phenomena systematically. They noted that 14 cats out of 71 showed abnormally large increases in body weights in the 1st postoperative month. A control group of 26 randomly selected cats showed an average weight increase of about 11% in 40 days, while the weight gaining limits in the hyperphagic group was an increase in weight of over 30% in 40 days and just under 30% in 8 days postoperatively. Increases in weight of up to 50% were seen in the first postoperative month in some of the animals (15). Unfortunately, there was no exact control...
of the diet. They found that the most consistently injured region to be near the junction of the basal and lateral nuclei of the amygdala. These data do not agree with ours in that our animals did not begin to show hyperphagia until the 2nd postoperative month. Also, the rate of weight gain in our animals was not as great. However, out of 17 cats that were confirmed histologically to be bilaterally amygdalectomized all became hyperphagic, though in varying degrees. Since our lesions were made by the open suction method the entire anterior piriform lobe was removed together with large amounts of the different amygdaloid nuclei. Hence, we are not able to locate precisely the area of the amygdala responsible for the hyperphagia. In all our cases, however, the area of junction of the basal and lateral nuclei was totally removed along the overlying piriform cortex. We have not yet seen a case of hypersexuality in our animals, although seven were males and sustained extensive damage to the piriform cortex below the basal nuclei of the amygdaloid complex. This is in striking contrast to the findings of Green et al.

Wood (4) has reported a marked increase in food intake in four of nine cats with stereotaxic lesions of the central and medial nuclei of the amygdala. He noted that the daily food intake was seen to quadruple in some of the animals, but no other quantitative data are given. This study does not agree with our anatomical data since many of our cats showing marked hyperphagia sustained no damage to the medial or central nuclei of the amygdala.

In dogs, Fuller, Rosvold and Pribram (5) report that after open removal of the piriform-amygdala-hippocampal complex, there results a doubling of the daily food intake in the first 2 postoperative weeks. This was associated with a temporary increase in weight. However, food intakes fell to normal in the second postoperative month. This, of course, does not agree with the present findings.

It is noteworthy that some clinical cases seen to show that a definite hyperphagia may result from temporal lobectomy involving the amygdaloid complex. Sawa et al. (6), Terzian and Ore (7), and Alajouanine et al. (8) all have reported such cases. In the case of Terzian and Ore the patient was said to have an insatiable appetite and to eat as much as four normal persons. No detailed food intake or weight studies were made on any of these cases, however.

It is interesting that MacLean and Delgado (16) have suggested that the medio-basal portion of the temporal lobes is basically concerned with the organization of the oral activities of the animal as they pertain to feeding and to the vocalization, defense and attack involved in obtaining food. In a more recent report (17) MacLean notes that this region is primarily concerned with functions that insure self-preservation, these responses falling into two general groups. One category includes responses of an alimentary nature, such as licking, chewing, eating, etc. In the other category are the responses associated with the animal's search for food. That the area of the amygdala plays an integral part in the organization of eating activities of the animal seems definite. The mechanisms of how this control is exerted awaits further elaboration.

We conclude that this region in the cat is concerned with food intake and body weight and apparently acts normally as a satiety center in the terminology of Brobeck (18). Thus, removal of this area results in increased food intake and body weight. Our results do not in any way confirm the hypothesis presented by Bard and Mountcastle that the amygdala acts as a rage suppressor center since none of our animals showed the slightest lowering of rage thresholds in the face of noxious stimuli. Nor was there any indication of a 'taming' effect or 'placidity' following amygdalectomy. We maintain, however, that these latter changes are most difficult to assess in an animal such as the cat which tames under ordinary laboratory conditions.

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REFERENCES