TASTE REACTIVITY IN RATS WITH VENTROMEDIAL-HYPOTHALAMIC
AND SEPTAL LESIONS

by

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Thanks also go to Dr. Lawrence M. Schoen for the development of the computer summation program which made the data analysis possible.

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TABLE OF CONTENTS

TABLE LISTINGS

FIGURE LISTINGS

INTRODUCTION

A large body of literature has characterized animals with particular brain lesions as hyperresponsive to taste stimuli. Ventromedial hypothalamic (VMH) preparations, for example, typically overeat and become obese on high-fat diets (Corbit & Stellar, 1964) or sweetened diets (Teitelbaum, 1955). In contrast, these same animals show hypophagia and weight loss, relative to controls, on diets adulterated with bitter substances (Graff & Stellar, 1962). This relative overconsumption of palatable foods and underconsumption of unpalatable foods is known as "positive" and "negative finickiness", respectively (Weingarten, 1982).

Animals with lesions of the septum also show finicky eating behavior. For example, Beatty and Schwartzbaum (1968) found that septal preparations showed an increased intake of sucrose solution relative to controls, and others have demonstrated a relative decrease in the intake of quinine hydrochloride solution (Carey, 1971; Donovick, Burright, & Zuromski, 1970).

The results of initial studies, though informative, are not conclusive because the experiments did not control for two relevant variables: the influence of postingestive factors and body weight on ingestion (Weingarten, 1982). Most of the above studies explicitly or implicitly assume that the duration and amount of ingestion are primarily determined by oropharyngeal

1

sensations; the hypothesis is that VMH and septal animals have heightened orosensory responses to food which lead to increased or decreased intake depending upon the nature of the foodstuff. In focusing on oropharyngeal factors, many studies have neglected the possibility that the lesions may affect intake by altering behavioral responses to postingestive and/or postabsorptive cues (Weingarten, 1982). For example, an animal with a lesion could conceivably overconsume because it lacked the capability to respond to gastric distension or underconsume because of a disrupted central mechanism which responds to afferent chemoreceptor signals from the gut.

Additionally, many studies have employed ad libitum feeding, allowing rats with lesions to become obese prior to tests of intake. In such studies, one cannot discount the possibility that taste hyperresponsivity, though existant, may result secondarily as a consequence of obesity and not from the lesions per se (Weingarten, 1982). Indeed, some researchers have found that nonobese rats with VMH lesions do not underconsume quinine-adulterated foodstuffs and, when maintained on a diet of this type, they regulate their body weight at a level near that of control subjects (Ferguson & Keesey, 1975).

An approach which circumvents the above problems is to use a sham-feeding technique in conjunction with restricted- or pair-feeding. Sham-feeding involves the

2

use of a gastric fistula which allows the contents of the stomach to drain freely during ingestion, eliminating postingestive influences. This type of procedure was employed by Weingarten (1982). In a 30-minute intake test, he found that sham-fed VMH animals ingested significantly more 18% and 30% sucrose solution (14 ml and 18 ml more, respectively) than sham-fed controls. Using this same technique, Weingarten, Chang, and Jarvie (1983) found that VMH animals did not ingest significantly less sucrose solution (.0025 and .005 M) or wet mash adulterated with quinine hydrochloride, than control animals. These researchers concluded that rats with VMH lesions do display positive finickiness due to oropharyngeal factors, but do not show negative finickiness compared to control rats.

Though the sham-feeding technique represents an advance in isolating and investigating the influence of oropharyngeal factors on ingestion, its use has not been without drawbacks. In the study above on negative finickiness, it is probable that the amount of food ingested was influenced by more than just the amount of quinine adulteration because the foods also contained sucrose or wet-mash. Since it is suspected that VMH animals have a heightened responsivity to palatable foods, it is quite possible that the presence of positive hedonic gustatory components, such as the sweet taste of sucrose,

3

could have masked the effect of quinine on ingestion.

A more general critique of intake measures is that they are only able to gauge finickiness in terms of the amount of food ingested. The processes of deciding when and how much of a food to ingest are ongoing, and involve continuous integration of positive and negative gustatory and olfactory sensations (Grill & Berridge, 1985) along with postingestive information (Schwartz & Grill, 1984). Intake measures are not capable of separating the influences of these components.

The taste reactivity test, developed by Grill and Norgren (1978), is an alternate method of measuring taste responsivity in which a tastant is infused into an animal's mouth via an intraoral fistula. The infusion process typically lasts one minute during which time the animal's responses to the tastant are filmed for later frame-by-frame analysis. The resulting behavior sequences, called "fixed action patterns", are highly stereotypic and form the basis of the taste reactivity analysis.

Grill and Norgren have divided the behavioral responses into two groups: ingestive and aversive. Ingestive responses are consummatory in nature and include mouth movements (MM), tongue protrusions (TP), lateral tongue protrusions (LTP), paw licks (PL), lip flairs (LF), and swallowing (SW). In contrast, aversive responses

4

often serve to expel a tastant from the oral cavity and include gaping (G), chin rubbing (CR), head shaking (HS), face washing (FW), forelimb flailing (FF), paw pushing (PP), fluid expulsion (FE), and passive drip (PD). Locomotion (LO) is also included as an aversive response in some analyses. A brief description of the behaviors follows. (For an illustration of the responses, see Figure 1).

Mouth movements consist of "low amplitude, bilaterally symmetrical movements of the mandible that occur rhythmically (6.6 cycles/sec)" (Grill & Norgren, 1978, p. 267 & 268). Tongue protrusions are rhythmic protrusions of the tongue which break the plane of the upper incisors and occur at a rate of approximately 8.8 cycles/sec. Swallowing invariably involves a tongue protrusion and is analogous to the head-lunging seen in dogs and other species while they lap a liquid. Lateral tongue protrusions usually follow MM and TP and involve "the tongue emerging from the side of the mouth, extending the upper lip laterally as... [it] moves forward" (p. 270). LTPs typically last from 85-215 msec. Lip flairing is an indentation or flairing of the portion of the lips overlying the upper incisors, and generally occurs at a frequency of approximately 1/sec. Paw licking consists of rhythmic extensions of the tongue on the midline toward the forepaws; the paws may be held in front

5

of the nose or on the floor while they are being licked.
Paw licking is less frequently observed than other
ingestive responses but may be idiosyncratic to certain
rats (Flynn, 1985).

<div align="center">

Insert Figure 1 about here

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Gapes often occur at the beginning of a response
sequence and entail rapid retraction of the corners of the
mouth which consequently forms a triangular shape,
exposing the upper and lower incisors. Gapes occur in
bursts of two to six and last approximately 166 msec.
Chin rubbing involves lowering the head and bringing the
mouth in contact with the floor, followed by extension of
the neck and head in a forward swooping motion; the
behavior lasts approximately 500 msec. Chin rubbing is
often followed by head shaking which is the rapid
side-to-side movement of the head at a speed faster than
60 cycles/sec, the resolution limit of the film analysis
(Grill & Norgren, 1978). It is analagous to "wet-dog
shaking" except that it is confined to the head. Head
shaking may cause fluid to be expelled from the mouth, in
which case fluid expulsion is scored. Face washing,
sometimes categorized as grooming (GM), is the only FAP
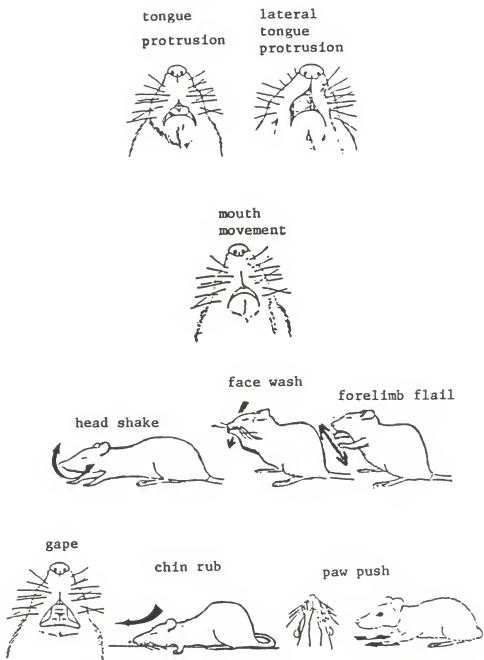which is observed without prior gustatory stimulation.

Figure 1. Taste reactivity responses. Top row: ingestive responses (tongue protrusion, lateral tongue protrusion); second row: neutral response (mouth movement); third and bottom rows: aversive responses (head shake, face wash, and forelimb flail; gape, chin rub, and paw push) (after Grill & Norgren, 1978).

7

It involves the animal simply wiping the front paws over the face while rearing on its hindquarters.

Forelimb flailing is the rapid side-to-side movement of the front paws at a speed faster than 60 cycles/sec, and it follows face washing in response to some tastants. Paw pushing is the final behavior of the rejection sequence and involves rubbing the front paws on the floor in an alternating pattern: as one paw is being retracted, the other is being extended; extension of each forelimb lasts from 166-250 msec (Grill and Norgren, 1978). Passive drip may occur after a prolonged sequence of aversive behaviors. In this case the animal ceases to react and lets fluid drip from its mouth to the floor.

The present study utilized the taste reactivity test to determine the nature of taste hyperresponsivity in rats with VMH and septal lesions. The taste reactivity test is well suited to investigate finickiness in these preparations for several reasons: First, it can be employed in a way which circumvents the confounding effect of postingestive influences because animals need only respond to 1 ml of a tastant. It can also be used in conjunction with a weight-restriction procedure to nullify obesity as a confound. It is relatively precise because it separates consummatory and rejective behavior into individual responses and allows one to analyze differences in the ways that various preparations process gustatory

information. Finally, the taste reactivity test is particularly useful in detecting negative finickiness because, unlike intake measures, it is not constrained by a floor effect. In intake measures increased aversiveness is indicated by decreased consumption which has a lower limit of zero. Therefore, using an intake measure, an animal might be unable to indicate increased aversion above a certain level. In the taste reactivity test, increasing aversiveness is indicated by an increase in the number of aversive responses which are free to increase up to the point of physical limitation (in the present study, this limit was not reached with the concentrations used).

The study addressed two main questions: 1) What is the magnitude of taste hyperresponsivity in septal and VMH animals relative to controls? 2) Is the hyperresponsivity in rats with VMH lesions qualitatively the same as that in rats with septal lesions? Septal and VMH preparations were investigated because of the extensive literature on finickiness in these animals discussed above.

<div align="center">METHOD</div>

Subjects

Twenty-two male Holtzman albino rats, weighing between 513 and 674 g at the outset of the experiment, were individually housed in a room with a 12 hour light/dark (7 a.m. / 7 p.m.) cycle; all testing took place during the light phase. Each animal was handled 10 times

<div align="center">9</div>

preoperatively and 4 times postoperatively to decrease the hyperresponsiveness which follows lesions of both the VMH and septum, and to reduce responses to the novelty of the test chamber. Preoperative handling consisted of removing each animal from its cage, placing it in the test chamber for approximately 10 minutes, and returning it to its home cage. Postoperative handling entailed the above procedure along with the actual infusion of 1 ml distilled water into the oral cavity while the animal was in the chamber.

Surgery

The rats were deprived of food and water for 24 hr prior to surgery. All animals were anesthetized with sodium pentobarbital (Nembutal, 60 mg/kg, IP) and mounted in a stereotaxic instrument with the incisor bar positioned 5.0 mm above the interaural line.

Septal lesions were produced by passing a 1.5-mA anodal dc current through the 0.5-mm uninsulated tip of a stainless steel insect pin (size 00) for 20 seconds, with the electrode tip located 1.5 mm anterior to bregma, 0.5 mm lateral to the midsagittal suture, and 6.0 mm ventral to the surface of the cortex. VMH lesions were produced by using a 2-mA anodal dc current for 15 seconds with the electrode tip located 2.5 mm posterior to bregma, 0.5 mm lateral to the midsagittal suture, and 0.8 mm dorsal to the base of the skull.

All groups of animals were then implanted

10

unilaterally with an intraoral fistula made of polyethylene (PE 100) tubing, placed just anterolateral to the first maxillary molar. The fistula was inserted through an incision in the cheek and brought up subcutaneously to exit on the top of the skull where it was secured with dental acrylic (see Phillips & Norgren, 1970, for details). This allowed for the later attachment of PE 160 tubing which was connected to a syringe pump during tastant infusion. (For an illustration of the fistula in situ, see Figure 2.) Following surgery, all rats were given 150,000 units of bicillin im (Benza-pen: 75,000 units Benzathine, 75,000 units Procaine).

Feeding

On postoperative days 1-3, rats were maintained on a wet mash diet with water ad libitum. On day 4 they started a liquid diet which consisted of sweetened/condensed milk (Borden's) and water in equal proportions, plus a multi-vitamin supplement (Poly-Vi-Sol). On days 8-16, when testing took place, the feeding tubes were removed from the cage 2 hr prior to testing and replaced with a 12-ml portion of liquid diet, which was generally consumed within 10 minutes. After the test period, animals were given a 35-ml portion of liquid diet which was available overnight.

The feeding regimen was maintained to ensure that each animal's gastric content was approximately the same

11

during the test period. The diet provided 74
kilo-calories per day, and previous research has shown
that it affords adequate nutrition and hydration for
normal body maintenance (Schwartz & Grill, 1984).
Apparatus and Testing

Testing took place in a cylindrical acrylic testing
chamber which was 22.2 cm in inside diameter and 25.4 cm
high. The chamber rested on a glass base and was mounted
over a mirror so that the animal's ventral side could be
videotaped during infusion (see Figure 3).

Insert Figures 2 and 3 about here

Before each trial, a syringe was used to flush 1 ml
of distilled water through the animal's fistula to remove
any debris which may have accumulated. Air was then blown
through the fistula to remove the water. Deadspace in the
tubing was minimized by filling it with tastant prior to
attaching it to the animal's fistula. The tubing was then
attached to the infusion pump and the rat was placed in
the chamber for a 5-minute adaptation period.
Unrestricted movement was allowed in the test chamber.

Testing proper consisted of videotaping the animal's
responses to the infusion of 1 ml of a tastant delivered
at the rate of 1 ml/min. Videotaping was done with a
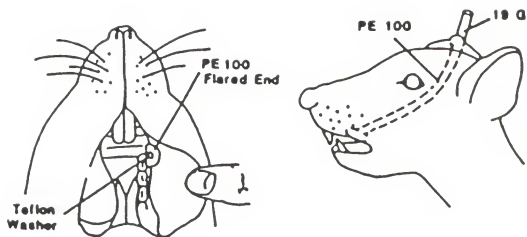Panasonic WV-460 camera outfitted with a Cannon 15-150 mm

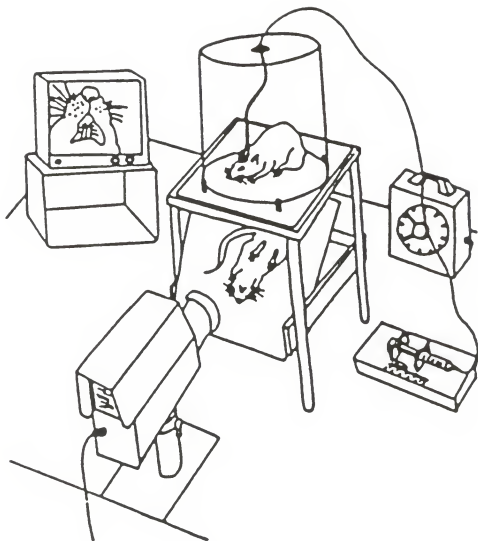Figure 2. A diagram of the intraoral fistula (after Grill & Berridge, 1985).

Figure 3. A diagram of the testing apparatus (after Grill & Berridge, 1985).

zoom-lens, and connected to a Sony model video cassette recorder with a time code generator. The pump and an electronic timer were turned on simultaneously so that the start of infusion marked the start of the trial. Taping continued for approximately 80 sec to ensure the procurement of at least 60 sec of scoreable behavior.

Animals were tested on solutions of sucrose (0.01, 0.03, 0.3, and 1.0 M) and quinine hydrochloride (QHCl) (0.003, 0.0003, 0.00003, and 0.000003 M). The solution concentrations were selected so that the lowest concentration was detectably different from distilled water and that there would be observable differences in taste reactivity between concentrations. More specifically, Schwartz & Grill (1984) demonstrated that tongue protrusions and lateral tongue protrusions increased linearly using the above concentrations of sucrose, and that gapes and chin rubs increased exponentially with the above concentrations of QHCl.

The presentation order of the solutions was designed to detect an order effect and/or a recovery-of-function effect (see Table 1). Because each concentration of sucrose solution was preceded by each concentration of QHCl solution at least once, one could determine if a strong concentration of QHCl solution affected the response to sucrose solution on the subsequent trial. A recovery-of-function effect could be detected by

15

determining if the response to any given tastant changed throughout the eight-day period of testing.

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Insert Table 1 about here

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Taste-Reactivity Scoring

The responses of 18 animals to the 8 tastants were videotaped, yielding 144 trials which were scored using frame-by-frame analysis in conjunction with a micro-computer program designed for this purpose (Schoen, 1986). The program facilitated the division of each 60-second trial into 600 .1-second blocks. The videotape was advanced, frame-by-frame (32 frames per second), and each block was filled with one of the 16 different FAP components which occurred during that .1 second in the trial (as indicated by the elapsed time at the bottom of the monitor). "No Response" (NR) or "No Data" (ND) could also be entered. NR was entered each .1 second that the rat was in full view and motionless; ND was entered when, due to locomotion, the animal's head was not in the picture. Scoring began with the appearance of the first response (usually within 5 seconds after the infusion pump was started) and ended exactly 600 blocks (60 sec) later. A trial consisted of 60 seconds of data in which no more than 10 seconds were scored as "no data" (mean ND was 2.4 seconds). This criterion insured that large numbers of

Table 1

Presentation order of the taste solutions (S = sucrose, Q = Quinine; 1 = lowest concentration, 4 = highest concentration).

				Day				
Animal	1	2	3	4	5	6	7	8
1	q1	s4	q4	s2	q2	s3	q3	s1
2	q2	s1	q1	s3	q3	s4	q4	s2
3	q3	s2	q2	s4	q4	s1	q1	s3
4	q4	s3	q3	s1	q1	s2	q2	s4
5	q1	s1	q2	s2	q3	s3	q4	s4
6	q2	s2	q3	s3	q4	s4	q1	s1

responses were not missed in the frame-by-frame analysis.
The raw data for each trial were then summed by
micro-computer and the sums for each of the 18 different
responses were statistically analyzed. Intra-rater
scoring consistency on the first 20 trials was found to be
96%; that is, 96% of the .1 second blocks were scored with
the same response on the second scoring as on the first
scoring. Ten randomly-selected trials were also rescored
blindly to insure that scorer knowledge of the taste
solution did not affect the scores. Blind-scoring
reliability was found to be 97%.

The data were analyzed using a repeated-measures 3 X
2 X 4 (Group by Tastant by Concentraton within Tastant; n
= 6) ANOVA. In addition, the data were broken down by
tastant and analyzed in 3 X 4 (Group by Concentration)
ANOVAs to provide more specific information.

Three dependent variables were derived by grouping 14
responses into 3 categories as follows: Ingestive (I) =
LTP + TP + SW + LF; Neutral (N) = MM + FW + GM; Aversive
(A) = PD + FF + HS + FE + G + CR + PP. (See the
Introduction section for a description of each response.)

Categorization of responses into ingestive and
aversive types was based primarily on previous research
(Schwartz & Grill, 1984) and on our own experience.

Mouth movements, face washing, and grooming were
combined to form a separate "Neutral" category for the

18

following reasons: Mouth movements were categorized as neutral because they were considered to be a general sampling response to a tastant, and not indicative of ingestion or expulsion. Face washing and grooming were considered neutral because they occurred in the absence of any tastant (often initially after the animal was placed in the test chamber). Furthermore, mouth movements, face washing, and grooming were all negatively correlated with both sucrose and quinine concentration, having correlation coefficients that ranged from -.14 to -.40 (an exception was the correlation of mouth movements with QHCl concentration, with a coefficient of 0). The decrease in neutral responses with increasing solution concentration occurred because other behaviors, directly determined by the solution concentration, increased in frequency and dominated the behavioral repetoir as concentration increased.

Locomotion (LO) was analyzed separately as a fourth dependent variable because it was measured in units of time, while the other components were all measured in frequency.

Histology

Rats were sacrificed with an overdose of anesthetic and perfused intracardially with physiological saline, followed by 10% formal-saline. The brains were removed, stored in formal-sucrose (approx. 30% conc.), and later

embedded in albumin matrix.

Brains were frozen and cut into 40-um sections and every fifth section in the area of the lesion was saved. Brain sections were mounted and stained with cresyl echt violet and lesion placement was microscopically verified.

RESULTS

Histological Findings

Lesions of the septum were found to be homogeneous in form and location but were generally about 0.5 mm anterior to the desired coordinates (see Figure 4). As such, all of the lesions destroyed the anterior 1/2 to 2/3 of the septum but spared parts of the posterior septum, ending at the beginning of the point where the anterior commissure crosses. In most cases the lesions encroached slightly on the ventral midline cortex anterior to the septum and on the olfactory bulbs. Bulb damage was confined to only the most posterior dorsal area. Of the seven animals in the Septal Group, one sustained only minimal damage to the septum and its data were not included in the analysis, leaving six animals in this group.

Lesions of the VMH (Figures 5 and 6) generally destroyed 3/4 to all of this nucleus but were variable in shape and often not bilaterally symmetrical. Because of their variability, they often encroached onto other nuclei: In two animals, part of the anterior hypothalamic area was destroyed, and in two others, the anterior

portion of the mammillary bodies was destroyed.
Additionally, the dorsomedial hypothalamus was damaged in
one animal and the arcuate nucleus was damaged in another.
Lesions in two animals destroyed less than half of the VMH
and their data were not included in the analysis, leaving
six rats in the VMH Group.

Insert Figures 4, 5, and 6 about here

Body Weights

Several statistical analyses were performed to
determine that the animals' body weights were adequately
controlled throughout the experiment. Three one-way
ANOVAs showed that there were no significant differences
between groups in preoperative (just prior to surgery),
pretest (the first day of taste reactivity testing), or
post-test (the last day of taste reactivity testing)
weights. This result indicates that random assignment
produced groups that were adequately homogeneous in body
weight, and that no group gained or lost more weight than
another throughout the experiment. Additionally, the
feeding regimen held weights at a relatively constant
level throughout the test period. Mean pretest/post-test
weights for each group were as follows: Control Group,
627.2/614.7 g; Septal Group, 574.7/578.8 g; VMH Group,
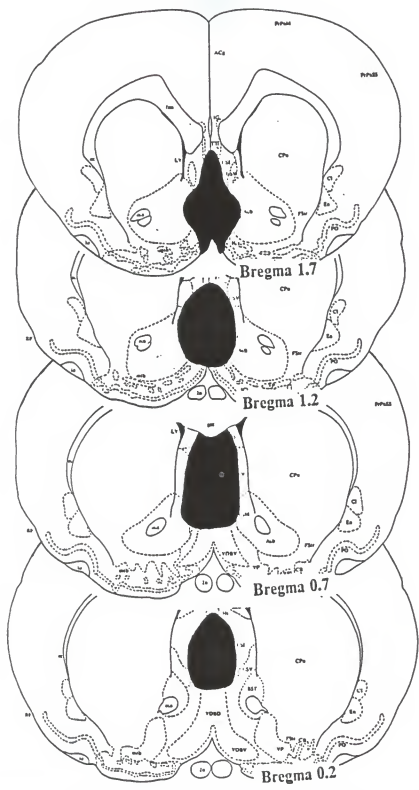586.7/573.3 g. A related-samples t-test showed that, for

Figure 4. Reconstruction of a representative lesion in
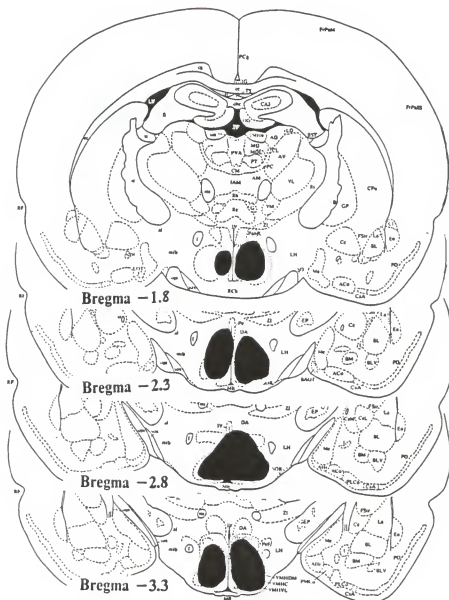the Septal Group (diagram adapted from Paxinos & Watson,
1982).

22

Figure 5. Reconstruction of a representative anterior
lesion in the VMH Group (diagram adapted from Paxinos &
Watson, 1982).

Bregma −2.8

Bregma −3.3
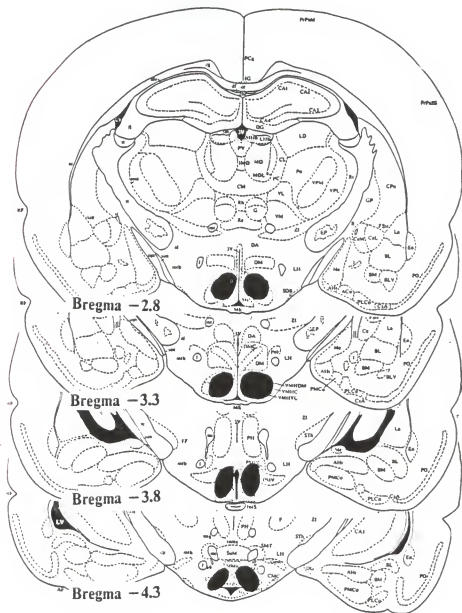
Bregma −3.8

Bregma −4.3

Figure 6. Reconstruction of a representative posterior
lesion in the VMH Group (diagram adapted from Paxinos &
Watson, 1982).

24

the VMH Group, the pre-/post-test weight difference was
significant at the .05 level, although the difference was
in the opposite direction from the dynamic weight gain
often seen in VMH animals. That is, the animals actually
lost a mean of 13.4 g throughout the test period.

Taste Reactivity

Paw licking (PL) and "no response" (NR) were not used
in the analysis. NR was not used because it can be caused
by factors not associated with taste reactivity (e.g., a
fear response to the test chamber) and does not yield
unambiguous information concerning gustatory processes.
PL was not used because post hoc examination of the data
revealed that there were two types, one associated with
ingestion and the other with the rejection of a solution.
The "ingestive" type consisted of licking the front paws
while they were held in front of the face with little
movement. The "aversive" type consisted of rhythmic
downward stroking of the front paws over the protruding
tongue, usually followed by forelimb flailing. The
responses were similar enough that a differentiation was
not made until after the scoring procedure. For this
reason paw licking was removed from the analysis.

Further analysis showed that for the sucrose
solutions, PL did not change significantly with
concentration, nor did the three groups show differences
in frequencies of displaying this response. For QHCl

solutions, PL decreased as concentration increased,
$F(3,45) = 3.21$, $p < .03$, but frequencies did not differ
between groups. Because there were no group differences,
removing PL from the analysis should not have
differentially affected aversive or ingestive scores in
the three groups.

"No response" (NR) did not differ in occurrence
between groups in response to solutions of sucrose or
QHCl, but for all groups its frequency decreased as
concentration of sucrose solution increased, $F(3,45) = 9.40$, $p < .0001$.

Figures 7-10 show frequencies of ingestive and
aversive responses to sucrose and QHCl for Septal, VMH,
and Control Groups. Note that different Y-axes were used
to accommodate varying ranges of values of the four
dependent variables.

A comparison of Figures 7 and 8 shows that rats
exhibited more ingestive responses to sucrose than to
QHCl, Quasi-$F(1,7) = 7.14$, $p < .025$. Figure 7 indicates
that the Septal and VMH Groups displayed more combined
ingestive responses over all sucrose concentrations than
did the Control Group, $t(69) > 1.99$, $p < .05$. This figure
also shows that ingestive responses were
concentration-dependent, increasing as sucrose
concentration increased, $F(3,45) = 21.58$, $p < .0001$.
Figure 8 shows that ingestive responses decreased as QHCl

26

concentration increased, $F(3,45) = 3.68$, $p < .02$. This figure also shows that the Septal Group showed more ingestive responses than the VMH or Control Group at the .000003 M concentration of quinine, $t (15) > 2.13$, $p < .05$).

A comparison of Figures 9 and 10 shows that rats displayed more aversive responses to QHCl than to sucrose, $Quasi-F(1,7) = 8.71$, $p < .025$. These figures also show that aversive responses were concentration dependent, increasing as QHCl concentration increased, $F(3,45) = 6.50$, $p < .001$, and decreasing as sucrose concentration increased, $F(3,45) = 21.43$, $p < .0001$. Additionally, they show that all groups displayed similar increases in aversive reponses as QHCl concentration increased, and similar decreases as sucrose concentration increased.

Insert Figures 7, 8, 9, and 10 about here

Figures 11-14 show frequencies of neutral responses and amount of locomotion per trial for Septal, VMH, and Control Groups. There was no statistically significant difference between sucrose and quinine solution in the number of neutral responses elicited. Figure 11 shows that neutral responses decreased as sucrose concentration increased, $F(3,45) = 8.33$, $p < .0002$. Figure 12 shows that neutral responses decreased as the concentration of
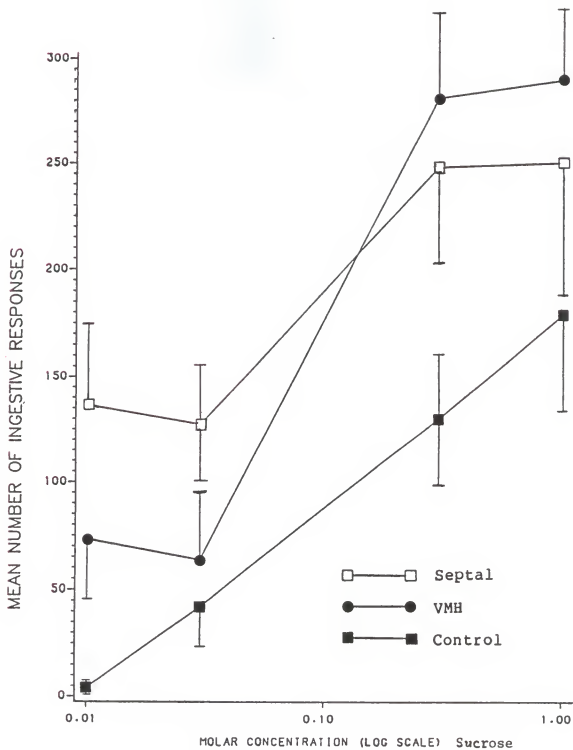
27

Figure 7. Mean number of ingestive taste reactivity responses (± S.E.M.) elicited during 1 min of intraoral sucrose infusion in the Septal, VMH, and Control Groups.

28

Figure 8 . Mean number of ingestive taste reactivity
responses (± S.E.M.) elicited during 1 min of intraoral
quinine hydrochloride infusion in the Septal, VMH, and
Control Groups.

Figure 9. Mean number of aversive taste reactivity responses (± S.E.M.) elicited during 1 min of intraoral sucrose infusion in the Septal, VMH, and Control Groups.
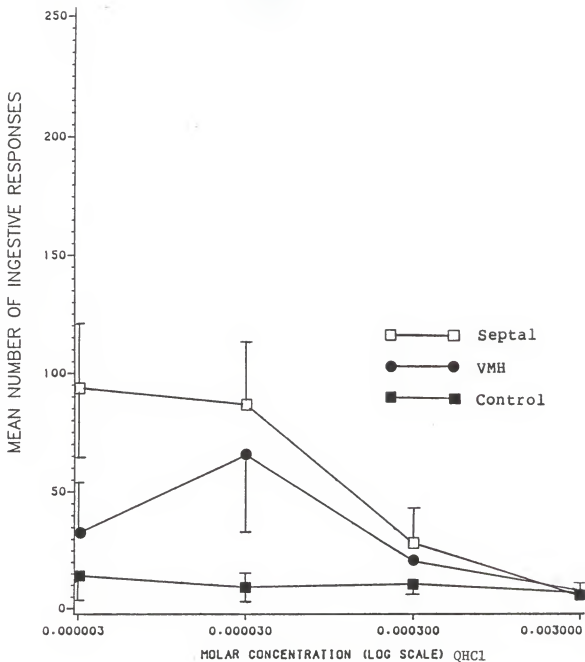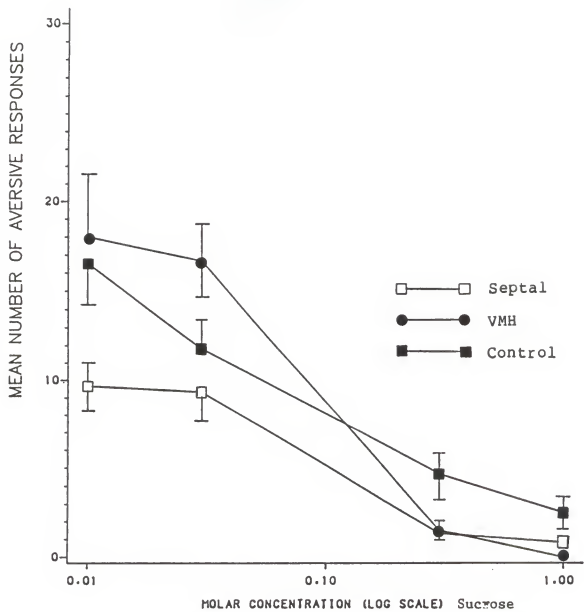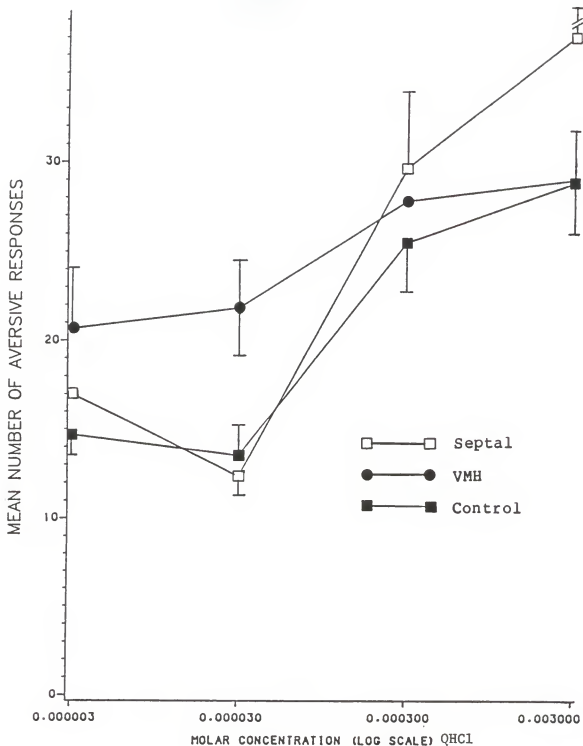
Figure 10. Mean number of aversive taste reactivity responses (± S.E.M.) elicited during 1 min of intraoral quinine hydrochloride infusion in the Septal, VMH, and Control Groups.   31

QHCl increased, $F_{(3,45)} = 2.60$, $p < .06$.

A comparison of Figures 13 and 14 shows that rats displayed more locomotion in response to QHCl than to sucrose, Quasi-$F_{(1,7)} = 8.99$, $p < .025$. These figures also show that locomotion increased with increasing concentration of quinine, $F_{(3,45)} = 8.71$, $p < .0001$, and decreased as concentration of sucrose increased, $F_{(3,45)} = 13.66$, $p < .0001$.

Both the Septal and VMH Groups showed less locomotion than Controls while responding to sucrose solution, $t_{(69)} > 1.99$, $p < .05$, and the VMH Group alone showed less movement while responding to quinine solution, $t_{(69)} > 1.99$, $p < .05$.

With regard to sucrose, the differences in locomotion may be explained by the fact that the two Lesion Groups showed more ingestive responses to all concentrations of sucrose, and often remained motionless in the chamber while reacting to it. The Control Group also showed this "immobilization" but only at the .3 and 1.0 molar concentrations of sucrose solution.

High concentrations of QHCl produced what appeared to be escape-oriented movement. In this case, the lower level of locomotion exhibited by the VMH Group may have reflected a general lesion-produced torpor.

DISCUSSION

The primary purpose of the present study was to determine the magnitude of taste hyperresponsivity in rats with septal and VMH lesions. The results indicated that the Septal and VMH Groups displayed more combined ingestive responses across concentrations of sucrose than the Control Group (refer to Figure 7). Furthermore, aversive responses decreased similarly for all groups as sucrose concentration increased; this indicates that the lesions did not produce a generalized increase in all FAP components in response to sucrose. Although it is difficult to ascribe a precise value to the relative magnitude of finickiness in animals with septal or VMH lesions, the finding that the lesion groups displayed significantly more ingestive responses to sucrose solution, while showing similar numbers of aversive responses, provides strong evidence of positive finickiness.

While the data did indicate positive finickiness, it failed to show evidence for negative finickiness: The Septal and VMH Groups did not display significantly more aversive responses than the Control Group to quinine.

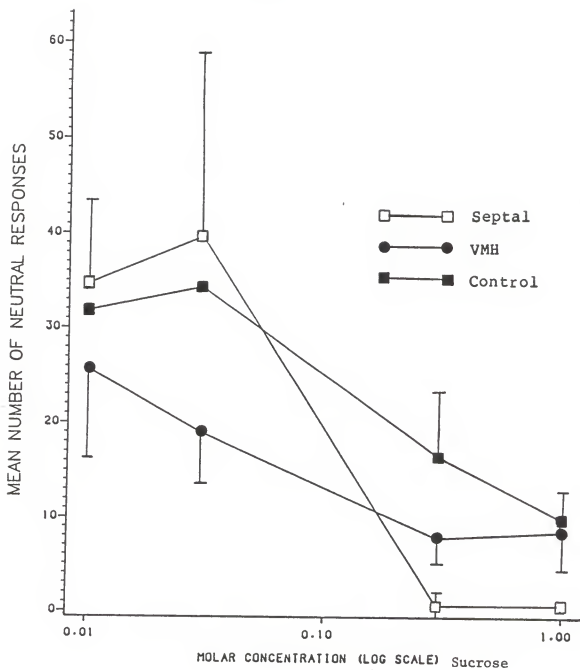The evidence of positive finickiness and lack of

33

Figure 11. Mean number of neutral taste reactivity
responses (± S.E.M.) elicited during 1 min of intraoral
sucrose infusion in the Septal, VMH, and Control Groups.
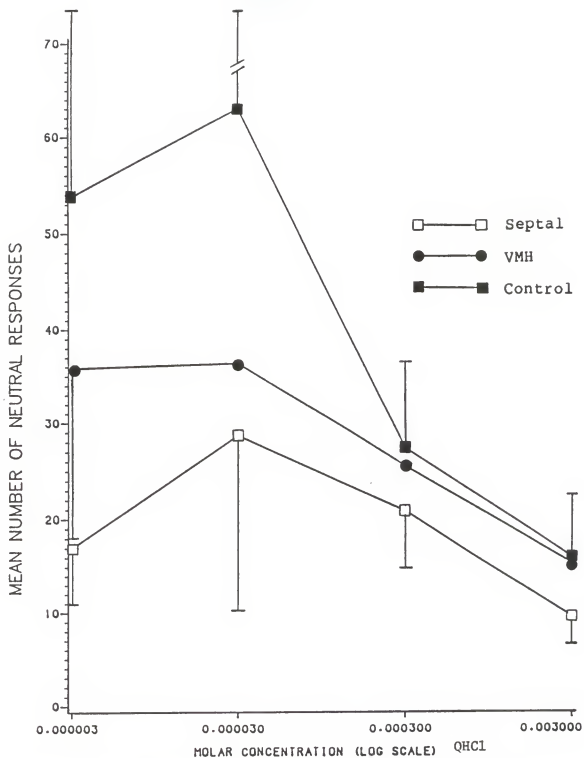
Figure 12. Mean number of neutral taste reactivity responses (± S.E.M.) elicited during 1 min of intraoral quinine hydrochloride infusion in the Septal, VMH, and Control Groups.

35

Figure 13. Mean amount of locomotion per trial (± S.E.M.) during 1 min of intraoral sucrose infusion in the Septal, VMH, and Control Groups.
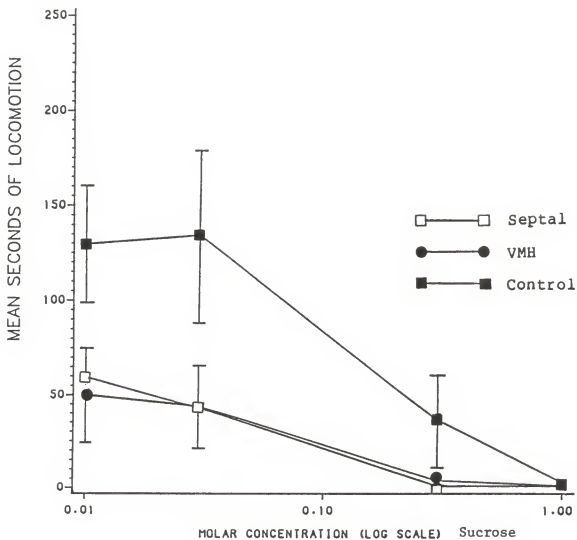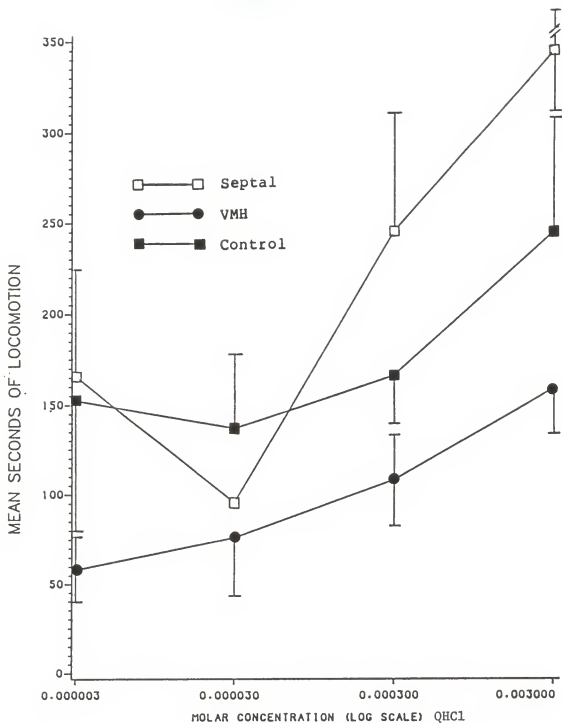
Figure 14. Mean amount of locomotion per trial
(± S.E.M.) during 1 min of intraoral quinine
hydrochloride infusion in the Septal, VMH, and Control
Groups.   37

evidence of negative finickiness corroborates and clarifies the findings of Weingarten (1982) and Weingarten et al. (1983). The taste reactivity test, as used in this experiment, eliminated the potential confounds of postingestive effects, floor effects, obesity, and compound tastants on taste evaluation. Thus, the present results unambiguously indicate that oropharyngeal factors were responsible for the differences between Control and Lesion Groups in taste reactivity.

A second goal of the present study was to determine whether taste hyperresponsivity in VMH and septal animals was qualitatively the same; that is, did septal animals increase ingestive responses to a palatable tastant in the same manner as VMH animals?

The issue of similarity of hyperresponsiveness was addressed by using the method of least-squares to fit straight lines to the ingestive FAP data in response to sucrose solution (refer back to Figure 7). By comparing the slopes of the lines for the Septal and VMH Groups, one could determine if ingestive responses increased with sucrose concentration at the same rate.

While both lesion groups showed more ingestive responses than Controls at all concentrations, Figure 15 shows that the slopes of the lines for Septal and Control Groups were approximately the same. In contrast, the slope for the VMH Group was markedly steeper than that of

the Septal Group. This indicates that the nature of taste
hyperresponsivity in the two lesion groups might have
qualitatively different. The septal lesion appeared to
produce a constant increase in responsivity (relative to
control rats) across the sucrose concentrations tested,
while the VMH lesion produced a multiplicative increase in
responsivity as sucrose concentration increased.

Insert Figure 15 about here

The additive effect of the septal lesion on ingestive
FAP components corroborates data on licking activity
(Beatty and Schwartzbaum, 1968) and could reflect a
heightened responsivity to all sensory stimuli. Animals
with septal lesions have been found to be hyperresponsive
to puffs of air, pokes with a stick, foot shock, shifts in
ambient temperature, light, and sound (Isaacson, 1982).
They also show high distractability and disrupted
sequences of feeding (Flynn et al., 1986) and maternal
(Fleischer & Slotnick, 1978) behavior.

In contrast to septal lesions, VMH lesions may
produce a more circumscribed hyperresponsivity to the
sensory characteristics of food. Powley (1977) proposed
that VMH hyperphagia is produced by heightened cephalic
phase responses to food. Cephalic phase responses of
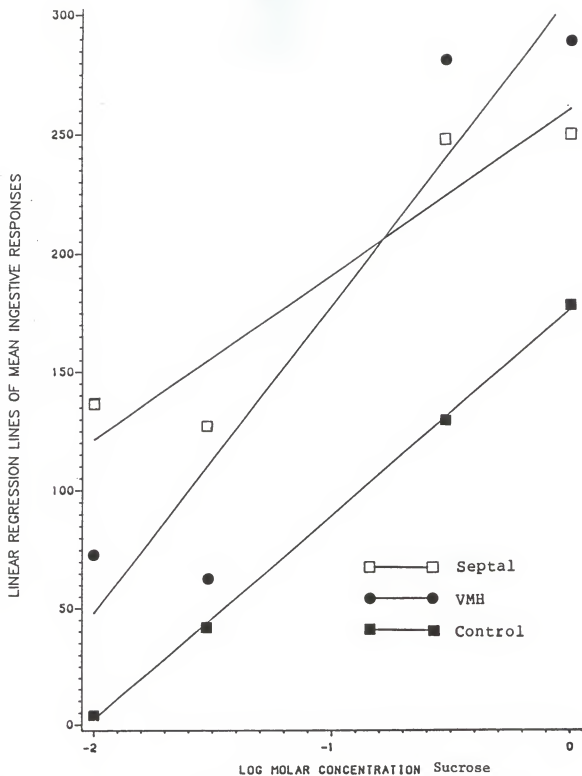digestion are autonomic and endocrine reflexes involved in

39

Figure 15. Linear regression lines for the mean number of
ingestive taste reactivity responses elicited during 1 min
of intraoral sucrose infusion in the Septal, VMH, and
Control Groups.

40

the metabolism of food that are triggered by sensory contact with a foodstuff. In the case of the animal with a VMH leson, hyperphagia may occur because the sight, smell, and taste of a palatable food elicit a relative hyperresponsivity in insulin release, gastric secretion, and gluconeogenesis.

The increased number of ingestive responses to sucrose by the VMH Group may thus be uniquely food oriented, whereas the parallel behavior displayed by the Septal Group may have no exclusive relationship to food stimuli.

The above interpretation of the regression data must be tempered by the fact that an ANOVA on the individual slopes of all animals failed to show statistically significant slope differences between the Control, Septal, and VMH groups; this was due to the large amount of variation in the individual slopes of animals within a group. The issue of whether septal and VMH lesions do in fact produce different types of taste hyperresponsivity, could best be settled by experimental replication.

A final issue of discussion concerns the classical conditioning, which occurred in the test chamber. On several trials where sucrose was the tastant and the the animal on the previous day had received the .003 M concentration of QHCl, it would display several gapes in response to the sound of the VCR being switched on. The

41

gapes would then be followed by mouth movements and tongue
protrusions as the sucrose solution was infused into the
oral cavity. Because it took one or two seconds for the
tastant to reach the tongue, these initial behaviors could
not be in response to gustatory stimulation and, thus,
they were not included in the response sums for the trial.
The fact that the behaviors occurred before any possible
gustatory stimulation indicates that they must have been
the result of classical conditioning in which the sound of
the VCR served as the conditioned stimulus, paired with
the unconditioned stimulus of the taste of quinine
hydrochloride. Although this relatively circumscribed
conditioning to the sound of the VCR did not affect the
taste reactivity data, it is quite feasible that a more
generalized conditioning to the test chamber or the
experimental room in which it was housed, could have
affected the results of the experiment. The exact nature
of the conditioning would have depended upon the order in
which the animal received the taste solutions.

For example, if one considers that only the two
strongest concentrations of each type of taste solution
elicited a clearly ingestive or aversive sequence, it is
evident that the fourth animal in each experimental group
would have experienced a markedly different taste sequence
than the fifth (refer back to table 1). Animal number
four received Q4, S3, Q3, S1 as its first four taste

42

solutions, while animal number five received Q1, S1, Q2, S2 as its first four solutions. It is conceivable that animal number four, upon experiencing the bitter tastes of Q4 and Q3, could have acquired a weak conditioned aversion to the test chamber, while in the case of animal number five, the chamber could have acquired positive secondary reinforcing value. This classical conditioning could have differentially affected cephalic phase responses and made the results less clear than they might have been had all of the sucrose solutions been presented first, followed by all of the quinine solutions. In the design of future experiments, due consideration should be given to the effect of learning on taste evaluation.

In conclusion, the present results strongly support the idea of positive finickiness in septal and VMH rats. Because obesity and postingestive factors were eliminated in the taste reactivity analysis, one can have confidence that the differences in ingestive responses were due to oropharyngeal factors. Both septal and VMH lesions appeared to change the hedonic value of a palatable solution and make it more palatable and/or rewarding, but the shift in palatability appeared to be different for the two groups: The Septal Group showed a constant increase in palatability across concentrations; the VMH Group showed a multiplicative increase.

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TASTE REACTIVITY IN RATS WITH VENTROMEDIAL-HYPOTHALAMIC
AND SEPTAL LESIONS

by

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ABSTRACT

The taste reactivity test was used to evaluate the
magnitude of taste hyperresponsivity displayed by rats
with lesions of the ventromedial hypothalamus (VMH) and
septum. Septal, VMH, and Control Groups (n=6) received
1-ml intraoral infusions of solutions of sucrose (.01,
.03, .3, 1.0 M) and quinine hydrochloride (QHCl) (.00003,
.00003, .0003, .003 M). The resulting stereotyped
behavioral responses or "fixed action patterns" were
videotaped and analyzed frame-by-frame.

Responses were divided into three categories:
ingestive, aversive, and neutral (neutral responses were
tnose which decreased as the concentration of sucrose and
QHCl inceased). The lesion groups showed no evidence of
negative finickiness; there were no sigificant differences
between groups in numbers of aversive responses to quinine
hydrochloride. The Septal and VMH Groups did display
significantly more ingestive responses to sucrose than
Controls, and all groups of animals showed similar
decreases in aversive responses as sucrose concentration
increased.

These results support the notion of positive
finickiness in animals with septal and VMH lesions.
Additionally, ingestive responses were plotted against
sucrose concentration and it was found that, while the
Septal Group showed more ingestive responses than Controls

at all concentrations, the slopes of the lines for the two groups were approximately the same. In contrast, the slope for the VMH Group was markedly steeper than that of the Septal Group. This indicates that the nature of taste hyperresponsivity in septal animals might be different from that in VMH animals: The septal lesion appeared to produce a constant increase in responsivity across the sucrose concentrations tested; the VMH lesion appeared to produce a multiplicative increase in responsivity as sucrose concentration increased.