AN INVESTIGATION OF THE ROLE OF ANTERIOR AND LATERAL CONNECTIONS OF THE VENTROMEDIAL HYPOTHALAMUS IN CONTROL OF FOOD INTAKE, ACTIVITY, FOOD MOTIVATION AND REACTIVITY TO TASTE

by

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ABSTRACT

The "hyperphagic syndrome" consists of a number of behavioral changes which normally occur along with the increased weight gain following VMH lesions in the rat. The present study explored the possibility of separating these behavioral changes from the increased weight gain. The behavioral dimensions sampled were activity, tendency to work for food and reactivity to diet adulteration. Three different methods were employed to produce the increased weight gain: electrolytic VMH lesions, cuts lateral to the VMH, and cuts anterior to the VMH. The results confirmed the decreased activity, decreased tendency to work for food, and increased reactivity to taste manipulation previously reported for VMH-lesioned animals. Cuts lateral to the VMH were indistinguishable from lesions on each of these measures. Cuts anterior to the VMH resulted in no change in normal activity level or in the tendency to work for food but did result in increased reactivity to taste manipulation. The results are consistent with the notion of a medial-lateral system controlling some facit of energy balance and of an anterior system underlying a sex dependent part of the weight gain in female rats.

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INTRODUCTION

The hyperphagic syndrome caused by lesions of the ventromedial hypothalamus (VMH) is characterized by increased food intake (Anand, 1961; Brobeck, Tepperman & Long, 1943), viciousness (Wheatley, 1944), decreased spontaneous activity (Hetherington & Ranson, 1942; Teitelbaum, 1957), elevated reactivity to taste properties of food ("finickiness"; Teitelbaum, 1955), a decreased tendency to work for food (Miller, Bailey & Stevenson, 1950; Teitelbaum, 1957), altered lipid metabolism (Han, 1967), a decrease in growth hormone secretion (Han, 1967), and a disturbance of sex hormone secretion (Sawyer & Robinson, 1956). The work of a number of investigators seems to suggest that the various aspects of the syndrome are due to a collection of independent disturbances which do not invariably occur together. Ingram (1952), Skultety (1966) and Anand, & Brobeck (1951) have each noted a lack of correlation between obesity and viciousness. Graff and Stellar (1962) have found the lesions which produce maximal hyperphagia to be different from those which produce maximal finickiness. Both Hetherington and Ranson (1942) and Brooks (1946) have shown that the decrease in spontaneous activity is not invariably associated with either hyperphagia or obesity. The altered lipid metabolism and disturbance of growth hormone also do not appear to be invariant features of the syndrome. It has been reported that only 30% of the VMH-lesioned animals accumulate excess fat when pair-fed with non-lesioned controls (Han & Young, 1964) and that the interruption of growth due to the disturbance of the hormone can be alieviated by replacement injections without altering the obesity (Han, 1967).

While this evidence seems to make clear the likelihood of multiple disturbances underlying the hyperphagic syndrome, the evidence is limited and largely of a correlational nature. There have been no systematic

investigations into which of these effects are due to entirely separate causes. This is not surprising since the lesion technique does not lend itself to the separation of overlapping neural systems underlying different behaviors.

Recently the complementary technique of brain cuts has been used to study the hyperphagic syndrome. Cuts anterior (Albert, Storlien, Albert & Mah, 1971) or lateral (Albert & Storlien, 1969; Sclafani & Grossman, 1969; Gold, 1970a,b) to the VMH have both been found to produce increased weight gains. The only evidence on behavioral accompaniments of increased weight gains following cuts is that of Sclafani and Grossman (1969). They found no difference between cuts lateral to the VMH and VMH lesions on measures of reactivity to taste. However, their results must be treated with caution because, unlike other authors (Graff & Stellar, 1962) they also found no difference between VMH lesions and controls.

The present experiments are concerned specifically with whether the weight gain produced by VMH lesions can be separated from the behavioral changes which might interact with it. The behavioral characteristics investigated are lowered spontaneous activity, decreased tendency to work for food, and increased reactivity to taste manipulation. The design allows for comparison along these dimensions when the weight gain is produced by three different methods: electrolytic lesions of the VMH, cuts lateral to the VMH, and cuts anterior to the VMH. The comparison of the behavioral accompaniments produced by lesions and cuts is of particular interest because whereas lesions destroy both cell bodies and fibers of passage in the region of the VMH, cuts primarily interrupt only pathways passing in a particular direction. The comparison of the effects of anterior cuts to those of lateral cuts and of lesions is also of special interest because the weight gain following the anterior cuts appears to

be due to an imbalance of female sex hormones (Albert, Storlien, Albert & Mah, 1971) and might therefore involve quite a different syndrome than that resulting from VMH lesions or lateral cuts.

METHOD

The subjects were experimentally naive, female hooded rats (Quebec Breeding Farms) weighing between 160 and 260 grams at the time of surgery. Three types of surgery were performed: electrolytic lesions of the VMH, cuts at the lateral or anterior borders of the VMH, and cutter guide stab wounds for the operated controls.

Lesions were produced electrolytically using an electrode of twisted (0.25 mm dia.) stainless-steel wire, insulated at all but the cross-sectional tip. The lesion circuit was completed through a cathode connected to the tail. The D-C lesioning current was 2 ma for 15 seconds at coordinates A 5.8, L$\pm$0.5, and H 9.0 (deGroot Atlas coordinates; deGroot, 1959).

Brain cuts were made with a device which has been described previously (Albert, 1969). The cutter consists of an outer guide cannula (21 gauge stainless-steel hypodermic tubing) which is stereotaxically lowered into the brain of an anesthetized animal. With the guide cannula in place, a 26 gauge insert with a cutting blade of stainless-steel wire (0.15 mm dia.) is lowered into the cannula. When the cutting blade reaches the lower slit, it extends and cuts through the neural tissue as it is lowered further. The cutting blade is then removed and the guide cannula raised out of the brain.

Surgical procedure for the operated controls was similar to that for the cut animals. The cutter guide was lowered into the brain but no cut was made.

Upon completion of the experiments the subjects were killed, the brains fixed in formal-saline, sectioned at 40 microns, and every fifth section stained with thionin.

TESTING PROCEDURE

Post-operatively, subjects were maintained on free access to Purina Lab Chow pellets and tap water. The Lab Chow pellets were placed on the floor of the cage as well as in the food hoppers. Also available was one tin (40 grams dry weight) of wet mash per day. Subjects were weighed daily for 5 to 7 days. At the end of this period animals in the lesion and two cut groups which were gaining weight at a rate above the control level were selected to continue on in the study. Previous results obtained in our laboratory have shown that animals with badly misplaced lesions or cuts extending into the anterior hypothalamus or lateral hypothalamus gain weight at or below the control level (Albert & Storlien, 1969).

Activity Measurement. Activity cages and running wheels were used to assess activity in the animals which met criterion for weight gain. The activity cages consisted of across 7" by 10" by 7" deep Wahmann Individual cages, with a single photocell placed across the width. The photocell beam was 1 1/4" above the floor and equidistant from either end. Each beam breakage activated a Rustrack recorder and the activity score represents counts made from the Rustrack chart paper. The running wheels were LaFayette Instrument Co. Model A34 activity wheels, with an attached counter, which indicated the number of wheel turns.

Activity measurement was carried out over 8 days under both food-deprived and non-deprived conditions. On the first 2 days the animals were run in the activity cages for 2 hours followed by 2 hours in the running wheels with free access to food and water. On day 3 food was removed from the home cages. On days 4 and 5 the animals were again run as on the first 2 days with the exception that they were 19 hours deprived at the beginning

of activity measurement. On day 4, following the test periods, they were fed for 1 hour in their home cages. Free access to food was reinstated following the test conditions on day 5, and day 6 was spent free feeding in the home cages. The final two days, days 7 and 8, the animals were again run in the activity wheels for 2 hours per day and had free access to food. These final two days acted as a control for learning effects in the running wheels. Water was available continuously throughout.

Bar Pressing for Food. Following the final day in the activity wheels subjects were food-deprived for 24 hours. They were then placed in Scientific Prototype Model A 106 lever-press boxes. Arbitrarily, each animal was required to press 100 times for food before being removed from the lever-press box. Most animals accomplished this within 1 hour and all did so within 24 hours. No difference was noted between any of the groups in time taken to learn the required response.

After learning the bar-press response, 4 days were allowed for stabilization on a fixed ratio schedule in which the animal received one pellet of food (45 mg Noyes pellet) for each bar-press (FR 1). On each pair of days thereafter, the animal was successively required to make more bar-presses for each food reward. The reinforcement schedules used were FR 4, FR 16, FR 64 and FR 128. Bar pressing for food was allowed for 1 hour per day throughout the experiment. In order to maintain the animals at a fairly stable weight, 1 hour of free feeding in the home cage on Purina Lab Chow pellets was allowed per day. This period of free feeding always began 1 hour after termination of the lever-pressing.

Diet Adulteration. Following the experimental tests of bar-pressing for food the effect of the cuts and lesions on food intake was assessed. The animals were first habituated to a liquid diet (commercially

available Metrecal[1]) for 4 days. Following this their intake was measured when the diet was adulterated with each of the following: 1.0% saccharin, 0.5% saccharin; 0.05% quinine hydrochloride, and 0.025% quinine hydrochloride. With 2 days of unadulterated Metrecal between each presentation, each subject received each level and type of adulteration in a counterbalanced design controlling for order effects. In all cases consumption was measured over a 24-hour period, with water available at all times.

It was found that through handling and movement of the cages 3-4 grams of Metrecal was lost per bottle per day. This amount lost was not subtracted from any score. It was also found that outside of transient cases of diahorrea, the animals maintained themselves well on the Metrecal and with 2 exceptions (the data of which are discarded) the Metrecal did not coagulate within the 24-hour period.

Statistical Analysis. An analysis of variance followed, where appropriate by Neuman-Keuls test for post-hoc comparisons (Winer, 1962) was used throughout for statistical evaluation of the results.

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1. The Metrecal used in the present experiments was generously supplied by Mead-Johnson Canada Limited.

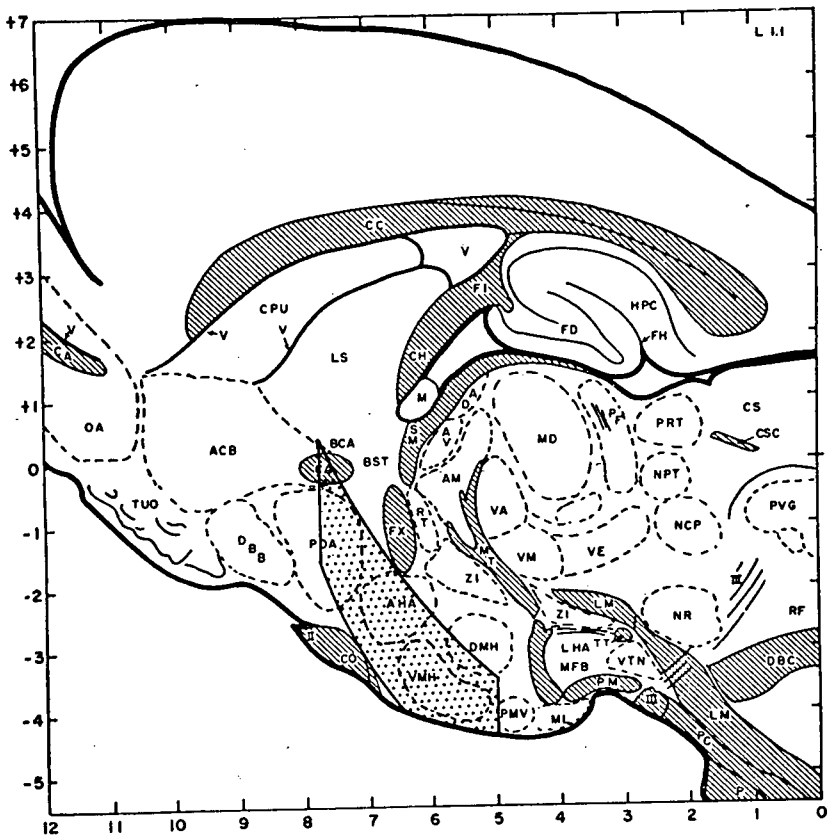
RESULTS

Animals were selected for inclusion in the VMH lesions, Lateral Cut and Anterior Cut groups on the basis of histological examination. This was done so that the groups included only animals which had cuts and lesions that previous results (Anand & Brobeck, 1951; Albert & Storlien, 1969; Albert et. al., 1971) have shown produce maximal weight gains. The criterion for inclusion in the VMH-lesion group was that the lesions destroy the entire VMH bilaterally. Damage to the dorsomedial nucleus was allowed but not destruction anterior to the VMH or lateral to the fornix. Existing evidence indicates that anterior or lateral hypothalamic damage may attenuate the effect of VMH lesions (Skultety, 1966; Anand & Brobeck, 1951). Animals in the Lateral Cut group had cuts (Fig. 1a,b) which extended the entire length of the VMH. The cuts did not intrude into the VMH nor extend more than 0.5 mm lateral to the fornix. The lateral cuts also extended dorsally above the most dorsal aspect of the ventromedial nuclei and ventrally to the base of the brain. Animals in the Anterior Cut group had cuts (Fig. 2a,b) which were in the coronal plane between the anterior tip of the VMH and 0.5 mm anterior to this point. These cuts extended approximately 2 mm lateral at the level of the anterior commissure and narrowed to 0.5 mm lateral at the base of the brain.

The magnitude of postoperative weight gains (Fig. 3) and the group differences are in good agreement with previous results (Albert & Storlien, 1969; Albert, Storlien, Albert & Mah, 1971). Animals in the VMH lesioned group (N=6) gained an average of 70 g., significantly more ($p<.01$) than any other group. The Anterior Cut (N=6) and Lateral Cut (N=7) groups gained an average of 35 and 36 g respectively. Both groups gained significantly more ($p<.01$) than either the Operated Control (8 g; N=8) or Unoperated Control (10g; N=9) groups. The control groups themselves did not differ statistically ($p>.20$).

Figure 1. (a) A saggital section (reporduced from de Groot, 1959) showing the approximate extent of the lateral cuts in relation to the VMH. The actual location of the cut is lateral to the VMH.

(b) A coronal section (40 microns thick, thionin stain) at the level of the VMH showing the two lateral cuts in an animal which gained 34 grams in 5 days post-operatively. Arrows indicate extent of cuts.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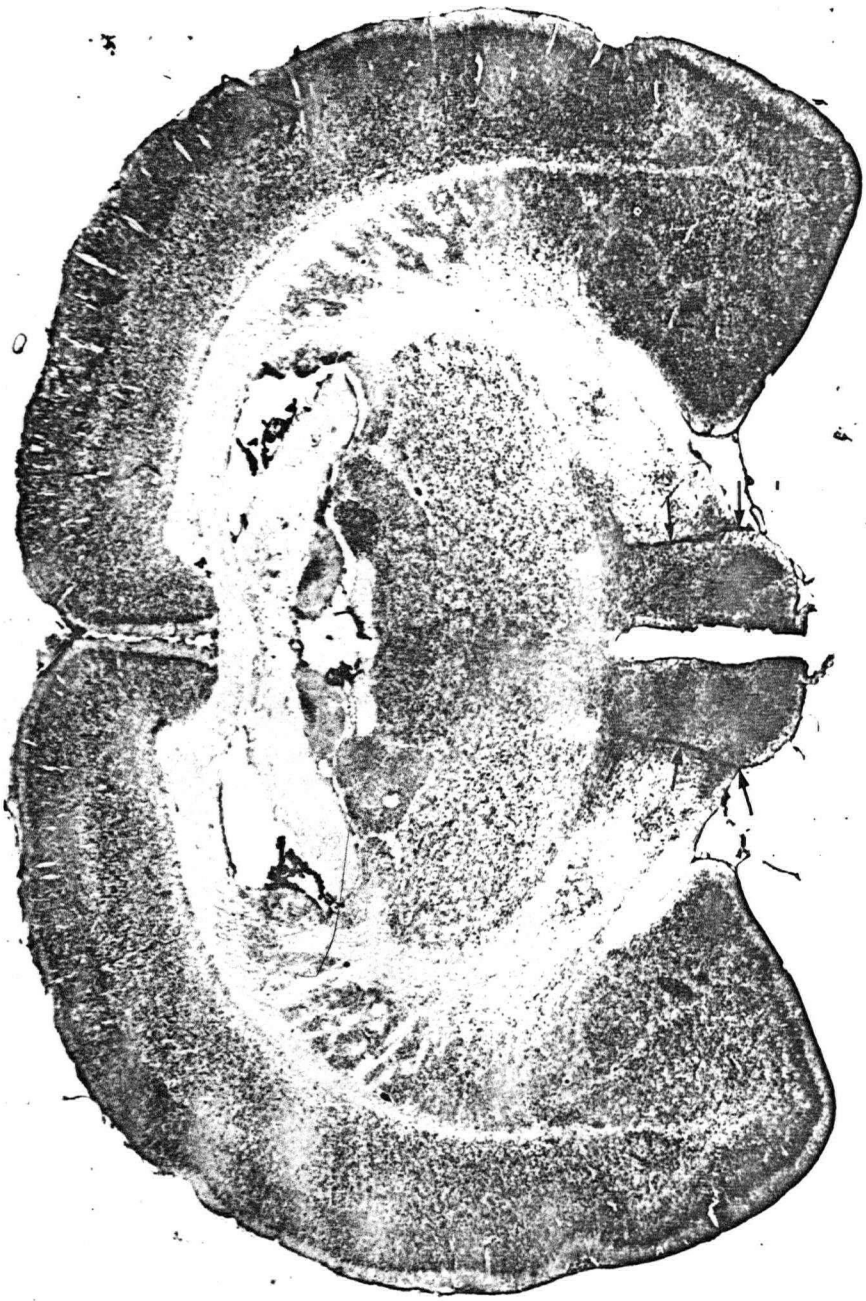

Figure 2. (a) A coronal section (reproduced from de Groot, 1959) showing the approximate extent of the anterior cuts. The actual cut is slightly anterior to this section.

(b) A horizontal section through the VMH showing the anterior cuts in an animal which gained 32 grams in the 5 days postoperatively. The anterior cuts normally opened slightly along the ventricle. Outer arrows indicate ends of each cut.

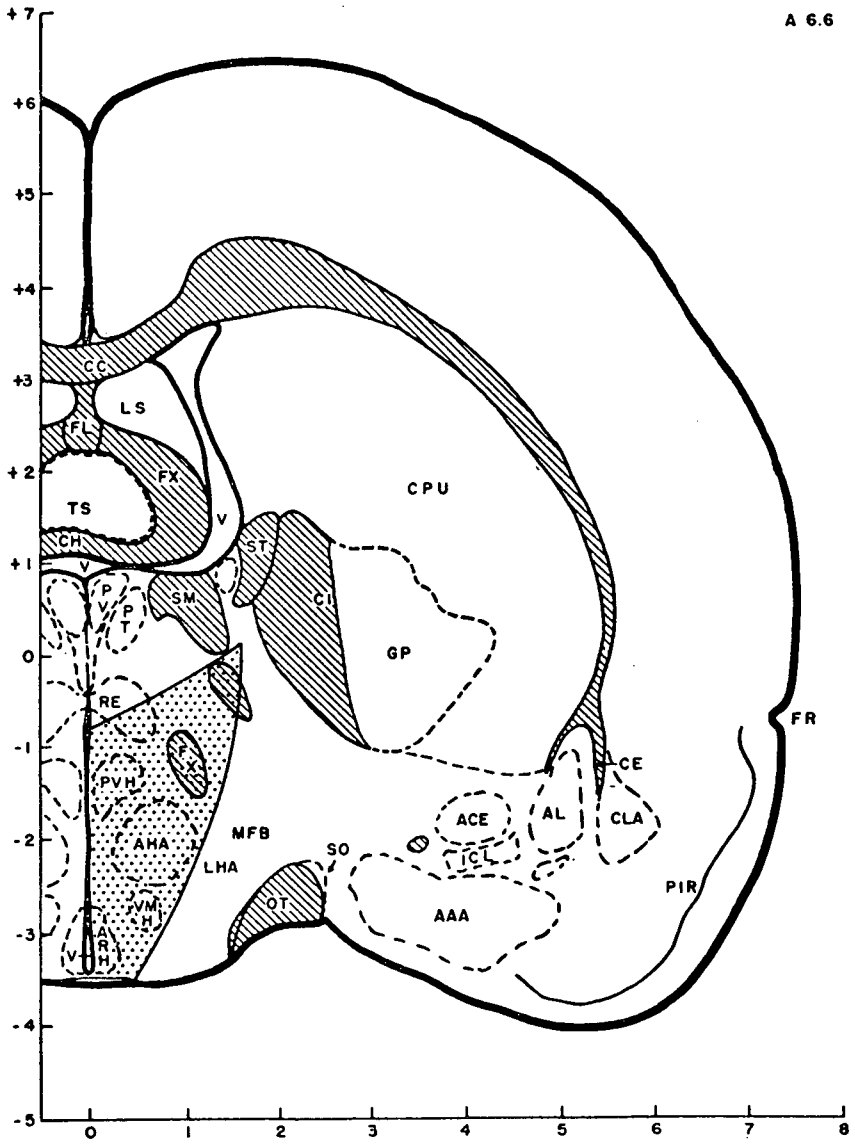
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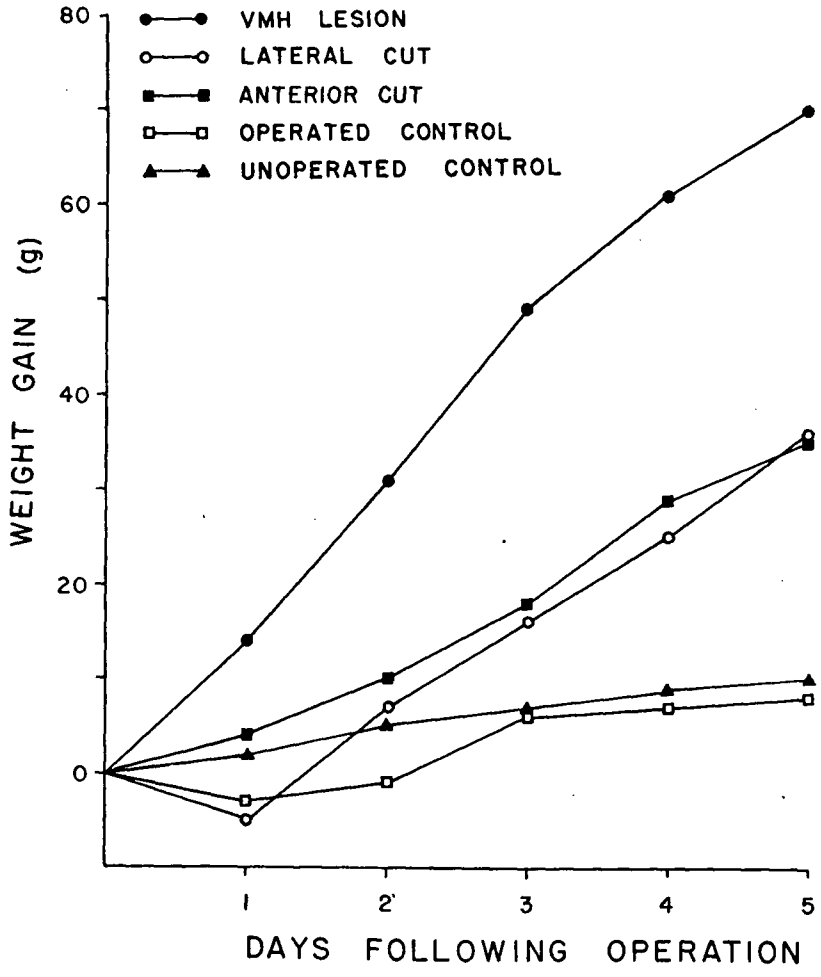
Figure 3. Mean weight gain (g) for each of the experimental groups over the first five days postoperatively.


VMH LESION
LATERAL CUT
ANTERIOR CUT
OPERATED CONTROL
UNOPERATED CONTROL
WEIGHT GAIN (g)
80
60
40
20
0
1
2
3
4
5
DAYS FOLLOWING OPERATION

Activity Measurement. The results of the measurement in the activity cage and running wheel are shown in Table 1. With the activity cages, the two scores per group represent the mean number of photocell beam breakages over the 2 days non-deprived and the 2 days deprived respectively. The first 15 minutes was not included in an attempt to minimize any influence on the results due to handling. Since a comparison between the operated and unoperated control groups showed that the differences between these two groups did not approach statistical significance ($p>.20$), the scores for these were combined for the overall between groups analysis.

There were significant ($F=14.2$; $p<.001$) differences between groups with respect to level of postoperative activity. The VMH Lesion and Lateral Cut groups were significantly ($p<.01$; $p<.05$) less active overall than the Control group. No other pair differences were significant. Food deprivation did increase activity overall ($F=20.0$; $p<.001$) but since the operative treatment by deprivation interaction did not approach significance ($F=1.0$), it would appear that all groups reacted with similar increased activity to deprivation.

Table 1 also contains the results of the wheel running measure of activity. The "non-deprived" scores are the means over 4 days of no food deprivation and the "deprived" scores are the means over the 2 days deprived. The total two hour session was used for each score since the effects of handling were minimized by allowing the animals to quiet in the side cage for approximately 10 minutes before opening the sliding door which permitted access to the wheel. Again, operated and unoperated control groups did not differ significantly ($p>.20$) and were combined for the between group analysis.

As with the activity cage, there were significant ($F=3.4$, $p<.05$)

Table 1

Mean number of activity responses per test period for each group averaged over the total number of days spent in each condition (standard deviation in brackets). Non-deprived indicates ad llb. access to food. Deprived indicates prior 19 hours spent with no food available. Activity Cage: Mean number of photocell beam breakages in the last 1 3/4 hours of the 2 hour test period. Activity Wheel: Mean number of activity wheel revolutions in the total 2 hour test period. Standard deviations for each mean score appear in brackets following the mean.

Group	N	Activity Cage		Activity Wheel	
		Non-deprived	Deprived	Non-deprived	Deprived
VMH lesion	6	54 (19.2)[a]	142 (64.6)[a]	7 (7.5)[b]	11 (11.6)[b]
Lateral cut	7	104 (33.6)[a]	125 (38.5)[a]	9 (6.2)[b]	14 (15.1)[b]
Anterior cut	6	122 (46.8)	162 (27.0)	79 (71.1)	119 (139.4)
Control	17	165 (45.6)	224 (74.1)	67 (59.9)	152 (123.4)

a differ from Control group, $p < .05$

b differ from Control and Anterior Cut groups, $p < .05$

differences between groups with respect to level of postoperative activity. Both the VMH Lesion and Lateral Cut groups were significantly ($p<.05$) less active than either the Anterior Cut or Control groups. The striking suppression of activity following lesions and lateral cuts is shown in Table 1. No other pair differences were significant. As with the activity cage, there was a significant increase in activity with food deprivation ($F=8.1$, $p<.01$) but the operative treatment by deprivation interaction again did not approach significance ($F=1.0$).

Bar-Press for Food. Fig. 4 contains, for each group the mean bar-press rate over the last 2 days at FR 1 and over the 2 days on each of the other schedules. The most striking result with the bar-press is that while all groups pressed at similar rates for FR1, FR4 and FR16, there is a dramatic drop in rate of pressing on FR 64 and FR 128 schedules for both the VMH Lesion and Lateral Cut groups. In contrast, the Anterior Cut and Control groups all continued to increase press rate up to and including FR 128. The VMH Lesion and Lateral Cut groups differed from all other groups ($p<.01$) at FR 128. These same differences approached significance at FR 64. No other pair comparisons were significant.

Diet Adulteration. The effect of saccharin and quinine hydrochloride adulteration on intake of the liquid diet is shown in Table 2. The Operated and Unoperated Control groups again did not differ significantly ($p>.20$) on any measure and were combined for analysis.

Three aspects of the results are important. First, the VMH Lesion and Lateral Cut groups stabilized at significantly higher intakes of unadulterated Metrecal than the Control group ($p<.05$). The stabilized intake of the Anterior Cuts group was also greater than that of the Control group but this difference did not reach statistical significance ($p>.10$). Second, the VMH Lesion ($p<.05$; $p<.05$), Lateral Cut ($p<.01$;

Figure 4. Mean number of responses per hour as a function of FR schedule. The mean for FR 1 was obtained over the last 2 days of training and the means for the remaining schedules are taken over both days on that particular schedule.

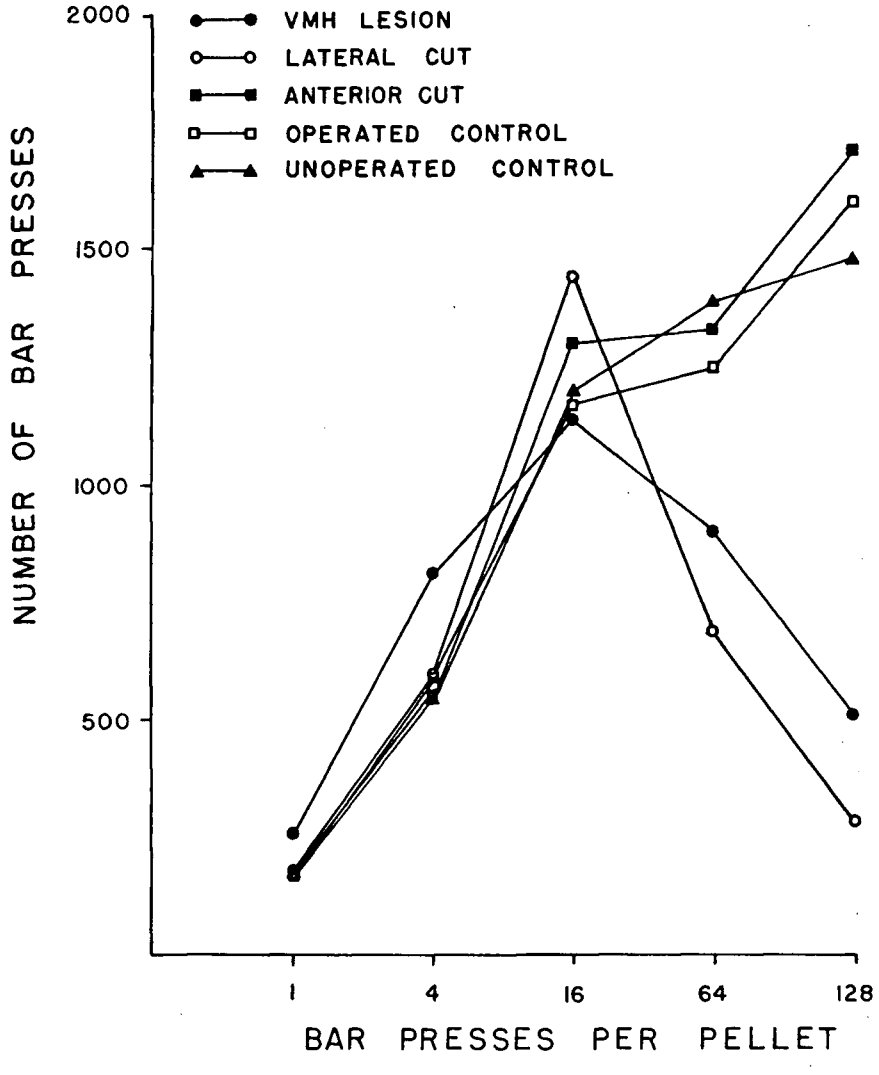

VMH LESION
LATERAL CUT
ANTERIOR CUT
OPERATED CONTROL
UNOPERATED CONTROL
NUMBER OF BAR PRESSES
2000
1500
1000
500
1
4
16
64
128
BAR PRESSES PER PELLET

Table 2

Mean daily intake (g) of the liquid diet. The stabilization score represent the mean 24 hour intake averaged over the last 2 days of inadulterated Metrecal prior to the first presentation of adulterated diet. All other scores are the mean 24 hour intakes for the single presentation of each type of level of adulterating substance. Standard deviations for each mean score appear in brackets following the mean.

			Saccharin		Quinine hydrochloride	
Group	N	Stabilize	1.0%	0.5%	0.025%	0.05%
VMH Lesion	6	57 (9.1)[a]	28 (7.7)[a,b]	35 (12.2)[b]	11 (5.1)[a,b]	7 (5.7)[a,b]
Lateral cut	7	63 (9.7)[a]	39 (15.1)[b]	38 (12.0)[b]	9 (3.5)[a,b]	9 (3.0)[a,b]
Anterior cut	6	53 (9.6)	39 (14.5)[b]	55 (11.6)	8 (5.2)[a,b]	8 (1.9)[a,b]
Control	17	46 (7.8)	44 (9.5)	50 (12.5)	30 (11.4)[b]	18 (7.9)[b]

a differ from Control group, $p < .05$

b differ from stabilized intake, $p < .05$

$p<.05$), and Anterior Cut ($p<.01$; $p<.05$) groups all ate less than controls on both the 0.025% and 0.05% quinine conditions respectively. The third important aspect of the results was the tendency to reduced intake with saccharin adulteration for the Lesion and both cut groups. This proved significant ($p<.01$) for both levels of adulteration for the VMH Lesion and Lateral Cut groups and significant ($p<.05$) for the Anterior Cut group at the 1.0% saccharin level of adulteration.

Summary of Results. Both the VMH Lesion and Lateral Cut groups were significantly different from the controls on each behavioral measure. Both groups gained more weight postoperatively than controls, were less active, slowed bar-press rate dramatically on increasing FR reinforcement schedules, and reacted more to both sweet and bitter adulteration of diet. The Lateral Cut group was not significantly different from the Lesion group on any measure except for a slower rate of postoperative weight gain. The Anterior Cut group differed from the controls only with respect to its higher postoperative rate of weight gain and its greater reactivity to taste.

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DISCUSSION

The most surprising aspect of the present experiments is the lack of difference in behavioral characteristics between animals with cuts lateral to the VMH and those with VMH lesions. Animals with each of these disturbances showed decreased activity, increased reactivity to taste, and decreased willingness to work for food. These effects are similar to those obtained previously with VMH lesions (Miller et. al., 1950; Teitelbaum, 1955 and 1957) except for the results with saccharin adulteration. The animals in the present experiments tended to decrease food intake when the liquid diet was sweetened above its baseline level, instead of increasing food intake in response to the increased sweetness. The reason for this may be that the normal Metrecal diet was already quite sweet and that further saccharin tended to decrease its palatability by making it too sweet. The only difference found between the VMH lesioned animals and those with lateral cuts was that the lesioned animals showed a significantly greater postoperative weight gain.

The failure to find behavioral differences between VMH lesioned animals and those with lateral cuts does not exclude the possibility of a series of independent effects caused by the interruption of several distinct pathways by the same lesion or cut. The results do, however, offer substantial support for those theories which account for both the increased weight gain and some behavioral changes in terms of damage to one fiber system. One such explanation is in terms of a medial-lateral system controlling energy intake. If the medial hypothalamic area is conceived of as an area monitoring energy balance, cuts lateral to it or lesions of it could result, via the mechanism of a lateral hypothalamic system, in the organism constantly behaving as though it were experiencing

an energy deficit. From this point of view, the increased weight gain can be looked upon as a result of excess food intake in response to a neural system that is constantly signalling an energy deficit and the decreased tendency to work for food could occur as a result of an attempt to conserve energy. The apparent increased sensitivity to taste in spite of increased hunger might be explained in terms of Jacobs' (1966) findings of increased finickiness with increased deprivation. Alternatively, the increased sensitivity to taste may represent a rather non-specific disturbance of the feeding system; disturbances of many parts of the brain associated with food intake have been observed to result in changes in sensitivity to taste [amygdala (Kemble and Schwartzbaum, 1969), septum (Beatty and Schwartzbaum, 1967), lateral hypothalamus (Booth and Quartermain, 1965)]. A closely related possibility is that the increased weight gain following VMH lesions is due to an increased tendency to store fat (see Teitelbaum, 1961). Assuming this tendency is very severe, the result would be a deficit in energy expendible for other purposes and the behavioral effects would be in terms of an actual energy deficit.

There is at least one other interpretation of the increased weight gain following VMH Lesion which tends to be supported by the present findings. This is the suggestion of Grossman (1966) that all of the effects including the increased weight gain are due to an increase in affective responsivity. Thus, reduced activity and decreased willingness to work for food are interpreted in terms of the negative aspects of these behaviors while increased food intake and consequent increased weight are attributed to the positive response to the taste properties of palatable food.

The findings with anterior cuts while less surprising are also important. The present experiments find an increased weight gain as has

been reported previously (Albert et. al., 1971). However, in contrast to the effect with lateral cuts, there are no significant differences from controls in activity level or tendency to work for food. There is an increased sensitivity to diet adulteration. These findings are an interesting contrast to those with lateral cuts and VMH lesions because they show that an increased rate of weight itself does not necessarily cause the various behavioral changes and in fact could account for the fact that lesions in the region of the VMH sometimes seem to cause an increased weight gain but little behavioral change (see introduction). It has been suggested that the increased weight gain with anterior cuts is due to a disturbance of female sex hormones (Albert et. al., 1971). The present findings are generally consistent with that point of view in that both anterior cuts and ovariectomy (Wade & Zucker, 1970) seem to cause an increased sensitivity to diet adulteration.

Albert, D. J. A simple method of making cuts in brain tissue. Physiology and Behavior, 1969, 4, 863 - 864.

Albert, D. J., and Storlien, L. H. Hyperphagia in rats with cuts between the ventromedial and lateral hypothalamus. Science, 1969, 165, 599 - 600.

Albert, D. J., Storlien, L. H., Albert, J. G., and Mah, C. J. Obesity following disturbance of the ventromedial hypothalamus: a comparison of lesions, lateral cuts, and anterior cuts. Physiology and Behavior, 1971, in press.

Anand, B. K. Nervous regulation of food intake. Physiological Review, 1961, 41, 677 - 708.

Anand, B. K., and Brobeck, J. R. Hypothalamic control of food intake in rats and cats. Yale Journal of Biology and Medicine, 1951, 24, 123 - 140.

Beatty, W. W., and Schwantzbaum, J. S. Enhanced reactivity to quinine and saccharin solutions following septal lesions in the rat. Psychonomic Science, 1967, 8, 483 - 484.

Booth, D. A., and Quartermain, D. Taste sensitivity of eating elicited by chemical stimulation of the rat hypothalamus. Psychonomic Science, 1965, 3, 525 - 526.

Brobeck, J. R., Tepperman, J., and Long, C. N. H. Experimental hypothalamic hyperphagia in the albino rat. Yale Journal of Biology and Medicine, 1943, 15, 831 - 853.

Brooks, C. McC. The relative importance of changes in activity in the development of experimentally produced obesity in the rat. American Journal of Physiology, 1946, 147, 708 - 716.

De Groot, J. The rat brain in stereotaxic coordinates. N. V. Noord-Hollandsche Uitgevers Maatschappij, Amsterdam, 1959.

Gold, R. M. Hypothalamic hyperphagia produced by parasaggital knife cuts. Physiology and Behavior, 1970a, 5, 23 - 25.

Gold, R. M. Hypothalamic hyperphagics: males get just as fat as females. Journal of Comparative and Physiological Psychology, 1970b, 71, 347 - 356.

Graff, H., and Stellar, E. Hyperphagia, obesity, and finickiness. Journal of Comparative and Physiological Psychology, 1962, 55, 418 - 424.

Grossman, S. P. The VMH: a center for affective reactions, satiety, or both? Physiology and Behavior, 1966, 1, 1 - 10.

Han, P. W. Hypothalamic obesity in rats without hyperphagia. Transactions of the New York Academy of Science, 1967, 30, 229 - 243.

Han, P. W., and Young, T. K. Obesity in rats without hyperphagia following hypothalamic operations. Chinese Journal of Physiology, 1964, 19, 143 - 156.

Hetherington, A. W., and Ranson, S. W. The spontaneous activity and food intake of rats with hypothalamic lesions. American Journal of Physiology, 1942, 136, 609 - 617.

Ingram, W. R. Brainstem mechanisms of behavior. Electroencephalography and Clinical Neurophysiology, 1952, 4, 397 - 406.

Jacobs, H. L. in Proceedings of the 7th Internation Congress on Nutrition 1966, Vol. 2. Regulation of Hunger and Satiety, pages 17 - 29.

Kemble, E. D., and Schwartzbaum, J. S. Reactivity to taste properties of solutions following amygdaloid lesions. Physiology and Behavior, 1969, 4, 981 - 985.

Miller, N. E., Bailey, C. J., and Stevenson, J. A. F. "Decreased hunger" but increased food intake resulting from hypothalamic lesions. Science, 1950, 112, 256 - 259.

Sawyer, C. N., and Robinson, B. Separate hypothalamic areas controlling pituitary gonadotropic function and mating behavior in female cats and rabbits. Journal of Clinical Endocrinology, 1956, 16, 914 - 915.

Sclafani, A., and Grossman, S. P. Hyperphagia produced by knife cuts between the medial and lateral hypothalamus in the rat. Physiology and Behavior, 1969, 4, 505 - 507.

Skultety, F. M. Changes in calorie intake following brainstem lesions in cats. III Effects of lesions of the periaqueductal gray matter and rostral hypothalamus. Archives of Neurology, 1966, 14, 670 - 680.

Teitelbaum, P. Sensory control of hypothalamic hyperphagia. Journal of Comparative and Physiological Psychology, 1955, 48, 158 - 163.

Teitelbaum, P. Random and food-directed activity in hyperphagic and normal rats. Journal of Comparative and Physiological Psychology, 1957, 50, 486 - 490.

Teitelbaum, P. Disturbances in feeding and drinking behavior after hypothalamic lesions. In Nebraska Symposium on Motivation; M. R. Jones, Ed. Lincoln: University of Nebraska Press, 1961, 39 - 69.

Wade, G. N., and Zucker, I. Hormonal modulation of responsiveness to an aversive taste stimulus in rats. Physiology and Behavior, 1970, 5, 269 - 273.

Wheatley, M. O. The hypothalamus and affective behavior in cats. Archives of Neurology and Psychiatry (Chicago), 1944, 52, 296 - 316.

Winer, B. J. Statistical Principles in Experimental Design. New York: McGraw-Hill, 1962.