THE NORMALIZING EFFECT OF LATERAL AMYGDALAR LESIONS UPON THE DORSOMEDIAL AMYGDALAR SYNDROME IN DOGS

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Abstract. Two types of bilateral amygdalar lesions were performed successively. The first operation, involving the dorsomedial part, produced aphagia and subsequent hypophagia, vomiting and apathy. Consequently the body weight was reduced. After a few weeks partial recovery occurred. The second operation, involving the lateral part of the amygdaloid complex, produced hyperphagia, increase of body weight and restoration of general arousal. These results indicate the existence of two antagonistic systems: excitatory in the dorsomedial amygdala and inhibitory in the lateral amygdala. Damage of the inhibitory system reverses almost completely the syndrome produced by an initial lesion of the excitatory system.

INTRODUCTION

We showed previously (6, 8, 9, 14, 17, 18) that in dogs lesions situated in the dorsomedial part of the amygdaloid complex produced aphagia with subsequent hypophagia and apathy. We suggested that this syndrome is caused by inhibitory influences prevailing over excitatory alimentary ones, rather than by the disruption of the neuronal substratum for alimentary mechanisms (11, 14).

One of the sources of this inhibition may be the lateral part of amygdala. It has been shown that stimulation of the lateral amygdaloid nucleus produced inhibition of food intake (4, 7, 15, 16, 22), while a lesion in the same region caused hyperphagia, increase of body weight and lively behavior (10, 13, 14, 19, 20, 23).

In view of these results it was important to study how lateral amygdalar lesions would affect the symptoms produced by dorsomedial lesions. If the syndrome of dorsomedial amygdalar aphagia is caused primarily by an impaired, inhibitory–excitatory balance between these amygdala complexes (i.e., the global prevailing of inhibitory influences upon the other
parts of the alimentary system), the effect of the dorsomedial lesion should be ameliorated by the lateral lesion. If, however, the neuronal substratum for the patterns of food intake are substantially impaired by the dorsomedial damage, the increase of excitation produced by removal of the source of inhibition in lateral amygdala would not reverse the symptoms.

METHOD

Experiments were performed on 12 male, mongrel dogs weighing 10–14 kg. The animals were individually housed in cages. Daily measurements of food intake were made twice a day at about 8 AM and 2:30 PM. At that time the dogs were allowed to eat and drink ad lib. The food consisted of cooked cereal mixed with broth and meat. In between these meals, no food or water was available. Before the operation the measurements were made daily for a period of 10–20 days. After the operation the food intake was measured for 10 days and then again for 10 days after about 1 month and once again before the second operation. In between these periods the dogs were not fed ad lib., but received once a day a standard portion of food weighing about 1.5 kg. As a rule the second operation was performed 6–9 weeks after the first (seven dogs). However, in order to exclude the coincidence of the recovery period and the effect of the second operation, in three dogs (AD9, AD11, AD12) the second operation was performed after only 3–4 weeks, and in two others (AD1, AD2) after 3 and 4-month periods, respectively. In the latter dogs, additional measurements of the food consumed ad lib. were made between the two operations. After the second operation the same procedure for measuring food intake was used as after the first operation, i.e., for 10 days starting with the first postoperative day, and then 1 month later. In two dogs it was also measured 2 months after the operation. Body weight was measured every 5th day.

Surgical procedures

Lesion of dorsomedial amygdala (DMA). Surgery was performed aseptically under Nembutal anesthesia (35 mg/kg). The dorsomedial amygdala region (DMA) was electrocoagulated by means of electrodes introduced stereotaxically. Electrodes consisted of stainless steel needles 0.5 mm in diameter insulated by enamel except for 0.5–1.0 mm at the tip. A direct anodal current of 4.0 ma and 2–6 v was applied for 1 or 2 min. An indifferent electrode was connected to the skin of the head. The stereotaxic coordinates used were based on the atlas of Lim et al. (21) with our own corrections for the dimensions of the dogs’ skulls. Two points were coagulated on each side 1.5–2 mm distant each of other in anterior–posterior plane.
**Lesion of lateral amygdala (LA).** The procedure was the same as that described above for DMA lesions. The electrodes were aimed at the lateral part of the amygdala (LA). Two points were coagulated on each side 2 mm distant each of other in sagittal plane, and each lesion was enlarged dorsally by moving the electrodes 2 mm vertically upwards. As a result eight points were coagulated, four of which, in most dogs, made one combined lesion on each side.

**Histology**

After the experiments were completed, the dogs were anesthetized and perfused by formalin. Their brains were embedded in paraffin and cut in frontal sections 20 μ thick. Every 10th section was stained alternately by Klüver's and Nissl's methods in order to determine the anatomical limits of the lesions.

**RESULTS**

**The effects of lesions of the dorsomedial amygdala (DMA)**

**Food intake.** After the operations all the dogs were aphagic (Table IB). They refused to take food, and resisted feeding (Fig. 5bc). They also did not drink water. The total aphagia lasted in various dogs from 1–16 days.

**Table I**

Food intake in amygdala dogs. Mean from 10 meals ad lib. feeding. A, before operation normal state; B, during 1–5 days after DMA operation; B1, during 6–10 days after DMA operation; C, during 5 days before LA operation; D, during 1–5 days after LA operation; D1, during 6–10 days after LA operation.

<table>
<thead>
<tr>
<th>Dog</th>
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<th>C</th>
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<th>D1</th>
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<td>260</td>
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<td>260</td>
<td>960</td>
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<td>AD6</td>
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<td>0</td>
<td>0</td>
<td>1900</td>
<td>2090</td>
<td>1950</td>
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<td>1750</td>
<td>2276</td>
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<td>0</td>
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<td>1652</td>
<td>1850</td>
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</tr>
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* Decrease of food intake in this dog was produced by mild signs of epileptic seizures and somnolence produced by luminal treatment.
During this period the dogs had to be fed either by stomach intubation, or in case of those dogs which were able to swallow the food, by putting the food deeply into their mouths. Comparison of 10 days food intake before and 10 days after DMA lesion showed an evident decrease for the whole group. This decrease was highly significant for the whole group and for each individual dog (p < 0.001, Student t-test).

Later, all the dogs started to eat spontaneously; but most of them were hypophagic, had to be baited in order to aproach the food and showed food preferences. Similar effects of lesions of the dorsomedial amygdala were described in our previous papers (6, 9, 18). These symptoms lasted for several weeks. Afterwards, in most of the dogs restoration of the alimentary behavior occurred and the food intake reached, or nearly reached, the preoperative level (Fig. 1A–C). Nevertheless, the dogs seemed to be uninterested in food in spite of the fact that they ate the normal amount of food if served. In five dogs (AD1, AD4, AD5, AD7, AD9) pronounced hypophagia persisted until the second operation (Table I). The most persistent hypophagia was observed in dog AD1. In this dog, after a few days of initial restoration of food intake, consumption decreased again; and during 3 months of observations, i.e., until the second operation, it oscillated between 0 and 400 g of daily intake (Fig. 7).

Vomiting. Vomiting was observed in six dogs (AD2–AD6, AD12). The dogs vomited during the period of aphagia when forced fed and also in the period of hypophagia when they consumed the food voluntarily. Vomiting usually occurred within 20–30 min after the meal. Sometimes the dogs vomited also in between meals. During the first few days after the operation the dogs vomited several times a day although they were fed only twice daily. Progressively vomiting became less and less frequent with time, did not follow each meal and stopped completely after a few (3–6) weeks. Two dogs continued vomiting until the second operation.

Body weight. Body weight consistently dropped after DMA damage. This decrease was highly significant in all dogs if we compare their weight the day before the operation and that on the 10th day after the operation (p < 0.001, Student t-test). On Table IIA and B we see that the decrease of body weight was consistent in all dogs. Most of the dogs which started to eat a normal amount of food, after a few weeks continued nevertheless, to be underweight until the second operation (Table IIC and Fig. 2ABC).

General behavior. All the dogs just after the surgery spent most of the time lying somnolently. Sometimes they got up and walked around in a stereotyped manner. After 3–7 days they did not sleep more than before the operation but were still apathetic and depressed. Placed in new surroundings, they did not explore them but stood in one position where they were placed (Fig. 5a). The dogs seemed not to recognize the experimenter,
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Table II

Body weight (kg) in amygdala dogs. A, normal state before DMA operation; B, 10th day after DMA operation; C, before LA operation; D, 10th day after LA operation; E, 2 months later.

<table>
<thead>
<tr>
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<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
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<td>12.500</td>
<td>11.000</td>
<td>12.200</td>
<td>13.400</td>
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<td>12.000</td>
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<td>10.800</td>
<td>11.200</td>
<td>12.100</td>
<td>12.700</td>
</tr>
<tr>
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<td>10.300</td>
<td>11.700</td>
<td>13.900</td>
<td>14.800</td>
</tr>
<tr>
<td>AD7</td>
<td>13.300</td>
<td>12.900</td>
<td>11.500</td>
<td>13.900</td>
<td>15.800</td>
</tr>
<tr>
<td>AD9</td>
<td>11.000</td>
<td>10.500</td>
<td>10.100</td>
<td>13.000</td>
<td>13.100</td>
</tr>
<tr>
<td>AD10</td>
<td>12.900</td>
<td>11.800</td>
<td>11.200</td>
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</tr>
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<td>AD11</td>
<td>13.500</td>
<td>11.800</td>
<td>12.200</td>
<td>12.300</td>
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</tr>
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<td>11.400</td>
<td>10.100</td>
<td>11.800</td>
<td>13.400</td>
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</tr>
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</table>

for they did not greet him by jumping up and licking as they used to do before the operation. They did not obey commands; if called they did not react at all or approached slowly, unwillingly. Only two dogs became lively and gay before the second operation.

In five dogs (AD2, AD4, AD5, AD8, AD10) the lesion of dorsomedial amygdala affected also their emotional behavior in such a way that they became more fearful, irritable and aggressive, in spite of being in general apathetic and indifferent. These dogs were in particular very sensitive to confinement in the harness and tried by all means to free themselves, whining and barking during these attempts. The vocalization of these dogs differed strikingly from that of normal dogs. The sounds were something in between barking, screaming and whining and reminded one of the sounds of frightened ducks. It was colloquially called by technicians “quacking”. This “quacking” was observed when the dog was fastened in the experimental chamber. In their home cage, most often late in the evening or at night, they started to whine. It was very prolonged, toneful, wolflike whining, called by technicians “singing”. Similar vocalization was observed when the lesions involved only the most dorsal part of amygdaloid complex (5).

Other changes. The general appearance of the animals changed. The dogs looked dirty, their coats were thin, dusty and sheenless. Several weeks after the operation the dogs looked thin, not only because they lost weight, but also because their hair fell out in great amounts and was thinner and shorter than before the operation. Some dogs had bold or half-bold island on the trunk. The skin was thin and flexible, partly as
a result of loss of underlying adipose tissue and partly caused by some changes within it. Some of the dogs, although more inert and unreactive than before the operation, were nevertheless hypersensitive to touching of the skin. In general the animals gave the impression of sadness and depression. Their eyes were dull; their ears drooped; their tails were curled under their bodies; and their legs were slightly bent.

Effects of subsequent damage to the lateral amygdala (LA)

Food intake. On the next day after the LA operation, all dogs showed an increased interest in food. Except for dog AD9, which was not fully awakened from anesthesia the day following the operation, and dog AD5, which was treated to prevent seizures, the dogs started at once to eat voraciously. Dogs AD6, AD7, AD8 and AD10, ate more than 2 kg on the day after the operation. Dog AD1, which for 2 months before the second operation did not eat more than 200–400 g, consumed 1300 g on the 1st postoperative day. In the other dogs, the increase in food intake appeared a few days later.

If we compare the means for the 5-day period before and 5-day period following LA operation, there is an evident increase in food intake. Only in two dogs (AD5, AD8) did it decrease instead (Table ID). This was caused by the fact that dog AD5 was somnolent or asleep for 4 days following the operation, as he was treated with luminal to prevent seizures which appeared on the 1st postoperative day. In dog AD8 total recovery from the effect of the first (DMA) operation occurred before the second operation, which consequently produced no further increase in food intake. Also in dog AD6 there was no evident increase in food intake. Very marked effect of the LA operation was on these dogs which did not recover completely after the DMA operation (AD1, AD2, AD4, AD7, AD9, AD12),

The peak of hyperphagia occurred usually a few days after the operation, when the effect of surgery itself passed. Therefore, if we compare the mean values for 5 days preoperatively with the mean for 5 days taken from the period of 6–10 days after the operation (Table ICD), the increase of food intake is more evident for all dogs than that observed during the first 5 days directly after the operation (Table ICD). The increase of food intake in the period between the 6th and the 10th day after the operation was for the whole group, highly statistically significant in comparison with the level before LA operation (p < 0.001, Student t-test). This increase, however, differed in individual dogs. For example dogs AD1, AD4, AD7, and AD9 ate 2–4 times more than before the LA operation; while dogs AD6 and AD8, although they consumed the food voraciously, ate about the same portion as before the operation. In the remaining dogs the increase of food intake was about 25–70% of the
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preoperative level. The comparison of the normal state in unoperated dogs and the period between 6–10 days after LA damage shows that seven dogs (AD3, AD4, AD7-AD11) ate even more than they did in the normal state before both operations (Table I). Afterwards the food intake in some dogs continually decreased; while in the others it remained at a constant level after an initial decrease, which took place about 10–14 days postoperatively. Daily food intake showed fluctuations caused by the fact that overeating produced sometimes gastrointestinal disturbances, which as a consequence limited food intake during the next days. Evident differences between the preoperative and postoperative state appear if we compare the maximal amount of food consumed ad lib. during one single meal. Dogs AD1, AD4, AD7 and AD9 ate a maximum of 2–4 times more than before the LA operation, and dogs AD4 and AD9 consumed at one feeding a much greater amount of food than in their normal unoperated state. The increase in maximal food intake as compared with the period between MA and LA operations was significant for the whole group ($p < 0.001$, Student t-test). As seen from Fig. 4 the frequency distribution of the amount of ad lib. food intake was before LA operation skewed to the right; whereas after LA operation it was skewed — to the left. This shows once more that the dogs were able to consume bigger meals postoperatively then before LA operation.

The most striking changes occurred in the general alimentary behavior of the dogs. As mentioned above the dogs after the first (DMA) operation were indifferent towards food. Even if their food intake reached the preoperative level, they seemed to be uninterested in it. In contrast, after the second operation (LA), the dogs again started looking for food (Fig. 5d), followed the technician preparing food, jumped up to the food bowl and passed obstacles to reach it. When food was presented, they came to the food bowl at once (Fig. 5e) and devoured the food quickly and voraciously. Even in those dogs in which the amount of food intake did not increase significantly, the attitude toward food changed dramatically.

General behavior. The behavior of the dogs after LA operation remained one of their normal behavior before the first operation. They again became lively, gay and interested in the surroundings. They returned to their old habits of play and exhibited the friendly attitude towards the experimenters which they used to have before both operations, such as holding the leash, jumping on and licking the technician's face, rolling on the back for play, etc.

The behavior of some of the dogs was similar to that described previously after bilateral damage of LA alone. i.e., they were more playful and lively than in the normal state (13). The other dogs returned to the
Fig. 1. Food intake of amygdala dogs. Bars represent mean value for the whole group of the amount of food eaten in 10 meals of ad lib. feeding before DMA operation (A), 10 meals after the DMA operation (B), the next 10 meals (B1), 10 meals 1 month later (C), 10 meals just before the LA operation (C1), 10 meals just after the LA operation (D), the next 10 meals (D1), and 10 meals 1 month later (E). Perpendicular lines, standard errors.

Fig. 2. Body weight of amygdala dogs. Blocks represent mean value for the whole group in the unoperated state before the DMA lesion (A), on 10th day after the DMA operation (B), before the LA operation (C), on 10th day after the LA operation (D), and 2-3 months later at the end of the observation period (E). Perpendicular lines, standard errors.
Fig. 3. Food intake and body weight (in kg) before and after DMA and LA lesions in the representative dog AD7.
same forms of behavior which were observed before both operations. Emotional symptoms of fear and aggression were completely abolished in three dogs and greatly diminished in two others. The specific vocalization observed after DMA operation was preserved only in two dogs, and even in those dogs it appeared only occasionally.

Vomiting. Vomiting was observed only in two dogs (AD2, AD11), which vomited also during the whole period between the two operations. The LA operation enhanced vomiting in these dogs for a few days. Then, for 2–3 weeks the dogs vomited occasionally; and later vomiting no longer occurred.

Body weight. The body weight increased after lateral amygdala lesions (Table IIICD). This increase was significant in all dogs ($p < 0.001$, Student t-test). Body weight continued to increase further in most of the dogs, even in those in which food intake gradually reduced (Fig. 2E). Figure 3 shows the curves of food intake and body weight in a representative dog, AD7. Although the two parameters increased directly after the LA operation, the body weight increased further even in the period when the dogs limited their food intake. In some dogs the LA operation affected only the body weight. The food intake stayed at about the preoperative level, and the only effect of the LA operation was the increase of body weight.

Other changes. Lesion of La affected the general appearance of the
Fig. 5. Dog AD4 after DMA operation (a, b and c) and after LA operation (d and e). Notice apathetic look, no interest in food “cafeteria” (a) and withdrawal from feeding (b and c) after DMA operation, in contrast with interest in food and spontaneous eating after subsequent LA operation (d and e).
Fig. 6. Reconstruction of the DMA and LA lesions, based on histological sections of the brain of representative dog AD1. Cross-hatched areas, first operation; blackened areas, second operation; stripped areas, degeneration in optic tract.
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Fig. 7. Changes in food intake in three dogs after the DMA and subsequent LA damage, when the second operation was performed in three different periods of time after the first one.

animals in a way opposite to that caused by lesion of DMA. The dogs gained weight, and thus addipose tissue underlying the skin became thick and compact. The skin itself became thick and elastic. The hair grew luxuriantly. For example, in dog AD2, in which the hair was short (about 1 cm) and straight before the LA operation, became long (3–4 cm), thick, glittering and curled after the LA operation. The dogs again stood erect, wagging their tails; and their ears were held up and reactive to external stimuli.
Anatomical verification

In all the dogs, the first operation invaded the dorsomedial part of the amygdala. In most of the dogs, the central and medial nuclei were damaged bilaterally. Dog AD2 had damage in the dorsomedial amygdala region in the right hemisphere while on the left side the lesion involved the internal capsule neighboring the dorsal amygdala but did not reach the amygdala itself. In another dog (AD8), the lesion involved only unilaterally the upper part of the medial nucleus and cut bilaterally the internal capsule. Also in this dog the posterior hypothalamus and nucleus subthalamicus and premammillary nucleus were touched by the lesion. The hypothalamus was also damaged unilaterally in two other dogs (AD4, AD7) on its lateral left part. The optic tract was damaged unilaterally or bilaterally in seven dogs.

The second operation impaired in all the dogs the lateral amygdala. In most of the dogs the lesion involved its ventral posterior part. In three dogs (AD4, AD6, AD7) the ventral hippocampus was also touched bilaterally and in one dog (AD2) unilaterally. In most of the dogs, the adjacent part of the temporal lobe, lateral or ventral to the amygdala, was also reached unilaterally by the lateral extension of the lesions. Figure 6 shows a reconstruction of the lesion in the representative dog AD1.

A more detailed analysis of the neuroanatomical verification will be the subject of another paper.

DISCUSSION

Our results have shown that lateral amygdala lesions (LA) reverse completely or almost completely the syndrome produced by previous damage of the dorsomedial amygdala (DMA). This seems to show that aphagia and apathy produced by dorsomedial lesions are not caused by impairment of the neuronal basis for alimentary mechanisms, but are due to prevailing inhibitory influences deriving from the basolateral part of the amygdala. When the source of inhibitory influences is abolished by additional lesions, the normal homeostasis (excitatory–inhibitory balance) is restored. As a consequence, general arousal is increased and alimentary functions regain their normal level.

The problem arises as to whether the recovery of alimentary function is really due to the damage of inhibitory mechanisms or is simply produced by the compensation which usually occurs with time after most brain lesions. In fact, in five of our dogs such a compensation had already taken place to a great extent before the second operation was performed. Therefore, the effect of the second operation may be seeming, and can be
ascribed to the spontaneous recovery of feeding mechanisms. For various reasons we do not think this is the case. First, the second LA operation was performed at different times after the first DMA operation; and, therefore, it seems unlikely that it coincided with the day of spontaneous recovery. In most of the dogs partial recovery of food intake was stabilized at a certain level before the second operation (Fig. 2) and did not increase further. In two dogs the second operation was purposely, performed later than in the remaining dogs (3 and 4 months after the first one). In one of these dogs (AD2) the food intake was restored almost to the preoperative level before the LA operation; whereas, in the second one (AD1) hypophagia persisted and even increased with time. Nevertheless, in both dogs the effect of the lesion of the lateral nucleus of the amygdala was the same as in the remaining dogs, i.e., an increase of food intake and body weight. Three other dogs (AD9, AD11, AD12) were subjected to the second operation about 4 weeks after the first one. In these dogs the effect of the lesion was even more striking (Table I and II). Figure 7 demonstrates that the effect of the LA operation does not depend on the period in which it is performed after the DMA operation.

In most dogs obvious changes in emotional aspects of alimentary behavior occurred parallel to the increase of the food intake. Immediately following the second operation the dogs showed an increased interest in food. They not only consumed a greater amount of food than before the operation but ran to the food bowl and devoured the food voraciously. Such a sudden onset of voraciousness was not observed if the recovery proceeded without the second operation.

Another interesting fact is that LA lesion reversed also the effects of the DMA operation on the general behavior and other aspects of the emotional state of the dogs. After the first operation the dogs were apathetic, showed somnolence and indifference. They lost their friendly relations with humans. In a few weeks after the first operation although they later began to eat spontaneously, and most of them even, in sufficient amounts, they still had altered personalities. Immediately after the LA operation, or at least within a few days, the behavior of all dogs changed in the opposite direction. They again became lively and interested in everything. Their old habits which they used to have in their normal state before the DMA operation, like for example play and friendly attitude toward humans were restored fully after the second operation. This restoration was spontaneous and did not need any special retraining. It was contrast sharply with their behavior after the first and before the second operation. In some of the dogs these emotional changes were much more striking than the increase in the amount of food consumed.
The evidence of opposite effects from two successive operations damaging two areas of the amygdala explains why many authors, including ourselves, found no such obvious changes after a large ablation of the amygdaloid complex. When in one operation two antagonistic systems were simultaneously excluded the net effect was balanced to almost null or was minimal. These results clearly indicate that damage limited to the crucial points is more effective.

The fact that the effect of the dorsomedial lesion may be reversed seems to indicate that the alimentary "centers" of the amygdala do not consist of neurons indispensable for patterns of alimentary reactions, but rather are control centers which send excitatory (dorsomedial part) or inhibitory (lateral part) impulses. The decrease of excitatory impulses which is the result of dorsomedial amygdala lesions, and the following excess of inhibitory impulses suppresses alimentary function but does not damage it irreversibly. This suppression is not limited only to alimentary mechanisms but involves also the general state of arousal and positive aspect of emotions.

After damage of the inhibitory system all these symptoms are reversed. The source of excessive inhibition is abolished by LA damage and the excitatory–inhibitory balance of the whole alimentary system is restored. Together with alimentary functions, vitality and positive motivational behavior return. The animals again enjoy eating, strive for food, play, and show affection for humans. They look as normal as before the first operation.

The interesting fact is that general arousal and high motivational state are connected with such basic and important biological functions as those connected with feeding. The DMA operation abolished mostly the positive aspects of drive. The dogs after this operation ceased to be friendly and gay, and some of them became more fearful, sad or aggressive, after LA operation friendliness and interest in the environment were restored together with general arousal and alimentary drive. Negative reactions—fear and aggression—decreased or disappeared. This coincidence seems not to be accidental. It is possible that the two systems: positive emotional and alimentary, are in anatomical proximity to each other and are therefore damaged simultaneously by the same lesion, although they may be quite independent functionally. As shown by us (11, 12, 17, 26, 28), similar effects are seen in the hypothalamus, i.e. lateral hypothalamic aphagia in dogs is also connected with apathy and depression. It seems therefore that accidental parallelism in two different anatomical areas is much less probable. Therefore the physiological relations between positive emotions and feeding mechanisms seems to be more basic. In addition, the lateral hypothalamic aphagia and depression may also be re-
versed by lateral amygdala lesion as shown by our preliminary results (11, 12). The inhibitory effects of the lateral amygdala upon the lateral hypothalamus have been proven by electrophysiological studies (24, 25). Recently we have also started electrophysiological experiments in order to study whether such antagonistic relations exist also between the lateral and dorsomedial amygdala (R. Tarnecki, E. Fonberg, E. Mempel and J. Łagowska, in preparation).

We should also discuss the fact that both DMA and LA lesions strongly affected general metabolism as it is reflected in body weight. It is interesting to follow the parallel changes of the body weight and food intake after both operations. The dorsomedial amygdalar lesions produced aphagia, further hypophagia and significant decrease of body weight, which in most dogs remained at a low level even during the period of compensation in food intake. After the second operation producing damage of the lateral amygdalar nucleus, the increase in food intake was followed by increase in body weight. After a few weeks the food intake decreased; but the body weight increased further; and in four dogs it not only exceeded the level from before the second operation but even that of the normal, unoperated state. These facts seem to indicate that the increase of body weight is not only due to the excess of food consumed, but also to some metabolic changes produced by the operation itself. These changes are more persistent than the increase of food intake.

These data are consistent with the results of our previous work concerning the effects of single operations on the lateral amygdala. In that report the increase of food intake was also transient and limited to the first phase, and the increase in body weight persisted for a longer time (10, 13). Similar phases were described by Brobeck et al. (2), Teitelbaum (30, 31), Teitelbaum and Campbell (33), after lesions of the ventromedial hypothalamus in rats, and by Rożkowska and Fonberg in dogs (27). As shown by other authors (2, 3) with regard to hypothalamic hyperphagic rats, the increase of body weight is produced by fat storage. Whether it is true also for amygdalar hyperphagia we do not know. The dogs never become as fat as the rats do. The increase of body weight in our amygdala dogs usually did not exceed a value of 20–30%. This increase was not due to reduced energy output, as may be the case in hypothalamic animals (see 1), since our dogs were much more lively and motile than before the LA operation.

As pointed out by Teitelbaum (30–32) with regard to obese rats after ventromedial hypothalamic lesions, obesity inhibits further food intake. So, the animals must somehow detect body weight in order to regulate their food intake. This may be regulated by the cells in ventromedial hypothalamus, which in our experiment remain intact. According to Tei-
telaum the food intake of the hyperphagic hypothalamic rats drops only when the body weight is much higher than before operation. The same holds true if the lateral amygdala lesions are made in normal dogs (13). However, if lateral amygdala damage was added to the previous dorso-medial lesion, as it was in this study, the food intake usually dropped before the body weight reached the normal level from before both operations.

The appearance of the hair and skin after both operations also speaks in favor of profound metabolic changes. As a consequence of DMA damage the skin becomes thin and flexible and the hair weak and dull. Similar changes were observed by Schwartz and Kling (29) in amygdalectomized aphagic rats. After the subsequent LA operation the hair grew, shone and sometimes curled. However, further careful metabolic investigations are needed to elucidate the complicated problem of the interplay of arousal, emotions, food intake and metabolic balance.

In conclusion we suggest that within the amygdala there exist definite areas connected with alimentary functions and belonging to two opposite systems: one — excitatory in the dorsomedial part, the other — inhibitory in the lateral part. These systems consist of neither the anatomical substratum of neurons patterning the alimentary reactions nor the pathways of alimentary reflex arcs. They exert respectively excitatory or inhibitory effects on the other parts of the alimentary system. These two systems are antagonistic with a reciprocal negative correlation. Selective damage of each of them impairs the balance of the alimentary functions in opposite directions. This explains why the disturbances in food intake and alimentary motivation produced by impairment of the excitatory (DMA) part might be abolished by subsequent lesion of the inhibitory (LA) part. The possibility of improving pathological functions by damage to the system antagonistic to the impaired one may furnish practical implications for human clinic.

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