Physiology & Behavior, Vol. 19, pp. 389–395. Pergamon Press and Brain Research Publ., 1977. Printed in the U.S.A.

Aphagia, Adipsia, and Sensory-Motor Deficits Produced by Amygdala Lesions: A Function of Extra-amygdaloid Damage[1]

D. M. DACEY AND S. P. GROSSMAN

Biopsychology Committee, University of Chicago, Chicago, IL 60637

(Received 6 June 1977)

DACEY, D. M. AND S. P. GROSSMAN. *Aphagia, adipsia, and sensory-motor deficits produced by amygdala lesions: a function of extra-amygdaloid damage.* PHYSIOL. BEHAV. 19(3) 389–395, 1977. – Small bilateral electrolytic lesions confined to the region of the central nuclei of the amygdala failed to affect food and water consumption in male rats. More medially-placed lesions which damaged portions of the internal capsule, entopeduncular nucleus and globus pallidus adjacent to the central nucleus produced transient aphagia and adipsia accompanied by sensory-motor impairments that interfered with the act of feeding, as evidenced by a large postoperative increase in the amount of food spilled. In some cases lesions restricted to the internal capsule and entopeduncular nucleus produced these sensory-motor deficits but no aphagia or adipsia. Small lesions damaging primarily ventral portions of the posteromedial pallidi resulted in aphagia and adipsia but no apparent sensory-motor impairment. The results indicate that at least two, possibly independent, deficits in food intake may be produced by lesions in the immediate area of the dorsomedial amygdala. It is suggested that the effects on ingestive behavior which several investigators have observed in rats with dorsomedial amygdaloid damage may in fact be due to incidental destruction of adjacent tissues.

Aphagia Adipsia Sensory-motor deficits Internal capsule Globus pallidus Amygdala

IT IS WELL established that lesions in some portions of the amygdala of the rat [7,19] cat [16,31] dog [12,15], and monkey [34,40] result in hyperphagia. There is less agreement concerning the exact location of the effective lesions. Most investigators report that ventral and lateral portions of the amygdaloid complex must be involved, and there are several reports (e.g., [1, 9, 37]) of hyperphagia after damage to periamygdaloid cortex which did not involve nuclei of the amygdala proper. Electrical stimulation of electrode sites in many aspects of the amygdala exert inhibitory effects on food intake in the rat [19,45], cat [13], dog [10], and monkey [36], but the specificity of these effects is often inadequately demonstrated. On balance, the literature nonetheless supports the conclusion [18] .that the amygdala contains diffusely represented neural mechanisms which exert inhibitory influences on food intake.

There is less agreement concerning the possibility that the amygdaloid complex may also be the source of excitatory influences on feeding behavior. Only very brief periods of aphagia or hypophagia have been reported in monkeys [44] and cats [3,21] after often extensive amygdaloid lesions. Some investigators [16,29] have reported more persistent effects in the cat but in these cases the aphagia or hypophagia is accompanied by catalepsy, catatonia, and general deterioration and the specificity of the effect on food intake is in doubt. In the rat [4, 6, 35, 38, 39] and dog [5, 11, 14], persisting aphagia or hypophagia have been reported by several investigators but it is unfortunately not clear whether these effects are due to the destruction of amygdaloid tissues rather than incidental damage to adjacent portions of the internal capsule or striatopallidal system as Stevenson [42] and others have suggested. Published descriptions of the effective lesions indicate that their principal focus lies in the medial segment of the amygdala and most investigators agree that the dorsomedial amygdala, particularly the region of the central and medial nuclei, may be critical. However, lesions which destroy most of this region almost invariably involve the globus pallidus, caudate nucleus, and lateral portions of the internal capsule. In the rat, the published descriptions of effective lesions indicate without exception that significant damage to some or all of these structures occurred. In the dog, the literature is similar,

[1] This research was supported by Grant MH 26934 from the U.S. Public Health Service to S. P. Grossman. Send reprint requests to S. P. G. We gratefully acknowledge the generous assistance of L. Grossman and K. Dacey.

although Fonberg [11] has reported aphagia in two animals with dorsomedial amygdaloid lesions which appear to produce little damage to adjacent tissues. Even here, the published evidence does not entirely rule out incidental interference with some possibly critical extra-amygdaloid pathways. Fonberg's report unfortunately fails to make clear whether the effect observed in these two animals persisted for more than two days (the duration of aphagia for all experimental animals is said to range from two to fourteen days) and what role, if any, the apparently severe arousal and motor dysfunctions which such lesions produced may have played.

The literature on the effects of electrical stimulation of the amygdala does not offer substantial support for the hypothesis that the amygdala might be the source of excitatory influences on food intake. Licking, chewing, and biting have been observed during electrical stimulation of electrode sites in the amygdala of cats, dogs, and monkeys [23, 30, 46], but feeding itself has been seen only in a few instances in the monkey [36] and never in the rat [19], cat [8] or dog [10,13]. Norepinephrine injections into the amygdala potentiate (but do not elicit) feeding in deprived rats [17] but this may reflect a blockade of inhibitory mechanisms rather than excitation of faciliatory outputs [41].

We [20] have recently investigated some of the behavioral effects of small tubular lesions which were largely confined to each of the six major nuclei of the rat amygdala or adjacent regions of the temporal lobe. None of these animals were aphagic at any time after the first postoperative day even though some of the nuclei which have been implicated in food intake regulation by other investigators were largely destroyed. The central nuclei which Box and Mogenson [4] believe to be responsible for aphagia after medial amygdaloid lesions were destroyed almost completely in ten animals which ate and drank normally but died a few weeks after surgery of unknown causes. Six additional rats with lesser damage (average destruction 50%to this area survived for months without showing any evidence of hypophagia. Ten animals with extensive damage to the medial nuclei (average destruction 75%; range 60–100%), which have been implicated by the work of Fonberg [11] and others, also ate and drank normally after the first postoperative day. Due to the multiple electrode technique that was used to produce our lesions, none of our rats sustained significant damage to extra-amygdaloid tissue and it appears likely that this may account for our observation of unchanged food and water intake. The present experiments were designed to test this hypothesis by comparing the effects of lesions restricted to the central nucleus of the amygdala with more medially-placed lesions damaging only adjacent portions of the internal capsule or globus pallidus, leaving the amygdala intact.

METHOD AND SURGICAL PROCEDURE

Forty adult male albino rats (Sprague-Dawley Strain, Holtzman Co.) ranging in body weight from 325–400 g were used. The animals were maintained in an air-conditioned colony (21–24° C) under a 12-hr light-dark cycle (0600–1800 hr light). Each rat was individually caged and allowed to feed ad lib a standard pellet diet (6% fat) placed on the floor of the cage. Fresh water was always available from a drinking tube.

Following surgery, food and water intake and amount of spillage were monitored daily for ten days. Animals were considered aphagic or adipsic if they failed to initiate feeding or drinking behavior. Rats that attempted to feed but did not ingest normal amounts due to an increase in spillage were considered to have sensory-motor impairments. The effect of the sensory-motor deficit on food intake was recorded in terms of an efficiency index generated by dividing the amount of food eaten by the total amount of food handled (spillage plus amount eaten). This measure was used to quantify recovery of sensory-motor control of food intake after spontaneous ingestive behavior reappeared. Those animals that did not initiate feeding within 48 hr after surgery were fed a liquid diet intragastrically until spontaneous food intake reappeared.

Fifteen animals under Nembutal anesthesia received bilateral electrolytic lesions aimed stereotaxically at the central nucleus of the amygdala according the coordinates [33] AP = 5.5, H = −1.8, L = 4.2. The remaining 25 rats received lesions in the internal capsule 1 mm medial and slightly dorsal to the central nucleus (AP = 5.5, H = −1.5, L = 3.2). Lesions were produced by passing 0.8–1.0 mA anodal DC for 8–20 sec through a stainless steel insect pin (No. 1) insulated with Teflon except for 0.5 mm at the tip. At the conclusion of the experiment all lesions were verified histologically using a standard frozen tissue technique and cresyl violet cell stain.

RESULTS

Of the fifteen animals sustaining damage to the amygdala, histological verification showed that in 9 rats a major portion of the central nucleus was destroyed without disturbing extra-amygdaloid tissue. Damage to other amygdaloid tissues was slight: in 3 animals the stria terminalis was damaged, in 4 others the lesion impinged slightly on the lateral nucleus. The 6 other animals of this group had lesions that were either assymetric or did no significant damage to the central nucleus. The data from these 6 rats were discarded.

The postoperative food and water intake (Figs. 1A, 2) of the 9 rats with central nucleus damage was not significantly different from their preoperative baseline except for a slight depression during the first 24 hr after surgery which reflects the effects of anesthesia and general surgical trauma. No motoric disabilities or general malaise were observed and food spillage was within the normal range as revealed by our measure of feeding efficiency (see Fig. 1B).

Histological examination of the 25 brains with electrodes aimed slightly medial to the central nucleus showed assymetrical or inappropriate placement in 7 of them; data from these animals were excluded from further consideration. The 18 remaining animals could be divided into three groups on the basis of differences in the area of principal damage and behavioral deficits. Group 1 (n = 5) animals were aphagic and adipsic for 2–4 days (Figs. 1A, 2) after surgery but showed no deficit in efficiency when feeding behavior reappeared (Fig. 1B). Postural aberrations or significant activity changes that might indicate sensory-motor alterations were not apparent in these animals. Histological analysis of this group revealed significant damage, at least unilaterally, to the ventral posteromedial pallidum but little damage to the capsule. An example of such a lesion is shown in Fig. 3.

The animals of Group 2 (n = 8) exhibited a profound drop in feeding efficiency beginning the first day post-

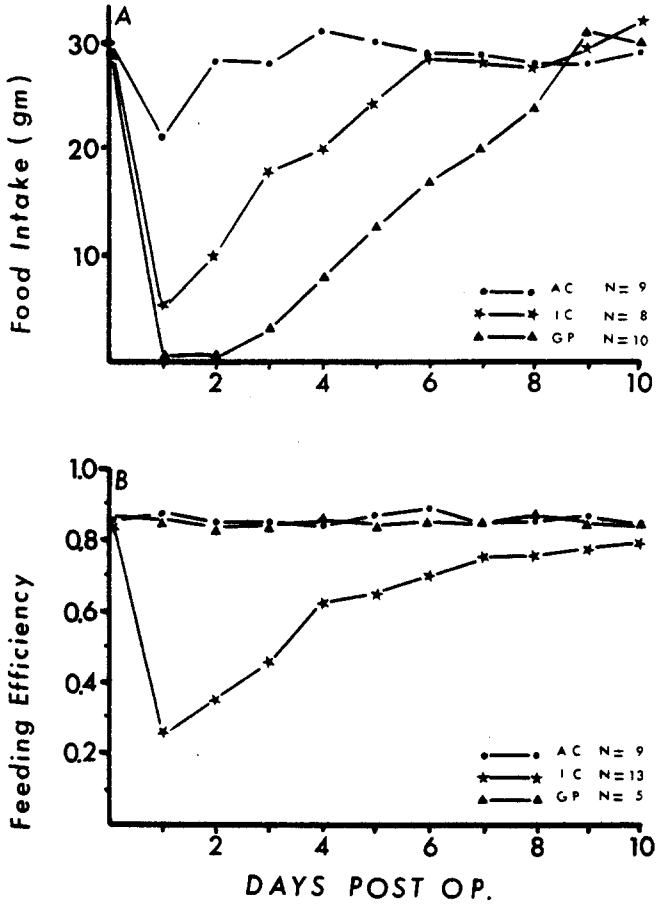

FIG. 1. (A) Mean daily food intake for rats with lesions in the region of the dorsomedial amygdala. AC = lesions of the central nucleus (Group 0), GP = lesions of the globus pallidus (Groups 1 and 3). Group 3 animals sustained damage to the internal capsule as well as to the globus pallidus. IC = lesions restricted to the internal capsule (Group 2). (B) Feeding efficiency index (1.0 = 100% efficiency or zero spillage) for the same animals. In this plot GP represents only those animals (Group 1) that sustained damage restricted to the globus pallidus. IC = all remaining animals with internal capsule damage (Groups 2 and 3).

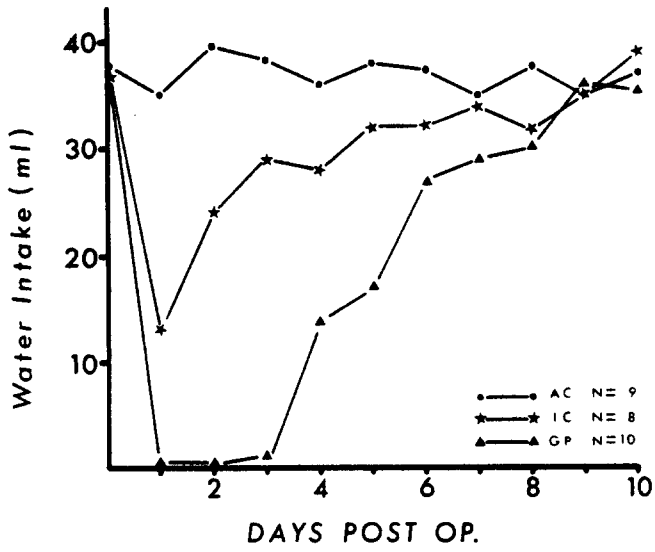

FIG. 2. Mean daily water intake for rats with lesions in the region of the dorsomedial amygdala (see Fig. 1A for abbreviation).

surgery. The animals were clearly not aphagic at any time after surgery and their apparent hypophagia may well be due entirely to sensory-motor disability. For example, one rat in this group spilled 100 g of food but ingested only 3 g during the first two days after surgery. This is a vivid illustration of the fact that, unlike the aphagic rats of Group 1 which did not initiate feeding after surgery, these animals repeatedly and vigorously attempted to feed but were incapable of doing so. Histological examination of the brains of the animals that were hypophagic as a result of sensory-motor dysfunctions revealed damage to the capsule and entopeduncular nucleus but virtually no damage to the globus pallidus.

The animals of Group 3 (n = 5) were aphagic and adipsic for 2–7 days and demonstrated severe impairments in feeding efficiency when spontaneous food intake reappeared. Efficiency gradually returned to normal levels over the next 3–7 days. The pattern of recovery of the rats in Group 3 seems to indicate that the aphagia/adipsia and the sensory-motor impairment are two independent effects. This is indicated by the observation that the rats which displayed the most persistent aphagia and adipsia (5–7 days) showed less severe and persistent sensory-motor deficits upon recovery of voluntary ingestive behavior than did those that had resumed feeding after only 3–4 days. This suggests that recovery from the sensory-motor dysfunction may proceed concurrently with but independent of recovery from aphagia and adipsia. The intake records of Groups 1 and 3 are considered together in Fig. 1A to provide a picture of what we believe to be the motivational component of the deficit. The contribution of the sensory-motor impairments to the feeding deficit is shown in Fig. 1B, which summarizes the average feeding efficiency of all 13 rats (Groups 2 and 3) with motoric disabilities. Histological data from the animals in Group 3 revealed lesions that were more extensive than those of Groups 1 and 2, damaging the globus pallidus, adjacent tissue of the internal capsule, and the entopeduncular nucleus (see Fig. 4).

DISCUSSION

Our observation that lesions in the region of the central nucleus of the amygdala failed to produce aphagia or adipsia in the rat replicates earlier findings from this [20] as well as other [25] laboratories. This supports the hypothesis that the influence of the amygdaloid complex on ingestive behavior may be entirely inhibitory in nature.

The apparent conflict of our results and conclusion with several published reports of aphagia after dorsomedial amygdaloid damage may be resolved by our consistent replication of these effects in animals with lesions in adjacent structures (posteroventral portions of the globus pallidus, ventral striatum, internal capsule, and entopeduncular nucleus). Inspection of the histological data presented by other investigators who have reported aphagia after amygdaloid damage indicates that their lesions almost certainly invaded some of these structures. The possible contribution of sensory-motor deficits to the aphagia-

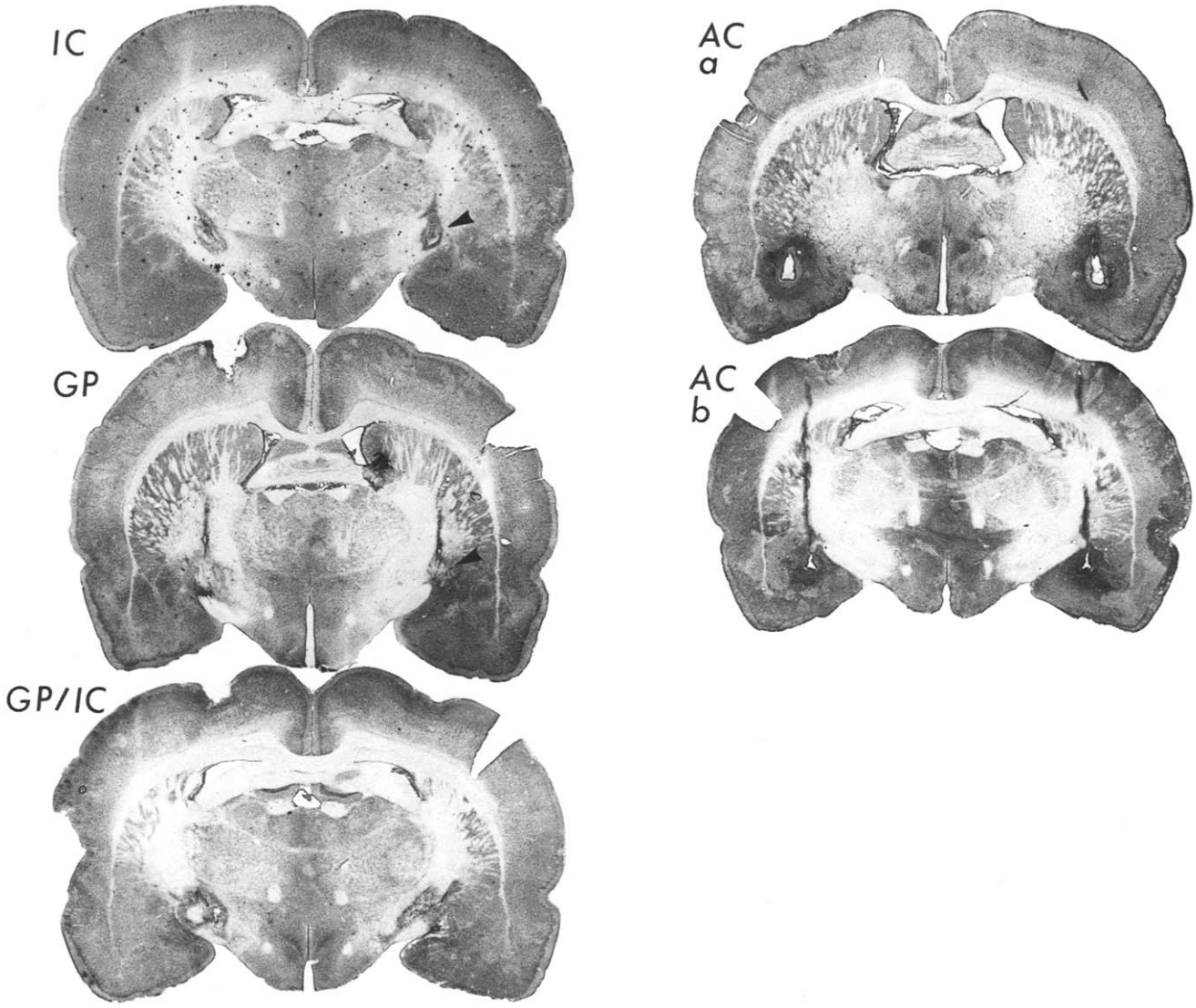

FIG. 3. Photomicrographs of sections through the plane of maximal damage in rats with lesions in and around the dorsomedial amygdala. Lesions of the central nucleus of the amygdala (AC) did not impinge on extra-amygdaloid tissue. Shown are examples of individual lesions in the rostral portion of the central nucleus (a) and another more caudally placed lesion (b). Lesions in adjacent structures – globus pallidus (GP), internal capsule (IC), globus pallidus and internal capsule (GP/IC) – did not invade the amygdala.

adipsia syndrome is unfortunately not explicitly discussed in most relevant publications.

An interesting exception is a recent paper by Box and Mogenson [4] which describes precisely the same compound syndrome – transient aphagia and adipsia followed by severe sensory-motor disturbances that interfere with the recovery of normal ingestive behavior – that we observed after lesions in the internal capsule. Box and Mogenson attributed these effects to damage to the central nucleus of the amygdala but explicitly recognized that their lesions infringed on the internal capsule. Their published histological material suggests, in fact, that the damage to this structure may have been extensive. Box and Mogenson argue that capsular damage should not have been a factor in the etiology of the aphagia syndrome because their lesions spared the medial-most aspects of the capsule that contain the nigrostriatal projection system which has recently been implicated in the aphagia/adipsia syndrome typical of rats with lateral hypothalamic lesions [43]. Our histological results are in general agreement with the conclusion that lesions which spare the medial aspects of the capsule, (and thus, presumably, most of the nigrostriatal projections) produce the syndrome described by Box and Mogenson but indicate that it is due to capsular and/or pallidal rather than amygdaloid damage.

The effects of these lesions are, in fact, quite different from those seen in rats with lateral hypothalamic lesions or chemical destruction of the nigrostriatal projections [43]. The latter preparations display pronounced akinesia, stupor, and lack of arousal responses to all but the most intense environmental stimuli and it has been suggested that a lack of endogenous activation [28] or general arousal [43] may be responsible for the initial aphagia and adipsia which characterizes these animals. Rats with lesions re-

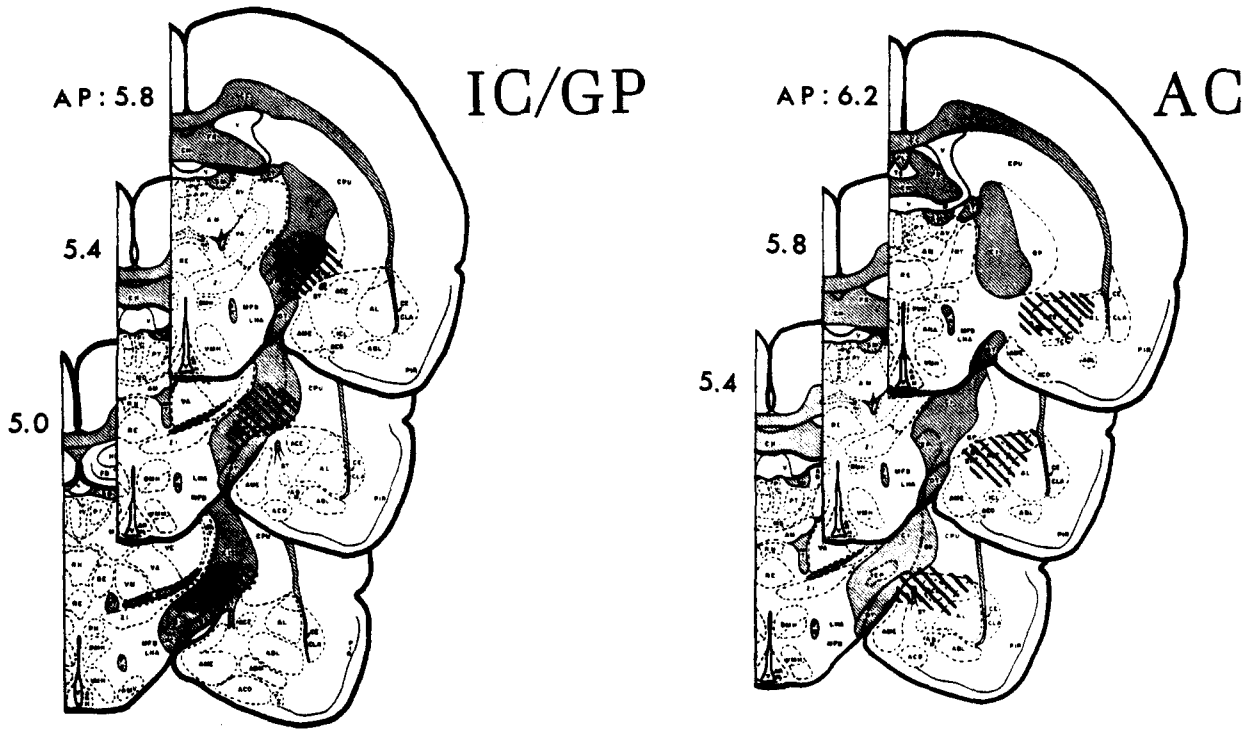

FIG. 4. Tracings of all individual lesions at the point of maximal damage were superimposed at the appropriate plane of section in a single composite representation shown above. All individual lesions were considerably smaller than and are contained within the crosshatched area. AC represents the total extent of amygdala damage for the 9 rats in Group 0. IC/GP encompasses the lesions of all 18 animals with extra-amygdaloid damage (Groups 1, 2, and 3).

stricted to the internal capsule (Group 2) do not display these arousal deficits and show a much more limited sensory-motor deficit which appears to affect food intake mainly because it interferes with the control of oral musculature used to chew, swallow, etc. It is not known, however, whether these effects are due specifically to damage to descending pyramidal fibers, ascending sensory input, of perforating pallidal connections. In this regard it can be noted that our results are reminiscent of the effects of entopeduncular and/or globus pallidus lesions described by Schwartzbaum and associates [26,27]. Similar but more severe dysfunctions have also been observed in rats that recovered voluntary food intake after a period of aphagia following orbitofrontal cortex lesions [24]. It is possible that an interruption of higher-order trigeminal projections may contribute to the effect. Ziegler and Karten [47] have reported transient aphagia (without adipsia) in rats with brainstem lesions that interrupt these sensory projections from the region of the mouth.

That a sensory-motor impairment is responsible for the hypophagia observed in Group 2 animals is shown clearly in our analysis of feeding efficiency. Animals with lesions restricted to the entopeduncular nucleus and internal capsule consumed little food during the initial postoperative period due to an incapacity to ingest food (as reflected in spillage) rather than a lack of food motivation. The extremely large amounts of shredded food under the cages clearly indicates a strong disposition towards feeding behavior.

The effects of our lesions that infringed on the posteroventral aspects of the globus pallidus suggest that a second, probably independent, syndrome of deficits in ingestive behavior can be obtained by lesions in the general area of the dorsomedial amygdala. These lesions typically resulted in adipsia (as well as aphagia) and there was no indication of debilitating sensory-motor disabilities (i.e., no impairment in feeding efficiency) during the relatively brief (1–2 day) period of hypophagia and hypodipsia that preceded recovery of normal ingestive behavior. We observed neither gross arousal nor activation difficulties or overt motor impairments during the initial period of aphagia and adipsia in these animals and are of the opinion that the effects of these lesions may be significantly different not only from those of the capsular lesions described above but also from chemical or electrolytic lesions of the nigrostriatal afferents [43]. The pattern of effects seen in this portion of our experiment does appear similar to, although less severe than, that reported by Alheid *et al.* [2] after extensive undercuts of the striatum and globus pallidus which transected a major portion of its afferent and efferent connections but spared a large percentage of the dopaminergic nigrostriatal inputs to the system. Since pallidofugal fibers pass through and around this region of the capsule, it is probable that all of our lesions medial to the amygdala at least slightly interrupted these systems. The extent of damage to pallidal connections could not be assessed in this study, but transection of functionally specific afferent and/or efferent pallidal connections may, at least in part, be responsible for the differential effects on behavior observed. That an interference with striatal and/or pallidal connections (as well as capsular elements of unknown origin and destination) may account for the effects of large amygdaloid or smaller dorsomedial amygdaloid lesions on ingestive behavior is in agreement with

Korczynski and Fonberg's [25] recent observation that lesions in the dorsomedial amygdala of the rat produced only minor disturbances in food intake unless the damage extended into the region dorsal to the amygdala proper.

Recent anatomical and electrophysiological evidence [32] indicates that the central nucleus receives tertiary gustatory afferents from the brainstem. The results of our investigation suggest that these inputs are not essential for the initiation and maintenance of daily food intake but this does not, of course, rule our more subtle influences on ingestive behavior, particularly in situations where taste is a critical variable.

REFERENCES

1. Adey, W. R., N. C. R. Merrillees and S. Sunderland. The entorhinal area: Behavioral, evoked potential, and histological studies of its relationships with brain-stem regions. *Brain* 79: 414–439, 1956.
2. Alheid, G. F., L. McDermott, J. Kelly, A. Halaris and S. P. Grossman. Deficits in food and water intake after knife cuts that deplete striatal DA or hypothalamic NE in rats. *Pharmac. Biochem. Behav.* 6: 273–287, 1977.
3. Anand, B. K., S. Dua and G. S. Chhina. Higher nervous control over food intake. *Indian J. med. Res.* 46: 277–278, 1958.
4. Box, B. M. and G. J. Mogenson. Alteration in ingestive behavior after bilateral lesions of the amygdala in the rat. *Physiol. Behav.* 15: 679–688, 1975.
5. Brutkowski, S., E. Fonberg, J. Kreiner, E. Mempel and B. Sychowa. Aphagia and adipsia in dogs with bilateral complete lesion of the amygdaloid complex. *Acta Biol. exp., Vars.* 22: 43–50, 1962.
6. Collier, D. B. and F. P. Gault. Aphagia and adipsia following lesions of the amygdala. *Psychon. Sci.* 17: 41–42, 1969.
7. Czech, D. A. Effects of amygdalar lesions on eating and drinking and saline preference in the rat. *Physiol. Behav.* 10: 821–823, 1973.
8. Delgado, J. M. R. and B. K. Anand. Increase of food intake induced by electrical stimulation of the lateral hypothalamus. *Am. J. Physiol.* 172: 162–168, 1953.
9. Entingh, D. Perseverative responding and hyperphagia following entorhinal cortex lesions in cats. *J. comp. physiol. Psychol.* 75: 50–58, 1971.
10. Fonberg, E. Inhibitory role of amygdala stimulation. *Acta Biol. exp., Vars.* 23: 171–180, 1963.
11. Fonberg, E. Effects of small dorsomedial amygdala lesions on food intake and acquisition of instrumental alimentary reactions in dogs. *Physiol. Behav.* 4: 739–743, 1969.
12. Fonberg, E. Hyperphagia produced by lateral amygdalar lesions. *Acta Neurobiol. exp., Vars.* 31: 19–32, 1971.
13. Fonberg, E. and J. M. R. Delgado. Avoidance and alimentary reactions during amygdala stimulation. *J. Neurophysiol.* 24: 651–664, 1961.
14. Fonberg, E. and B. Sychowa. Effect of partial lesions of the amygdala in dogs. I. Aphagia. *Acta Biol. exp., Vars.* 26: 35–46, 1968.
15. Fuller, J. F., H. D. Rosvold and K. H. Pribram. The effect on affective and cognitive behavior in the dog of lesions of the pyriform-amygdala-hippocampal complex. *J. comp. physiol. Psychol.* 50: 89–96, 1957.
16. Green, J. D., C. D. Clemente and J. DeGroot. Rhinencephalic lesions and behavior in cats. *J. comp. Neurol.* 108: 505–545, 1957.
17. Grossman, S. P. Behavioral effects of chemical stimulation of the ventral amygdala. *J. comp. physiol. Psychol.* 57: 29–36, 1964.
18. Grossman, S. P. Neurophysiologic Aspects: Extrahypothalamic factors in the regulation of food intake. *Adv. Psychosom. Med.* 7: 49–72, 1972.
19. Grossman, S. P. and L. Grossman. Food and water intake following lesions or electrical stimulation of the amygdala. *Am. J. Physiol.* 205: 761–765, 1963.
20. Grossman, S. P., L. Grossman and L. Walsh. Functional organization of the rat amygdala with respect to avoidance behavior. *J. comp. physiol. Psychol.* 88: 829–850, 1975.
21. Kling, A. and N. B. Schwartz. Effect of amygdalectomy on feeding in infant and adult animals. *Fedn Proc.* 20: 235, (Abstract), 1961.
22. Koikegami, H. Amygdala and other related limbic structures: Experimental study on the anatomy and function. *Acta med. biol. Jap.* 12: 74–266, 1964.
23. Koikegami, H., S. Fuse, T. Yokoyama, T. Watanabe and H. Watanabe. Contributions to the comparative anatomy of the amygdaloid nuclei of mammals with some experiments of their destruction or stimulation. *Folia psychiat. neurol. Jap.* 8: 336–370, 1955.
24. Kolb, B. and A. J. Nonneman. Prefrontal cortex and the regulation of food intake in the rat. *J. comp. physiol. Psychol.* 88: 806–815, 1975.
25. Korczynski, R. and E. Fonberg. Instrumental reactions and food and water intake in medial amygdala rats. *Acta Neurobiol. Exp.* 36: 667–686, 1976.
26. Levine, M. S. and J. S. Schwartzbaum. Sensorimotor functions of the striatopallidal system and lateral hypothalamus and consummatory behavior in rats. *J. comp. physiol. Psychol.* 85: 615–635, 1973.
27. Levine, M. S., N. Ferguson, C. J. Kreinick, J. W. Gustafson and J. S. Schwartzbaum. Sensorimotor dysfunctions and aphagia and adipsia following pallidal lesions in rats. *J. comp. physiol. Psychol.* 77: 282–293, 1971.
28. Levitt, D. R. and P. Teitlebaum. Somnolence, akinesia, and sensory activation of motivated behavior in the lateral hypothalamic syndrome. *Proc. Natl. Acad. Sci.* 72: 2819–2823, 1975.
29. Lewinska, M. K. Changes in eating and drinking produced by partial amygdalar lesions in cat. *Acta Biol. exp., Vars.* 15: 301–305, 1967.
30. MacLean, P. D. and J. M. R. Delgado. Electrical and chemical stimulation of fronto-temporal portion of limbic system in the waking animal. *Electroencephalogr. Clin. Neurophysiol.* 5: 91–100, 1953.
31. Morgane, P. J. and A. J. A. Kosman. Rhinencephalic feeding center in the cat. *Am. J. Physiol.* 197: 158–162, 1957.
32. Norgren, R. Taste pathways to the hypothalamus and amygdala. *J. comp. Neurol.* 166: 17–30, 1976.
33. Pellegrino, L. J. and A. J. Cushman. Stereotaxic atlas of the rat brain. New York: Meredith, 1967.
34. Pribram, K. H. and M. Bagshaw. Further analysis of the temporal lobe syndrome utilizing frontotemporal ablations. *J. comp. Neurol.* 99: 347–375, 1953.
35. Pubols, L. M. Changes in food-motivated behavior as a function of septal and amygdaloid lesions. *Expl. Neurol.* 15: 240–254, 1966.
36. Robinson, B. W. and M. Mishkin. Alimentary responses evoked from forebrain structures in Macaca mulatta. *Science* 136: 260–262, 1962.
37. Ross, J. F., L. L. Walsh and S. P. Grossman. Some behavioral effects of entorhinal cortex lesions in the albino rat. *J. comp. physiol. Psychol.* 85: 70–81, 1973.
38. Schwartz, N. B. and A. Kling. The effect of amygdaloid lesions on sexual behavior and reproductive capacity in the male rat. *Fedn Proc.* 20: 335, (Abstract)1961.
39. Schwartz, N. B. and A. Kling. The effect of amygdaloid lesions on feeding, grooming and reproduction in rats. *Acta neuroveg.* 26: 12–34, 1964.

40. Schwartzbaum, J. S. Some characteristics of amygdaloid hyperphagia in monkeys. *Am. J. Physiol.* **74:** 252–259, 1961.
41. Singer, G. and R. B. Montgomery. Neurohumoral interaction in the rat amygdala after central chemical stimulation. *Science* **160:** 1017–1018, 1968.
42. Stevenson, J. A. F. Neural control of food and water intake. In: *The Hypothalamus*, edited by W. Haymaker, E. Anderson and W. J. H. Nauta. Springfield: C. C. Thomas, 1969, pp. 524–621.
43. Stricker, E. M. and M. J. Zigmond. Recovery of function following damage to central catecholamine-containing neurons: A neurochemical basis for the lateral hypothalamic syndrome. In: *Progress of Psychobiology and Physiological Psychology*, Vol. 6, edited by J. M. Sprague and A. N. Epstein. New York: Academic Press, 1976.
44. Weiskrantz, L. Behavioral changes associated with ablation of the amygdaloid complex in monkeys. *J. comp. physiol. Psychol.* **49:** 381–391, 1956.
45. White, N. M. and A. E. Fisher. Relationship between amygdala and hypothalamus in the control of eating behavior. *Physiol. Behav.* **4:** 199–205, 1969.
46. Wood, D. C. Behavioral changes following discrete lesions of temporal lobe structures. *Neurology* **8:** 215–220, 1958.
47. Zeigler, H. P. and H. J. Karten. Central trigeminal structures and the lateral hypothalamic syndrome in the rat. *Science* **186:** 636–637, 1974.