

REGULATION OF WEIGHT LEVEL WITH
PALATABLE FOOD AND FLUIDS
IN RATS WITH LATERAL HYPOTHALAMIC LESIONS

by

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Bilateral lesions of the lateral hypothalamic area (LH) produce starvation (Anand & Brobeck, 1951) and failure to drink (Teitelbaum & Stellar, 1954). If the animal is maintained by force feeding, however, it will begin to eat again, and recover apparently normal feeding and drinking through a series of stages of recovery, the "lateral hypothalamic syndrome" (Teitelbaum & Epstein, 1962). In Stage I, aphagia and adipsia, the animal with LH damage refuses all food and water. In Stage II, the animal eats small amounts of highly palatable foods (anorexia), but refuses water. In Stage III, the intake of food increases, and the animal can regulate food intake, but water is refused. In Stage IV, the animal can be weaned to plain water and dry food. Once the animal maintains body weight while eating dry food and drinking water, it is considered to be recovered.

Until recently, the ability of animals recovered from LH damage to regulate body weight had not been investigated. Powley and Keesey (1970) proposed that LH lesions lower the level at which the animal regulates its body weight. Under their interpretation, LH damage produces effects opposite to those produced by damage to the ventromedial hypothalamus (VMH), i.e., a lower setpoint for weight rather than obesity. Furthermore, they proposed that the reduction in weight is proportional to the amount of LH destruction, just as the weight increase is proportional to the amount of VMH damage (Hoebel, 1969). Small bilateral LH

lesions (1 ma. for 4 sec.) lower the setpoint to 94% of the weight of unlesioned animals. Larger lesions produced by 1 ma. for 7 and 10 sec. lower the setpoint to 88% and 81%, respectively.

As further proof of their hypothesis, Powley and Keesey starved animals to 80% of the weight of control animals before making lesions (4 and 7 sec.). Prestarved animals ate vigorously when lesions were made until they reached the appropriate level (94% and 88%), at which point they regulated body weight. The lowered weight level was maintained for as long as 250 days.

The recovered animal in Stage IV of the lateral hypothalamic syndrome is far from normal in its behavioral responses to homeostatic challenges: a recovered animal does not eat or drink in response to alterations in blood glucose, cellular hydration, or blood volume (Epstein & Teitelbaum, 1964, 1967; Stricker & Wolf, 1969; Teitelbaum & Epstein, 1962; Williams & Teitelbaum, 1959). Further, animals with LH damage are finicky throughout all stages of the lateral syndrome, resembling animals with VMH lesions. The palatability of foods and liquids is the most important determinant of ingestion (Epstein & Teitelbaum, 1962). Animals recovered from LH damage do not drink water except during meals ("prandial drinking"), reject water with as little as .005% quinine, and reject food with as little as .05% quinine or with 25% cellulose (Williams and Teitelbaum, 1959; Teitelbaum, 1961; Teitelbaum & Epstein, 1962).

These striking deficits have tended to overshadow more subtle responses to diets of low palatability which are still

eaten by recovered animals. For example, Baillie and Morrison (1963) found that animals with LH lesions entering Stage II would feed themselves via a nasogastric tube before orally ingesting a specially prepared diet, but Rodgers, Epstein and Teitelbaum (1964) pointed out that the diet used by Baillie and Morrison was of low palatability, and showed that recovering animals would orally ingest highly palatable diets before they would feed themselves the same diet intragastrically.

A major aspect of the LH syndrome is the refusal of animals to drink water. Animals recovered from LH damage do not drink water in response to the injection of hypertonic saline, in response to a hot environment, or in the absence of dry food (Teitelbaum & Epstein, 1962) and show an aversion to water even when they must drink to avoid or to escape electric shock (Williams & Teitelbaum, 1959).

Powley and Keesey weaned their animals from moistened chocolate chip cookies to moistened ground chow, and, then to dry ground chow and water. Animals were maintained on dry ground chow and water for the duration of the experiment. Since it has been shown that finicky hyperphagic (VMH) animals gain less weight on dry ground chow than on a high fat diet (Corbit & Stellar, 1964; Rehovsky & Wampler, in press), the long-term lowering of body weight observed by Powley and Keesey in animals recovered from LH damage may have been the result of maintaining them on an unpalatable diet. Alternatively, since water is aversive to recovered animals (Williams & Teitelbaum, 1959), the

lowered level of body weight may reflect hypodipsia with secondary hypophagia. Either or both of these alternatives could produce the effects seen by Powley and Keeseey. Recovered animals, if maintained on a highly palatable diet such as egg-nog, are hyperphagic over short periods (Williams & Teitelbaum, 1959), although Powley and Keeseey (1970) did not find hyperphagia to eggnog over long periods. The purpose of the present study was to test whether the unpalatability of the ground food and/or hypodipsia were responsible for the results obtained by Powley and Keeseey (1970).

Method

Subjects

A total of 34 female albino rats (Carworth CFE strain), ranging in weight from 230 to 270 gm., served as subjects in the experiment. Twenty-five animals were given bilateral lesions, nine served as sham-lesion control subjects.

Procedure

Groups. Three experimental groups were formed: (a) eleven animals in the 7-sec. Group were given bilateral LH lesions (1 ma. dc for 7 sec.), (b) five animals in the 4-sec. Group were given smaller bilateral lesions (1 ma. dc for 4 sec.), (c) nine animals in the 7-sec. Prestarved Group were starved to 80% of their normal weight and then given bilateral LH lesions (1 ma. dc for 7 sec.). All experimental and control groups were matched for body weight.

There were three control groups: (a) five animals in the Average Deprived Group were given sham lesions, and offered 10 gm. of cereal-milk mixture for the mean duration of aphagia observed in animals in the combined 4-sec. and 7-sec. Groups (2.1 days). They were deprived of water for the average duration of adipsia observed in the combined 4-sec. and 7-sec. Groups (2.5 days). They were given 6 ml. and 8.6 ml. of water on the two days following water deprivation, representing the average intake of the combined 4-sec. and 7-sec. Groups on these days. (b) Two animals (Normal Control Group) were given access to both food and water immediately after the sham-lesion operation. (c) Each of two animals (Prestarved Control Group) was matched for weight with one of two animals in the 7-sec. Prestarved Group and deprived of food before operation. Following sham lesions, these two animals were given 10 gm. of the cereal-milk mixture until their counterpart animal in the 7-sec. Prestarved Group began to eat (1 or 3 days). Similarly, no water was given until the counterpart animal began to drink water (3 or 5 days).

Preoperative procedure. Animals were housed in individual wire mesh cages and given ad lib. access for a 2-wk. period to high fat diet (Corbit and Stellar, 1964) placed in 8-cm. diameter glass sponge cups and to water in calibrated glass founts. All animals were exposed for one 24-hr. period to eggnog (Teitelbaum and Epstein, 1962) and to a cereal-milk mixture (Gerber's Baby Cereal [2 parts] and Similac [1 part] moistened to make a slurry). Papers were placed under the cages to catch spillage. Animals

were exposed before surgery to the diets offered after surgery to maximize the recovery of feeding (DiCara, 1970).

After the 2-wk. exposure to the high fat diet, animals were assigned to experimental and control groups based on body weight. Shortly thereafter, surgery was performed on animals in the groups which were not prestarved. The animals in the Pre-starved Groups were on restricted food (3 gm/day Purina Lab Chow pellets) and ad lib. water access until 80% of normal weight was reached (26 days), at which time surgery was performed.

Surgery. Surgery was performed under Equithesin anesthetic (.25 - .3 ml/100 gm.) using a Kopf stereotaxic instrument. The animal's head was positioned so that the frontal bones of the skull were parallel to the table of the stereotaxic instrument. A stainless steel wire (.014 in. diameter), insulated with Formvar enamel except for a cross section of the tip, was used to make lesions (2.5 mm. posterior to bregma, 1.7 mm. lateral to the midline sinus, and 8 mm. below the dura). A rectal cathode completed the circuit. Sham-lesion animals were treated as experimental animals, except that the electrode was lowered 4 mm. below the dura and no current was passed.

Postoperative procedure. Animals with LH lesions were offered only water for the first 24 hr. after surgery; they were offered the high fat diet and water beginning 24 hr. after surgery. If water was rejected, the animal was considered to be in Stages I, II, or III. If the high fat diet was also rejected, the animal was considered to be in Stage I or II. If high fat

diet was rejected, the cereal-milk mixture was offered, and the animal was observed for several hour-long periods each day. If the mixture was not eaten (Stage I), attempts were made to coax the animal to eat: the mixture was brought near the mouth on a spatula, placed in the mouth, etc. These procedures were repeated several times daily until the animal began to eat. Once the cereal-milk mixture was accepted, eggnog and high fat diet were given. The animals were weaned to water over a period of several days. Animals in Stage I were maintained by 3-ml. injections of .9% saline given twice daily, administered intraperitoneally and subcutaneously in alternation. Body weight and food and water intake were measured daily for at least nine days or until the animals began drinking water (Stage II).

After the postoperative periods of aphagia and adipsia or hypodipsia and anorexia, food and fluid intakes were measured every three days until 87 days postoperatively. During this period, various solutions were offered the animals. For the 4-sec. and 7-sec. Groups and the Normal Control and Average Deprived Groups, water was available until the ninth postoperative day; then 6% sucrose (weight/volume) was available until Day 51; water was available on Days 52-66; 6% sucrose was available again on Days 67-72; 1% saccharin was available on Days 73-81; .2% saccharin was available on Days 82-87. For the animals in the 7-sec. Prestarved and the Prestarved Control Groups, water was available until the fifth postoperative day; then 6% sucrose was available until Day 51; water was available Days 52-63; 6%

sucrose was available again on Days 64-69; 1% saccharin was available on Days 70-78; .2% saccharin was available on Days 79-87.

Histology

The animals were perfused with .9% saline followed by 10% formalin while under Equithesin anesthesia. The brains were removed immediately after perfusion, hardened in acid formalin, dehydrated, and embedded in celloidin. Sections were cut at 40 μ , every fourth section was stained with cresylecht violet and was mounted for examination.

Results

Histology

Figure 1 shows the lesions at the anterior-posterior coordinate of maximum damage for animals in the 7-sec. Group. The brains of five of the nine animals in the 7-sec. Prestarved Group have been examined, and the lesions resemble those of the 7-sec. Group in terms of placement and size. The 7-sec. lesions destroyed most of the LH on both sides, but spared the internal capsule, fornix and VMH and surrounding areas. The only exception was subject 74 with asymmetrical lesions which destroyed most of the LH on one side, and a portion of the internal capsule on the other side. This animal was 3 gm. below the mean weight of the animals in the 7-sec. Group on day 87, less than one standard deviation from the mean.

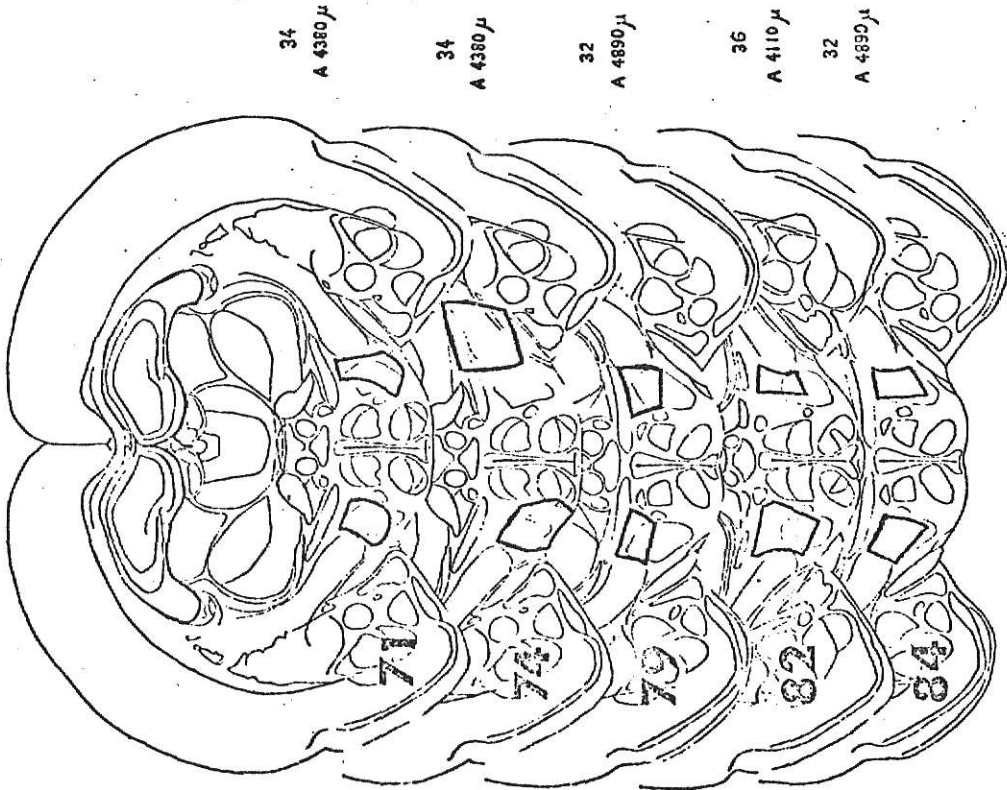
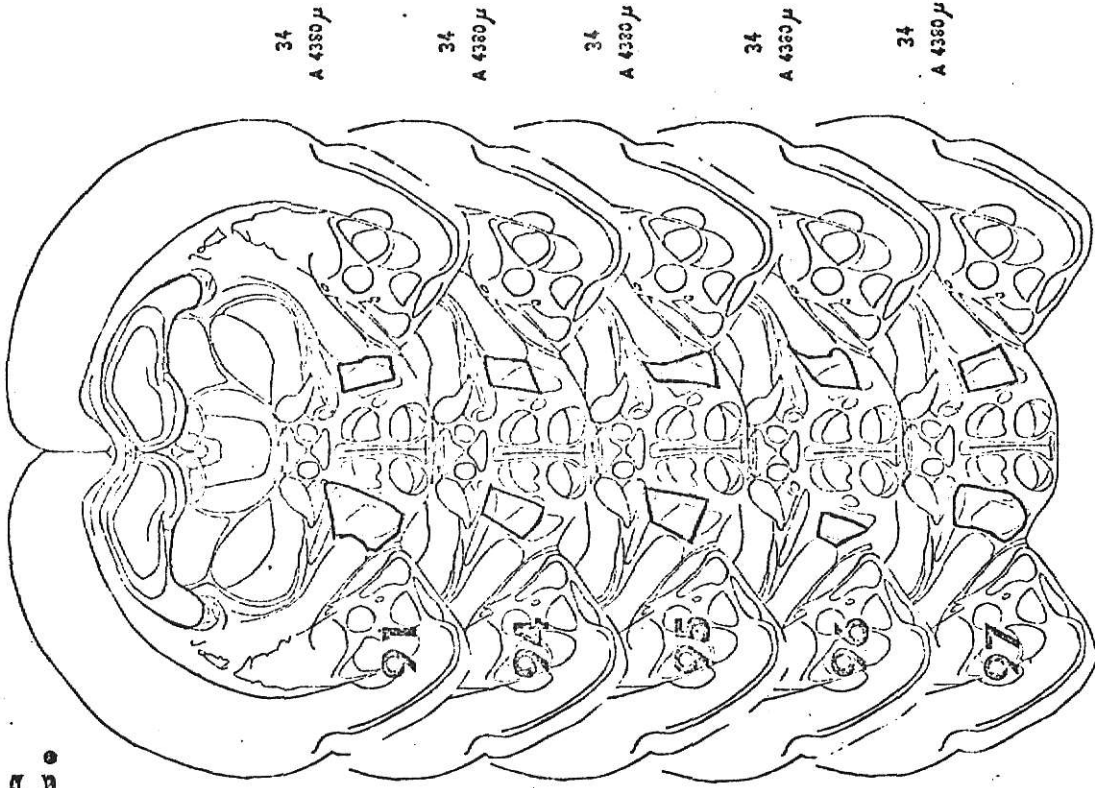
Figure Caption

Fig. 1. Lesions in animals in the 7-sec. Group at the anterior-posterior section of maximum damage. Diagrams are modified from König and Klippel (1963).

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7-SEC.



The 4-sec. lesions shown in Figure 2 destroyed large portions of the LH on both sides. As in the 7-sec. Group, there was no damage to the internal capsule, fornix, or the VMH and surrounding areas. The exception was subject 85 with slight internal capsule damage.

Aphagia and Adipsia

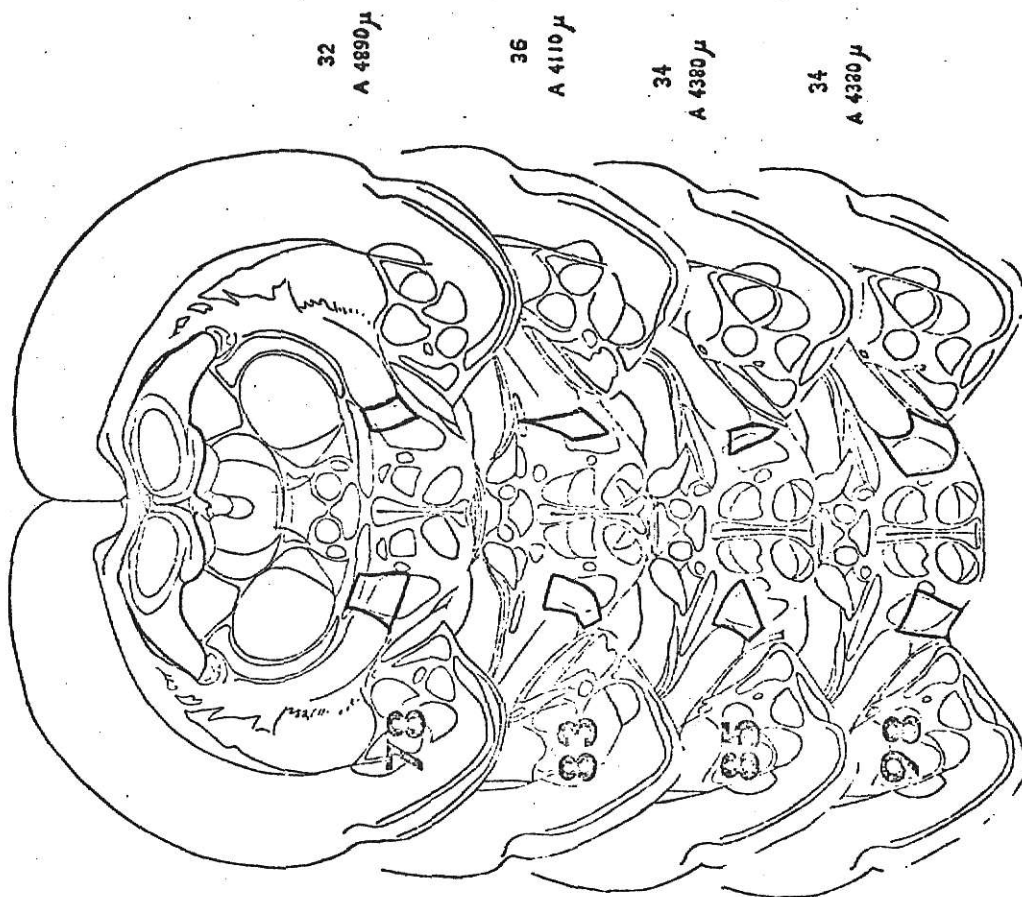
All lesioned animals exhibited the classic LH syndrome of aphagia and adipsia. Animals in the 7-sec. Group were aphagic for an average of 2.4 days; those in 4-sec. Group were aphagic for 1.8 days (not significantly different). Animals in the 7-sec. Prestarved Group were aphagic for .8 days and adipsic for 3.4 days. The period of aphagia was significantly shorter than in the 4-sec. Group ($t=2.840$, $df=12$, $p<.02$) and in the 7-sec. Group ($t=3.369$, $df=18$, $p<.01$). The animals in the 7-sec. group were adipsic for 2.5 days; those in the 4-sec. Group were adipsic for 2.6 days; those in the 7-sec. Prestarved Group were adipsic for 3.4 days. The duration of adipsia was not significantly different between animals in any group. During the period of aphagia and adipsia, there was a steady decrease in body weight which continued through Stage II (anorexia and adipsia).

Animals in the 7-sec. Group showed anorexia for an average of 2.4 days; animals in the 7-sec. Prestarved Group showed anorexia for an average of 1.9 days (not significantly different). Animals in the 4-sec. Group showed 1.4 days of anorexia. There was a significant difference in duration of anorexia between animals in the 7-sec. Group and those in the 4-sec. Group

Figure Caption

Fig. 2. Lesions of animals in the 4-sec. Group at the anterior-posterior section of maximum damage. Diagrams are taken from König and Klippel (1963).

4-SEC.



($t=2.150$, $df=14$, $p<.05$), but there was no significant difference between the animals in the 4-sec. Group and the 7-sec. Prestarved Group. Once the animals began to eat the cereal-milk mixture, full recovery followed in a few days. All animals with lesions were drinking water when switched from water to sucrose at Day 9 (4-sec. and 7-sec. Groups) or Day 5 (7-sec. Prestarved Group), with one exception in the 7-sec. Prestarved Group.

Animals in the Normal Control Group showed no aphagia or adipsia after the sham-lesion operation. Animals in the Average Deprived and the Prestarved Control Groups lost weight during the postoperative deprivation, but all seven ate the cereal-milk mixture as soon as it was offered 24 hr. after surgery. Animals in the Normal Control Group drank water in the first hours after the operation and ate within 24 hr. All postoperatively deprived control subjects showed a rapid increase in weight when high fat diet was available ad lib.

Although prelesion starvation reduced the average period of aphagia from 2.4 days (7-sec. Group) to .8 days (7-sec. Prestarved Group), animals in the 7-sec. Prestarved Group did not show hyperphagia and an immediate weight gain following lesioning; rather, the prestarved animals remained anorexic for several days. These data are summarized in Table 1.

Regulation of Body Weight

Following periods of aphagia and adipsia, animals with lesions steadily returned towards the body weight of animals in

Table 1

High Fat Diet Intake (gm.) in 7-sec. Prestarved Group

Over the First Nine Postoperative Days

Subject	1	2	3	Postoperative Day					8	9
				4	5	6	7	6% Sucrose		
			Water							
110	.4	5.7	5.4	7.6	3.4	9.9	8.3	13.0	26.0	
111	.4	0	.4	5.8	3.9	7.6	9.3	11.0	22.1	
101	2.2	.5	4.9	7.0	3.7	9.6	12.9	10.3	21.0	
115	.7	1.3	6.6	8.6	3.4	9.2	10.9	9.9	19.9	
108	0	4.3	4.6	4.0	7.5	13.3	13.6	17.4	16.0	
109	0	7.5	8.2	6.9	11.3	19.2	16.0	25.7	21.0	
70	12.8	13.8	18.1	14.5	18.9	16.3	11.0	17.1	7.3	
99	2.1	5.0	12.8	16.3	17.1	17.0	16.0	14.9	15.6	
50	0	0	0	12.2	12.3	12.0	12.5	13.0	12.0	
\bar{M}	2.1	4.2	6.7	9.2	9.1	12.7	12.3	14.7	17.9	

control groups. Figures 3 and 4 present the average weight of animals in each group over days. On Day 87, the average weight of animals in each experimental group was higher than the body weight of animals in the pooled control groups. These comparisons are summarized in Table 2.

The average weight of animals in the 7-sec. Group surpassed that of animals in the Average Deprived Group 24 days after surgery, and weight remained greater than that of animals in the Average Deprived Group until the experiment was terminated. The average weight of animals in the 4-sec. Group did not surpass that of animals in the Average Deprived Group until 39 days after surgery. Beyond this point, animals in the 4-sec. Group were always heavier than those in the Average Deprived Group. Animals in the 7-sec. Prestarved Group were always heavier than those in the Prestarved Control Group, and were heavier than animals in the Average Deprived Group beyond the twelfth day after surgery. The weights of animals in the lesioned groups did not differ from one another, nor did the weights of animals in any control group differ significantly from those of any other control group.

Effects of Various Solutions on Ingestion and Body Weight

Animals in the three control groups showed no consistent differences in food or fluid intake when the measures for the last three days on each solution were compared. The data for these animals were pooled and compared to the data from animals in each experimental group. The average daily intake for animals

Table 2

Terminal Weight of Animals in Experimental
and Pooled Control Groups

Group	Mean terminal weight in gm. (S.E.)	\bar{t}^a	<u>df</u>	<u>p</u>	Percent of weight of pooled control animals
4 sec.	320.9 (16.0)	1.802	12	<.1	110
7 sec.	329.2 (12.2)	2.878	18	<.05	113
7 sec. Prestarved	313.6 (11.4)	1.783	16	<.1	108
Pooled controls	290.6 (5.7)	-	-	-	100

^aTwo-tailed t test, weights of experimental animals in each group compared to weights of pooled control animals.

Figure Caption

Fig. 3. Body weight for the 4-sec. and 7-sec. Groups and the Average Deprived and Normal Control Groups over 87 post-operative days.

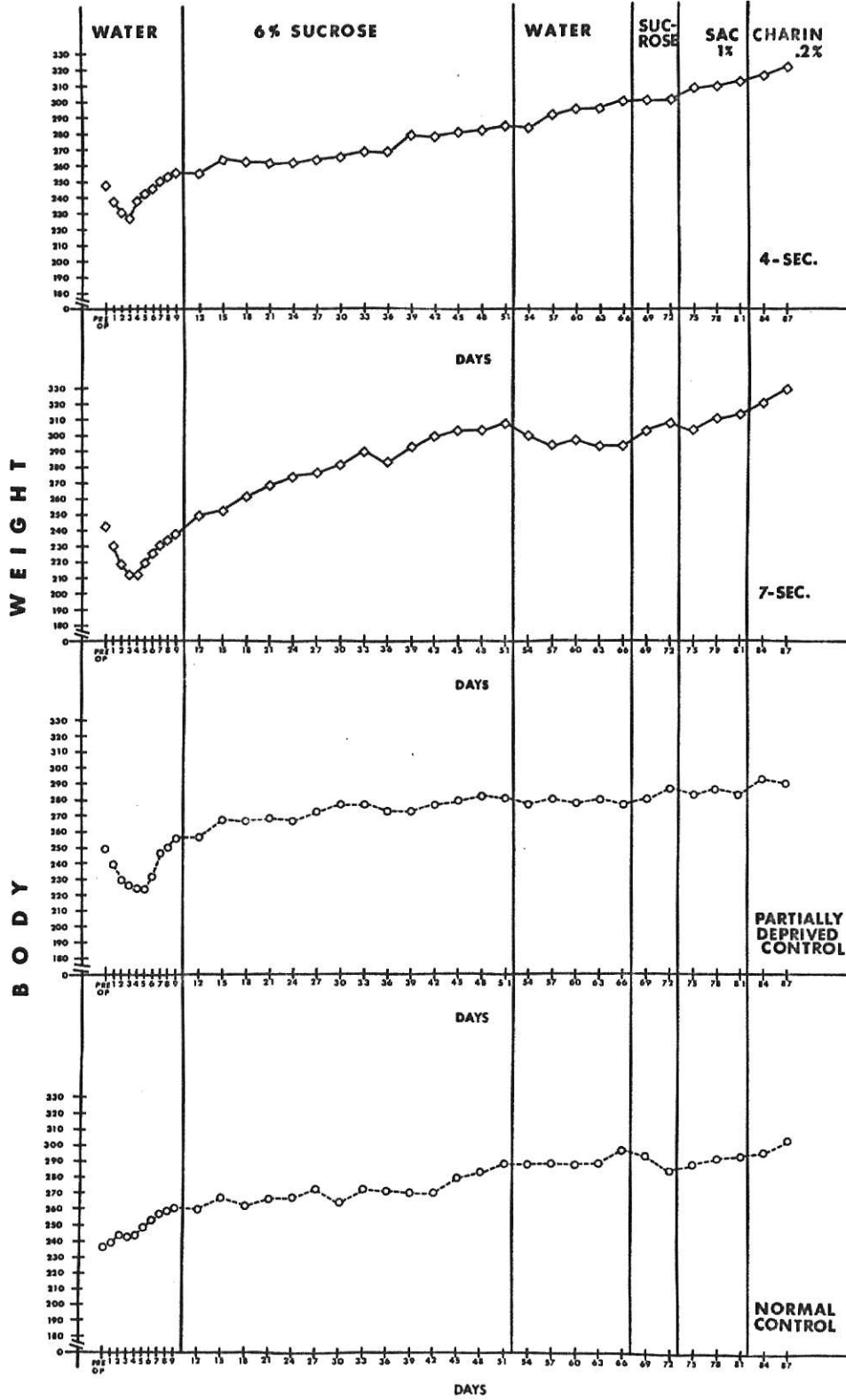
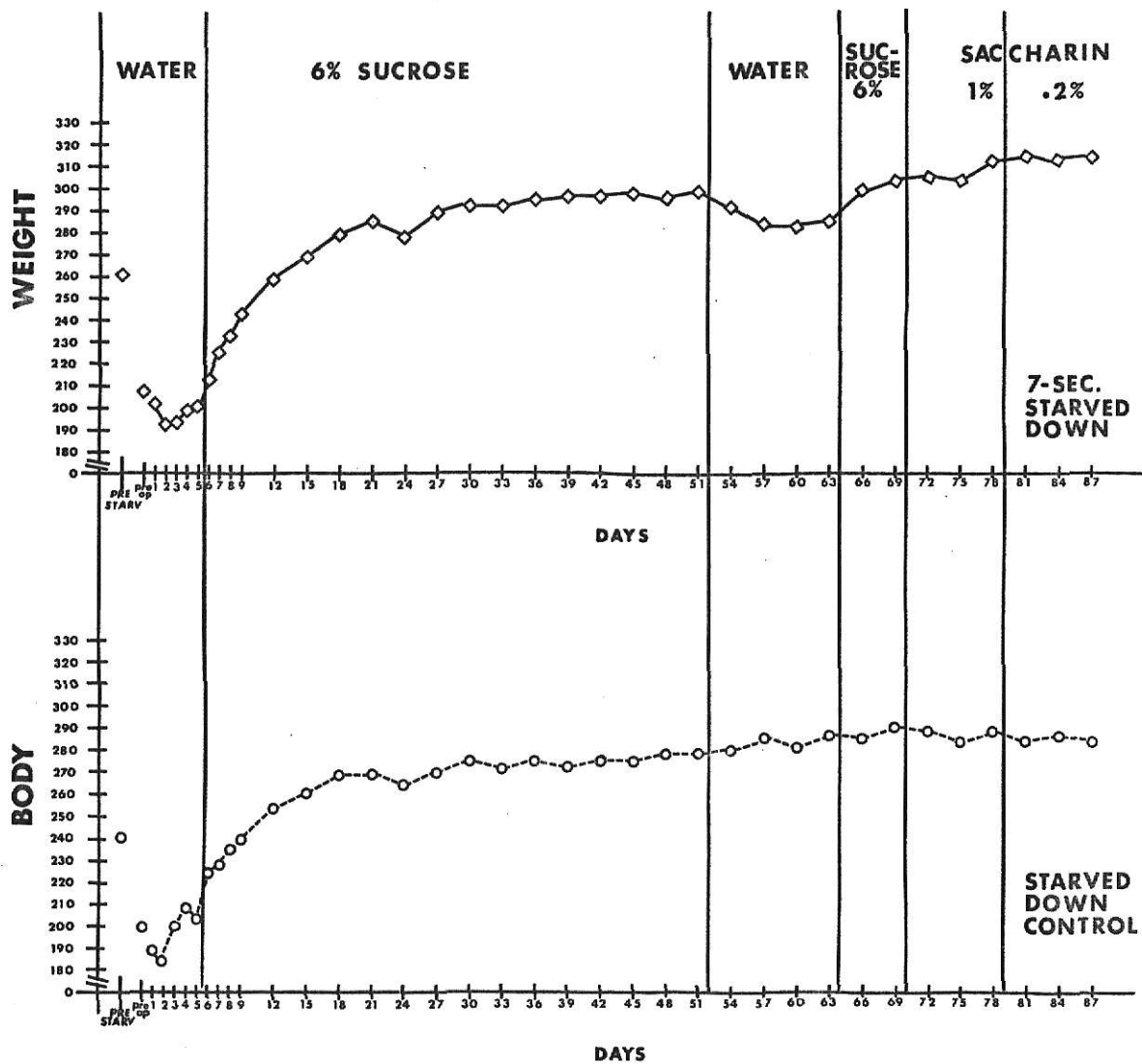


Figure Caption

Fig. 4. Body weights for the 7-sec.
Prestarved Group and the Prestarved Control
Group over 87 postoperative days.



in the experimental groups and the intake for animals in each of the experimental groups and pooled control groups are shown on Table 3, along with the results of t tests on intake data from animals in experimental and the pooled control groups. In general, the animals with LH lesions in the experimental groups ate more of the high fat diet than those in the control groups, but drank less of all solutions.

Animals with LH damage ate and drank enough to show a weight gain in the first period on water. When 6% sucrose was available, this gain in weight continued and animals with LH damage ate more high fat diet over the last three days on sucrose than did control animals. Food intake decreased when water was offered a second time, but body weights in experimental animals did not decline below those of control animals. Animals with smaller 4-sec. lesions did not show a decrease in body weight and even increased food intake during the second period on water. When 6% sucrose was returned, intake of food increased. Food intake remained higher in experimental animals than in control animals when non-nutritive saccharin solutions were given.

Discussion

The hypotheses of Powley and Keesey (1970) that (a) LH lesions produce a lower level of weight regulation and that (b) LH lesion size is inversely related to the level of weight regulation were not supported by the results of the present experiment. Once the average weight of animals in each of the

Table 3

Average Daily Food and Fluid Intake on the Last Three Days of Various Fluids						
Group	Fluid Available ^a					
	Water	Sucrose (6%)	Water	Sucrose (6%)	Saccharin (1%)	Saccharin (.2%)
Food intake (gm.)						
4-sec.	12.3	9.9*	11.1	8.6	10.8	12.2
7-sec.	12.5	11.2***	8.9	11.1	10.2	14.0**
7-sec. Prestarved	9.0	9.8*	8.5*	11.9***	12.9***	10.9
Pooled controls	12.0	7.7	10.1	9.0	9.9	10.9
Fluid intake (ml.)						
4-sec.	17.0*	57.1	13.0***	54.4*	30.5	39.9
7-sec.	16.5**	43.7***	10.0***	40.8***	19.7	27.3**
7-sec. Prestarved	6.3***	42.8*	8.9***	47.4***	25.6	23.0***
Pooled controls	24.8	76.7	22.8	84.6	26.4	52.0

^aAll comparisons are between data from lesioned group and pooled control groups (t test).

* $p < .05$, two-tailed test

** $p < .02$, two-tailed test

*** $p < .01$, two-tailed test

three groups with lesions surpassed that of the appropriate control animals, weight remained greater than the weight of control animals until the termination of the experiment, regardless of the fluid offered. Furthermore, the terminal weights of the animals in the experimental groups with different lesion sizes were not significantly different. The contradiction in results between the present study and that of Powley and Keesey (1970) may come from either or both of two sources: the difference in the diet available following Stages I and II of the LH syndrome in each study and/or differences in the relative degree of hydration of the recovered animals in each study.

The highly palatable diet offered all animals in the present experiment was eaten in greater quantities by animals with 7-sec. lesions than by control animals, regardless of the sweetened fluid available. When water was available, the animals with lesions ate and drank enough to show a rapid weight gain in the first days after lesioning; however, when water was available again after sucrose, animals in the 7-sec. Group and 7-sec. Pre-starved Group lost weight for the first days and then started to gain. Animals in the 4-sec. Group continued to gain weight across the period when water was available. The greater intake of high fat diet by experimental animals was the obvious source of the increased weight on Day 87. The animals appeared to be regulating caloric intake since intake of food with non-nutritive saccharin solutions was higher than when sucrose was available.

The greater food intake of animals with LH lesions and their ability to maintain a greater body weight than control animals depends on the state of hydration of the recovered animals. The large drop in water intake accompanied by a drop in food intake and body weight of animals in the 7-sec. Group and 7-sec. Prestarved Group suggests that proper hydration was crucial in keeping food intake of animals with larger lesions above the intake of control animals and, therefore, in maintaining their body weights above those of control animals. "Finickiness" to water remains in recovery, and the extremely low intake of water on the days succeeding the change to water after sucrose indicates that water was aversive to animals with extensive LH damage. The fact that animals in the 4-sec. Group did not lose weight suggests that these animals were less finicky about water, i.e., that larger lesions made water more aversive than smaller lesions. The steady increase in weight in animals with lesions when non-nutritive saccharin was offered indicates that the animals with lesions were attempting to regulate at a higher body weight, as long as they were sufficiently hydrated. Furthermore, the animals with larger lesions (7 sec.) were regulating as well as those with smaller lesions (4 sec.).

The present study also failed to show voracious eating in all animals in the 7-sec. Prestarved Group after LH lesions. Rather, prestarved animals were aphagic and adipsic, and did not immediately eat the cereal-milk mixture when it was available 24 hr. after surgery. They either ignored the food entirely or

approached it, took a few licks and went to sleep next to the dish. The first meal did not occur until the evening of the second day (36 hr. after surgery) at the earliest, and, as indicated in Table 1, eating was neither vigorous nor sustained. This difference in results did not seem to be the result of surgery, per se, since control animals ate immediately upon presentation of the food. It may have been the result of an interaction between a disturbance of the cortex (spreading depression) produced by electrode penetration and LH damage, as was suggested by Powley and Keesey. Even if such an interaction occurred, however, the hypothesis of altered weight level predicts that when the prestarved animals, now further underweight, began to eat, such eating should have been vigorous. This was not observed until the ninth day in the present experiment.

The present experiment showed that body weight in female rats with LH lesions is greater than control body weight; the research of Powley and Keesey showed a decrease in body weight of male rats following LH lesions. There is no evidence that there are sex differences in the effects of LH lesions. Powley and Keesey used, as support for their hypotheses, single animal data from other research reports (Montemurro & Stevenson, 1957; Rodgers, Epstein & Teitelbaum, 1965; and Teitelbaum & Epstein, 1962) in which females were used as subjects.

The discrepancy in level of body weight stabilization between the two sets of data seems to result from different approaches to the problem of finickiness: the present study

maximized palatability of foods and fluids by the use of sweet fluids (nutritive and non-nutritive) and high fat diet; the studies by Powley and Keesey maximized unpalatability by the use of water and dry ground chow. The differences between these approaches were emphasized most strongly when animals with lesions were switched from sucrose to water in the present experiment: the animals with the larger lesions (7 sec.) lost weight; the animals with smaller lesions (4 sec.) did not, nor did the control animals.

The present data suggest that an animal maintained on an unpalatable diet with only water to drink would remain below normal weight. Many of the contradictions in the data on weight regulation following LH lesions, like the contradictions which have occurred in weight data from animals with VMH lesions (Rehovsky & Wampler, in press) are problems of finickiness: the less palatable the substances available (foods or fluids), the lower the body weight at which the lesioned animal stabilizes. Conversely, highly palatable diets or fluids will increase the level at which body weight stabilizes in lesioned animals.

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REGULATION OF WEIGHT LEVEL WITH
PALATABLE FOOD AND FLUIDS
IN RATS WITH LATERAL HYPOTHALAMIC LESIONS

by

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Female rats given either 4-sec. or 7-sec. 1-ma. bilateral lesions of the lateral hypothalamus (LH) were maintained after recovery (Stage IV) on high fat diet and water or sweetened fluids. Over 87 days, the lesioned rats reached a weight level which was higher than that of control animals. This higher weight level was maintained regardless of the fluid available. Animals with larger lesions reached and surpassed the weight of control animals before animals with smaller lesions.

When animals were starved to 80% of their normal weight and then LH damage (7 sec.) was made, the period of aphagia was shorter than if lesions were made at normal body weight. However, the prestarved animals did not eat vigorously and gain weight rapidly; rather, the periods of anorexia and adipisia following LH lesions were the same as that observed in normal weight animals with 7-sec. lesions.

These data do not support the hypothesis that LH damage effectively lowers the setpoint of weight regulation. Rather, the finickiness of LH rats appears to result in a smaller intake of unpalatable foods and water which, in turn, results in stabilization of weight at a level below that of control animals. Highly palatable foods are taken in larger quantities by LH animals than by control animals, resulting in a stabilized weight above that of control animals.