

CHANGES IN REINFORCING PROPERTIES OF STIMULI FOLLOWING ABLATION OF THE AMYGDALOID COMPLEX IN MONKEYS¹

J. S. SCHWARTZBAUM²

Institute of Living, Hartford, Connecticut

It has now been demonstrated that many of the behavioral disturbances found by Klüver and Bucy (1939) after bilateral temporal lobectomy in monkeys have reference to functions subserved by structures in the vicinity of the amygdaloid complex. Bilateral ablation of the amygdaloid complex and adjacent temporal polar cortex results in the tameness, reduced dominance, oral and "hyper-metamorphotic" tendencies, and altered sexual and dietary activities observed after the more radical temporal lobectomy (see Rosvold [1959] for a recent summary of this work).

How these symptoms of the Klüver-Bucy syndrome are interrelated poses a problem that can be attacked both by anatomical fractionation of the syndrome (e.g., Green, Clemente, & de Groot, 1957) and by systematic behavioral analysis of each of the symptoms. In this way it should be possible to specify both the general and specific functions involved. To date, relatively few studies have dealt with the dietary changes.

In both primates and carnivores, resection of the amygdaloid complex results in hyperphagia (Morgan & Kosman, 1957) and, what is perhaps more unique, seemingly indiscriminate dietary behavior (Pribram & Bagshaw, 1953; Schreiner & Kling, 1953). Amygdalectomized monkeys, e.g., will eat normally rejected foods such as meat or fish, exhibit coprophagia, and may even prefer inedible

¹ This experiment is part of a dissertation submitted in partial fulfillment of the PhD degree in the Department of Psychology at Stanford University. The research was supported by a grant, No. M-546(C), from the United States Public Health Service, National Institutes of Mental Health. A report of the findings was presented at the 1958 meeting of the Eastern Psychological Association. The author wishes to express his appreciation to Karl H. Pribram for his help and encouragement throughout the study. Acknowledgment is also due Douglas H. Lawrence and William A. Wilson, Jr. for their assistance in the preparation of the thesis.

² Now in the Department of Psychology, University of Wisconsin.

over edible objects (Pribram & Bagshaw, 1953). These modifications in dietary patterns represent in one sense a change in the reinforcing properties of the objects. Certain objects that formerly had little or no positive reward value become reinforcing to the amygdalectomized animal. They are now responsible for the recurrence of responses directed toward the objects.

Such a conceptualization of the dietary changes may be useful insofar as the changes reflect a more general form of disturbance in the reinforcing properties of stimuli. In the present experiment, an operant form of response was utilized to study the performance of normal and amygdalectomized monkeys for different amounts of a normally accepted food reward. Each of the amounts served in turn as a periodic reinforcement for bar-pressing.

METHOD

Subjects

The Ss were eight preadolescent rhesus monkeys, five males and three females, without previous test experience. Their body weights at the outset of the experiment ranged from 3.6 to 6.4 lb. All were housed in the same room, one or two to a cage. Precautions were taken in grouping the Ss to insure that any change in social status following the brain lesions did not interfere with their feeding behavior in the home cages.

Surgery

Four Ss (397, 405, 438, 442), two males and two females, received bilateral lesions in the anteromedial region of the temporal lobes. The lesions included the amygdaloid complex and part of the surrounding temporal polar cortex. The other four Ss (439, 441, 443, 447) served as operative controls by undergoing a sham operation in which the amygdaloid region was made visible but not resected.

All surgery was performed aseptically in a single stage under Nembutal anesthesia (0.6 cc/kg intrahepatically). A description of the procedure is contained in Pribram and Bagshaw (1953). A part of the temporal bone was removed and the underlying dura excised to expose the temporal polar region. The amygdala could be seen by retracting the temporal lobe and was removed by means of subpial aspiration using a small-gauge sucker. In closing the wound, several

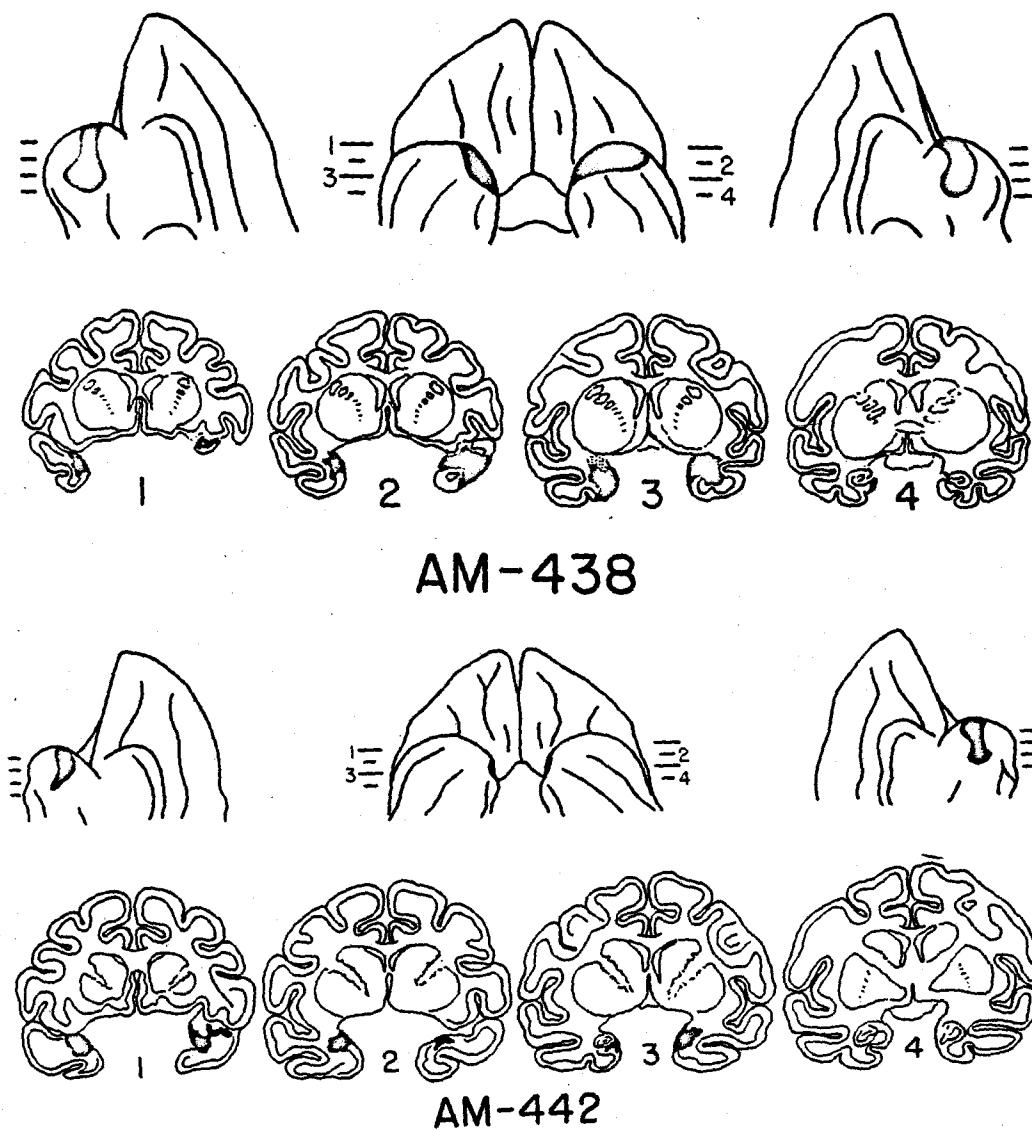


FIG. 1. Reconstructions and cross sections of the smallest and largest lesions in the amygdalectomized animals. (Ventral and medial views are shown in the reconstructions. Cross sections are taken 2 mm. apart.)

layers of interrupted silk sutures were used to approximate the deeper structures and a continuous suture was used to approximate the subcuticular tissue.

Anatomy

After completion of experimentation, the animals with lesions were sacrificed, their brains embedded in celloidin, and the blocks serially sectioned at 50 μ . Every tenth section was stained with aniline thionin, and every fourth stained section was used to make an orthogonal projection onto graph paper. Sections were examined microscopically for evidence of tissue damage.

The anatomical reconstructions and cross sections in Figure 1 show the minimal and maximal extent of the lesions³. In three Ss, excluding AM-438, there was slight sparing unilaterally of the basal part of the amygdala. The removal of the amygdala was in other respects complete. The hippocampal complex suffered minimal damage, with the lesion extending at most to only the anterior tip of Ammon's formation and a small part of the adjoining subiculum and entorhinal cortex. There was additional damage to the temporal poles,

³ Copies of the reconstructions and cross sections of all lesions are available from the author.

especially on the medial surface of the left temporal lobe during the second half of the surgery. In two Ss (AM-397 and AM-405), the lesion on one side invaded cortex along the depths of the superior temporal sulcus. In AM-438 the anterior part of the insula was partially destroyed. There was no evidence in any of the brains of retrograde degeneration in either the lateral geniculate nuclei or the pulvinar of the thalamus.

Apparatus

All tests were carried out in a sound-insulated box whose interior dimensions were 36 $\frac{1}{2}$ in. high, 21 in. wide, and 18 $\frac{1}{2}$ in. deep. The S was confined in a mesh-wire carrying cage (20 in. by 17 $\frac{1}{2}$ in. by 14 $\frac{1}{2}$ in.) that fitted securely on a wooden frame within the box. The animal had access to a lever and a food cup. The lever was a metal bar, 0.5 cm. in diameter and 3.6 cm long, attached to a telephone switch. It was centered 11 $\frac{1}{2}$ in. above the floor of the carrying cage and 4 $\frac{1}{2}$ in. above the food cup, and required a force of at least 60 gm. to open a contact on the switch. A belt-feeder magazine (Ralph Gerbrands Co.) was used to dispense the reward so that the number and size of the food pellets could be varied. A 7 $\frac{1}{2}$ -w. frosted bulb mounted on the ceiling of the box illuminated the interior of the test chamber. With the delivery of a reward, this light went off and a second light mounted above the food cup simultaneously went on for approximately 1 sec. An intake fan circulated air continuously through the test chamber. The relay panels, timers, and counters used to record the behavior and to program the reward were situated outside the test room and thus provided no cues to the animal. An "impulse shortener" prevented S from accumulating responses by holding the lever down continuously.

Procedure

Preliminary training. The animals were trained preoperatively to press the lever on a 2-min. fixed-interval (FI) schedule of reinforcement. Preliminary training required one week of daily tests. At first each response was reinforced. On successive days the reward contingency was shifted to a 20-sec. FI, then a 40-sec., an 80-sec., and finally to a 2-min. FI schedule. Each session continued until S had accumulated 30 to 40 reinforcements. During this time, the daily food ration consisted of four Purina Laboratory Chow pellets and a quarter of an orange.

Preoperative control tests. Each animal received preoperatively a total of nine test sessions with the 2-min. FI schedule; adequate to obtain relatively stable levels of bar-pressing. The tests were spaced on alternate days, excluding Sundays, so as to fall three times a week. Each session lasted 60 min. The reward was programmed so that the first bar-press would be reinforced, marking the start of the 2-min. contingency. A $\frac{1}{2}$ -gm. lab food tablet (P. J. Noyes and Co.), measuring 1.0 cm. in diameter and 0.5 cm. in thickness, served as the reward. Except for the reward, the test conditions described above were kept constant throughout the experiment.

With the start of the preoperative control tests, the daily diet of the Ss was fixed at 8 to 10 Purina Lab

Chow pellets and a quarter of an orange, supplemented by eight unshelled peanuts on the nontest days. They were fed in their home quarters 2 to 3 hr. after each test session, and 24 hr. before the start of the next session. The diet was designed to allow a gradual increase in body weight of about 1 to 2 oz. per week. Weights were taken prior to each test so that the food ration could be adjusted when necessary to maintain this schedule.

Surgery and recovery. The surgery followed the last control test by one to three days. Assignment of the Ss to the two groups was based upon performance on the last three control tests. A two-week recovery period ensued during which all Ss initially received a bland diet of soft foods and later, the regular ration. The recovery of the amygdalecomized animals followed a typical pattern described by Weisskrantz (1956). One monkey ignored its food and had to be assisted in eating for the first two days postoperatively.

Postoperative control tests. A series of nine control tests were carried out under exactly the same conditions of deprivation and reward as existed preoperatively. Two Ss in each group received an additional month of tests in which deprivation conditions were varied, the amount of reward remaining constant. Their control levels of performance were reestablished for six sessions before proceeding with the shifts in reward. The other Ss began the reward tests immediately after the postoperative control series. This difference in experience did not seem to affect the test performance.

Shifts in amount of reward. Three numerically different shifts in the amount of reward were selected so as to include both increased and decreased amounts. The sequence was the same for all Ss. It was hoped that this procedure of shifting the rewards with the same set of Ss would enhance the differential effects that the amount had on performance (e.g., Schrier, 1958). The sequence began with an increase in reward from a $\frac{1}{2}$ -gm. pellet, used in the control tests, to three $\frac{1}{2}$ -gm. pellets. Each reinforcement consisted of this large reward for six consecutive sessions. Repeated observations were obtained in order to describe the time course of any lesion effect. The amount of reward was then reduced markedly to a small 45-mg. lab food tablet, measuring 0.40 cm. by 0.33 cm. (P. J. Noyes and Co.), which had substantially the same composition as the large pellet. The shift in conditions once again lasted for six consecutive sessions. Finally, the control conditions of $\frac{1}{2}$ -gm. pellets were reinstated for six sessions, representing a sizable increase in the level of reward.

Extinction. Upon completion of the last series of tests, data were obtained on the extinction of bar-pressing as part of a different sequence of conditions. The results from the initial 60-min. extinction session are relevant to the present findings. In this test the food reward was omitted but other events associated previously with its presentation continued with the same temporal contingency as before.

RESULTS

Performance on the postoperative control tests, using the preoperative levels as a base

line, was not affected by the amygdalectomy. The total number of bar-presses made by *S* on each postoperative control test was expressed as a percentage change of its mean total responses in the last three preoperative sessions. Preoperative values ranged from 292 to 782 responses per session, with no significant difference between groups. Bar-pressing decreased postoperatively by about 15% to 20%, but the change as evaluated by an analysis of variance was not related to the operative treatment. The analysis failed to indicate any over-all group difference or any group \times session interaction effect.

With the shifts in reward, a closer analysis was made of the performance within each of the test sessions by recording the number of bar-presses for successive 10-min. periods of each session. These results are given in Figure 2 for the large reward and antecedent control conditions. An over-all analysis of variance was performed on the percentage changes in performance for each of these periods, using the corresponding control data as a base line.

It can be seen in Figure 2 that amygdalectomy affected the response to the large reward as a function of the intrasession period. This interaction is significant at the .01 level ($F = 3.90$ for 5 and 30 *df*). In addition there are evident intrasession effects that obtained for both groups ($F = 3.17$ for 5 and 30 *df*, $p < .05$). This analysis did not indicate any over-all group differences averaging across intrasession periods or any change in the intrasession lesion effect with the repeated test sessions.

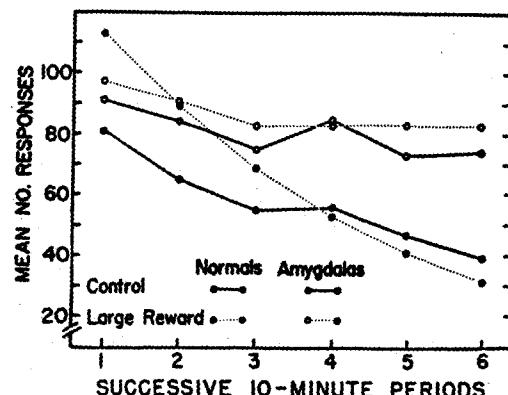


FIG. 2. Mean number of responses during successive 10-min. periods of the test sessions with the large reward and of the three preceding control sessions.

Examination of the changes in performance shows that amygdalectomy attenuated the increase in bar-pressing for the large reward during the first few 10-min. periods of these sessions. The normal animals averaged a 40% increase and the amygdalectomized animals 20%. Separate analysis of the response data for the large reward yields results which in terms of significant group \times intrasession interaction ($p < .001$) and intrasession effect ($p < .001$) support the analysis of the changes in performance.

The two groups also differed markedly in the reduction of bar-pressing during succeeding periods of the large-reward sessions. The brain-damaged animals exhibited minimal decrements in performance whereas the normal animals dropped sharply from the initially enhanced rates to below control levels. A percentage comparison of the number of responses made during the last half of these sessions relative to the first half yields a *p* of .028 by a two-tailed Mann-Whitney *U* test. Indeed, a similar, though less marked, phenomenon is apparent in the control conditions (*p* = .028). The amygdalectomized animals appear to exhibit more sustained behavior.

Amygdalectomy was also found to attenuate the decrease in responding following a sizable reduction in reward. Figure 3 plots the performance of the two groups within the first session with the small reward. Although the amygdalectomized animals were not insensitive to such a large change in reward, they made proportionately more responses in the last half of this session relative to the

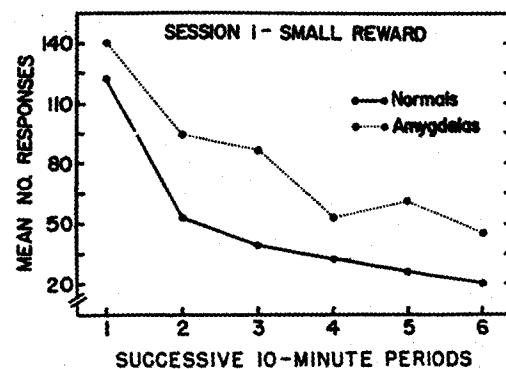


FIG. 3. Mean number of responses during successive 10-min. periods of the first test session with the small reward.

first half than did the normal animals. The difference in decline of bar-pressing is significant at the .028 level by the *U* test. Furthermore, as shown in Figure 4, this attenuating effect of the lesion tended to persist throughout the repeated tests. Although the output of both groups continued to decline after the first session, the asymptotic levels of performance still appeared to differ. The over-all difference between the groups in the percentage changes is significant at the .05 level by the *U* test. There was also a tendency for the brain-damaged animals to adjust somewhat more slowly to the altered conditions. The generally marked decline in bar-pressing produced by the reduction in reward is noteworthy.

A fixed-interval type of schedule also generates typically a temporally ordered pattern of bar-pressing between reinforcements (Skinner, 1938), which provides an additional measure of performance for the altered rewards. The response distributions are plotted in Figure 5 in such a way as to control for changes in output. The proportion of the total nonreinforced responses made during the last half of the 2-min. intervals was used as an index of the temporal patterning. It can be seen that the normal animals were much more reactive to the reduction in reward. In place of the progressively increasing rate of response with the passage of time between reinforcements, there occurred a less organized distribution approximating a linear function. The difference between the groups

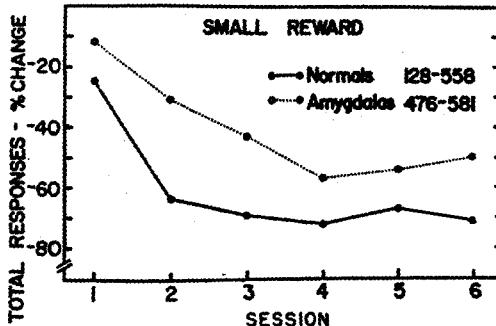


FIG. 4. Mean percentage changes in total responses in test sessions with the small reward. (The values in the legend refer to the range of total responses for the three preceding large-reward sessions on which the percentage changes are based; group differences are not significant statistically.)

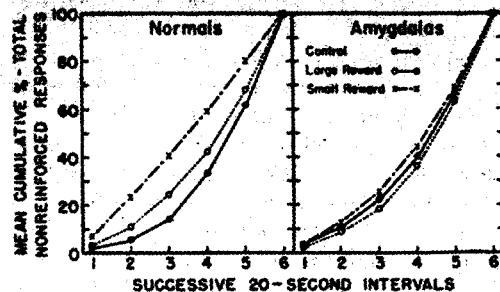


FIG. 5. Distribution of total nonreinforced responses within the 2-min. fixed intervals between reinforcements for each of the reward conditions. (Responses made during successive 20-sec. intervals were separately recorded and accumulated throughout each test session. These values were transformed to percentages, and are plotted as cumulative percentages. The results are based on all test sessions with a given condition. "Control" refers to the three sessions which preceded the large reward.)

in shift of response distributions is significant at the .05 level by *t* test. These results substantiate the findings with output of responses.

The amygdalectomized monkeys were also less responsive to the final increase in reward. They increased their output by 66% over that of the last three sessions with the small reward. The corresponding value for the normal group was 161%. These results are significant statistically ($p = .05$ by *U* test), but it should be noted that the base-line values also differed for the two groups.

By contrast, no effect of the lesion was discernible in the partial extinction of bar-pressing. Both groups reduced their output from about 80 responses during the first 10-min. period of the extinction session to about 20 responses during each of the last three 10-min. periods. These changes represented an over-all decrease of approximately 40% from the preceding level of performance.

DISCUSSION

The present findings demonstrate that, at least under certain conditions, ablation of the amygdaloid complex modifies rate of response for different amounts of food reward. Amygdalectomized monkeys were consistently less responsive, though by no means insensitive, to increased or decreased amounts, provided the rewards were not omitted entirely. These effects persisted over a considerable number of test sessions. The underreactivity to shifts

in reward was also reflected in a more stable temporal patterning of responses between reinforcements. Indeed, the patterning of behavior appeared to be an especially useful measure with decreased reinforcement. Under these conditions, a rate measure may to a greater degree confound emotionally induced responses and the like with responses controlled by the concurrent reinforcement.

These changes in performance after amygdalectomy are not secondary to an increase in hunger drive, as might be inferred from the hyperphagic effects of the lesion (Morgane & Kosman, 1957). Three sets of evidence oppose the interpretation: First, the postoperative control data, consistent with the findings of Weiskrantz (1956), and the extinction data gave no indication of such an increase. Weiskrantz, however, did report slower extinction of a food-reinforced response in amygdalectomized monkeys. Second, an increase in hunger would not account for the depressed response to the enlarged reward. Third, amygdalectomy attenuates the response to prolonged deprivation of food (Schwartzbaum, *in press*). An increase in hunger would imply exactly the opposite result. Nor do increases in hunger simply produce a more sustained pattern of bar-pressing as observed in the amygdalectomized monkeys (Schwartzbaum, *in press*).

The present findings suggest that the disturbances in dietary behavior are part of a more general change in the reinforcing properties of food stimuli, relating to both their qualitative and quantitative characteristics. This conceptualization may also apply to the less discriminating sexual behavior of amygdalectomized animals (Green et al., 1957; Schreiner & Kling, 1953). We cannot, however, state to what extent the effects obtained are specific to the amygdaloid complex without testing other ablation preparations.

The extension of the findings to a quantitative dimension of a normally accepted food reduces the likelihood that the dietary disturbances can be explained adequately in terms of gustatory or olfactory types of disorders. Previous studies (Fay, Miller, & Harlow, 1953; Hutt, 1954) have shown that the qualitative properties of a reward, as might be affected by such disorders, influence

performance within limits independently of its quantitative properties. Manipulation of the taste properties of a reward did not, e.g., affect the functional relation between performance and the amount of the reward (Hutt, 1954). Since the lesion effects are not selective in this respect, an impairment of taste or smell would not seem to be a sufficient cause.

Two mutually compatible interpretations of the results seem tenable in the light of other available data. With shifts in reward, the relationships that exist among the rewards may assume critical importance (e.g., Schrier, 1958). The effective reinforcing value of a given amount becomes contingent upon other rewards which are made available in the situation. Thus, a reward may have less reinforcing value if it is preceded by a larger amount than if it is not. The marked decline in performance for the small reward provides evidence of such "contrast effects." Monkeys tested with this size reward under nonshift conditions show much higher rates (unpublished data, K. H. Pribram).

It is therefore possible, on the one hand, that the changes in reinforcing values relate to a reduction in emotional responsiveness (King & Meyer, 1958) or, similarly, to an impairment in the conditioning of emotional responses (Brady, Schreiner, Geller, & Kling, 1954). The contrast effects obtained with shifts in reward have been assumed by some workers to represent essentially emotional phenomena (Crespi, 1944). If this is true, then a dampening of emotional responsiveness would give rise to less differentiated reward values. However, it still remains questionable whether an affective type of disorder is sufficient to account for the dietary changes (Weiskrantz, 1956). This reservation is strengthened by the fact that the lesion acts in a relatively selective fashion with respect to shifts in reinforcing conditions (Schwartzbaum, 1960) and food-preference patterns (unpublished data of the author and W. A. Wilson, Jr.).

On the other hand, the effects obtained may reflect a failure of the amygdalectomized animals to "interrelate" different reinforcing events, i.e., to respond to one set of events in terms of some other set. Insofar as the diversity

of reinforcing values is contingent upon such a process, as would appear to be the case, amygdaleotomy may act to constrict the range of these values. The difficulty would not seem to lie in the discriminability of the reinforcing stimuli as judged by performance on visual discriminations (Mishkin, 1954; Pribram & Bagshaw, 1953) and in paired-comparisons tests with different amounts of food (unpublished data of the author and W. A. Wilson, Jr.). The impairment may, instead, be related to processes which are necessary if the organism is to generalize appropriately among stimuli and utilize its experience when confronted with "new" events. For example, the shifts in "amount" of reward involved changes in size and number of pellets, concentration of taste stimuli, and in duration of consummatory response. In these terms, amygdalectomized animals can be said to *undergeneralize*.

The apparently "indiscriminate" feeding, as well as sexual, behavior of amygdalectomized animals might seem inconsistent with this notion. But the reasoning is basically the same. It assumes that the reinforcing values that are normally attached to such stimuli are based in part upon the relationships of the stimuli to existing habits of reinforcement. A failure to make these associations would tend to produce less differentiated reinforcing values, and thus overgeneralized behavior, if the stimuli were at least potentially reinforcing in terms of taste, smell, texture, ingestibility, etc. This notion is not inconsistent with the view that amygdalectomized animals are unable to "recognize" or to "identify" reinforcing stimuli (Weiskrantz, 1956). But the present hypothesis would relate these effects to experimental conditions which maximize the importance of generalization processes. If this hypothesis is valid, then the pre- and postoperative conditions which determine the novelty of a stimulus or its surrounds would be of special importance.

SUMMARY

The experiment was intended to determine whether the changes in dietary behavior after removal of the amygdaloid complex are symptomatic of a more general disturbance in reinforcing properties of stimuli. Eight

monkeys received either bilateral lesions of the amygdaloid complex or an equivalent sham operation. They were tested in a bar-pressing situation, using a fixed-interval schedule of reinforcement, with different amounts of a normally accepted food reward. Each of the amounts was presented for a number of sessions to all animals. The shifts included both increases and decreases in reward.

It was found that amygdaleotomy depressed responsiveness to shifts in amount of reward. The amygdalectomized monkeys showed less of an increase in bar-pressing with enlarged rewards, and somewhat less of a decrease with a substantially reduced amount. These effects were also reflected in the temporal patterning of responses between reinforcements. The lesion also attenuated satiation-like decrements in performance within test sessions, especially with the large reward. Minimal effects were obtained with uniform conditions of reinforcement and with extinction. Two possible interpretations of the results were considered.

REFERENCES

- BRADY, J. V., SCHREINER, L., GELLER, I., & KLING, A. Subcortical mechanisms in emotional behavior: The effect of rhinencephalic injury upon the acquisition and retention of a conditioned avoidance response in cats. *J. comp. physiol. Psychol.*, 1954, **47**, 179-186.
- CRESPI, L. P. Amount of reinforcement and level of performance. *Psychol. Rev.*, 1944, **51**, 341-357.
- FAV, J. C., MILLER, J. D., & HARLOW, H. F. Incentive size, food deprivation, and food preference. *J. comp. physiol. Psychol.*, 1953, **46**, 13-15.
- GREEN, J. D., CLEMENTE, C. D., & DE GROOT, J. Rhinencephalic lesions and behavior in cats. *J. comp. Neurol.*, 1957, **108**, 505-545.
- HUTCHINS, P. J. Rate of bar-pressing as a function of quality and quantity of food reward. *J. comp. physiol. Psychol.*, 1954, **47**, 235-239.
- KING, F. A., & MEYER, P. M. Effects of amygdaloid lesions upon septal hyperemotionality in the rat. *Science*, 1958, **128**, 655-656.
- KLÜVER, H., & BUCY, P. C. Preliminary analysis of functions of the temporal lobes in monkeys. *Arch. Neurol. Psychiat.*, 1939, **42**, 979-1000.
- MISHKIN, M. Visual discrimination performance following partial ablations of the temporal lobe: II. Ventral surface vs. hippocampus. *J. comp. physiol. Psychol.*, 1954, **47**, 187-193.
- MORGANE, P. J., & KOSMAN, A. J. Alterations in feline behavior following bilateral amygdaleotomy. *Nature*, 1957, **180**, 598-600.
- PRIBRAM, K. H., & BAGSHAW, M. Further analysis of the temporal lobe syndrome utilizing fronto-

- temporal ablations. *J. comp. Neurol.*, 1953, **99**, 347-375.
- ROSVOLD, H. E. Physiological psychology. *Ann. Rev. Psychol.*, 1959, **10**, 415-454.
- SCHREINER, L., & KLING, A. Behavioral changes following rhinencephalic injury in the cat. *J. Neurophysiol.*, 1953, **6**, 643-659.
- SCHRIER, A. M. Comparison of two methods of investigating the effect of amount of reward on performance. *J. comp. physiol. Psychol.*, 1958, **51**, 725-731.
- SCHWARTZBAUM, J. S. Response to changes in reinforcing conditions of bar-pressing after ablation of the amygdaloid complex in monkeys. *Psychol. Rep.*, 1960, **6**, 215-221.
- SCHWARTZBAUM, J. S. Some characteristics of "amygdaloid hyperphagia" in monkeys. *Amer. J. Psychol.*, in press.
- SKINNER, B. F. *The behavior of organisms*. New York: Appleton-Century, 1938.
- WEISKRANTZ, L. Behavioral changes associated with ablation of the amygdaloid complex in monkeys. *J. comp. physiol. Psychol.*, 1956, **49**, 381-391.

(Received June 28, 1959)