EFFECTS OF TEGMENTAL LESIONS ON MOTIVATED BEHAVIOR IN RATS

by

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Submitted to the Faculty of Graduate Studies and Research of McGill University in partial fulfillment of the requirements for the degree of Doctor of Philosophy, April, 1960.

Daniel Ehrlich designed and constructed
the test apparatus and prepared the figures;
his assistance is gratefully acknowledged.

C O N T E N T S

Clinical reports of pathological hunger and obesity resulting from brain injury go back at least as far as the nineteenth century, but systematic attempts to localize the crucial area (or areas) by producing the effect experimentally in animals did not begin until approximately forty years ago; and, with respect to localization, real success has been achieved only during the last twenty. The interest of psychologists in pathological hunger is of still more recent origin and dates from an experiment by Miller, Bailey, and Stevenson (1950) in which the hypothesis tested was that lesions known to increase ad lib. food intake would also lead to better performance on behavioral tasks motivated by hunger. Surprisingly, the results were in the opposite direction from the prediction. Rats made obese by destruction of the ventromedial nuclei of the hypothalamus ate significantly more food than normals when fed ad lib., but they performed significantly worse than normals on food-motivated tasks. In the first experiment reported here, lesions in the midbrain tegmentum produced a similar decrease in food motivation, and hence a review of the literature on brain lesions and hunger is appropriate. The effects of hypothalamic lesions will be discussed first, since more is known about the effects on hunger of lesions in this area than about lesions in any other part of the brain.

Effects of Hypothalamic Lesions on Hunger

Mohr is generally credited with having published the first clinical report (in 1840) of pathological obesity in man resulting from a tumor at the base of the brain (cited by Brobeck, 1946). Thereafter, although similar cases appeared in the literature from time to time, no particular interest was shown in localizing the effective lesion until the early nineteen hundreds when a heated dispute arose as to whether damage to the pituitary or to the hypothalamus was responsible. Numerous reports of experimentally-produced hypothalamic obesity in animals appeared in the literature between 1920 and 1940, but most of these (with the possible exception of Bailey and Bremer's work in 1921) were received with skepticism because the operative procedure used made it unlikely that the pituitary had been completely spared. The difficulty was not overcome until the early nineteen thirties when the method of making discrete electrolytic lesions by means of the stereotaxic instrument came into general use. At that time, several preliminary reports appeared in which this method was used (Ranson, Fisher & Ingram, 1938; Hetherington & Ranson, 1939), but the most conclusive demonstration that hypothalamic lesions alone were responsible for obesity was furnished by Hetherington and Ranson's experiment in 1940. The possibility that pituitary

damage played even a partial role was ruled out by hypophysectomizing rats before (Hetherington, 1943) and after (Hetherington & Ranson, 1942b) hypothalamic lesions. The results showed that obesity occurred after hypothalamic lesions whether the pituitary was intact or not, and removal of the pituitary in no way altered the hypothalamic symptoms.

In a further series of experiments, the most effective area within the hypothalamus was shown to be the ventromedial nucleus and the area immediately adjacent to it (Hetherington; 1941; 1944; Hetherington & Ranson, 1942c). That obesity follows ventromedial lesions was quickly confirmed in several species-- rats (Tepperman, Brobeck, & Long, 1941),cats (Wheatley, 1944), and monkeys (Brooks, Lambert, & Bard, 1942). In mice, obesity following ventromedial hypothalamic lesions has been demonstrated by two different methods--electrolytic lesions (Mayer, French, Zighera, & Barrnett, 1955) and goldthioglucose poisoning (Marshall, Barrnett, & Mayer, 1955).

It was fairly soon established that the cause of hypothalamic obesity was overeating or hyperphagia. Hetherington was inclined at first to attribute the weight gain to underactivity and metabolic disturbances (1941), but paired feeding experiments with normal controls as well as measurements of food intake, metabolic rate,and activity level showed that the changes in activity and metabolism were minor by comparison with the enormous increase in food intake

(Tepperman, et al., 1941; Brobeck, Tepperman, & Long, 1943; Brobeck, 1946; Brooks, 1946; Brooks, Marine, & Lambert, 1946). With electrolytic lesions, the hyperphagia is apparent as soon as the animal comes out of the anesthetic (Brobeck, et al., 1943; Brooks, Lockwood, & Wiggins, 1946), and it continues for a period of several months (dynamic phase) during which the animal may double its original weight. Eventually, a plateau is reached (static phase) and the food consumption tapers off to normal. Obese animals can be dieted down to a normal weight, but they again become obese when fed ad lib. (Brobeck, et al., 1943; Brooks & Lambert, 1946; Kennedy, 1950). During the dynamic phase, resection of most of the stomach does not appreciably alter the hyperphagia (Brooks, et al., 1946). Additional lesions of the lateral hypothalamus, however, not only eliminate the hyperphagia but also cause the animal to stop eating altogether (Anand & Brobeck, 1951a).

Once it had been established that animals with ventromedial hypothalamic lesions become obese primarily because they overeat, it became important to discover the cause of the overeating, and this proved to be more difficult. Brobeck has suggested that these lesions disturb some hypothalamic mechanism which normally maintains a balance between energy needs and food intake (1946), but this is not so much an explanation as a description of a

generally recognized phenomenon--hyperphagic animals are deficient in the ability to adjust to changed caloric requirements. Thus, although they are less active than normal animals (Hetherington, 1941; Hetherington & Ranson, 1942a), hyperphagic animals that are in the dynamic phase consume about twice as much food as normals (Tepperman, et al., 1941). Also, hyperphagic animals that are in the static phase (when food consumption is approximately normal), if fed a diet adulterated with some inert substance such as kaolin or cellulose (Kennedy, 1950; Teitelbaum, 1955; Larsson & Ström, 1957) or subjected to low temperatures (Fregly, Marshall, & Mayer, 1957), do not compensate for the decreased calories or the increased need for heat production by increasing their food intake as normals do under these conditions. It should be noted, however, that the failure of obese hypothalamic hyperphagic animals to eat more of a cellulose adulterated diet can also be explained on the grounds that hyperphagic animals are more responsive than normals to alterations in the taste and texture of food; this will be discussed in more detail later.

The overeating which follows ventromedial hypothalamic lesions has also been attributed to a change in emotionality. Wheatley pointed out that lesions in this area make animals savage (1944); this prompted Brooks, Lockwood, and Wiggins to suggest in 1946 that the voraciousness of hypothalamic

hyperphagic animals was actually a substitute for attack, but the explanation does not seem to have been generally accepted. It does not account for the overeating which results from lesions in other areas such as the caudal hypothalamus and caudal thalamus where taming effects have been noted (Hetherington & Ranson, 1942a; Patton, Ruch, & Walker, 1944; Brobeck, 1946), and this objection was cited by Brooks a year later when he repudiated his own earlier position (1947).

Perhaps the most popular explanation is that hyperphagia is a "release phenomenon." Hyperphagic animals overeat not because they are "hungrier" but because, lacking a "satiety" mechanism, they are unable to stop eating once they have started (Kennedy, 1950; Miller, Bailey,& Stevenson, 1950; Anand & Brobeck, 1951b; Teitelbaum, 1955). How this satiety mechanism operates in normal animals is not generally agreed upon. According to Brobeck, the ingestion of food causes an almost immediate increase in heat production which in turn causes thermal receptors in the hypothalamus to inhibit eating (1957). Mayer, Vitale, and Bates (1951) and Kennedy (1953) have suggested that the control of food intake depends on chemoreceptors situated in the hypothalamus which are sensitive to changes in blood composition. Whatever the mechanism involved, the evidence for a satiety center derives from a number of sources, and

these will be considered separately.

One line of evidence concerns the difference between the eating patterns of normal and hypothalamic hyperphagic animals. Increased consumption by hyperphagic animals is reflected mainly in meals which are larger than normal (Brooks, Lockwood, & Wiggins, 1946; Larsson & Strom, 1957; Teitelbaum & Campbell, 1958). On a solid or liquid diet, hyperphagic animals, unlike normals, consume as much during the day as at night (Brooks, 1946; Anliker & Mayer, 1956; Teitelbaum & Campbell, 1958), and on a diet of wet mash hyperphagic animals eat most just after the food has been changed regardless of the hour (Brobeck, Tepperman, & Long, 1943; Brooks, et al., 1946).

A second line of evidence is related to the dissociation between two measures of food drive in hypothalamic hyperphagic animals. These animals do not work as hard as normals on food-motivated tasks, but they overeat when fed ad lib. (Miller, et al., 1950; Teitelbaum, 1957; Ingram, 1958), and these findings have been interpreted to mean that ventromedial hypothalamic lesions lower food drive but impair the mechanism which inhibits eating. The degree of the performance deficit is a function of the amount of work involved. In an experiment in which food was obtained by pushing the lid off a box, hyperphagic animals ate more than normals when the lid was unweighted but less than normals

when the lid was weighted (Miller, et al., 1950). In another experiment in which the total daily food supply was obtained by bar pressing during a 12-hour period in a Skinner box, hyperphagic animals pressed more and obtained more food than normals when the ratio of non-reinforced to reinforced responses was low, but they pressed less and obtained less food when the ratio was high (Teitelbaum, 1957).

The third line of evidence concerns changes in food preferences shown by animals with ventromedial hypothalamic lesions. According to Teitelbaum, the "release" brought about by these lesions consists of an over-reactivity to certain positive and negative stimulus characteristics of the diet (1955). On the positive side are the observations of Hetherington and Ranson (1942a) that hyperphagic animals gained more on a soft diet than on pellets; those of Miller, et al., (1950), that a high fat synthetic diet significantly increased the food intake of operated rats which had not appeared to be hyperphagic on a diet of dry chow; and those of Teitelbaum (1955), that the addition of dextrose to a powdered diet significantly increased the food intake of obese hyperphagic animals. On the negative side, the addition of quinine to the diet in an amount which did not disturb normal animals resulted in a significant decrease in food intake by obese hyperphagic rats (Miller, et al., 1950; Teitelbaum, 1955). The rejection by obese hyperphagic animals of diets adulterated with cellulose or kaolin can also be explained

on this basis, as noted earlier.

An important point raised by both Kennedy (1950) and Teitelbaum (1955) is that hyperphagic animals which have not yet become obese are not as responsive as obese animals to changes in the taste and texture of food. Kennedy found that the addition of kaolin to the diet was readily compensated for by recent operates but not by rats that had already become obese. Teitelbaum found that, whereas both groups overreacted to cellulose adulteration, the decrease in food intake was more extreme in the obese group. Similarly, the addition of quinine to the diet in an amount which caused the obese group to limit its intake severely had as little effect on the non-obese as on the normal group. Both Kennedy and Teitelbaum related these findings to the release concept in the following manner: While ventromedial hypothalamic lesions make animals over-responsive to food, in the dynamic phase it is the positive qualities which are more important in determining the response while in the obese phase it is the negative qualities which are predominant. According to Teitelbaum, the change occurs because the laying down of fat deposits alters the internal environment, a possibility that is indirectly supported by Fuller and Jacoby's finding that the responses to dietary changes of genetically obese mice with no hypothalamic dysfunction resemble those of obese hyperphagic animals (1955).

We turn now to a group of experiments in which hypothalamic lesions produced a totally different effect on eating. Injury to the lateral hypothalamus at the same rostro-caudal level as the ventromedial nucleus has been found to result in a condition known as "aphagia", which is characterized by refusal to eat and loss of weight. In many cases death by starvation occurs, although recovery is possible if the animals are tube-fed during the period (which may be several months) when they refuse to eat spontaneously (Teitelbaum & Stellar, 1954; Morrison & Mayer, 1957a). Large lesions which encompass both the ventromedial nucleus and lateral area result in aphagia and, similarly, lateral lesions in animals previously made hyperphagic by ventromedial lesions result in aphagia (Anand & Brobeck: 1951a; 1951b).

Aphagia, like hyperphagia, has been demonstrated in a variety of animal species--rats and cats (Anand & Brobeck: 1951a; 1951b; Teitelbaum & Stellar, 1954), monkeys (Anand, Dua, & Shoenberg, 1955), and chickens (Feldman, Larsson, Dimick, & Lepkovsky, 1957). According to some authorities, the effective area within the lateral hypothalamus is extremely limited. Anand and Brobeck found that complete rejection of food resulting in death followed lesions of only a circumscribed area in the lateral hypothalamus; deviations by 1 mm. or less in any direction produced only temporary aphagia

(1951b). Morrison and Mayer further showed that merely lowering an electrode at the same coordinates used by Anand and Brobeck did enough damage to produce temporary aphagia (1957b). This makes it difficult to account for Montemurro and Stevenson's failure to affect food intake with small lesions in roughly the same area (1955).

Anand and Brobeck have proposed a dual-center theory to account for the effects of medial and lateral hypothalamic lesions at the tuberal level. They suggest that an "excitatory" or "feeding" center is located in the lateral hypothalamus which initiates the feeding response when the animal is stimulated by the sight or smell of food. A corresponding "inhibitory" or "satiety" center, located in the ventromedial nucleus, stops the response when the animal has satisfied its physiological need for food (Anand & Brobeck: 1951 a; 1951b; see also Anand, Dua, & Shoenberg, 1955). The theory accounts for some but not all of the data. Its strongest support derives from the finding that stimulation and ablation of the ventromedial nucleus and of the lateral hypothalamic area have reciprocal effects. Thus, stimulation of the inhibitory center results in a decrease in food consumption (Smith, 1956; Olds, 1958) but removal of the inhibitory center results in an increase (Hetherington & Ranson, 1940). Similarly, stimulation of the excitatory center increases food intake (Delgado & Anand, 1953;

Larsson, 1954; Smith, 1956; Olds, 1958), but its ablation causes cessation of eating (Anand and Brobeck: 1951a; 1951b). It has been further shown that the eating elicited by stimulation of the lateral hypothalamus is "motivated" and not merely reflexive. Satiated animals, so stimulated, will perform a variety of instrumental responses to get food. Coons and Miller trained rats to push back a hinged door and to bar press (unpublished experiment, cited by Miller, 1957) and Wyrwicka, Dobrzecka, and Tarnecki trained goats to place the foreleg on a food tray (1959). The data which do not fit so readily into Anand and Brobeck's scheme are derived from two sources: Teitelbaum's work on aphagia and experiments implicating other parts of the hypothalamus in the regulation of food intake.

Teitelbaum's experiments have shown that the cessation of eating produced by lateral hypothalamic lesions is not an all-or-none phenomenon. Recovery was hastened in rats and cats which were offered preferred foods like milk or chocolate (Teitelbaum & Stellar, 1954). Furthermore, rats offered a liquid diet immediately after operation ate normally and maintained their weight level, although they starved to death if offered an ordinary laboratory diet. Even more surprising was the finding that "recovered" aphagic animals fed an ordinary diet and also the ones fed a liquid diet became obese (Williams & Teitelbaum, 1959). Instead of being separate disorders, hyperphagia and aphagia may actually re-

present different degrees or different stages of the same disorder, an exaggerated responsiveness to the taste and texture of food. In the dynamic phase, hyperphagic animals eat most foods readily, but as they gain weight they resemble aphagic animals in that they become more fussy and refuse foods accepted by normal animals. On the other hand, aphagic animals refuse virtually all foods at first; there is an intervening period when they accept preferred foods, and later they overeat and become obese on an ordinary laboratory diet. The fundamental similarity between hyperphagia and aphagia is most easily demonstrated by the reversal of symptoms which occurs when the ordinary diet is changed. Obese hyperphagic animals lose weight when fed a quinine-adulterated diet and aphagic animals which are starving eat and become obese when fed a liquid diet.

Whether this similarity between hyperphagic and aphagic animals extends to other phenomena such as activity level and performance on food-motivated tasks is not known at present, but this is one line of investigation which might profitably be followed. Also, in view of Teitelbaum's theory that the fussy eating habits of obese hyperphagic animals are related to the accumulation of fat, it would be useful to know something about the reactions of "recovered" obese aphagic animals to food adulterated with quinine or cellu-

lose. The nature of the taste changes which occur in newly operated aphagic animals also should be clarified. The fact that animals with lateral hypothalamic lesions will die of starvation although literally surrounded by food raises the possibility that the food has not merely lost its stimulus value but has acquired aversive properties.

The other and perhaps less serious criticism of Anand and Brobeck's theory is that the region of the hypothalamus involved in the regulation of food intake may be more extensive than these experimenters believed. The present discussion has so far been confined to experiments dealing with injuries or stimulation at the tuberal level, but there are a few experiments which implicate other parts of the hypothalamus in feeding behavior, and these will now be described briefly.

Lesions placed rostral to the ventromedial hypothalamic nucleus in rats, cats, and monkeys were found to have no effects on food intake (Hetherington, 1944; Anand & Brobeck, 1951b; Anand, Dua, & Shoenberg, 1955) but, in dogs, obesity resulted from anterior hypothalamic lesions which destroyed the paraventricular nuclei (Biggart & Alexander, 1939; Heinbecker & White, 1942; Heinbecker, White, & Rolf, 1944). With respect to lesions placed in the caudal hypothalamus, the evidence is confusing. There are several studies in which lesions in the region of the mammillary bodies produced aphagia

(Ingram, Barris, & Ranson, 1936; Ranson, 1939; Clark, Magoun, & Ranson, 1939). Hetherington and Ranson produced obesity in rats by ablating a part of the lateral hypothalamus dorsolateral to the mammillary bodies, and they attributed their results to destruction of fibers from the ventromedial nucleus which pass through this region on the way to the midbrain (1942c). In other studies, caudal hypothalamic lesions in rats and cats (Anand & Brobeck, 1951b) and in cats and monkeys (Anand, et al., 1955) produced no change in food intake. To confuse the issue further, in one study in which caudal hypothalamic lesions eliminated spontaneous eating, it was reported that the animals chewed and swallowed voraciously if food was forced into their mouths (Ingram, et al., 1936).

Recent studies utilizing the stimulation technique have also implicated parts of the hypothalamus not previously believed to be involved in the regulation of food intake. Cats stimulated in the dorsal hypothalamic nucleus or in the paraventricular hypothalamic nucleus performed a learned response to obtain food (Grastyán, Lissák, & Kékesi, 1956). Also, eating was induced by stimulating the mammillary bodies, the premammillary region (Ruch, Maire, & Patton, 1956), and the area caudo-lateral to the mammillary bodies (Larsson, 1954). Some of these data are of doubtful reliability because no histological verification of placements has been presented. It is also possible that some of the results can be "explained

away" on the basis of current spread to the "feeding" or "satiety" centers. Nevertheless, until the issue is resolved by further research, the possibility that other parts of the hypothalamus function in the regulation of food intake cannot be entirely ignored.

Effects of Extra-Hypothalamic Lesions on Hunger

In this section, we shall discuss only those instances of increased intake following brain lesions which bear some resemblance to hypothalamic hyperphagia and omit those which occur in conjunction with increased energy requirements, as in the case of frontal lobe lesions (Richter & Hawkes, 1939). Also omitted are the few reports of aphagia following extra-hypothalamic lesions (Bailey & Davis, 1942; Kennard, 1955), since the behavioral descriptions suggest that motor or intellectual deficits were responsible for the feeding change.

It should be noted that a great deal of the material which follows was derived from experiments in which increased food intake was reported only incidentally, and consequently, in many cases, systematic records of food intake and of weight gain were not presented. This admittedly limits the conclusions which can be drawn about the effects of lesions in any one area, but, taken as a group, these experiments are important because they call attention to a topic which has not yet been adequately explored, the part played by

areas other than the hypothalamus in the regulation of food intake.

Discrete lesions in several thalamic nuclei have been reported to increase food intake. Schreiner, Rioch, Pechtel, and Masserman (1953) found that destruction of the dorsomedial thalamic nucleus resulted in a syndrome which was virtually identical to the one which follows ventromedial hypothalamic injury. Cats subjected to this operation were found to be hyerphagic, irritable, and underactive. Also, as was the case with ventromedial hypothalamic animals, there seemed to be a relation between eating and emotionality. When the cats were subjected to a mild stress, such as having their tails pinched, they gulped food. "It appeared as though the animals released their savage attacks upon the meat rather than turning to attack the offending stimulus as would be done, in most instances, by unoperated cats" (Schreiner, et al., 1953). That increased eating is caused by posterior thalamic lesions extending into the rostral mesencephalic tegmentum was noted incidentally by Ruch and his co-workers in the course of a series of experiments aimed at establishing the arcuate nucleus as the thalamic relay for taste. A few operate monkeys that showed only a mild and transient deficit in the ability to discriminate between quinine solutions and water were both hyperphagic and tame. Caloric intake was tripled, and these animals ultimately became obese (Ruch,

Blum, & Brobeck, 1941; Ruch, Patton, & Brobeck, 1942; Patton, Ruch, & Walker, 1944). The parallel between hypothalamic and thalamic lesions with respect to emotional changes is interesting; medial lesions in these structures produce hyperphagia and savageness (Wheatley, 1944; Schreiner, et al., 1953), while posterior lesions produce hyperphagia and tameness (Hetherington & Ranson, 1942a; Patton, et al., 1944).

Hyperphagia has also been reported in one monkey (Brown & Shäfer, 1888) and in one human patient (Terzian & Ore, 1955) following bilateral temporal lobectomy. The monkey was observed to devour different kinds of food indiscriminately, and a similar effect was noted in the human case.

> Some days after the operation the patient demonstrated an insatiable appetite and ate at least as much as four normal persons. He prepared himself for meals as for a ceremony. He would personally go to the kitchen and insistently ask for food at any hour. He would look for a secluded corner far from anyone, eat everything voraciously without preference for any certain food, lick the dish incessantly, and after 15 minutes asked for more food (Terzian & Ore, 1955).

Hyperphagia also results from more restricted damage to the temporal lobe. Ablation of the entorhinal area in the phalanger, an Australian marsupial, produced a temporary change. Although these animals are normally nocturnal, after operation they ate ravenously at any time of day, but the effect disappeared within about two weeks (Adey, Merrillees, & Sunderland, 1956).

Whether damage to the nearby amygdaloid area also produces hyperphagia is still disputed, although it has been reported not only following lesions in which the amygdala was damaged along with other temporal lobe structures, but also following lesions restricted to the amygdala. The effect has been produced in several animal species--monkeys (Brown & Schäfer, 1888), cats (Morgane & Kosman, 1957; Green, Clemente, & de Groot, 1957; Wood, 1958), and a baboon (Adey, 1958). However, with dogs only a temporary increase in eating was noted (Fuller, Rosvold, & Pribram, 1957), and, in one experiment no change was observed in rats and cats (Anand & Brobeck, 1952). There is also one instance in which amygdalectomized cats were found to be aphagic for periods ranging from five to twenty days (Schreiner & Kling, 1953). The failure of various experimenters to agree on how amygdalectomy affects food intake is reminiscent of the long-standing dispute concerning the effects of the same lesions on emotionality, and the possibility should be investigated that, in both cases, the discrepancy rests on localization within the amygdaloid complex.

The possibility that the same extra-hypothalamic lesions which increase food intake might also affect food-motivated behavior has not received much attention thus far. There are two instances in which amygdalectomy and cingulectomy reportedly lowered food motivation, but, unfortunately, in-

sufficient details are given to enable the reader to evaluate these reports (Masserman, Levitt, McAvoy, Kling, & Pechtel, 1958; Pechtel, McAvoy, Levitt, Kling, & Masserman, 1958).

The "Feeding System"

In 1921, when Bailey and Bremer described the effects of hypothalamic lesions on food intake, little was known about this structure, and they were careful to specify that it was a "region known to anatomists as the hypothalamus." They enthusiastically predicted that "...the time is not far distant when the neuropathologist will no more think of omitting to examine the hypothalamus than he would the motor cortex" (1921). Their prediction was accurate, and currently the pendulum has swung so far the other way that the hypothalamus has been credited with containing an excitatory and an inhibitory center for each type of motivation (Stellar, 1954). Some years ago, Teuber questioned the usefulness of such theorizing and commented on the "amazing burden of functions" attributed to the hypothalamus, a structure which he noted weighs four to five grams out of an average total brain weight of 1360 grams (1955).

The studies reviewed in the preceding section demonstrate that changes in food intake occur as a result of

ablating other parts of the brain, and it would seem, therefore, that the hypothalamus is only one part of a complex system involved in the regulation of food intake. Parts of the thalamus and temporal lobe are also involved, and there may be other areas whose importance has not yet been demonstrated. It should be noted that numerous connections exist between the hypothalamus, on the one hand, and the temporal lobe and thalamus, on the other. The amygdala, in particular, has direct connections with the ventromedial hypothalamic nucleus (Adey & Meyer, 1952). However, whether these areas participate in the regulation of food intake by virtue of their anatomical relations with the hypothalamus or whether they function independently is not clear on the basis of the available evidence. Some connections with the hypothalamus can be severed without altering food intake. For example, Hetherington and Ranson found that lesions involving the mammillothalamic tract or fornix produced no change (1942c), and Schreiner, Rioch, Pechtel, and Masserman reported similar negative findings following lesions of the anterior or intralaminar nuclei of the thalamus (1953). Far from ruling out the possibility that control is mediated via the hypothalamus, these data may only testify to the complexity of the circuits involved in the regulation of food intake. It is quite likely that the severity and persistence of the change produced by ablating any one part of the "feeding system" depend on the number of other pathways available.

THE PRESENT INVESTIGATION

The experiments to be reported grew out of a chance observation that some animals with lesions in the reticular formation of the midbrain (the tegmentum) were consuming unusually large quantities of food. With the view that the tegmentum might be part of the hypothetical "feeding system" already described, preliminary experiments were carried out in which tegmental operates were compared with normals with respect to weight gain and performance on a food-motivated task. Since the subsequent experiments were based on these preliminary findings, some of the data will be reviewed briefly.

Most of the experimental animals lost weight during the first few days after operation, when they were apparently both aphagic and adipsic, but the preoperative weight level was rapidly recovered when eating and drinking were resumed. In some animals that had lost a great deal of weight there was a brief spurt of overeating, but no animal became obese. It is interesting that Anand and Brobeck reported similar results in animals subjected to amygdalectomy (1952). Some of the tegmental operates that were aphagic were found to eat more readily if offered wet mash instead of pellets, and this procedure was followed in all subsequent experiments. Figure 1 shows the weight gain of 13 animals with

tegmental lesions and of 4 normal animals during an 18-day postoperative period when wet mash was always available. The tegmental group was lighter than the control group before operation, and the difference was maintained throughout the postoperative period. However, the rate of weight gain was approximately the same for both groups.

As a means of assessing the effects of tegmental lesions on food-motivated behavior, an attempt was made to teach nine of the tegmental operates to bar press in a Skinner box. Only one of the nine animals acquired the habit after four daily 20-minute sessions, so thereafter animals were trained to bar press before operation. Figure 2 is a graph showing the performance of five tegmental operates and of four normal animals trained preoperatively on a continuous reinforcement schedule and tested postoperatively on a fixed interval reinforcement schedule with reinforcement obtainable only once every five minutes. The groups were tested at 0, 24, 48, and 72 hours of food deprivation. This performance task was deliberately chosen to provide data comparable to those reported by Miller, et al., (1950), and it was found that the tegmental group showed the same sort of deficit demonstrated by these investigators in animals with ventromedial hypothalamic lesions. With continued food deprivation, the normal group increased its bar-pressing rate steadily, but the tegmental group remained at the same level throughout.

The preliminary data offered no support for the hypothesis that the tegmentum is one of the areas involved in the regulation of food intake, for the lesions produced no lasting change in the rate of weight gain. However, the results also showed that tegmental lesions alter food motivation and, therefore, two experiments were designed to explore further this phenomenon. In order to demonstrate that the effects obtained with tegmental lesions do not accompany every type of subcortical brain damage, two operate groups were used. In one group lesions were made in the tegmentum, and in the other group lesions were made in the area of the fornix and hippocampal commissure.

Since the preliminary work had shown that increasing the deprivation period did not alter the performance of the tegmental group, this factor was held constant and, in the first experiment, the effects of varying the amount of work involved in securing food were investigated. Work was varied in the Skinner box by increasing at each test session the number of bar presses necessary to secure a pellet of food. An additional test of food motivation, which consisted of running down a four-foot runway to food, was also employed. Since the four daily trials in the runway were completed in a minute or less (while bar pressing tests lasted 20 minutes), it seemed reasonable to assume that the runway involved less expenditure of effort than bar pressing. A measure was also

obtained of the amount of food eaten by hungry animals during a period of free access to wet mash.

The second experiment was designed to investigate the possibility that motivation to perform for water is affected by tegmental lesions. Work was varied in the Skinner box as in the previous experiment, and a measure was also obtained of the amount of water drunk by thirsty animals during a period of free access to water.

EXPERIMENT I: EFFECTS OF TEGMENTAL LESIONS ON FOOD-MOTIVATED BEHAVIOR

The subjects were 50 male hooded rats obtained from the Royal Victoria Hospital colony. They weighed between 200 and 230 grams at the start of the experiment and were housed two to a cage in standard laboratory cages measuring 10 by 12 by 9 inches.

The two pieces of apparatus used in this experiment were an enclosed runway and a Skinner box designed to deliver either food or water. The Skinner box was made of 1/2-inch unpainted pine and measured 10 1/2 by 10 by 6 inches. Three of its walls were made of wood, and the fourth wall and the floor were made of wire mesh. The bar was mounted 2 inches above the floor and could be depressed by a force of approximately 20 grams. The runway, which was made of 1/2-inch pine painted a flat grey, was 4 feet long, 4 inches wide, and 6 inches high; it contained no doors.

Procedure

During all tests involving hunger motivation, the animals were on a 24-hour deprivation schedule, and a period of one week was allowed for adjustment to this schedule before both preoperative and postoperative testing. Bar-pressing tests lasted 20 minutes, and each reinforced press de-

livered a 45-mg. food pellet. Except where noted, all tests were followed by a 20-minute feeding period. The diet consisted of wet mash (ground Purina Fox Chow mixed with water) when the animals were on feeding schedule and of Purina Fox Chow pellets when they were fed ad lib., except during the first 10 days after operation when they were fed wet mash. Water was always available in the home cage.

Before operation, hungry rats were trained to bar press for food in the Skinner box on a continuous reinforcement schedule. At the end of the training period, a preoperative bar-pressing score, representing the average number of bar presses per session after the habit had been acquired, was computed on the basis of performance during the last four of the seven training sessions. Animals with scores below 100 were eliminated, and the rest were divided into three groups with approximately equal means. Nineteen animals in the tegmental group, 16 in the normal control group, and 15 in the operate control group (with lesions in the area of the fornix and hippocampal commissure) survived the operation and completed all tests. The preoperative training period was followed by a three-day period during which the animals were fed ad lib., and then the operations were performed.

With the animals under Nembutal anesthesia, the skull was exposed and bilateral drill holes were made at the appropriate points. The location of the drill holes was determined by using two skull sutures, bregma and lambda, as

reference points. In the experimental group, bilateral lesions were made 6 mm. below the surface, 2 mm. in front of lambda, and 2 mm. lateral to the midline; in the operate control group, bilateral lesions were made 5 mm. below the surface, 2 mm. in front of bregma, and close to the midline. The lesions were made by inserting an insulated wire electrode, bare at the tip, into each drill hole, lowering the electrode to the desired depth, and passing a current of 2 ma. for 30 seconds. The scalp wounds were closed with interrupted silk sutures. The animals were allowed a 10-day recovery period, during which time records were kept of weight gain. At the end of the recovery period, they were again placed on a feeding schedule.

A four-day retest in the Skinner box on a continuous reinforcement schedule was begun 17 days after the operations. This served the purpose of retraining the animals and also provided a postoperative bar-pressing score comparable to the preoperative score. During the next six days, the animals were tested on a fixed ratio schedule which was increased day by day to increase the amount of work involved in securing a pellet of food. The following ratios were used: 3, 6, 12, 36, 72, and 144. An additional two-day bar-pressing test on a continuous reinforcement schedule was given about two months after operation in order to check on the permanence of the changes that were found.

In the runway, four trials per day were given for 10 days, and speed of running was recorded. A trial began when the animal was placed at that end of the runway which was farthest from the food (wet mash), and the trial ended when the animal reached the food dish. The animal was then allowed to remain at the goal for 10 seconds, after which it was placed at the start and another trial was begun.

To measure the amount of food consumed by hungry animals during a period of free access to food, each rat was placed in a feeding cage containing wet mash for one hour per day on three successive days. The difference between the animal's weight at the beginning and end of the hour was taken as an estimate of food eaten. No water was available during the test.

At the conclusion of the experiment, the operate rats were killed with ether and perfused with physiological saline followed by a 10% formalin solution. The brains were removed and placed in formalin for three days and then in 20% alcohol for at least one week. Frozen sections were cut at a thickness of 40 micra. In the region of the lesions, every fifth section was saved, mounted, and stained with cresyl violet.

Results

The data on weight gain during the 10-day recovery period resembled those obtained in the preliminary experiment. Both

operate groups lost weight for a few days after operation, and, although they remained lighter than the normal group, they gained at the same rate when eating was resumed.

Table I shows the means and standard deviations for the preoperative and postoperative bar-pressing scores on a continuous reinforcement schedule. Also shown in Table I is the mean postoperative change in each group. This measure was obtained by subtracting each animal's preoperative score from its postoperative score and then computing the group means. Comparing the groups with respect to degree of postoperative change revealed that the operations had significantly lowered the bar-pressing rates of both the tegmental ($p<.002$) and fornix groups ($p<.02$) by comparison with the normal group. (These, and the other probabilities reported here, were evaluated by means of the Mann-Whitney U Test.) Figure 3 shows the performance of the three groups on each of the four days of the postoperative bar-pressing test on a continuous reinforcement schedule. Although both operate groups showed a deficit in bar pressing, the fornix group improved with practice, so that by the fourth day it had reached a level which was very close to its preoperative mean. The tegmental group, on the other hand, showed no improvement over the four-day period.

Increasing the amount of work involved in securing food

in the Skinner box had markedly different effects on the two operate groups; as may be seen in Figure 4, the fornix group pressed at a much higher rate and the tegmental at a much lower rate than the normal group. For the six days on increasing ratio, the group means (based on total scores) were as follows: 4688 for the fornix group, 2580 for the normal group, and 1393 for the tegmental group. These means differ from one another at better than the .002 level of probability. Separate comparisons between the groups were also made for each fixed ratio value, and these p-values are shown in Table 2. Almost all comparisons yielded significant differences except at the very low and very high ratios. The extremely high rate of bar pressing reached by the fornix group is noteworthy. One animal pressed 2149 times in a single session when the ratio of non-reinforced to reinforced bar presses was 144, a rate which is comparable to that of animals bar pressing for electrical stimulation of the brain.

In Table 4 are shown the group means for the two-day bar-pressing retest on a continuous reinforcement schedule which was given about two months after operation. The tegmental group bar pressed significantly less than the normal group ($p < .02$), showing that the deficit found after operation was relatively long-lasting. The difference between the normal and fornix groups was not significant, and this

is consistent with the rapid improvement shown by the fornix group during the first four days of postoperative testing.

For each of the 10 days of the runway test, an individual animal's score was computed by averaging its four trials. In Figure 5, the daily means for each group are plotted. Since food motivation rather than speed of learning was the issue, the groups were compared only after their performance had reached an asymptote. Mean running times based on the combined scores of the last four days were as follows: 8.3 seconds for the fornix group, 13.2 seconds for the tegmental group, and 20.0 seconds for the normal group. All three groups differed significantly from one another at the .02 level of probability or better. The paradoxically superior performance of the tegmental group in this situation as compared with the Skinner box is interesting, for it suggests that the amount of work involved in obtaining food is a crucial variable. Tegmental animals are capable of performing better than normals on a short, simple task, but they are inferior to normals when sustained effort over a longer period is required.

Table 4 shows the mean weight gained by each group on each of the three test days during a period of free access to food. It will be noted that these values changed very little on successive days. The results of this test are congruent with those already described. When no work is re-

quired, animals with tegmental lesions do not differ from normals in amount of food consumed. Those with fornix lesions, on the other hand, not only work harder than the other groups, but they also eat more; for the three days combined, they differed significantly from normals at the .02 level and from tegmental operates at the .05 level of probability.

The performance data obtained in Experiment I can be summed up as follows: Rats with tegmental lesions are less motivated than normals to work for food in a situation involving sustained effort, work harder than normals on a short, simple task, and consume approximately the same amount of food when no work is required. On the other hand, rats with fornix lesions consume more food and are more motivated to work for food than normals.

EXPERIMENT II: EFFECTS OF TEGMENTAL LESIONS ON WATER-MOTIVATED BEHAVIOR

The subjects were 48 hooded rats divided into the following groups: 19 tegmental, 15 normal control, and 14 operate control with lesions in the area of the fornix and hippocampal commissure. The apparatus consisted of the same Skinner box used previously. The procedure was similar to that of Experiment I, but the following changes should be noted:

A period of four days was allowed for adjustment to the water deprivation schedule before both preoperative and postoperative testing. The preoperative training period lasted eight days. During the 10-day recovery period, all animals were offered pellets and mash for the first six days and only pellets for the remaining four days. Only three tests were used--bar pressing on a continuous reinforcement schedule before and after operation, postoperative bar pressing on an increasing ratio schedule similar to that used in Experiment I (except that it was terminated at 72), and amount of water drunk postoperatively during a period of free access to water. Bar-pressing tests lasted 10 minutes per day and were followed by a 40-minute period during which the animal had access to both water and pellets. The apparatus was adjusted to deliver .05 cc. per reinforced bar press. To obtain a

measure of the amount of water drunk by thirsty animals during a period of free access to water, each animal was placed in a cage containing pellets and a full water bottle for one hour per day on three successive days, and the loss in weight of the bottle was determined.

Results

Table 5 shows the means and standard deviations for the preoperative and postoperative bar-pressing scores of the three groups on a continuous reinforcement schedule. Degree of postoperative change was computed as in the previous experiment, and these means are also shown in Table 5. Comparison of the groups with respect to degree of postoperative change revealed that the bar-pressing rate of the tegmental group was significantly lowered by comparison with each of the other groups ($p<.002$). The difference between the fornix and normal groups was not significant.

As shown in Figure 6, increasing the amount of work involved in securing a drop of water produced a much greater rise in bar pressing in the normal and fornix groups than in the tegmental group. For the five days on increasing ratio, the group means (based on total scores) were as follows: 1499 for the normal group, 1393 for the fornix group, and 643 for the tegmental group. The tegmental group differed from the

other two groups at less than the .002 level of probability, but there was no significant difference between the fornix and normal groups.

When the amount of water drunk in one hour was measured on three successive days, it was found that animals with fornix lesions drank significantly more than animals in the other two groups ($p<.02$). The difference between the tegmental and normal groups was not significant. The group means for each of the three test days are shown in Table 6.

HISTOLOGY

Except for slight variations, the lesions of the experimental group used in Experiment I began 0.5 mm. caudal to the level of the posterior commissure and ended at the level which is 0.5 mm. rostral to the decussation of the brachium conjunctivum. The average lesion size rostro-caudally was 1.0 mm. The location of the maximum cross-section was 1.0 mm. or 0.5 mm. rostral to the level of the decussation of the brachium conjunctivum. The control lesions began in the septal area, 0.5 mm. or 1.0 mm. in front of the level of the anterior commissure, and ended at the level where the hippocampal commissure ends and the hippocampus begins. The average lesion size rostro-caudally was 1.8 mm. which was significantly larger than the size of the tegmental lesions ($p<.001$). The control lesions were largest at, or 0.5 mm. behind, the level of the anterior commissure. Figures 7 and 8 show some reconstructions at the levels where the lesions were largest.

In Experiment II, the lesions of both groups were slightly caudal to those in Experiment I; and, in the tegmental group, they were more medially placed than previously. The tegmental lesions began 1.0 mm. or 1.5 mm. behind the level of the posterior commissure and ended 0.5 mm. caudal to the level of the decussation of the brachium conjunctivum. The average lesion size rostro-caudally was 1.3 mm. Maximum destruction

was at the level of the decussation of the brachium conjunctivum. Most of the control lesions began in the septal area, 0.5 mm. or 1.0 mm. in front of the level of the anterior commissure; a few began at the commissural level. Most control lesions ended at the level of the hippocampal commisure and two extended as far back as the level of the anterior hippocampus. The average lesion size rostro-caudally was 1.5 mm., which did not differ significantly from the tegmental lesion size. The control lesions were largest at, or 0.5 mm. behind, the level of the anterior commissure. Figures 9 and 10 show reconstructions of some of these lesions.

The data from Experiment I were used to see whether, within each of the two operate groups, the animals having very high or very low bar-pressing rates could be distinguished from each other on the basis of specific structures destroyed, symmetry of lesions, or preoperative bar-pressing performance. In the control group, there was a tendency for high scorers (those most affected by the operations) to have larger lesions than low scorers. In the experimental group, these comparisons yielded uniformly negative results, and this type of analysis was not attempted with the data of Experiment II.

DISCUSSION

The main finding of the present study is that the rat's motivation to work for food or water is lowered by discrete lesions of the tegmentum, although no change occurs in the actual consummatory behavior and, except for a brief period after operation, there is no change in the rate of weight gain. Some characteristics of the performance decrement are that it is relatively long-lasting, that it is not reduced or eliminated by prolonging the deprivation period, and that it is related to the amount of work involved since, on a task requiring just a short burst of effort, tegmental operates perform better than normals.

There has been relatively little exploration of the effects of brain lesions on food- and water-motivated behavior; hence there are few comparisons that can be made between the present results and those of other investigators. Water intake in rats, cats, dogs, and goats is known to be altered by ablation of discrete areas of the hypothalamus (Strominger, 1947; Stevenson, 1949; Witt, Keller, Batsel, & Lynch, 1952; Teitelbaum & Stellar, 1954; Montemurro & Stevenson, 1955; Andersson & McCann, 1956); and, in the rat, water intake is also altered by lesions in the region of the posterior commissure (Gilbert, 1956). However, no one has studied the effects of such lesions, or of any other brain

lesions for that matter, on water-motivated behavior. With respect to food-motivated behavior, there is a parallel between the effects of tegmental lesions, as demonstrated in the experiments reported here, and the effects of ventromedial hypothalamic lesions, as described by other investigators (Miller, Bailey & Stevenson, 1950; Teitelbaum, 1957; Ingram, 1958). Animals with lesions in either the tegmentum or the ventromedial hypothalamic nucleus do not work as hard as normals to obtain food, even when food deprivation is continued up to 72 hours. However, the effortfulness of the task is a crucial variable, since animals with lesions in either of these areas perform as well as, or better than, normals when relatively little effort is required.

The behavioral deficit demonstrated in the present experiments does not occur simply because the animals have had a brain operation, nor is it the result of an impairment in learning, memory, or wakefulness. That locus, rather than size of lesion, is important is shown by the fact that the operate controls sustained larger lesions but performed significantly better than the experimental animals on all tests. That memory and learning were not impaired by the tegmental lesions is shown by the fact that the rate of bar pressing after operation was beyond the operant level in both experiments and the fact that the runway habit was readily acquired after operation in the

first experiment. Finally, somnolence and gross motor dysfunction are known to follow extensive tegmental lesions (Peterson, Magoun, McCulloch, & Lindsley, 1949; Lindsley, Schreiner, Knowles, & Magoun, 1950), but the relatively small lesions made in the present experiments did not result in such disturbances. A few animals had a tendency to circle when picked up and put down, but they were not the ones that performed most poorly in the Skinner box.

While it has not been demonstrated previously that tegmental lesions alter food-motivated or water-motivated behavior specifically, the present results have some precedents in the literature dealing with stimulation or ablation of the reticular formation. Emotional expression in humans and in animals is reportedly altered by large lesions of the rostral tegmentum (von Economo, 1929, cited by MacLean, 1955; Kelly, Beaton, & Magoun, 1946; Peterson, et al., 1949; Magoun, 1950). Except for von Economo's report of rage and antisocial behavior in humans, the general finding has been that such lesions have a taming effect. There is also a pronounced diminution of voluntary movement which, according to Magoun, is due not to a motor paralysis but to "a paralysis of volition" (1950). Motivational properties have also been ascribed to the reticular formation on anatomical grounds by MacLean (1955), Lindsley (1957), and Brady (1958); all of these authors emphasize the existence of numerous con-

nections between the limbic system, which is known to be involved in avoidance motivation, and the tegmentum. Olds has shown yet another type of relationship between the reticular formation and motivation. Using permanently implanted electrodes in rats, he has shown that various midbrain foci have positive and negative reinforcing properties when electrically stimulated (in Brazier, 1959, pp. 293-303).

Speculation about the way in which the reticular formation might affect emotional expression and motivated behavior is based largely on the finding that this system is capable of exerting a facilitatory effect on neural activity at both the cortical and spinal levels (Magoun, 1950). Lindsley (1951, 1952, 1957) and Hebb (1955) have pointed out that the psychological concepts of "drive," "emotion," and "motivation" all imply the existence of some system within the organism that energizes behavior but does not specifically guide its direction, and they note that the nonspecific arousal properties of the reticular formation qualify it as such an energizer of behavior. Lindsley (1952) and Hebb (1955) have further suggested that there is some optimal level of firing in the reticular formation at which learning and performance are most efficient. The present results can perhaps be explained on this basis. The interruption of a small part of the system which normally activates the cortex might result in a slight lowering of the arousal level. This would

not necessarily produce a performance decrement if the animal were highly motivated. For short periods, the loss of tissue might be compensated for by more energetic activity in remaining cells that are equipotential with the ones destroyed. However, after a time, the overworked cells would become fatigued and a performance deficit would appear. This sort of explanation accounts for the finding that tegmental operates were deficient in bar pressing but not in runway performance.

What is being suggested, then, is that the performance deficit had no special relation to food or to water motivation but would have occurred on any task of sufficient length irrespective of the motivator. There are several ways in which this could be tested. First, the use of EEG and other recording techniques would yield information about arousal level. Second, tests of avoidance and sex motivation would indicate the generality of the performance deficit. Some work has already been done on avoidance motivation, but the issue cannot be regarded as settled. The rats used in Experiment I were tested on a simple avoidance task (not reported here) but tegmental operates did not differ from either of the control groups; and Doty, Beck, and Kooi have reported similar findings in cats with large lesions in the rostral tegmentum (1959). However, in neither of these cases was sustained effort over an extended period of time required.

Let us return now to the similarity between the effects of tegmental and ventromedial hypothalamic lesions. As mentioned earlier, Miller, et al. (1950) and Teitelbaum (1957) attributed the deficit in food-motivated performance shown by animals with ventromedial hypothalamic lesions to lowered food drive, but the deficit can be explained as readily on the basis of lowered arousal level. This could result from destruction of connections between the ventromedial nucleus and the tegmentum. The fact that ventromedial hypothalamic lesions alter food intake has concentrated attention on food-motivated behavior, but there may be no relation between the change in eating and the change in performance. The first can be accounted for on the basis of destruction of part of the system which normally regulates food intake, but the deficit in food-motivated behavior may be a non-specific effect related to lowered arousal level. In this connection, it should be noted that activity level is greatly reduced by ventromedial hypothalamic lesions. The possibility that a deficit can be demonstrated with other motivators has not yet been adequately explored, though some preliminary data of Ingram on cats with ventromedial hypothalamic lesions suggest that avoidance as well as food-motivation is impaired (1958).

Additional support for this interpretation derives from the similarity between tegmental and ventromedial hypothalamic operates with respect to the condition of the skin and fur.

Hetherington and Ranson found that animals with large ventromedial hypothalamic lesions developed skin sores and lost hair (1940). In the present experiments the same effect was noted in all animals of the tegmental group but in not a single animal in the fornix group. According to Brobeck, the obesity of ventromedial hypothalamic animals prevents them from grooming properly and thus causes loss of hair and skin sores (1946). However, this explanation cannot be applied to tegmental operates. It seems more likely that operations that lower the arousal level decrease both motivated behavior and also spontaneous behavior such as grooming.

We turn finally to the effects of the control lesions on food- and water-motivated behavior. That brain lesions could improve performance was a surprising finding, and it is one which merits comment. Food-deprived rats with lesions in the area of the fornix and hippocampal commissure ran faster to obtain food and also bar pressed more than normals for food. However, while they ate more than normals in a situation where food was freely available following 24 hours of deprivation, their rate of weight gain after operation when they were on an ad lib. schedule did not differ from that of normal controls. A possible explanation for these data is that fornix lesions cause an increase in general activity. This would account for the improved performance on food-motivated tasks and also for the increase in food intake without a cor-

responding change in the rate of weight gain. The only serious objection to this explanation is that no significant difference was found between the operate and normal control groups in bar pressing for water. However, the test session was shorter in Experiment II and it is conceivable that this could account for the discrepancy.

The idea that activity level is increased by fornix lesions is supported by an additional finding, not discussed previously. The three groups used in Experiment I were given a single 20-minute bar-pressing test when satiated. Wet mash was available for one hour before testing. A fixed interval schedule with reinforcement obtainable only once every five minutes kept the number of reinforcements constant and at a minimum. The normal and tegmental groups pressed at a low rate; the means were 43.7 and 44.6 respectively, and these did not differ significantly. The fornix group, on the other hand, pressed at a high rate (M=176.2), and the differences between this group and the other two were significant ($p < .02$). These results indicate that the change in motivated behavior brought about by the control lesions was just as striking and of no less interest than the change produced by the experimental lesions.

SUMMARY

The effects of tegmental lesions on food- and water-motivated behavior in rats were investigated in a series of experiments. On tests in which food served as the motivator, it was shown that the amount of work involved is a crucial variable. In bar-pressing tests, where sustained effort was required over a 20-minute period, tegmental operates were inferior to normals; but, in a runway test, where trials were completed in one minute or less, tegmental operates were superior to normals. The bar-pressing decrement was relatively long-lasting and was not reduced or eliminated by food-depriving the animals up to 72 hours. Tegmental operates were also found to bar press less than normals when water served as the motivator. The amount of food or water consumed by tegmental operates did not differ from that of normals, nor did the rate of weight gain, except for a brief period after operation.

No such decrements in food- and water-motivated behavior were found in operate controls that sustained lesions in the area of the fornix and hippocampal commissure. These animals were superior to normals in the bar-pressing and runway tests when food served as the motivator, and they did not differ significantly from normals in bar pressing when water was the motivator. Operate controls ate and drank more than normals but, except for a brief period after operation, their rate of weight gain did not differ from that of normals.

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Table 1

Effect of Operation on Bar Pressing For Food on a Continuous Reinforcement Schedule

	Preoperative		Postoperative		
Group	Mean	S. D.	Mean	S. D.	Mean Change
Tegmental	128.2	19.0	96.7	32.0	-31.5
Fornix	127.4	21.1	111.9	31.5	-15.5
Normal	126.3	12.2	134.8	11.7	8.5[a]

[a] The normal group differs from the tegmental group ($p < .002$) and from the fornix group ($p < .02$).

Table 2

Bar Pressing For Food with Increasing Ratios of Reinforcement: P Values for Significance of Differences Between Group Means

Fixed Ratio

Groups	3	6	12	36	72	144	Total
Tegmental & Fornix	.002	.002	.002	.002	.002	.002	.002
Tegmental & Normal	.002	.002	.002	.02	-	-	.002
Fornix & Normal	-	.05	.002	.002	.002	.002	.002

Table 3

Bar Pressing For Food on a Continuous Reinforcement Schedule During Two-Day Retest Two Months After Operation

	Mean Number Bar Presses	
Group	Day 1	Day 2
Tegmental	93.1[a]	109.0[b]
Fornix	116.1	137.1
Normal	123.2	142.0

[a]The tegmental group differs from the other two groups ($p < .02$).

[b]The tegmental group differs from the fornix group ($p < .05$) and from the normal group ($p < .02$).

Table 4

Weight Gained (Grams) During Period of Free Access to Food

	Days			
Group	1	2	3	Mean
Normal	26.3	27.5	29.0	27.6
Tegmental	29.0	32.1	31.6	30.9
Fornix	35.6	34.5	34.8	34.9[a]

[a]The fornix group differs from the normal group ($p < .02$) and from the tegmental group ($p < .05$).

Table 5

Effect of Operation on Bar Pressing for Water
on a Continuous Reinforcement Schedule

	Preoperative		Postoperative		
Group	Mean	S. D.	Mean	S. D.	Mean Change
Tegmental	145.8	31.6	106.0	38.3	-39.7[a]
Fornix	151.2	29.0	169.1	29.3	17.8
Normal	149.1	24.7	164.8	26.1	15.6

[a]The tegmental group differs from the other two groups ($p<.002$).

Table 6

Amount of Water Drunk (Grams) During
Period of Free Access to Water

	Days			
Group	1	2	3	Mean
Normal	19.5	19.5	19.5	19.5
Tegmental	18.2	19.8	19.4	19.1
Fornix	22.6	22.2	25.1	23.2[a]

[a]The fornix group differs from the other groups ($p < .02$).

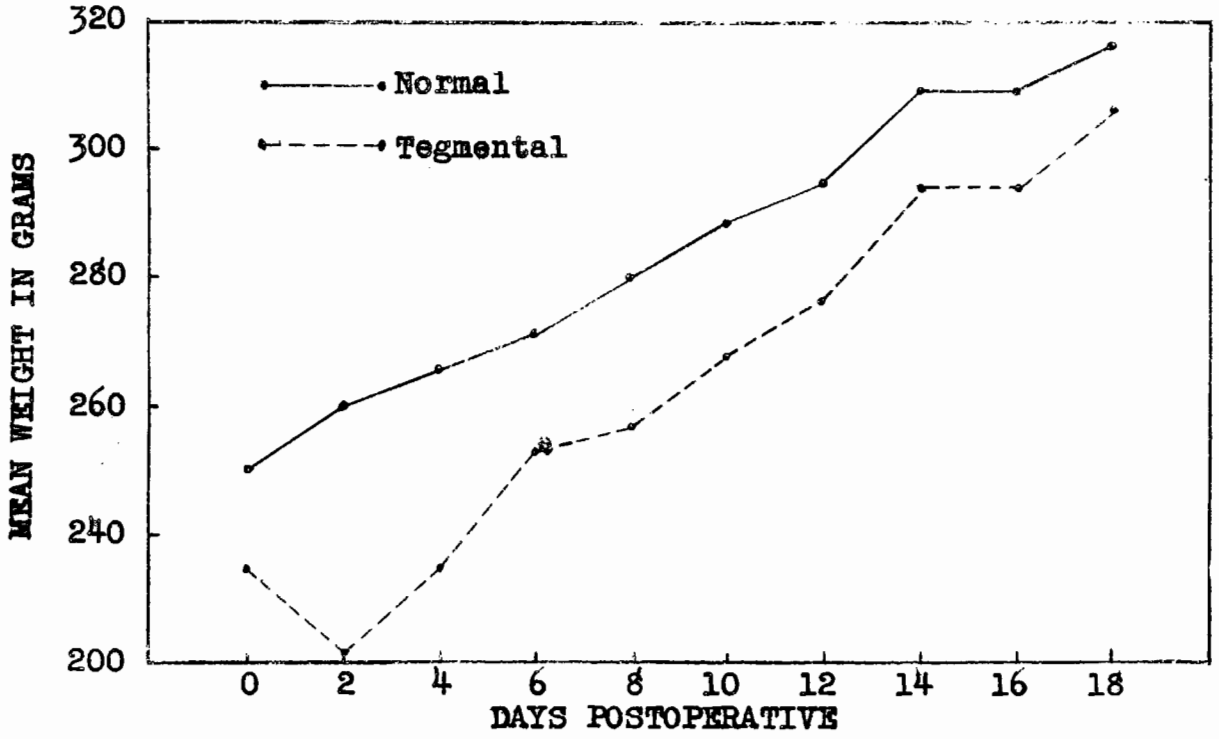

Fig. 1. Weight gain of tegmental operates and normals during 18-day postoperative period.

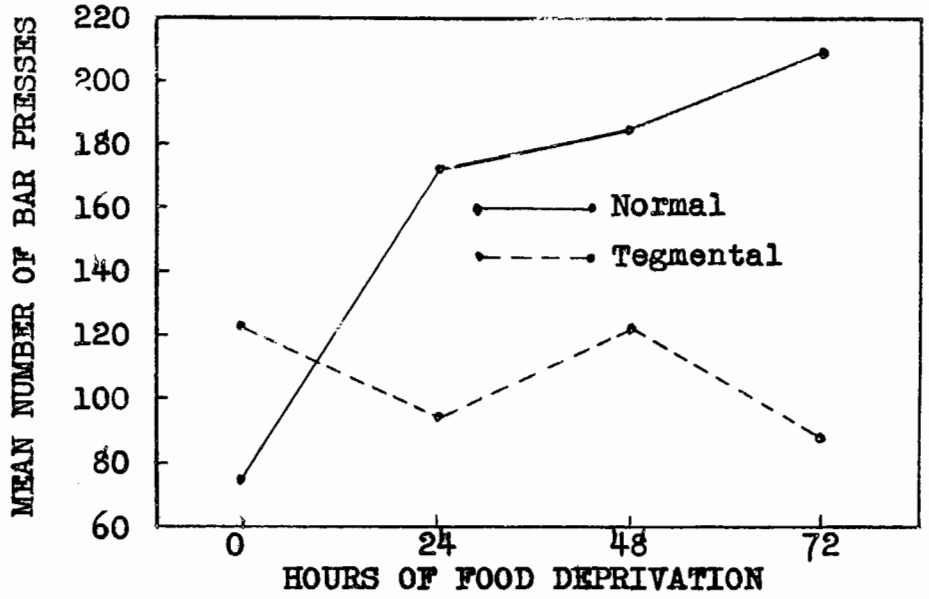

Fig. 2. Bar pressing for food on a fixed interval schedule with increasing amounts of deprivation.

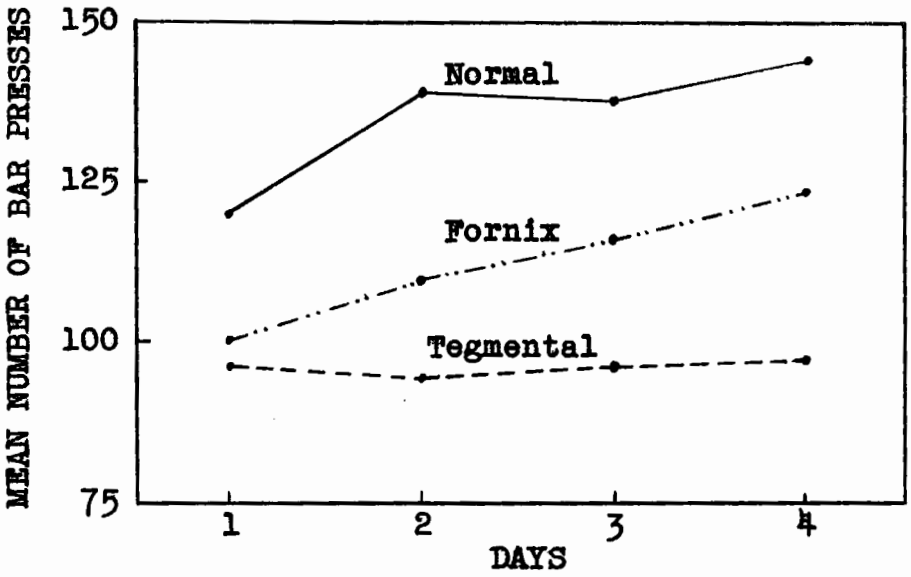

Fig. 3. Bar pressing for food on a continuous reinforcement schedule.

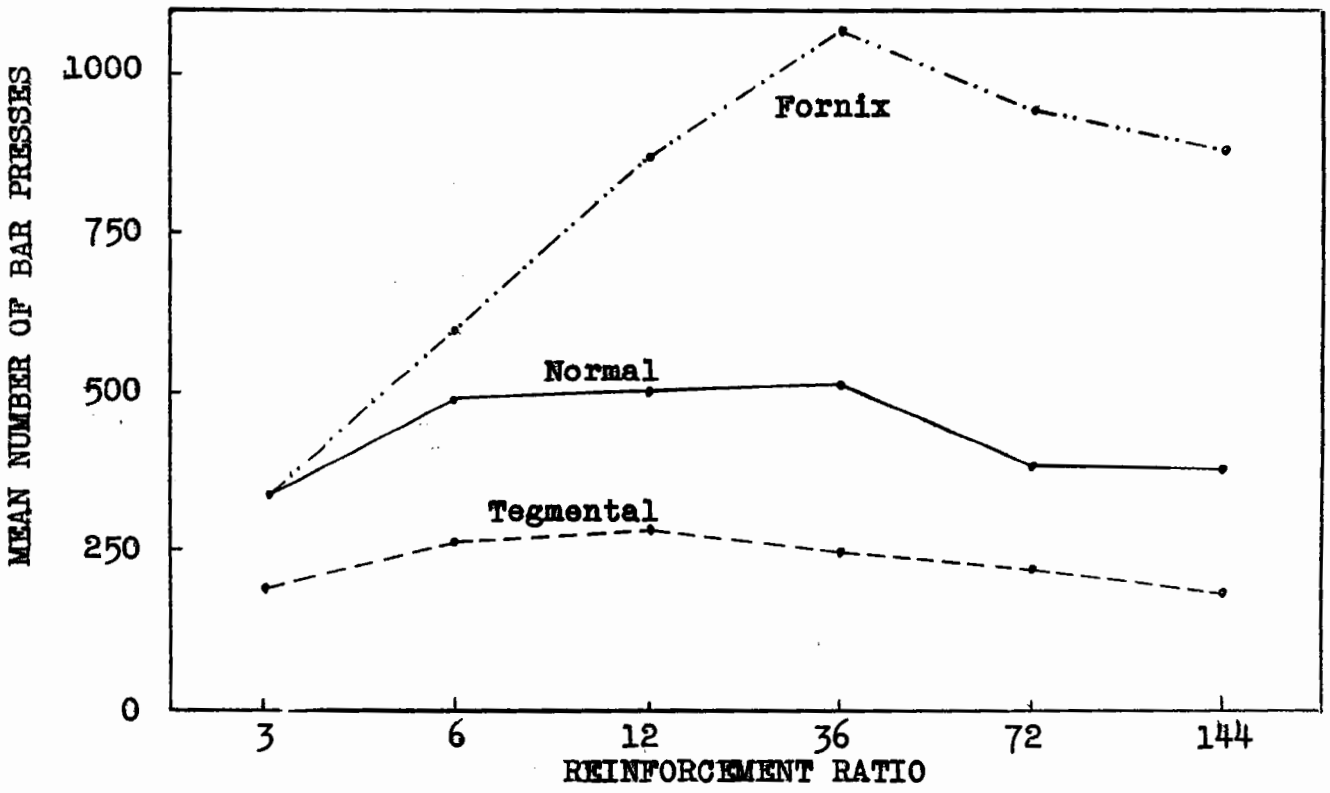

Fig. 4. Bar pressing for food with increasing ratios of reinforcement.

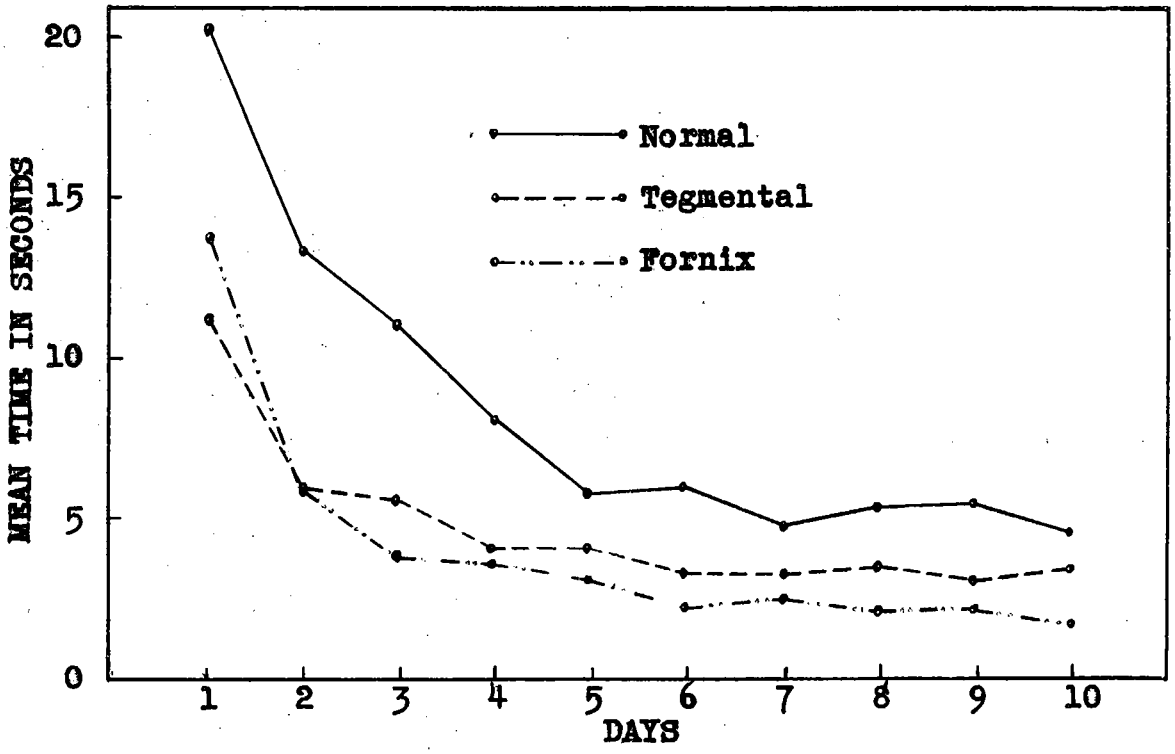

Fig. 5. Time to reach food in runway.

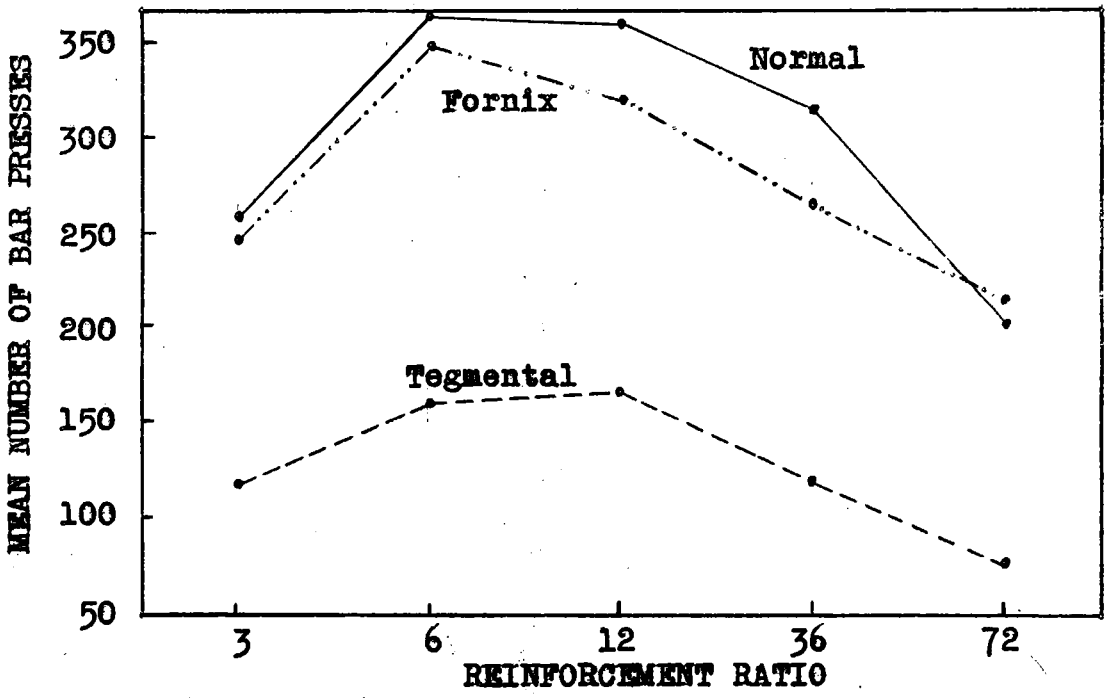

Fig. 6. Bar pressing for water with increasing ratios of reinforcement.

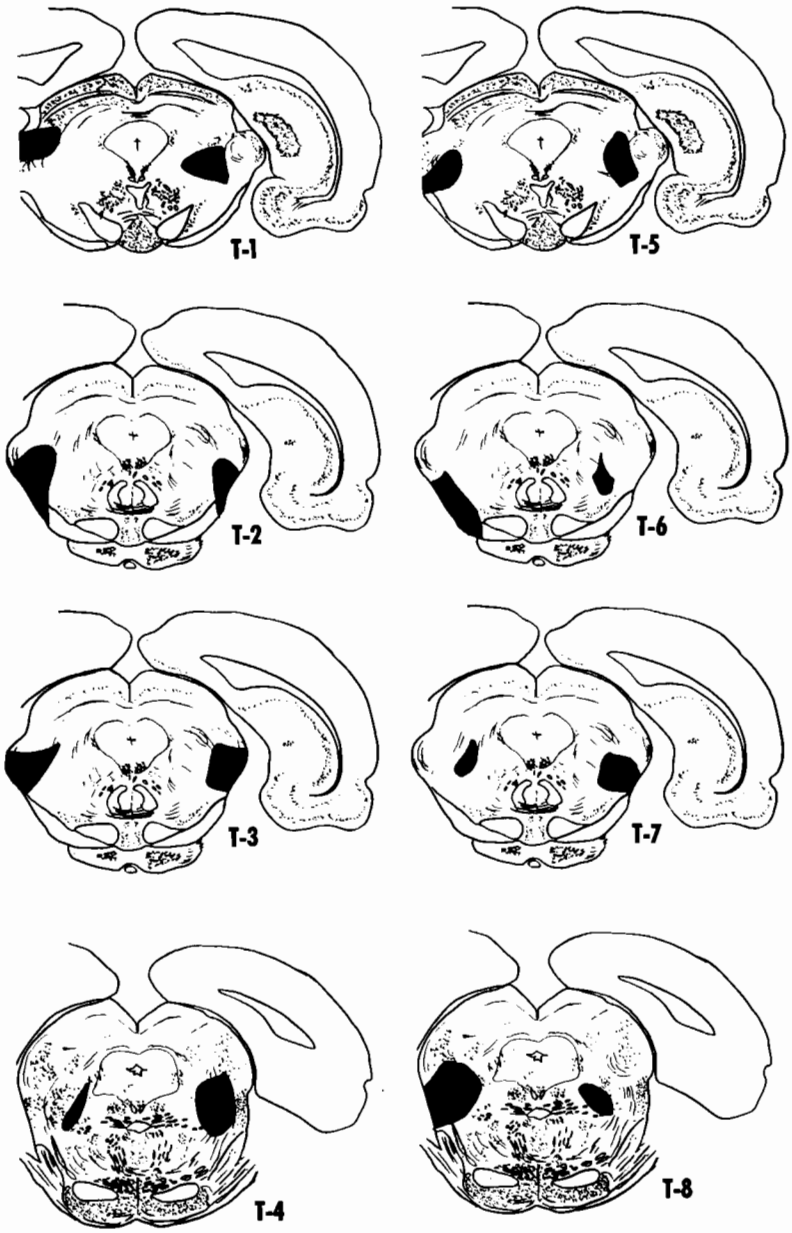

Figure 7. Reconstructions of typical lesions in experimental group of Experiment I. Each drawing shows the level at which the lesions were largest.

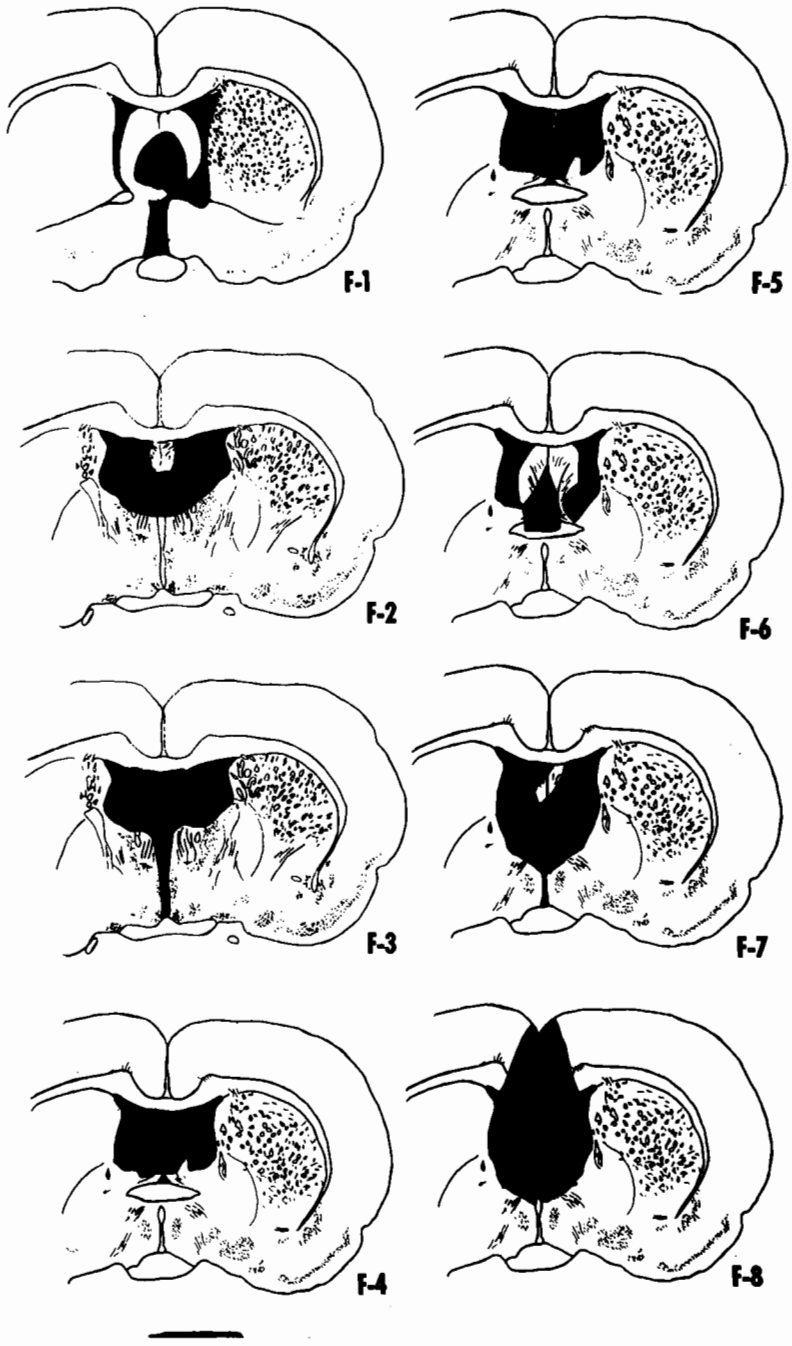

Figure 8. Reconstructions of typical lesions in operate control group of Experiment I. Each drawing shows the level at which the lesions were largest.

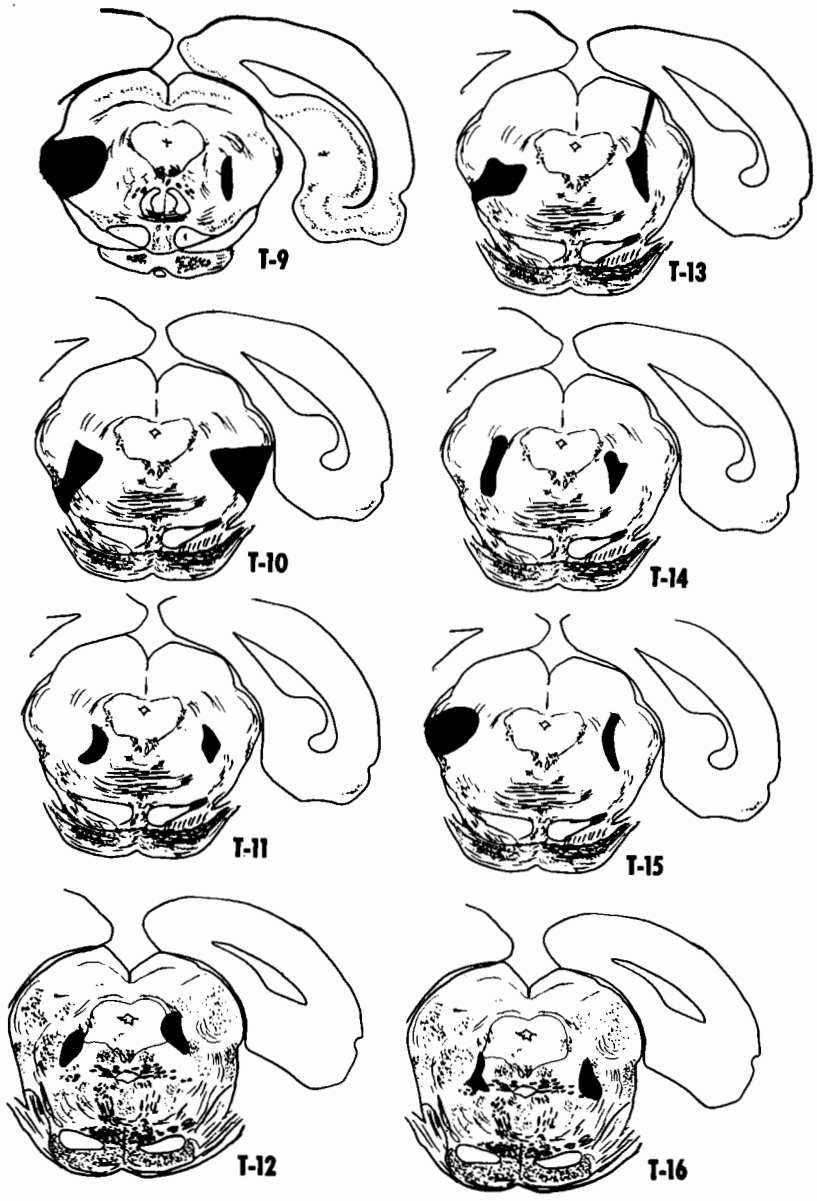

Figure 9. Reconstructions of typical lesions in experimental group of Experiment II. Each drawing shows the level at which the lesions were largest.

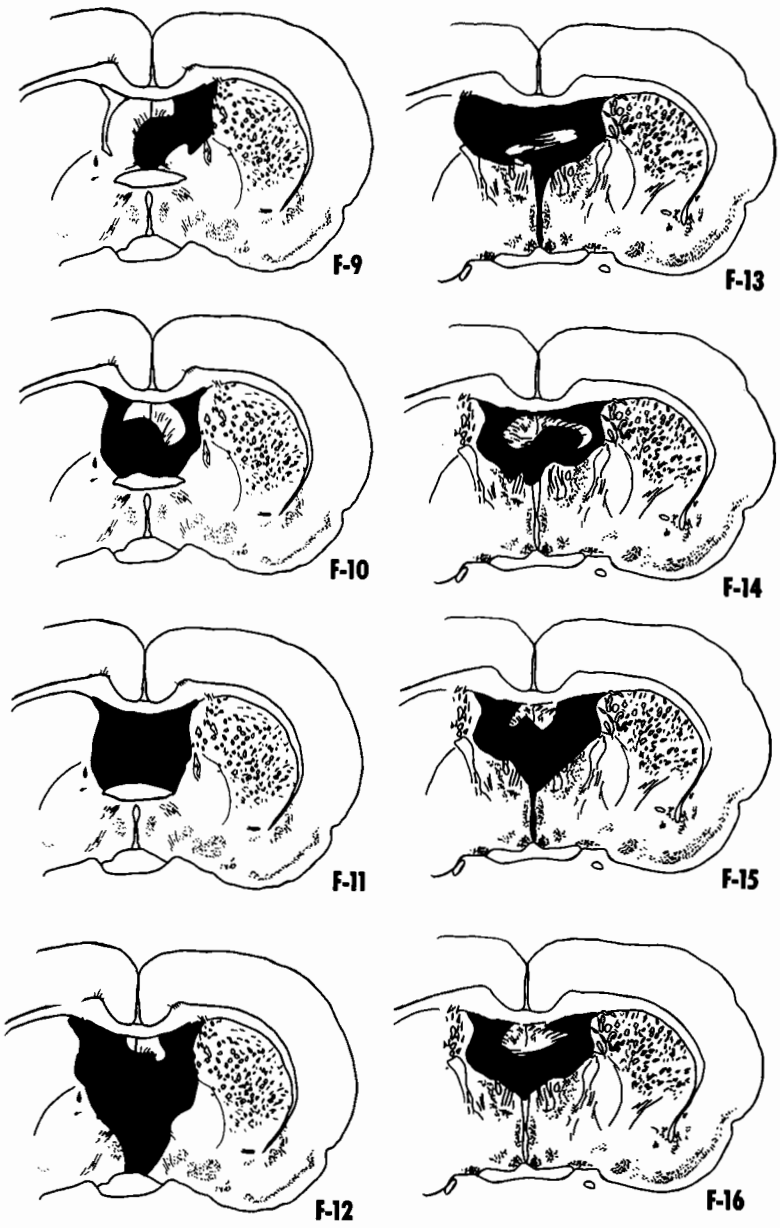

Figure 10. Reconstructions of typical lesions in operate control group of Experiment II. Each drawing shows the level at which the lesions were largest.