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A long-standing problem in understanding the neural substrates subserving behavior is to determine the extent that function can be precisely localized in the brain. The commonly asserted one-to-one relation between function and structure is directly challenged here and a more relativistic model is proposed. In brief, rats were rendered adipsic by a lesion in the lateral hypothalamus and, on careful measure, were found postoperatively to have a very low probability of drinking. At the same time, they showed a much higher probability of running. When the two behaviors were made contingent, such that in order to run the animal had to first drink, drinking immediately commenced. Yet, when the contingency was removed, no spontaneous drinking occurred. The results are consistent with the view that specific psychological processes, such as the motivation to drink, cannot be easily localized to specific brain structures. Rather, it would appear the entire cerebrum is involved in the process of determining motivational states.

INTRODUCTION

It has long been maintained by some that lesions (6, 17, 19) or electrical stimulation (2, 3) in the medial and lateral hypothalamic regions profoundly disturb the motivational state of an animal. Others have interpreted the resulting deficits in terms of a motor dysfunction (2, 3). In the present study it is demonstrated that neither interpretation appears correct for it is shown that animals with lateral hypothalamic lesions will drink if a more probable response, running, is made contingent on drinking. This would suggest that hypothalamic lesions do not disrupt motivational behavior but rather disturb the normal signaling and cueing systems serving in food and water regulation. It seems clear that the brain systems responsible for calculating the wide variety of motivated behaviors are a

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more general and widespread function of the entire cerebrum and not the exclusive localized function of the hypothalamus.

METHODS

Preoperative Training. In brief, use was made of the idea that a more probable response will reinforce a less probable response (14). With adipsia, rats ought to show a variety of behaviors more probable than drinking. In particular, running can be easily measured and compared to drinking. Would the adipsic rat drink in order to engage in the preferred behavior of running?

Two experimental groups were run. In Group I, baseline measures and contingency training were carried out both preoperatively and postoperatively. In Group II, the rats had no preoperative contingency training. All rats (albino males, 2 months old, 245–270 g) were placed in regular cages and given both food and water. They had access to a running wheel only 30 min per day during the testing session.

The testing apparatus consisted of a wheel connected to a retractable drinking tube. Two measures were used throughout the study. In the first, baseline measures were taken which allowed for free access to both the running wheel and the water. In the second, a drink-to-run contingency was available with all responses being automatically recorded.

The rats were first allowed to run for 30 min each day until a steady baseline count was established for each animal. Subsequently, Group I rats were tested 30 min a day on the drink-to-run contingency. This was arranged by locking the running wheel in place, making the water tube available to the rat for five licks which in turn would release the wheel for 10 sec. Only the actual time of running behavior was recorded. Absorbent paper surrounded the drinking apparatus to check on the possibility that the water might not have been ingested. There was no spilling or dripping.

Operative Procedures. All rats underwent bilateral stereotaxic lesions of the lateral hypothalamus according to the system of Albe-Fessard (1). Stainless steel electrodes (0.010 in. diameter) insulated with Formvar enamel, except for 0.5 mm at the tip, were used with a rectal cathode. Lesions were made with 0.5-ma direct current for 30 sec (21). After a recovery period of 3 days, the animals were tested. All animals avoided water and had to be force fed with wet mash throughout all postoperative testing (Table 1).

Postoperative Training. A new baseline was obtained postoperatively for each animal. Subsequently, contingency running was alternated daily with baseline running for 10 days. This allowed us to continuously monitor the separate probabilities of drinking vs. running during the

TABLE 1

Rat No.		Category[a] I	II	III	IV	V
		Group I				
1	Body wt (g)	467	432	376	296	258
	Food intake (g)	23	0	2	2.3	2.5
	Water intake (ml)	35	2	1.5	2.3	3.2
2	Body wt (g)	420	391	333	275	250
	Food intake (g)	28	0	2.3	2.0	4.2
	Water intake (ml)	45	2.5	4.2	4.8	3.7
3	Body wt (g)	505	446	363	311	279
	Food intake (g)	30	0	2.0	4.6	3.9
	Water intake (ml)	49	2.2	2.7	3.0	2.5
		Group II				
1	Body wt (g)	347	329	290	246	230
	Food intake (g)	27	0	1.3	1.6	4.2
	Water intake (ml)	29	0	1.1	2.3	2.7
2	Body wt (g)	352	331	293	263	223
	Food intake (g)	28	0	1.6	3.3	3.7
	Water intake (ml)	37	1	1.8	2.1	2.7
3	Body wt (g)	350	330	307	299	268
	Food intake (g)	34	0	1.0	3.3	4.0
	Water intake (ml)	32	2	2.4	1.2	5.1

[a] I: Average of 4 days preceding the lesion; II, III, IV: averages of 9 consecutive days following the lesion; V: average of last 4 days. All rats were force-fed with 10 ml of Metrecal a day. The recorded food data applies only to voluntarily consumed chocolate chip cookie mash.

course of recovery and to observe that drinking was always higher during contingency training (Figs. 1 and 2).

Histology. The animals were perfused with physiological saline solution followed by 10% formalin and their brains removed stereotaxically by replacing the head of the perfused animal in the stereotaxic instrument. After most of the skull was removed, a knife blade mounted in the electrode holder was used to make a vertical cut in the frontal plane at some distance away from the electrode tracks. The brain was sectioned in the same plane in which the electrodes were inserted. Thus, the entire electrode track could be seen in three or four of the 40-μm sections, facilitating deter-

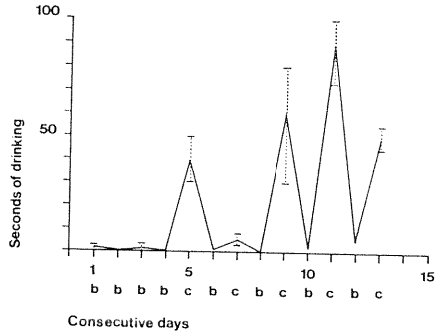

FIG. 1. Average postlesion performance of each rat in Group I. The broken line indicates the observed range. When drink-to-run contingency (C) is instituted, the probability of drinking increases over a baseline probability of drinking (B), and when contingency is removed, the drinking decreases to the baseline level.

mination of electrode placement and tissue destroyed by lesions. Frozen sections were cut at 40 μm with every third section mounted on gelatin-covered slides and stained with Luxol blue-Cresyl violet stain. The results for Group I are as follows. Rat 1, left side: in the zona incerta, medial to medial edge of the peduncle, 1.2 mm (dorsoventrally) $\times$ 1.1 mm (mediolaterally); right side: lateral hypothalamus, 1.5 mm $\times$ 1.1 mm. Rat 2, left side: lateral hypothalamus, 2.0 mm $\times$ 1.0 mm; right side: lateral hypothalamus, 1.5 mm $\times$ 1.1 mm. Rat 3, left side: anterior hypothalamus, 1.5 mm in diameter, ventral to mammilothalamic tract; right side: in the zona incerta, medial to the peduncle, 0.7 mm $\times$ 0.3 mm. Histology for the rats in Group II revealed the following: Rat 1, left side: lateral hypothalamus, 1.5 mm $\times$ 0.9 mm; right side: lateral hypothalamus, 1.3 mm $\times$ 1.2 mm. Rat 2, left side: dorsal to lateral hypothalamus on the border of the zona incerta and the peduncle, 1.0 mm $\times$ 0.7 mm; right side: lateral hypothalamus, extending dorsally to include ventromedial edge of the thalamus, 2.5 $\times$ 1.5 mm. Rat 3, both lesions 1.3 mm in diameter; left side: dorsolateral hypothalamus; right side: lateral hypothalamus. The variations in lesion size are due to some minor variability in the top exposure of the electrodes used. Also, body weight of the rat has an effect on lesion size; the greater the body weight, the higher is the resistance between the anode and the cathode, leading to a smaller lesion.

RESULTS

The results clearly show that rats with the general adipsic syndrome will nonetheless drink to run if running is a more probable response (Group I: Fig. 1; Group II, Fig. 2). Throughout all postoperative testing, the baseline data continued to show that little or no drinking behavior had reappeared. In addition, the animals in Group II that did not experience any contingency training prior to surgery readily performed the drink-to-run condition postoperatively.

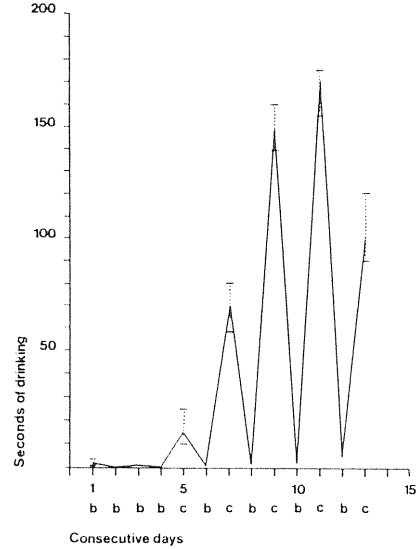

FIG. 2. Average postlesion performance of Group II. Broken line indicates the range, and B and C stand for the baseline and contingency, respectively.

DISCUSSION

These results are consistent with the view that motivated behavior such as drinking is not the exclusive and localized function of a particular neurological structure such as the hypothalamus. Drinking will occur in a rat bearing a lateral hypothalamic lesion if a more probable response is made contingent upon it. This result raises the question of whether it is productive to think in terms of "motivated" behavior as opposed to other kinds of behavior such as operant responses. If one accepts this distinction, the door is opened to an overly compartmentalized view of central nervous function. It would appear more useful to keep in mind the integrative nature of nervous function where motivation is viewed in terms of response probability and is dependent on a broad ranging set of neurological systems that take into account a variety of behavioral functions. In this light, it would appear unwise to tie down such a complex and pervasive behavioral function as "motivation" to a specific, peripheral, and relatively small neural network as the hypothalamus (5).

The present results are also of interest in terms of the problem of recovery of function following brain lesions. In this regard, there has been a variety of recent reports demonstrating neural growth following CNS lesions (12, 15, 16). It has been widely suggested that actual structural changes are at the foundation of recovery. Other more traditional views have urged that recovery comes about through diaschistic changes (20), through reeducation and functional reorganization (10), or through substitution (13) by having an unused neural system take on the lost function. Indeed, it has been suggested when recovery occurs in the rat with a lateral hypothalamic lesion, it follows the normal developmental course of encephalization (18). Other authors suggest specific structures take on the recovered ability (7), while still others have maintained that the recovery cycle better fits the model of denervation supersensitivity (8). The present view, however, urges the idea first advanced by Hughling Jackson (9) that recovery comes about by virtue of the fact that functions are multiply represented in the brain and it is a dangerous enterprise to try and isolate any of them to particular structures. Effecting such quick recovery by manipulating the response contingencies clearly suggests the capacity is instantly there following brain damage and that what occurs during the recovery period is a sorting out of the variety of response probabilities that govern most of our behavior.

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