Taste Preference and Discrimination with Cortical Lesions

ABLATION OF THE SOMATOSENSORY CORTEX FOR TASTE: EFFECTS ON TASTE PREFERENCE AND TASTE DISCRIMINATION BEHAVIOR

By

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A Thesis

Submitted to the Faculty of Graduate Studies

in Partial Fulfilment of the Requirements

for the Degree

Master of Arts

McMaster University

May 1967

MASTER OF ARTS (1967)
(Psychology)

McMASTER UNIVERSITY
Hamilton, Ontario

TITLE: Ablation of the somatosensory cortex for taste: effects on taste preference and taste discrimination behavior.

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NUMBER OF PAGES: iv, 99

SCOPE AND CONTENTS: Three groups of rats were tested both before and after bilateral ablation of the taste sensory cortex. The first group, exposed to quinine hydrochloride (QHCl) in a two-bottle preference situation, showed large deficits post-operatively, but these were considerably reduced by the fourth postoperative week. A second group, tested for sodium chloride (NaCl) discrimination in a modified signal detection situation, also showed significant postoperative impairment. A third group, QHCl discrimination, was discarded for failure to learn the detection task. The fourth group, NaCl preference, gave results which were very unclear compared with NaCl discrimination and QHCl preference. It is concluded that preference tests are unsatisfactory measures of taste sensitivity, unless the stimuli possess extreme aversive or preferred qualities.

ACKNOWLEDGEMENT

The author wishes to take the opportunity presented by the completion of this research to thank the following people for their encouragement and assistance:

Dr. G. R. Morrison, for his generous attention to the myriad and often mundane tasks of a thesis advisor.

Mr. Bodo Bilazewski, for his willing assistance in carrying out parts of the actual research.

CONTENTS

INTRODUCTION

Patton and Amassian (1952) electrophysiologically mapped the cortical projection zone of the chorda tympani nerve in the cat, locating it on the orbital surface of the hemisphere just rostral to the tip of the ectosylvian fissure. However, they did not equate this area with the cortical reception area for taste since it was possible that their electrical stimulation of the chorda was activating tactile tongue afferents as well as or instead of taste afferents. This has since been shown to be the case (Cohen et al., 1957; Landgren, 1957). These investigators agree with the localization reported by Patton and Amassian, and using microelectrodes to record from single cells in this area found two types of cells; those responding to a single modality, either thermal, tactile, or gustatory, and those responding to a combination of these. However, the preponderance of touch-responsive cells found in these studies, together with a report on separate cortical reception areas for touch and taste in the squirrel monkey (Benjamin and Emmers, 1960), would indicate that the true taste area in the cat was not tapped. Additional support for this view comes from ablation studies on rhesus monkeys (Ruch and Patton, 1946; Patton, Ruch, and

Fulton, 1946; Bagshaw and Pribram, 1953); taste deficits, as measured by preference tests for quinine hydrochloride, were observed only when the lesions invaded the buried opercular cortex. In the rat, with its lissencephalic cortex, attempts to localize the receiving area for taste have been much more successful, and it is known that the projection zones for taste and tactile afferents overlap a great deal (Benjamin and Pfaffmann, 1955). The taste nerve area occupies approximately four sq. mm. of cortex just dorsal to the rhinal fissure and beneath the middle cerebral artery, about four mm. caudal to the frontal pole.

At the thalamic level, information on the rat is again much more conclusive. Using the retrograde degeneration technique, Benjamin and Akert (1959) identified a morphologically distinct subdivision of n. ventralis medialis, the most ventral and posterior part, subjacent to n. parafascicularis, as the thalamic relay for taste. This conclusion is supported by later investigators (Emmers, Benjamin, and Ables, 1960; Emmers, Benjamin, and Blomquist, 1962; Prommer, 1961) whose electrophysiological recordings clearly show a further separation of function within the subnucleus itself. Taste afferents occupy the medial half of the subnucleus, the chorda tympani projecting anteriorly, and the glossopharyngeal nerve posteriorly; the lateral half receives lingual nerve

afferents, which mediate touch and temperature. That the relay nucleus for taste is located in this part of the thalamus not only for the rat, but for other species as well, is shown by the following investigators. Andersson and Jewell (1957a and b) report that lesions of n. ventralis posteromedialis (VPM) produce marked taste deficits in the goat. In the monkey, VPM lesions have the same effect (Patton, Ruch, and Walker, 1944; Bagshaw and Pribram, 1953), indicating that this nucleus, or some subdivision of it, is the thalamic relay for taste.

Several techniques have been used in the localization of these cortical and thalamic taste areas, but the only one which permits the unequivocal conclusion that the area of interest mediates taste sensation is the ablation technique, combined with pre- and postoperative measures of taste sensitivity. Most of this work has been done on the rat and has used as its sensitivity measure the two-bottle preference test as first described by Richter (1939). Normal rats exposed to quinine hydrochloride (QHCl) have aversion thresholds of approximately 15 x 10^{-6} M QHCl; after cortical lesions, these increase six- to eightfold (Benjamin and Pfaffmann, 1955; Benjamin, 1955b; Benjamin and Akert, 1959). The threshold elevation following destruction of the thalamic relay nucleus is much more dramatic, ranging from ten to twenty-five times the

normal (Ables and Benjamin, 1960; Oakley and Pfaffmann, 1962). Data on substances other than QHCl is sparse, but Oakley and Pfaffmann have reported that animals with thalamic lesions do not show the typical normal increase, then decrease, in intake when presented with increasingly concentrated solutions of sodium chloride and sucrose.

These results all come from nondeprived animals; the first indication that the aversion threshold is not a stable measure, and that taste discrimination is not a simple function referrable to one cortical area, comes from Benjamin (1955a and b). Quinine hydrochloride aversion thresholds obtained from animals on a low fluid deprivation regimen are significantly lower than those from highly fluid deprived animals. Moreover, ablation of the cortical taste area results in increased thresholds for low fluid deprived animals, but the highly deprived ones show no postoperative threshold change. In the case of these latter animals, expansion of the lesions to include most of the neocortex produces an increase in threshold. It appears, then, that under conditions of high fluid deprivation, areas other than the primary projection area function in taste discrimination, and to judge from the large lesion results, that these other areas are cortical.

One shortcoming of the preference technique, that concerning its sensitivity to degree of fluid deprivation, has just been mentioned. A more adequate demonstration of

the same fault is found in Richter's classic paper (1939) on the effect of adrenalectomy on the preference threshold for sodium chloride, and in various attacks subsequently made on this work. Richter found that adrenalectomized rats showed a much lower threshold for sodium chloride than did normals, and proposed that adrenalectomy lowers the sensory threshold, and that this effect is brought about by an increased sensitivity of the taste receptors. This is not the case; electrophysiological recordings from the chorda tympani show no difference between normal and adrenalectomized rats in their receptor sensitivity to sodium chloride (Pfaffmann and Bare, 1950). Other investigators have demonstrated that it is not adrenalectomy per se, but the high motivation resulting from adrenalectomy, that is responsible for the lowered thresholds of the operated animals (Carr, 1952; Harriman and MacLeod, 1953; Koh and Teitelbaum, 1961). Accordingly, a dual concept of threshold has been proposed. There is a stable sensory threshold relying on the capacity of the receptors and independent of motivational influences; the second is a preference threshold normally higher than the sensory threshold, but with its degree of separation adjustable through the influence of many other factors - in the case of adrenalectomized animals, through a metabolic need for sodium chloride. Thus, normal animals taste weak sodium

true sensory thresholds with electrophysiological techniques (Pfaffmann, 1955; Hagstrom and Pfaffmann, 1959), as "internally" motivated discrimination thresholds (Koh and Teitelbaum, 1961; Campbell, 1958), or as preference thresholds (Benjamin and Akert, 1959; Burright and Kappauf, 1963). Turning to sodium chloride, thresholds obtained with different techniques vary markedly. Electrophysiology and discrimination produce comparable results (Pfaffmann, 1955; Koh and Teitelbaum, 1961) which are much lower than those generated by a preference technique (Richter, 1939). This indicates that certain substances are inherently motivating by virtue of their taste qualities alone, and that this motivation, depending on degree, may be sufficient to reduce the "zone of indifference", or separation between sensory and preference thresholds, to a minimum. For the appropriate substances, then, a preference technique is a simple and accurate way of determining the sensory threshold.

This consideration of the preference technique leads to the conclusion that it is a valuable, though highly unreliable, method. For quinine, it gives a reliable estimate of the sensory threshold in normal nondeprived animals; this is not true for highly motivated animals. With hunger and shock motivation superimposed on the incentive derived from quinine itself, the preference threshold should approximate the sensory

threshold even more closely. On the other hand, fluid deprivation should have just the opposite effect. Previously avoided concentrations would be acceptable to an animal in a state of increased need, and the zone of indifference should increase. Conversely, the preferred quality of sodium chloride seems insufficient motivation to lower the value of the preference threshold to that of the sensory threshold in normal animals, though this does take place in highly motivated animals.

With these difficulties in mind, a reexamination of the ablation studies mentioned previously shows that all of them have used a preference technique, all have restricted their preference measures to quinine hydrochloride, other substances being ignored, and yet all have framed their conclusions in terms of sensory thresholds. This is a defensible position, however; quinine preference thresholds are reasonably good estimates of sensory thresholds, and all of these studies used nondeprived animals. In spite of this, a vast methodological improvement would have been the use of a technique which measures the sensory threshold directly.

Morrison and Morrison (1966) have developed such a technique, based on certain aspects of signal detection theory and methods. As such, it is a discrimination technique, but is uncontaminated by any of the complex motivational influences which can so distort preference

results. It does make use of hunger-motivated animals, but presumably the influence of this factor is small as compared to the known effects of fluid deprivation, and negligible when contrasted with the effects of a specific deficit like salt lack on thresholds for salt. The preferred or aversive qualities of the stimulus are given no chance to interfere, since the animals must sample a constant amount of each stimulus solution.

It is the purpose of this study to examine the effects of cortical taste area lesions in rats on taste discrimination as measured by two different techniques, and for two different substances. The techniques are the Morrison and Morrison method, and the two-bottle preference method; the substances are quinine hydrochloride and sodium chloride.

METHOD

The study consisted of two related experiments, one concerned with the effects of cortical ablation on taste preference behavior, and the other with the effects of similar lesions on taste discrimination. Preference and discrimination scores were both obtained for varying concentrations of quinine hydrochloride and sodium chloride.

Part I - Preference

Subjects and Apparatus

Nineteen male hooded rats were used as subjects, eleven in the quinine hydrochloride (QHCl) preference group, and eight in the sodium chloride (NaCl) group. They were individually caged in cylindrical wire mesh cages, 10 inches in diameter and 7 inches deep. Two 100 ml. graduate cylinders, fitted with corks and metal spouts, and hung on the outside of the cages so that their spouts projected through the mesh of the cage sides, were used as drinking bottles. They were positioned at opposite sides of the cage, with the food box located midway between them. The animals had continuous access

to food.

Procedure

The basic procedure was that of the two-bottle preference test previously described by Richter (1939) and by Benjamin and Akert (1959). For the first five days of the experiment, both drinking bottles contained tap water. Preference testing was then initiated by filling one of the two bottles with .1 mM QHCl; the other continued to hold tap water throughout the experiment. In the case of the NaCl group, testing was begun with a .1 M solution; again, the second contained tap water. These solutions were presented for 48 hours; after 24, and after 48 hours, the positions of the bottles were reversed, and the amount consumed of each solution recorded. At the end of each 48 hour period, the concentrations of the test solutions were reduced in .5 logarithmic steps, making the whole procedure a descending method of limits. Six concentrations of QHCl, ranging from .1 mM to .000316 mM, and seven concentrations of NaCl, from .1 M to .0001 M, were used. At the conclusion of these series, the animals were operated upon, according to the procedure described below, and were allowed a recovery period of seven to ten days. They were then retested exactly as outlined above, with the exception of repeating the series of concentrations to obtain measures at varying times postoperatively. At the

conclusion of the experiment, all animals were sacrificed, and their brains, after being fixed in formalin, were photographed.

For both the QHCl and the NaCl groups, 24 and 48 hour intake measures of water and of each solution concentration were obtained. These were combined to express solution intake as a percentage of total fluid intake at each solution concentration used, separate summaries being made for operated and control groups at each stage of each experiment.

Part II - Discrimination

Subjects

Twenty-seven male hooded rats served as subjects, thirteen in the NaCl discrimination group, and fourteen in the QHCl group. They were maintained at 75 to 80% of their *ad lib* weights, and had water available at all times in their individual home cages.

Apparatus

The test chamber, similar to that described by Morrison and Norrison (1966), was a plexiglass operant conditioning box 10" x 11" x 8" high, with one wall fitted with two rat levers on either side of a food cup connected to a Gerbrands pellet dispenser. The wall

opposite had a 3 inch long slot cut in the center of it 1 1/2 inches above and parallel to the grid floor. Outside the box, and evenly spaced around the rim of a disc of 8 inch diameter were ten plastic bottles fitted with corks and metal spouts. The spouts were wired into a drinkometer circuit, so that a rat standing on the grid floor and licking would complete the circuit. The disc was so positioned that a bottle spout could project through the slot in the box wall, and it was connected to a slow speed electric motor which could rotate it in either a clockwise or counterclockwise direction. Associated with this apparatus was the typical relay control circuitry.

Procedure

Training in the discrimination task can be divided into four stages. All animals had previously been trained to bar press in a single lever situation, so the first stage was the learning of a bar alternation. The two levers were randomly wired through a ten-point stepper to the reinforcement mechanism, with the stepper activated by a reinforced response. This procedure forced the animals to alternate between the two levers in order to be reinforced. Each animal was allowed 50 to 100 reinforcements per daily session, or, if he did not press,

was left in the box for one-half hour. These sessions continued until all animals were freely alternating between bars.

Stage II introduced the licking component of the response. All bottles were filled with tap water. Licking at the spout activated the motor to rotate the turntable counterclockwise 1/20 of a revolution and carry the spout out of the box. A bar press following this would either be reinforced or not, depending on which of the two bars was pressed. If reinforced, the motor would again rotate the turntable counterclockwise and the next spout would be presented; if not reinforced, the motor would reverse, turn the disc clockwise, and the same spout would be re-presented. The animals were 23 hours water deprived before the first three daily sessions of this stage; they were left in the box until they had received 50 reinforcements or for one-half hour. In this and subsequent stages, only the first bar press following the adequate licking response was counted. Bar pressing while the spout was in the box had no effect and these responses were not recorded.

For stage III, five of the bottles were filled with .1 M NaCl (or .0316 mM QHCl) and the other five with tap water. The stepper was wired so that a response on the right hand lever was reinforced in the presence of NaCl (or QHCl), and a response on the left hand lever

was reinforced in the presence of water. Otherwise this stage is identical to stage II. The QHCl discrimination group, after some initial progress, stabilized at a low level of accuracy, so the concentration of the solution was raised to .1 mM.

Stage IV is the same as stage III, except that both correct and incorrect bar press responses advanced the turntable. All animals were given 50 trials daily; if any one animal lagged noticeably behind the group, its daily session was increased to 100 trials until the deficit was made up.

All fourteen animals of the QHCl discrimination group were discarded as they could not successfully be trained. They were advanced from one stage to the next in the hope of some improvement, but their average accuracy score on the last day of training was only 63%, very poor as compared to the 87% achieved by the NaCl discrimination group on the concluding day of stage IV of training. Moreover, the QHCl group showed no improvement whatsoever during the last five weeks of training; their average accuracy score on the initial day of stage IV was 62%, and this level was maintained with surprising stability (range, 52% to 64%) until they were discarded. Stages I through IV of training for this group occupied better than four months, whereas the NaCl

group completed training in two and a half months.

At the conclusion of training, testing was begun, test days alternating with retraining days on which the solution concentration was .1 M NaCl. For each test day, the solution concentration was reduced by .5 logarithmic step, so that for the first five days, the following NaCl solutions were presented: Day 1 - .1 M, Day 2 - .0316 M, Day 3 - .1 M, Day 4 - .01 M, Day 5 - .1 M. The reduction of solution concentrations continued until performance accuracy sank to a stable value where discrimination could be said to have failed. Each series of solutions was presented twice, and following the second presentation, the animals were operated upon. After a seven to ten day recovery period, the animals were returned to the experimental situation, and given 400 retraining trials in four days, two sessions per day, 50 trials per session. Retesting was then begun, exactly as described above. At its conclusion, the animals were sacrificed and their brains fixed in formalin, and subsequently photographed.

Part III - Surgery

All operations were performed under Nembutal anaesthesia, 45 mg. per kg., with a supplementary dose of Chloral Hydrate if required. Skull holes were made with size 7 dental burr. Slight damage to the temporal

muscle which articulates the lower jaw was unavoidable, but all animals ate normally following the operation. The taste area of the somatosensory cortex, at the Krieg coordinates of anterior +3.0 and +3.5 mm., lateral 5.0 and 5.5 mm., and ventral 6.0 mm., was ablated by passing 3 ma. for 15 seconds through a monopolar needle electrode exposed for approximately 1 mm. at the tip. Each lesion was made by four separate electrode placements, with the electrode tip designating the four corners of a square. Control animals underwent sham operations, in which only scalp incisions and burr holes were made.

RESULTS

Quinine Hydrochloride Preference

Measures obtained from the QHCl preference group are plotted in Figure 1, with the concentrations, in millimoles, of the QHCl solutions used displayed along the X-axis, and the QHCl intake percentages along the Y-axis. The three pairs of curves represent operated and control groups at the preoperative and two postoperative stages of the experiment. That QHCl is a very aversive substance for the rat has been previously shown (Benjamin and Pfaffmann, 1955) and it is again apparent from the present results, with QHCl representing only 2 to 3% of the total intake preoperatively at the highest concentration used. As the solution concentration decreases, the amount consumed increases in the expected manner. An inspection of Table 1, a summary of the analysis of variance performed on these data, confirms this influence of concentration; the concentration factor (A) is significant at $<.001$ level. Returning to Figure 1, it can be seen that preoperative intake curves for the two groups are very similar, but that this resemblance disappears postoperatively. At all concentrations during postoperative test I, the operated

Figure 1. Quinine hydrochloride preference experiment. QHCl intake, expressed as a percentage of total fluid intake, is plotted against the millimolar concentrations of the QHCl solutions used.

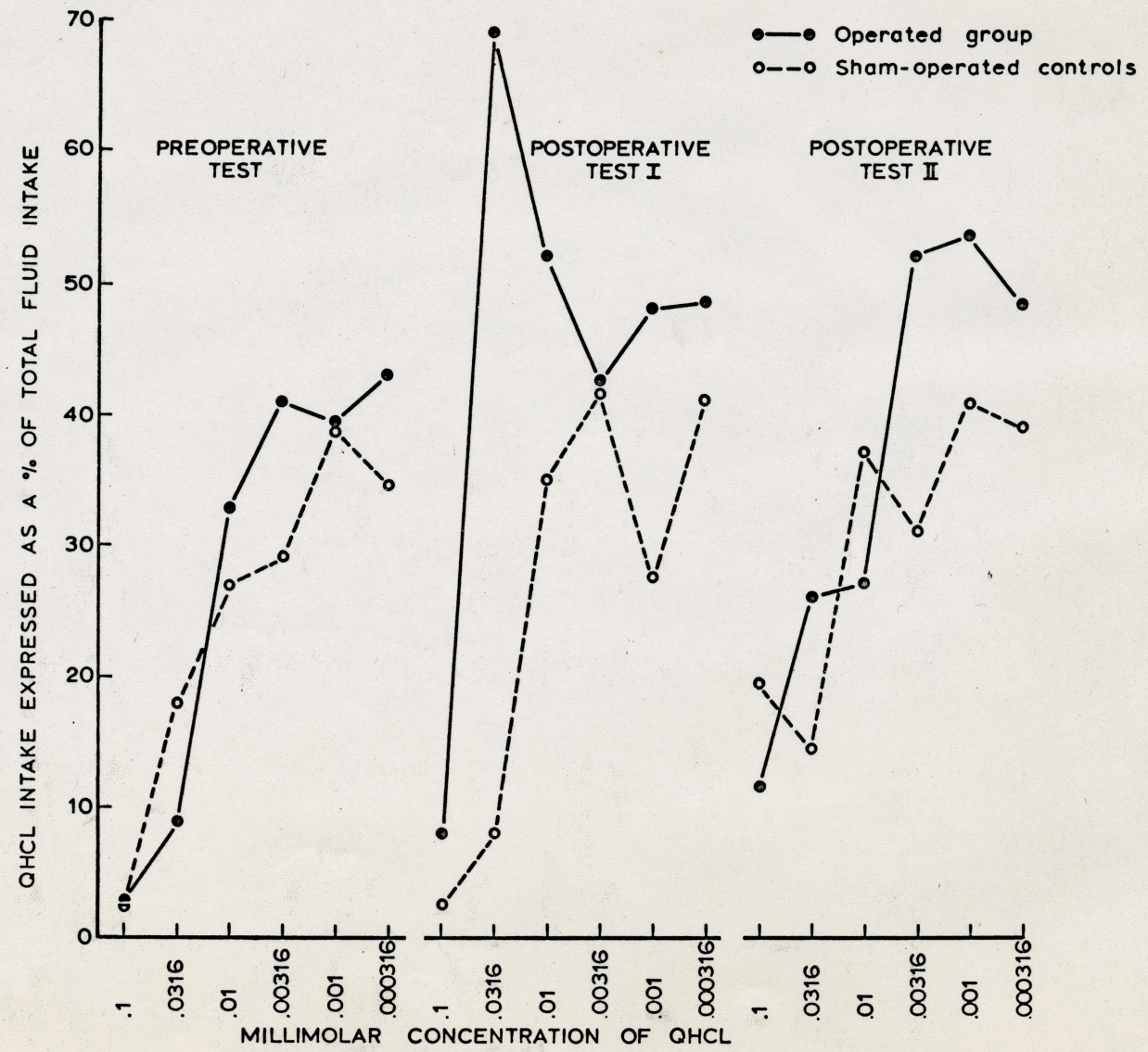

Operated group
Sham-operated controls
PREOPERATIVE TEST
POSTOPERATIVE TEST I
POSTOPERATIVE TEST II
QHCL INTAKE EXPRESSED AS A % OF TOTAL FLUID INTAKE
70
60
50
40
30
20
10
0
.1
.0316
.01
.00316
.001
.000316
MILLIMOLAR CONCENTRATION OF QHCL

TABLE 1

Summary of Analysis of Variance for Quinine Hydrochloride Preference Experiment

Source of Variation	SS	df	MS	F	p
Between subjects	19159.0397	21			
C (Group)	3403.0350	1	3403.0350	4.3197	-
error	15756.0047	20	787.8002		
Within subjects	95802.0017	374			
A (Concentrations)	30764.2545	5	6512.8509	44.5191	<.001
AC	1939.1743	5	387.8348	2.8062	<.025
error	13820.7117	100	138.2071		
B (Stage)	3778.2992	2	1889.1496	13.4511	<.001
BC	1349.1204	2	674.5602	4.8030	<.025
error	5617.8419	40	140.4460		
AB	6013.6551	10	601.3655	4.3180	<.001
ABC	4664.8909	10	466.4891	3.3495	<.001
error	27854.0537	200	139.2703		
		395 Total df			

animals drink more QHCl than do the controls, indicating that ablation of the taste cortex severely reduces sensitivity to QHCl. The results from postoperative test II, beginning twenty-five days after operation, show some reduction of this impairment, but a comparison of this curve with the comparable one for the control group shows that a substantial deficit still remains. If lesions affect preference behavior, this should lead to a significant stage by group interaction (BC), and if, as might be expected, they affect this behavior differentially at different concentrations, this should result in a significant concentration by stage by group triple interaction (ABC). The analysis of variance summarized in Table 1 shows that both these effects are present. Figure 1 shows that this result derives from the postoperative performance of the operated animals; the deficit is very slight for .1 mM QHCl in postoperative test I, and the three highest concentrations are treated normally during postoperative test II. It was expected that the concentration by stage (AB) and the concentration by group (AC) interactions would develop postoperatively, but would not be present before surgery. In the analysis of variance, this is confirmed by the presence of a significant triple interaction, and also by the significance of the two double interactions, AB and AC. Table 1 also shows that the stage effect (B) is significant, but the

BC summary table used in this analysis, to be found in the appendix, indicates that this is the result of the great increase in QHCl intake shown by the operated group on the postoperative tests. The group factor (C) is not significant.

Estimates of threshold values for QHCl can be obtained by interpolation at the 25% point on the curves in Figure 1. This procedure places the preoperative threshold for both operated and control groups at approximately .01 mM QHCl. Operated animals show threshold increases to approximately .1 mM and .0316 mM for postoperative tests I and II, respectively, while postoperative control thresholds remain stable at approximately .01 mM. These values agree well with those obtained by other investigators (Benjamin and Akert, 1959; Koh and Teitelbaum, 1961).

Sodium Chloride Preference

Unlike the QHCl preference experiment, this study is not a replication of previous work, but certain predictions were made about the NaCl results, based on extrapolation from those for QHCl. It was expected that the animals would prefer NaCl to water, and, within the range of concentrations used, that there would be an orderly relationship between solution strength and intake. Figure 2 plots the results for this group, with

the molar concentrations of the NaCl solutions along the abscissa and the percentage intake of NaCl along the ordinate. The two curves representing operated and control groups for the preoperative test show that the animals do prefer NaCl to water, but that the preference is evident only with the highest NaCl concentrations used. It is also apparent that the relationship between concentration and intake is much less orderly than that observed for QHCl, though Table 2, a summary of the analysis of variance, shows that the concentration factor (A) is significant.

It can also be seen from Figure 2 that preference for NaCl is steadily increasing over the three stages, for both operated and control groups, quite contrary to expectation. It was predicted that the effect of the ablations would be to decrease preference behavior, just as they produced a decrease in aversive reactions to QHCl. Another change which occurs in the period from the preoperative test to postoperative test II is an increase in the regularity of the relationship between solution concentration and intake, NaCl consumption being highest at the highest concentration and decreasing in an orderly fashion as the solutions get weaker. If the lesions were affecting preference behavior in the manner predicted, these changes would be in the opposite directions; preference should decrease for the operated animals, and the regularity

Figure 2. Sodium chloride preference experiment. Intake of NaCl, expressed as a percentage of total fluid intake, is plotted against the molar concentrations of the NaCl solutions used.

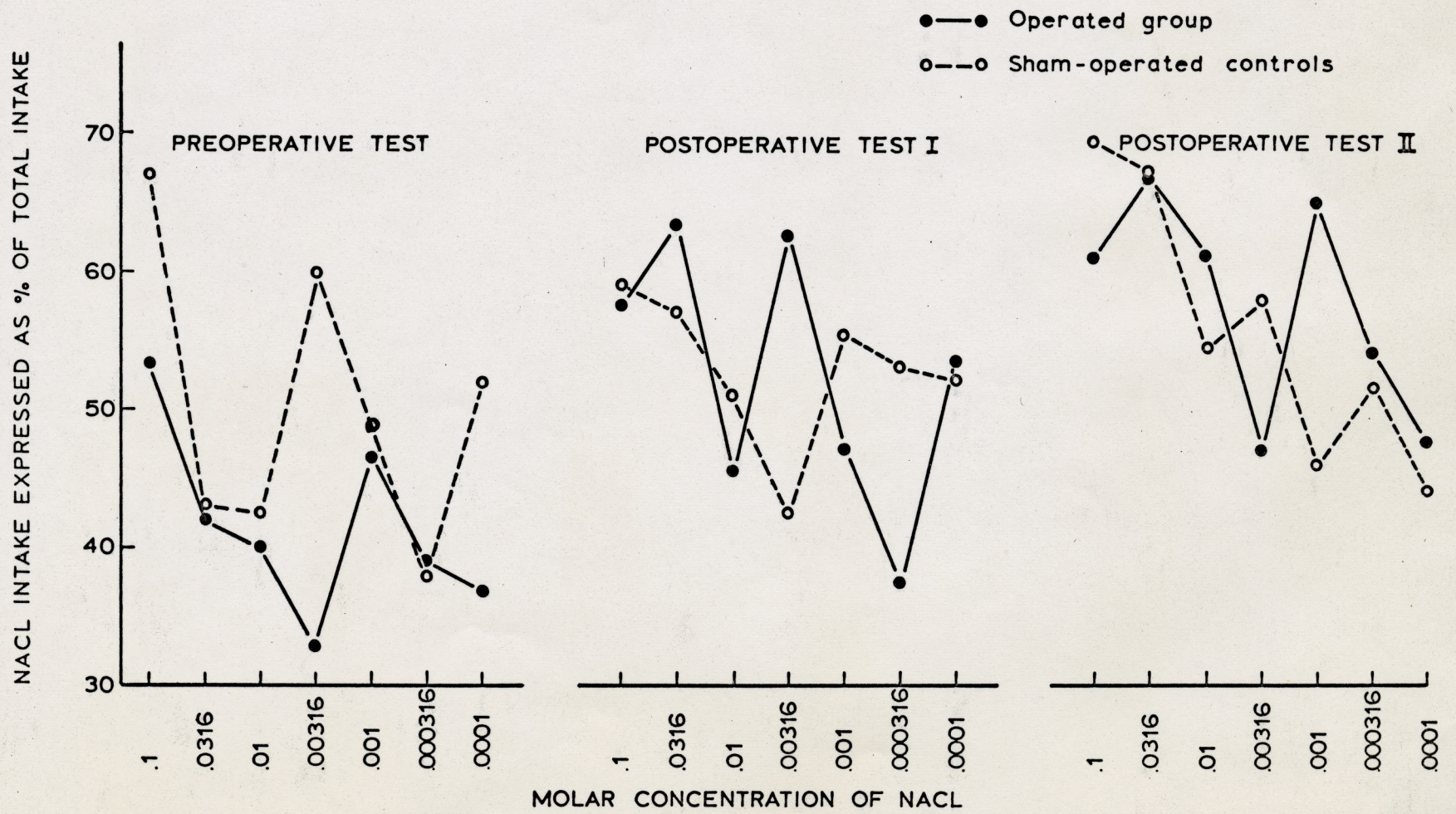

Operated group
Sham-operated controls
PREOPERATIVE TEST
POSTOPERATIVE TEST I
POSTOPERATIVE TEST II
NACL INTAKE EXPRESSED AS % OF TOTAL INTAKE
70
60
50
40
30
.1
.0316
.01
.00316
.001
.000316
.0001
MOLAR CONCENTRATION OF NACL

TABLE 2

Summary of Analysis of Variance for Sodium Chloride Preference Experiment

Source of Variation	SS	df	MS	F	p
Between Subjects	3241.7331	15			
C (Group)	239.6852	1	239.6852	1.1178	-
error	3002.0479	14	214.4320		
Within Subjects	41000.1111	320			
A (Concentrations)	3188.6119	6	531.4353	9.9817	$<.001$
AC	869.5669	6	144.9278	2.7221	$<.025$
error	4472.2275	84	53.2408		
B (Stage)	3854.8809	2	1927.4405	18.2205	$<.001$
BC	742.6439	2	371.3220	3.5102	$<.05$
error	2961.9495	28	105.7839		
AB	2486.2747	12	207.1896	1.8058	$\approx .05$
ABC	3148.6853	12	262.3904	2.2869	$\approx .01$
error	19275.2705	168	114.7338		
		335 Total df			

of the concentration-intake function should be disrupted. Since this is not so, it seems necessary to consider the influence of another factor totally different from ablations; this is a learning or practice effect, which would account for the increase in preference over stages, and also the increase in regularity of the concentration-intake relationship. The contribution of this factor, of course, was unsuspected, so it was not controlled for, and thus is confounded with the lesion effects in the analysis of variance. The stage by group interaction (BC) and the triple interaction (ABC) in Table 2 are significant, a result which would normally be interpreted as confirming the important influence of ablations on preference behavior. This conclusion, however, is impossible in this case, as the lesion effect is totally obscured by the learning effect.

Sodium Chloride Discrimination

The last of the three experiments was concerned with NaCl discrimination; the results of this experiment are plotted in Figure 3, with the molar concentrations of the NaCl solutions displayed on the X-axis, and the percentage of correct responses on the Y-axis. The operated and control groups contain four animals each due to the fact that one of the experimental rats was discarded at stage IV of training for failing to learn, and four others in the same group died following surgery.

The points on the curves of Figure 3 were obtained by averaging the percentage of correct responses for all animals in each group over test and retest cycles at both preoperative and postoperative stages. These cycles were collapsed since accuracy scores were similar for the two preoperatively and, more important, postoperatively. Preoperative accuracy scores for operated and control groups are markedly similar, and the effect of the decreasing solution concentrations is clear, response accuracy falling from about 85% at the strongest concentration to approximately 55% at the weakest. This effect is also apparent during the postoperative test. Statistical confirmation of the influence of concentration may be found in Table 3, which summarizes the analysis of variance performed for this group; the concentration factor (A) is significant at $<.001$ level of confidence. The postoperative curve for the control animals greatly resembles their preoperative performance, as would be expected if the sham operations have no effect on taste discrimination. This is not true, however, for the operated animals, whose accuracy score for .1 M, the strongest concentration used, is only 65%, and falls steadily as the solutions become weaker, dropping below chance level at .000316 M NaCl. This effect of the cortical lesions is reflected in two terms of the analysis of variance in Table 3. The stage by group interaction (BC) is significant, confirming the post-

Figure 3. Sodium chloride discrimination experiment. Accuracy scores for each group are plotted against the concentrations of the NaCl solutions used.

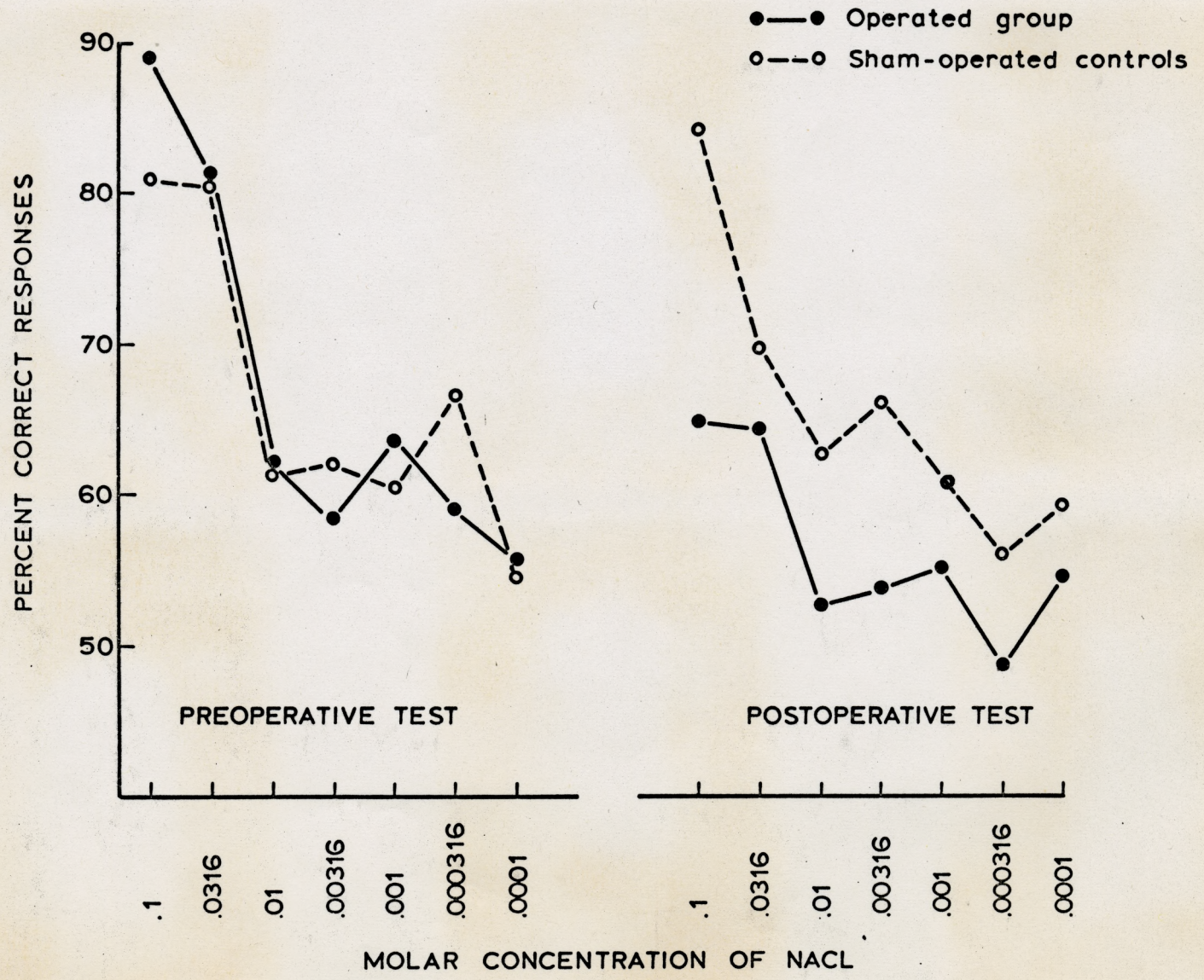

Operated group
Sham-operated controls
PERCENT CORRECT RESPONSES
90
80
70
60
50
PREOPERATIVE TEST
POSTOPERATIVE TEST
.1
.0316
.01
.00316
.001
.000316
.0001
MOLAR CONCENTRATION OF NACL

TABLE 3

Summary of Analysis of Variance for Sodium Chloride Discrimination Experiment

Source of Variation	SS	df	MS	F	p
Between Subjects	3247.3547	7			
C (Group)	542.5198	1	542.5198	1.2034	-
error	2704.8349	6	450.8058		
Within Subjects	14089.8393	104			
A (Concentrations)	8085.1160	6	1347.5193	36.1866	<.001
AC	180.7769	6	30.1295	.8091	-
error	1340.5714	36	37.2380		
B (Stage)	1035.1806	1	1035.1806	20.3967	<.005
BC	624.6453	1	624.6453	12.3077	<.025
error	304.5134	6	50.7522		
AB	809.0911	6	134.9985	3.7491	<.025
ABC	412.7768	6	68.7961	1.9106	-
error	1296.2678	36	36.0074		
		111 Total df			

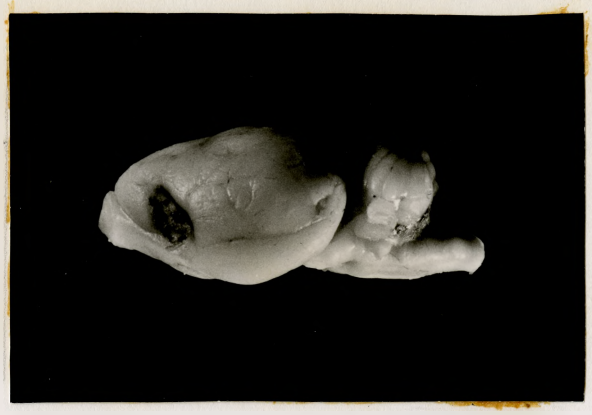
operative drop in accuracy of the operated animals, and the stage factor (B) is also significant, but provides little additional information as it is also produced by the impaired performance of the experimental animals following surgery. The absence of the concentration by group (AC) and triple (ABC) interactions indicates that the ablations impaired discrimination equally at all concentrations; that is, the effect was as large for high concentrations as it was for low ones.

Ablations

Photographs of the brains of three of the operated animals are shown in Figure 4. The top pair of pictures shows the largest lesions made, the bottom pair, the smallest, and the center pair, the intended lesions. As can be seen, the largest lesions encroached upon the somatic sensory area for the face, and in some cases extended rostral into frontal cortex. But in all cases, the lesions are located just dorsal to the rhinal fissure, and destroy the major part of the taste receiving area. The left hand photograph of the bottom pair shows the middle cerebral artery passing over the posterior third of the ablated area.

Benjamin and Akert (1959) also used lesions of the taste sensory cortex, but produced them by aspiration rather than the stereotaxic technique used in this study.

Figure 4. Photographs of brains from three operated animals.



Aspiration involves a direct approach to and exposure of the cortical area of interest and, in the case of the taste area, this means a complete separation of the temporal muscle from its insertion on the skull. Aside from the greater trauma and danger of infection which are a part of the aspiration technique, the animals are unable to eat normally following surgery since chewing movements are effected largely by the temporal muscle. Whatever advantages aspiration may have in terms of more precise localization or sparing of local blood supply are small when compared to its disadvantages. Besides, carefully made stereotaxic lesions are equally likely to be properly located, at least in the small brain of the rat, where direct visual guidance is of little help. The conclusion, then, is that the stereotaxic technique is superior to aspiration when it is desired to produce small cortical lesions in relatively inaccessible places with a minimum of trauma.

DISCUSSION

Pfaffmann and Bare (1950) urge that a clear distinction be made between preference and sensory thresholds, indicating that these two should be considered as separate functions. It is reasonable to believe that preference is a higher order function than discrimination, and depends on discrimination. The difference between two substances must be detected before an animal can begin to exhibit a preference for one or the other of these substances. The question, then, in view of the more complicated nature of preference behavior, is whether preference and discrimination are performed by the same area or areas of the brain, or by different ones.

It is known that ablation of the taste area of the sensory cortex (Benjamin and Akert, 1959) or ablation of the thalamic relay nucleus for taste (Oakley and Pfaffmann, 1962) impairs performance on the two-bottle preference test. A first working assumption, over-simplified, would be that the cortex is responsible for preference behavior, and that the thalamic lesions affect it by virtue of their interruption of the sensory pathway to the cortex. Is discrimination, then, performed at this same locus, or does it take place elsewhere in the brain? If the cortex is important, its removal should impair performance on a discrimination task. The results

of the sodium chloride experiment reported here show that this is the case. It is obvious that the cortex is not solely concerned with preference, but is also important for a discriminative or sensory function. It is unfortunate that the quinine hydrochloride animals could not be trained on this discrimination task, as their results, if consonant with those for sodium chloride, would considerably strengthen this conclusion.

The simple hypothesis outlined above, that the sensory receiving area for taste mediates both discrimination and preference behavior, is not adequate to account for all the available evidence. Figure 1 shows that the operated animals in the quinine hydrochloride preference experiment reacted with normal aversion to the highest concentration of quinine hydrochloride used, .1 mM, on the first postoperative test. This residual ability to taste very strong solutions after operation has also been mentioned by other investigators. Oakley (1965), using both a two-bottle preference test and a bar pressing task which was reinforced with sucrose, found that rats could still taste very strong sucrose solutions after lesions of the thalamic relay nucleus. Oakley and Pfaffmann (1962) have reported this same finding for sodium chloride, using a two-bottle preference test. Both the control and the operated groups avoided sodium chloride at the

strongest concentration used, which was 1.0 M. In the sodium chloride discrimination experiment reported here, this phenomenon was not observed, since the strongest solution used was .1 M, a concentration at which performance was impaired in the Oakley and Pfaffmann study. Thus, neither cortical nor thalamic lesions leave an animal totally unable to taste; in both these cases, some residual ability remains, and must depend on some brain area other than the primary sensory cortex or the thalamic relay nucleus.

An examination of the second postoperative test results for the quinine hydrochloride experiment, also shown in Figure 1, reveals that the operated animals have almost recovered from the operation. Their reaction to the three highest concentrations of quinine hydrochloride is very similar to that of the control group, indicating a return to normality for these concentrations. Since the second postoperative test began twenty-five days after surgery, it would be expected that most of the trauma sustained at operation, in the form of interruption would have dissipated by this time. The recovery, then, is assumed to be a true compensation. Benjamin and Akert (1959) have also reported a return to near normal preference behavior by the second month following cortical ablations. These results lead to the same conclusion as

that drawn in the preceding paragraph, namely that some brain area other than the primary receiving area plays a part in ability to taste. It is not yet possible to say whether this other area is functional in the normal intact animal, or whether it is active only under emergency conditions, such as would be caused by the removal of the cortical taste zone.

This recovery phenomenon was not observed in the operated sodium chloride discrimination animals, and for this reason data from the two postoperative tests was combined. Since the interval between surgery and the second postoperative test for this group was longer than for the quinine hydrochloride preference group, the animals presumably had completely recovered from surgical trauma. A possible explanation of this difference between the groups concerns the greater difficulty of the discrimination task. It is more complex than the preference test and so a greater degree of neural reorganization would be required for any compensation to be evident.

Benjamin (1959) reports that complete neodecortication produces larger deficits in preference behavior than do ablations of the cortical taste area alone. But, "no cortical lesion located outside this (taste nerve) area will produce impairment". (Benjamin and Akert, 1959, p. 237) The effects of removing cortex other than the primary taste area are manifest only in an

additive fashion, and there is no data available as to the duration of the deficit, or the amount or location of outside cortex which must be included in the removal to obtain deficits comparable to neodecortication. It is possible that the removal of a relatively restricted area in addition to the primary taste zone would be sufficient, indicating the existence of an additional taste area analogous to somatic sensory II. On the other hand, the effect may be a general one, much like the results of the mass action experiments (Morgan, 1951). On the whole, the effects of neodecortication would seem to indicate that the other brain areas important in taste are cortical. As mentioned in the Introduction, Benjamin (1955a and b) has conducted experiments with rats on low versus high fluid deprivation schedules, and his results tend to confirm the importance of cortical areas other than the primary receiving zone in taste preference.

Quinine hydrochloride aversion thresholds average approximately 15×10^{-6} M for normal intact animals. Lesions of the cortical taste area raise these thresholds to about 80×10^{-6} M, and direct coagulation of the thalamic relay nucleus for taste results in a further elevation to about 200×10^{-6} M. Moreover, the impairment produced by thalamic ablations is much more enduring, Oakley and Pfaffmann (1962) reporting that a substantial deficit still remains by the seventh month, in contrast to the near normal performance of cortically damaged animals by the second month.

One interpretation of these results has been offered by Ables and Benjamin (1960). Cortical ablations do not result in complete degeneration of the thalamic taste nucleus, and it is presumed that the small neurons which remain send axons to other subcortical structures. This explanation indicates the existence of diencephalic or telencephalic structures, exclusive of the thalamocortical pathways, which also function in taste discrimination. This interpretation does not account for the neodecortication results, but a minor modification of it would, namely the inclusion of the possibility of thalamic projections to cortex other than the primary taste cortex.

In summary, there are four findings which indicate that brain areas other than the primary taste cortex are functional in taste preference and discrimination. A. Animals subjected to cortical and thalamic ablations retain the ability to taste very strong solutions. B. Recovery of normal function after cortical ablations is nearly complete by the second postoperative month. C. Neodecortication results in larger deficits than does ablation of the primary taste cortex alone. D. Direct destruction of the thalamic relay nucleus produces even more severe impairment, which is also relatively permanent. These results point to two distinct, though not mutually exclusive possibilities for the location of these other areas

functional in taste, namely another cortical zone separate from the primary receiving area, or some portion of the subcortex. The studies to be reported next show that there is good evidence in support of both of these location possibilities, and also provide information on whether these accessory structures function in the intact animal or only in an emergency such as would be caused by damage to the primary system. However, neither these studies nor any work done in the area of taste itself permit any statements about which of these possibilities are the most likely.

Glees and Cole (1950) located the part of the motor cortex in monkeys which, when stimulated, gave rise to movements of the thumb. They then ablated this area and tested the monkeys postoperatively on a matchbox problem which required the animals to use apposition of the forefinger and thumb in order to open the box and obtain a food reward. The animals gradually recovered the use of their thumbs, and the strength of the tested grasping response increased steadily over time. A second operation was then performed; it was found that stimulation of cortex on the borders of the lesion would now give rise to thumb movements, whereas it did not in the original exploration. Ablation of this second area resulted in a return of thumb paralysis, and again, there was eventual recovery.

Maruyama and Kanno (1961) bilaterally ablated the auditory cortex in cats which had been trained on an avoidance task with tone intensity as the CS. They found a postoperative threshold increase of 5 to 20 db. above normal, and then explored the cortex for evoked potentials to the tone CS's. These were found in a restricted area of somatic sensory I, the ventral part of the posterior sigmoid gyrus, but only when the primary auditory cortex had been completely destroyed. This second auditory area did not develop if the original lesion was incomplete. Ablation of both the primary auditory cortex and the sigmoid gyrus resulted in a much more severe deficit, postoperative thresholds rising to 45 to 65 db. above normal.

Both these studies bear on the problem of recovery of function following brain damage, the same phenomenon as is shown by the operated quinine hydrochloride preference group in the experiments reported in this thesis. There is conclusive evidence in these two papers that compensation in the motor system and the auditory sensory system occurs at a cortical level. However, there is equally good support for the proposal that the subcortex is also operative in discrimination.

It is well known that a visual discrimination based on intensity differences is lost after the removal of the striate cortex, but it is also true that relearning

of this discrimination proceeds as easily and quickly as it does in normal intact animals (Lashley, 1929; Smith, 1937). The conclusion drawn from these results is that the visual cortex normally mediates the learning of intensity discriminations, but in its absence subcortical centers are capable of performing this function. This conclusion assigns to the subcortex a sort of emergency function. On the other hand, Katsuki assigns to the subcortex, and specifically to the medial geniculate nucleus, the normal function of discriminating pitch and intensity (Katsuki, Watanabe, and Maruyama, 1959), leaving to the auditory cortex the integration of these analyzed components. In other words, simple discriminations are performed at a subcortical level, while the cortex is responsible for the discrimination of timbre, melody, and so on. This question of whether the secondary area performs a normal or only an emergency function in discrimination, if indeed it performs such a function at all, is one which is also relevant to the cortex. Both Glees and Cole (1950) and Maruyama and Kanno (1961) find that their secondary areas are not active in a normal intact animal, but only develop when the primary sensory or motor cortex is missing.

A more outspoken proponent of the subcortex as the locus for not only simple discriminations, but in fact all discriminations, is Penfield, (1958, 1959).

He proposed a centrencephalic system, located in the higher brain stem, whose function is the integration and coordination of the activity of the two hemispheres. This system is "most intimately associated with the initiation of voluntary activity and with the sensory summation prerequisite to it..." (Penfield and Roberts, 1959, p. 21). Though it is not specifically asserted that discrimination is performed by the centrencephalic system, the phrase "sensory summation" would indicate that this is a reasonable assumption. Thompson (1965) makes this assumption much more explicitly in his review of the published findings concerned with the effect of various lesions on visual pattern and intensity discriminations. His conclusion is that both of these are performed by the centrencephalic system, and the occipital cortex acts only as another relay on the way to the true locus of the memory traces in the mesencephalon.

To summarize, the primary taste cortex is important in both preference and discrimination behavior, but it is not the only important structure. Other areas implicated are the subcortex and cortex separate from the taste zone. Unfortunately, all the work pointing to the influence of these additional structures has used a preference technique as its indicator of taste sensitivity, and these conclusions cannot be generalized to the discrimination case without reservation. Though

there are good discrimination techniques available (Carr, 1952; Harriman and MacLeod, 1953; Morrison and Morrison, 1966), none of these have been used in combination with ablations in an attempt to establish whether the preference results can be extended to discrimination, or to more precisely define other areas functional in these two tasks.

Turning now to the failure of the quinine hydrochloride discrimination animals to successfully complete the training period, several features of the training procedure and of the behavior of the animals may offer a partial explanation of this. The training procedure used here is a slight variation on the original developed by Morrison and Morrison (1966), which involved the use of tone (S^D) and no-tone (S^Δ) periods as additional cues to the animal of when reinforcement was or was not available. This was first superimposed on the bar alternation, then itself put under the control of the rat's licking, in the counterpart of what is here called stage III. That is, a predetermined number of licks turned on the tone, and a bar press terminated it, being reinforced or not depending on whether the solution was correctly identified. Moreover, the animals used in this experiment were highly practised in the discrimination situation, having been originally trained on sodium

chloride, and subsequently tested on sodium chloride, sucrose, and tartaric acid before ever encountering quinine. By contrast, the animals used in the experiments reported here were totally naive, and the only cue to the availability of reinforcement was the removal of the drinking spout from the testing box. Nevertheless, the animals trained with sodium chloride became proficient at the task, so the differences mentioned here cannot totally account for the failure of the quinine hydrochloride rats.

Observation of the behavior of both the sodium chloride and quinine hydrochloride animals while in the discrimination situation revealed several differences. As training progressed, the sodium chloride animals developed a very stereotyped response pattern, licking quickly and efficiently, then always turning away from the spout in the same direction. For any one animal, the direction of the turn could be either to the right or the left, but was always quite consistent. The animals would effectively circle the box, stop in front of the appropriate lever, and press it. By contrast, the quinine hydrochloride animals seemed quite random in their behavior, and never developed these stereotyped turning responses. There was no difference in the licking behavior of the two groups, both licking quickly and sometimes biting at the spout. This is surprising

in view of the fact that rats find quinine a very aversive substance, and this aversion could be expected to be evident in their sampling of the bitter solutions. The subjective impression gained from observation of these animals was that the aversive properties of quinine had generalized to the whole discrimination situation, and were not restricted to those trials on which the animal was forced to sample this substance. The animals seemed impatient to "get it over with", and uncaring whether they were reinforced or not, willing to settle for chance reinforcement. It seemed, in effect, that a competition between the noxious aspects of the situation and the hunger of the animals had been established, and that they were compromising between these two opposing tendencies.

Finally, the inconclusive nature of the results from the sodium chloride preference experiment must be dealt with. Motivation induced by the hedonic properties of the stimulus has been previously mentioned as a powerful influence on preference results. If it is strong, it acts to reduce the zone of indifference and lower the value of the preference threshold to approximately that of the sensory threshold. This motivation to obtain or avoid the substance itself is not nearly as strong in the case of sodium chloride as it is for sucrose or quinine, and consequently the two thresholds would remain well separated. It would be expected, then,

that a preference test would not be a good estimator of sensitivity to sodium chloride (Koh and Teitelbaum, 1961). This is indeed the case. A comparison of the results from the sodium chloride preference and discrimination experiments reveals that the former are very unclear, so much so that it is virtually impossible to conclude anything about sensory capacity from them. On the other hand, the discrimination results are unequivocal; there is order in the relationship between concentration and intake, whereas it is lacking for the preference animals, especially in the preoperative test, where the clearest relationship should be found. In general intake seems to decrease as solution concentration decreases, but these results do not permit a decision about the reason for this. It may be that rats prefer sodium chloride to water at the highest concentration used, but that the remaining weaker concentrations fall within the zone of indifference, and though they can be discriminated from water, are not preferred. Alternatively, there may be a genuine failure to discriminate. Both of these hypotheses would give rise to the results obtained, namely that the animals drink approximately equal amounts of sodium chloride and water. Moreover, it was expected that the lesions would make the operated animals unable to taste sodium chloride, and therefore they would drink approximately equal amounts

of both sodium chloride and water after surgery, in contrast to the control group, which was expected to maintain its preoperative performance. However, the preoperative test results are sufficiently irregular that it is difficult to evaluate any postoperative change. The effect of the lesions on discrimination is very clear, whereas their effect on preference, if any, is confounded with a general tendency toward increased salt intake on the part of both the control and operated groups.

To account for this, it seems necessary to postulate some influence other than the ablations, which would take the form of a learning or practice effect. Neither water nor sodium chloride are aversive to the rat; therefore, when the animals are thirsty and begin to drink at either bottle, they would presumably continue drinking until their thirst was satiated, since the contents of both are acceptable. It would then take some time, and perhaps contact with both bottles within a short period of time, for the animals to learn that the bottles contain different substances. Presumably, once the comparison and discrimination is made, the animals would immediately begin to prefer the sodium chloride to water. The overt behavior manifested before this learning takes place would be a continuation of drinking at the water bottle, once it was initiated, but after

the preference had been established, the animals would leave the water bottle and drink from the sodium chloride bottle. Over the course of strengthening this habit, the number of switches to the sodium chloride bottle from the water bottle would increase, and the latency of the switches would decrease. The total effect of this learning would be that a greater and greater percentage of the total fluid intake would be represented by sodium chloride. This is, in fact, what can be seen in Figure 2, for both postoperative tests I and II. The operated group are presumably somewhat impaired in their ability to taste, so this learning would proceed more slowly.

It is assumed that this learning would also take place if the fluids presented were quinine and water, but its time course would be so short that it would go unnoticed in the results. Quinine is an extremely aversive substance, and therefore the first contact with it would be a very brief one. A thirsty rat would not satiate his thirst at the quinine bottle, but would immediately stop drinking until his first accidental contact with the water bottle. In effect, the bitter quinine solution would force the animal to search for another means of satiating his thirst; the discrimination between water and quinine would be quickly made, even within the first session of testing. Some support for

this proposal comes from the first twenty-four hours of preoperative testing with .1 mM quinine hydrochloride. Seven animals drank only 1 ml., three animals drank 2 ml., and one animal drank 4 ml., in contrast with a mean intake of 47 ml. of water (range 32 to 65 ml.).

SUMMARY

Rats were tested both before and after bilateral ablations of the taste sensory cortex for sensitivity to two different substances. For the first two groups, the sensitivity measure used was the two-bottle preference technique; these groups will be referred to as the sodium chloride (NaCl) preference group and the quinine hydrochloride (QHCl) preference group. The third group was exposed to NaCl in a signal detection type discrimination task. A fourth group, QHCl discrimination, could not be successfully trained in this task and so was discarded.

Results and conclusions were as follows:

1. The QHCl preference animals show a large deficit on the first postoperative test, manifested as an increased intake of QHCl at all concentrations, and also as a breakdown in the normal relationship, seen preoperatively, between decreases in concentration and increases in intake. However, by the second post-operative test approximately one month after surgery, they perform almost normally, reacting with typical aversion to the three highest of the six QHCl concentrations used.

2. The results for the NaCl preference group are unclear compared to those for QHCl. No marked preference is evident on the preoperative test, nor do the lesions produce a difference between operated and control groups, as was predicted. Rather, there is a tendency toward increasing NaCl intake over the three stages of the experiment, and it overshadows any possible effect of the lesions. A practice or learning factor is proposed to account for this. Since NaCl is not so extreme in its hedonic properties as is the very aversive QHCl, the inconclusive nature of the results was not surprising; for NaCl, a preference test of the kind used here is not a reliable indicator of absolute sensitivity.

3. The NaCl discrimination group gives results which support the above conclusion, as the postoperative deficit is very clear, with performance accuracy decreasing at all concentrations. The fact that the lesions affect discrimination is also important, since it is known that preference and discriminative or sensory thresholds are different. This indicates two separate functions, of which preference is the more complex, depending on discrimination. These results establish that the cortical taste area is important in both, but also that it is not solely responsible. The rapid recovery of the QHCl preference animals points to auxiliary brain structures which function in taste

preference and discrimination. Information relevant to this is discussed, but no precise localization of these accessory structures is yet possible.

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APPENDICES

Summary tables used in analyses of variance.

A represents concentrations, A_1 being the strongest. B represents stages of the experiment, with B_1 the preoperative stage. C_1 are the operated group, and C_2 the control group. For the two preference experiments, the data is expressed as the percentage of total fluid intake represented by the solutions. For the NaCl discrimination experiment, the data is expressed as percentage of correct responses.

Quinine hydrochloride preference experiment

ABC summary table

	B_1		B_2	
	C_1	C_2	C_1	C_2
A_1	23.50	10.01	50.53	9.90
A_2	88.83	70.49	453.82	45.58
A_3	228.29	107.19	349.63	183.03
A_4	278.72	128.79	289.40	153.93
A_5	266.07	154.18	320.76	107.64
A_6	291.55	130.34	349.40	153.52

B_3

	C_1	C_2
A_1	88.02	63.75
A_2	180.86	63.21
A_3	208.68	151.15
A_4	349.18	120.52
A_5	380.87	154.77
A_6	333.86	161.19

AB summary table

	B_1	B_2	B_3
A_1	33.51	60.43	151.77
A_2	159.32	499.40	244.07
A_3	335.48	532.66	359.83
A_4	407.51	443.33	469.70
A_5	420.25	428.40	535.64
A_6	421.89	502.92	495.05

AC summary table

	C_1	C_2
A_1	162.05	83.66
A_2	723.51	179.28
A_3	786.60	441.37
A_4	917.30	403.24
A_5	967.70	416.59
A_6	674.81	445.05

BC summary table

	B_1	B_2	B_3
C_1	1176.96	1813.54	1541.47
C_2	601.00	653.60	714.59

Sodium Chloride preference experiment

ABC summary table

	B_1		B_2	
	C_1	C_2	C_1	C_2
A_1	279.11	201.14	254.83	187.28
A_2	211.92	120.22	316.52	166.85
A_3	212.20	127.00	239.86	149.74
A_4	157.29	176.40	313.33	126.99
A_5	225.15	157.32	231.87	169.60
A_6	192.96	112.79	188.58	155.00
A_7	188.57	157.09	253.18	156.66

	B_3	
	C_1	C_2
A_1	285.15	200.58
A_2	327.12	190.39
A_3	309.98	159.01
A_4	248.07	174.54
A_5	327.63	141.48
A_6	275.15	159.77
A_7	240.27	132.83

AB summary table

	B_1	B_2	B_3
A_1	480.25	442.11	485.73
A_2	332.14	483.37	517.51
A_3	339.20	389.60	468.99
A_4	333.69	440.32	422.61
A_5	382.47	401.47	469.11
A_6	305.75	343.58	434.92
A_7	345.66	409.84	373.10

AC summary table

	C_1	C_2
A_1	819.09	589.00
A_2	855.56	477.46
A_3	762.04	435.75
A_4	718.69	477.93
A_5	784.65	468.40
A_6	656.69	427.56
A_7	682.02	446.58

BC summary table

	B_1	B_2	B_3
C_1	1467.20	1798.17	2013.37
C_2	1051.96	1112.12	1158.60

Sodium chloride discrimination experiment

ABC summary table

	B_1		B_2	
	C_1	C_2	C_1	C_2
A_1	355.0	325.5	260.0	336.0
A_2	326.0	322.0	257.0	278.0
A_3	249.5	246.5	211.0	250.0
A_4	234.0	247.5	215.0	263.5
A_5	255.0	241.5	219.5	242.0
A_6	235.0	266.5	194.5	223.5
A_7	222.5	218.5	217.5	237.0

AB summary table

	B_1	B_2
A_1	680.5	596.0
A_2	648.0	535.0
A_3	496.0	461.0
A_4	481.5	478.5
A_5	496.5	461.5
A_6	501.5	418.0
A_7	441.0	454.5

AC summary table

	C_1	C_2
A_1	615.0	661.5
A_2	583.0	600.0
A_3	460.5	496.5
A_4	449.0	511.0
A_5	474.5	483.5
A_6	429.5	490.0
A_7	440.0	455.5

BC summary table

	B_1	B_2
C_1	1877.0	1574.5
C_2	1868.0	1830.0

Quinine hydrochloride preference experiment. Rats 1 to 6 are operated animals; rats 8 to 11 are sham operated controls. Figures indicate amount consumed to the nearest ml.

Preoperative test

.1 mM

	Day 1		Day 2	
Rat	H_2O	Q	H_2O	Q
1	65	4	59	1
2	45	2	36	2
3	46	1	53	1
4	39	1	35	1
5	32	2	35	1
6	33	2	49	1
7	44	1	46	1
8	46	1	36	1
9	53	1	31	1
10	57	1	40	2
11	60	1	47	1

.0316 mM

	Day 1		Day 2	
Rat	H_2O	Q	H_2O	Q
1	63	2	64	5
2	44	2	1	16
3	62	1	38	1
4	32	3	26	2
5	34	1	24	1
6	26	12	22	1
7	32	1	31	1
8	35	1	38	1
9	11	28	41	1
10	37	2	42	1
11	31	12	34	12

.01 mM

Rat	Day 1		Day 2	
	H_2O	Q	H_2O	Q
1	20	37	55	6
2	7	32	22	25
3	37	5	79	1
4	27	12	39	6
5	29	0	28	10
6	8	16	5	50
7	37	1	35	1
8	32	1	35	1
9	13	27	41	3
10	30	13	41	1
11	37	9	7	32

.00316 mM

Rat	Day 1		Day 2	
	H_2O	Q	H_2O	Q
1	3	56	62	1
2	34	13	25	19
3	1	53	53	1
4	19	20	40	5
5	26	6	11	24
6	24	17	6	38
7	35	4	43	1
8	15	17	32	1
9	1	42	40	1
10	7	17	35	1
11	59	1	37	13

.001 mM

Rat	Day 1 H_2O	Day 1 Q	Day 2 H_2O	Day 2 Q
1	1	63	58	0
2	22	18	31	14
3	16	31	22	24
4	36	5	39	1
5	30	5	13	21
6	34	17	30	16
7	31	5	13	26
8	12	19	38	1
9	8	34	28	18
10	28	11	38	1
11	27	17	18	22

.000316 mM

Rat	Day 1 H_2O	Day 1 Q	Day 2 H_2O	Day 2 Q
1	47	1	24	29
2	24	10	36	6
3	23	13	1	68
4	20	22	33	11
5	8	25	3	37
6	31	5	31	13
7	27	4	20	17
8	30	1	19	5
9	4	38	27	5
10	18	13	19	4
11	21	22	32	8

Postoperative test I

.1 mM

	Day 1		Day 2	
Rat	H_2O	Q	H_2O	Q
1	43	11	42	0
2	44	5	38	8
3	37	2	33	1
4	38	0	24	1
5	34	7	28	2
6	43	3	35	2
7	41	1	33	1
8	41	1	30	1
9	37	1	28	1
10	49	1	39	1
11	63	1	48	1

.0316 mM

	Day 1		Day 2	
Rat	H_2O	Q	H_2O	Q
1	1	46	1	44
2	21	14	3	33
3	13	24	19	18
4	14	14	4	28
5	17	1	1	27
6	23	14	4	32
7	1	32	27	1
8	26	1	24	1
9	11	10	12	4
10	34	1	25	1
11	47	1	48	1

.01 mM

Rat	Day 1		Day 2	
	H_2O	Q	H_2O	Q
1	1	46	1	38
2	25	17	13	6
3	18	16	14	12
4	42	1	24	8
5	2	29	14	20
6	18	12	6	11
7	25	13	16	4
8	48	2	44	13
9	31	19	6	33
10	44	7	1	25
11	1	48	52	5

.00316

Rat	Day 1		Day 2	
	H_2O	Q	H_2O	Q
1	10	40	1	53
2	6	37	41	1
3	32	2	36	1
4	48	1	27	18
5	23	12	9	24
6	35	7	26	13
7	37	3	1	33
8	29	2	20	1
9	17	21	14	15
10	11	31	15	4
11	20	38	29	12

.001 mM

	Day 1		Day 2	
Rat	H_2O	Q	H_2O	Q
1	35	17	1	46
2	1	42	37	1
3	7	30	20	17
4	43	1	11	35
5	17	14	22	6
6	20	16	13	29
7	33	1	28	8
8	45	10	20	17
9	24	26	13	31
10	37	2	35	2
11	46	1	42	8

.000316 mM

	Day 1		Day 2	
Rat	H_2O	Q	H_2O	Q
1	36	9	9	31
2	1	36	40	1
3	5	26	26	9
4	23	8	1	28
5	11	18	7	19
6	19	12	11	6
7	28	8	21	13
8	43	1	7	31
9	29	11	11	14
10	30	5	21	1
11	7	41	31	17

Postoperative II

.1 mM

	Day 1		Day 2	
Rat	H_2O	Q	H_2O	Q
1	25	3	48	1
2	32	1	41	2
3	20	7	32	1
4	15	5	30	16
5	22	3	33	1
6	9	5	40	4
7	33	1	23	1
8	34	1	41	1
9	40	1	15	19
10	35	1	30	1
11	36	38	28	2

.0316 mM

	Day 1		Day 2	
Rat	H_2O	Q	H_2O	Q
1	1	43	39	7
2	36	1	31	1
3	35	1	2	36
4	46	1	39	1
5	1	29	25	1
6	24	11	40	1
7	31	1	30	1
8	29	3	31	1
9	5	26	29	7
10	33	1	33	1
11	46	1	38	1

.01 mM

	Day 1		Day 2	
Rat	H_2O	Q	H_2O	Q
1	6	34	46	1
2	41	1	35	1
3	36	1	1	34
4	28	9	43	1
5	23	7	1	25
6	13	21	38	5
7	36	1	32	1
8	6	40	41	1
9	10	23	26	12
10	5	27	30	1
11	38	6	43	5

.00316 mM

	Day 1		Day 2	
Rat	H_2O	Q	H_2O	Q
1	1	40	27	5
2	1	36	31	1
3	35	1	1	39
4	20	22	25	12
5	18	10	10	15
6	12	1	2	14
7	24	28	14	17
8	39	1	30	8
9	1	34	33	2
10	31	1	23	3
11	40	1	1	39

.001 mM

	Day 1		Day 2	
Rat	H_2O	Q	H_2O	Q
1	2	5	22	22
2	35	1	1	39
3	11	39	64	9
4	9	12	41	1
5	5	15	18	26
6	11	14	36	2
7	5	17	25	17
8	8	8	24	13
9	5	2	1	39
10	26	3	28	1
11	36	39	45	1

.000316 mM

	Day 1		Day 2	
Rat	H_2O	Q	H_2O	Q
1	42	5	1	43
2	35	2	1	39
3	15	16	43	5
4	24	15	16	20
5	21	18	22	5
6	2	39	31	1
7	15	18	6	29
8	13	22	9	14
9	33	8	7	18
10	24	5	33	1
11	25	19	18	14

Sodium chloride preference experiment. Rate 1 to 5 are operated animals; rats 6 to 8 are operated controls. Figures indicate amount consumed to the nearest ml.

Preoperative test

.1 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	14	13	2	24
2	41	1	27	9
3	11	15	9	18
4	5	35	3	23
5	21	15	16	15
6	7	45	17	20
7	5	33	9	32
8	37	14	27	61

.0316 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	21	17	1	29
2	50	5	24	8
3	19	31	10	31
4	23	13	31	4
5	25	13	27	11
6	30	11	25	11
7	27	1	8	42
8	42	2	6	60

.01 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	11	10	8	15
2	33	6	7	7
3	12	10	28	13
4	22	10	17	13
5	20	16	10	10
6	26	11	3	6
7	35	3	23	29
8	60	25	22	40

.00316 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	43	7	1	34
2	57	9	10	36
3	39	11	35	1
4	40	1	30	1
5	12	37	34	2
6	18	42	35	9
7	21	52	12	34
8	45	27	13	51

.001 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	25	6	4	38
2	44	1	14	49
3	29	2	22	19
4	28	6	21	21
5	2	38	22	18
6	21	28	41	16
7	16	20	11	38
8	71	2	4	58

.000316 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	24	9	10	15
2	35	11	13	13
3	21	17	19	19
4	23	10	12	12
5	25	1	13	11
6	23	1	12	20
7	19	14	21	10
8	62	3	10	39

.0001 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	30	5	1	31
2	60	2	18	27
3	23	12	28	5
4	33	15	14	13
5	15	33	37	2
6	35	22	13	29
7	42	7	2	33
8	49	19	15	35

Postoperative I

.1 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	14	6	1	14
2	6	6	6	1
3	1	18	3	5
4	3	3	6	1
5	13	1	1	14
6	34	26	11	29
7	15	23	1	31
8	72	20	18	70

.0316 M

Rat	Day 1 H_2O	Day 1 NaCl	Day 2 H_2O	Day 2 NaCl
1	7	2	4	27
2	1	3	16	30
3	1	8	27	13
4	2	8	12	31
5	4	1	3	27
6	18	5	33	17
7	12	19	4	40
8	19	19	16	50

.01 M

Rat	Day 1 H_2O	Day 1 NaCl	Day 2 H_2O	Day 2 NaCl
1	25	1	7	20
2	5	24	26	6
3	17	15	4	3
4	21	3	1	6
5	12	3	1	12
6	32	9	12	22
7	22	11	5	18
8	44	3	3	55

.00316 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	11	4	1	32
2	17	9	4	37
3	4	8	27	10
4	1	12	9	27
5	4	1	1	34
6	22	5	27	21
7	20	3	2	45
8	33	10	23	34

.001 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	25	1	3	18
2	39	6	20	18
3	1	36	20	1
4	11	20	13	10
5	25	2	1	24
6	16	20	15	26
7	29	9	3	21
8	47	7	2	49

.000316 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	32	1	1	26
2	35	4	7	32
3	26	5	27	5
4	34	4	16	16
5	38	1	3	29
6	26	6	13	27
7	25	9	8	21
8	33	12	1	54

.0001 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	18	1	1	15
2	41	1	8	18
3	5	21	9	1
4	9	18	6	18
5	32	2	1	37
6	28	11	16	19
7	31	4	1	25
8	40	14	1	36

Postoperative II

.1 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	24	30	21	1
2	13	55	6	32
3	6	52	28	10
4	5	61	29	1
5	36	20	1	41
6	5	66	24	14
7	42	23	2	51
8	47	41	5	68

.0316 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	11	12	1	25
2	41	1	1	34
3	1	30	11	6
4	1	37	6	21
5	32	1	1	25
6	8	28	19	17
7	33	2	1	54
8	19	25	4	77

.01 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	24	1	1	18
2	1	41	19	13
3	3	36	31	1
4	1	27	3	26
5	41	2	1	30
6	16	23	24	12
7	33	9	18	23
8	16	39	10	34

.00316 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	24	1	1	21
2	41	1	31	1
3	1	24	7	15
4	13	17	8	14
5	28	3	1	32
6	11	20	13	26
7	17	15	10	28
8	38	2	5	60

.001 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	17	19	1	20
2	21	9	3	43
3	3	24	4	17
4	11	15	10	13
5	37	1	1	33
6	19	20	33	6
7	6	25	21	11
8	54	3	2	48

.000316 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	17	1	2	22
2	36	1	4	32
3	12	9	7	15
4	1	23	16	13
5	27	5	2	32
6	28	6	9	35
7	19	10	1	38
8	50	7	14	50

.0001 M

	Day 1		Day 2	
Rat	H_2O	NaCl	H_2O	NaCl
1	18	5	1	28
2	27	5	12	25
3	5	8	26	4
4	1	28	26	2
5	27	1	1	31
6	27	8	16	24
7	28	8	9	23
8	37	13	25	42

Sodium chloride discrimination experiment. Rats 1 to 4 are operated animals; rats 5 to 8 are sham operated controls.

Preoperative test

Cycle I .1 M

Rat	Correct responses R Bar	L Bar	Total Trials	% Reinf.
1	22	24	50	42
2	34	31	70	93
3	25	23	50	96
4	17	18	50	70
5	25	21	50	92
6	23	24	50	94
7	20	15	50	70
8	22	18	50	80

Cycle II

Rat				
1	25	23	53	91
2	31	32	64	98
3	23	23	50	92
4	19	20	50	78
5	25	18	56	77
6	22	24	50	92
7	10	21	50	62
8	22	20	50	84

.0316 M

Cycle I	Correct responses		Total	%
Rat	R Bar	L Bar	Trials	Reinf.
1	17	25	50	84
2	20	19	50	78
3	23	23	50	92
4	17	19	50	72
5	22	22	50	88
6	21	22	50	86
7	24	19	50	86
8	24	26	52	96
Cycle II				
1	32	35	80	84
2	24	23	50	94
3	22	23	50	90
4	17	12	50	58
5	14	19	50	66
6	22	24	50	92
7	8	21	50	58
8	16	20	50	72

.01 M

Cycle I				
1	17	19	50	72
2	14	12	50	52
3	11	17	50	56
4	19	22	70	59
5	11	21	60	53
6	12	19	50	62
7	19	11	51	59
8	9	21	51	59
Cycle II				
1	14	18	50	64
2	10	16	50	52
3	19	25	50	88
4	17	11	50	56
5	15	17	50	64
6	23	25	50	96
7	3	20	50	46
8	11	16	50	54

.00316 M

Cycle I	Correct responses		Total Trials	% Reinf.
Rat	R Bar	L Bar		
1	13	20	50	66
2	10	19	50	58
3	14	17	50	62
4	13	12	50	50
5	10	16	50	52
6	11	23	50	68
7	24	9	50	66
8	11	19	51	59
Cycle II				
1	7	18	50	50
2	7	13	50	40
3	15	27	55	76
4	17	16	50	66
5	13	18	50	62
6	15	23	50	76
7	5	21	50	52
8	12	18	50	60

.001 M

Cycle I				
1	12	21	50	66
2	10	21	50	62
3	14	21	50	70
4	12	23	50	70
5	11	18	50	58
6	9	21	50	60
7	22	13	50	70
8	12	16	50	56
Cycle II				
1	10	15	50	50
2	10	18	50	56
3	12	21	50	66
4	19	16	50	70
5	11	18	50	58
6	13	25	60	63
7	6	24	50	60
8	11	18	50	58

.000316 M

Cycle I	Correct responses			
Rat	R Bar	L Bar	Total Trials	% Reinf.
1	10	19	50	58
2	12	19	50	62
3	8	19	50	54
4	10	16	50	52
5	8	21	50	58
6	10	21	50	62
7	21	13	60	57
8	12	22	50	68
Cycle II				
1	10	20	50	60
2	14	17	50	62
3	10	19	50	58
4	17	15	50	64
5	20	16	50	72
6	19	24	50	86
7	7	24	50	62
8	14	20	50	68

.0001 M

Cycle I				
1	12	15	50	54
2	11	22	50	66
3	7	23	50	60
4	10	14	50	48
5	13	18	50	62
6	12	21	50	66
7	14	16	50	60
8	8	20	50	56
Cycle II				
1	15	29	70	63
2	9	13	50	44
3	13	18	50	62
4	13	11	50	48
5	6	8	50	28
6	13	22	51	69
7	4	23	50	54
8	7	14	50	42

Postoperative test

Cycle I — .1 M

Rat	Correct responses: R Bar	Correct responses: L Bar	Total Trials	% Reinf.
1	16	20	50	72
2	16	14	50	60
3	26	24	67	75
4	22	11	50	66
5	19	19	50	76
6	23	21	50	88
7	13	23	50	72
8	22	20	50	84

Cycle II

1	21	16	51	73
2	11	12	50	46
3	12	20	50	64
4	17	15	50	64
5	24	20	50	88
6	24	23	50	94
7	17	25	50	84
8	19	24	50	86

.0316 M

Cycle I

1	13	18	50	62
2	14	12	50	52
3	21	16	55	67
4	20	12	51	63
5	11	17	50	56
6	25	24	60	82
7	8	22	50	60
8	12	21	50	66

Cycle II

1	18	16	50	68
2	13	16	50	58
3	19	22	50	82
4	17	14	50	62
5	23	20	51	84
6	21	25	50	92
7	4	23	50	54
8	8	24	52	62

Cycle I — .01 M

Rat	Correct responses R Bar	L Bar	Total Trials	% Reinf.
1	13	17	50	60
2	34	20	105	51
3	13	13	50	52
4	18	9	50	54
5	12	18	50	60
6	18	23	50	82
7	5	21	50	52
8	19	18	50	74
Cycle II				
1	15	11	50	52
2	15	13	53	53
3	3	24	50	54
4	11	12	50	46
5	17	15	50	64
6	16	19	50	70
7	3	23	50	52
8	2	21	50	46
		.00316 M		
Cycle I				
1	10	10	50	40
2	15	9	50	48
3	9	16	50	50
4	17	10	50	54
5	18	14	50	64
6	22	26	54	89
7	11	22	50	66
8	10	21	50	62
Cycle II				
1	20	13	50	66
2	21	18	63	62
3	9	15	50	48
4	19	12	50	62
5	18	11	50	58
6	16	23	50	78
7	1	22	50	46
8	9	23	50	64

Cycle I — .001 M

Rat	Correct responses: R Bar	L Bar	Total Trials	% Reinf.
1	11	13	50	48
2	23	27	80	63
3	5	20	50	50
4	15	11	50	52
5	13	18	50	62
6	16	19	50	70
7	12	21	50	66
8	9	17	50	52
Cycle II				
1	12	11	50	46
2	18	15	50	66
3	18	12	50	60
4	15	12	50	54
5	12	16	50	56
6	15	20	50	70
7	4	23	50	54
8	5	22	50	54
Cycle I		.000316 M		
1	8	9	52	33
2	10	14	50	48
3	5	23	50	56
4	15	15	50	60
5	9	16	50	50
6	17	20	50	74
7	4	20	50	48
8	5	18	50	46
Cycle II				
1	14	17	50	62
2	11	10	50	42
3	2	16	50	36
4	13	13	50	52
5	15	20	50	70
6	11	20	50	62
7	4	24	50	56
8	1	19	49	41

.0001 M

Cycle I

Rat	Correct responses R Bar	L Bar	Total Trials	% Pref.
1	12	12	50	48
2	10	18	60	47
3	10	17	50	54
4	16	12	50	56
5	12	15	50	54
6	8	18	50	52
7	4	23	50	54
8	5	22	50	54

Cycle II

1	13	9	50	44
2	17	13	50	60
3	13	20	50	66
4	19	11	50	60
5	11	18	50	58
6	19	22	50	82
7	3	25	50	56
8	10	22	50	64