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SOME CHARACTERISTICS OF 'AMYGDALOID HYPERPHAGIA' IN MONKEYS

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Bilateral resections in the vicinity of the amygdaloid complex have been found to affect dietary activities. In both primates and carnivores, the amygdalectomized animals typically develop hyperphagia and, as a consequence, become obese.[1] The higher rate of weight-gain may persist for a considerable period of time after the operation.[2] The animals are also less selective about what they will eat, consuming foods and other objects that are normally rejected.[3] Both sets of effects are expressions of a more complex disturbance, as described by Klüver and Bucy, in temporal lobectomized monkeys.[4]

It is of some interest to determine whether the changes in dietary behavior reflect a more general increase in hunger or drive for food. Such an effect would correspond in its consequences to an increase in deprivation of food. It would be reflected in a disposition not only to consume more food than would normal animals, but also to respond more vigorously to food. In the present experiment, the rate of performance of a response periodically reinforced with food was studied under different conditions of deprivation and satiation.

EXPERIMENT I

Subjects. The *Ss* were eight preadolescent rhesus monkeys. As detailed elsewhere, four of the *Ss* (*AM* 397, 405, 438, 442) had received bilateral resections of the amygdaloid complex and adjacent anteromedial temporal cortex, while the others

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[1] K. H. Pribram and Muriel Bagshaw, Further analysis of the temporal lobe syndrome utilizing frontotemporal ablations, *J. comp. Neurol.*, 99, 1953, 347-375; J. L. Fuller, H. E. Rosvold, and K. H. Pribram, The effect on affective and cognitive behavior in the dog of lesions of the pyriform-amygdala-hippocampal complex, *J. comp. physiol. Psychol.*, 50, 1957, 89-96; J. D. Green, C. D. Clemente, and J. de Groot, Rhinencephalic lesions and behavior in cats, *J. comp. Neurol.*, 108, 1957, 505-545; P. J. Morgane and A. J. Kosman, Alterations in feline behaviour following bilateral amygdalectomy, *Nature*, 180, 1957, 598-600.

[2] Morgane and Kosman, *op. cit.*, 599.

[3] Pribram and Bagshaw, *op. cit.*, 355-359; Leon Schreiner and A. Kling, Behavioral changes following rhinencephalic injury in the cat, *J. Neurophysiol.*, 6, 1953, 643-659.

[4] Heinrich Klüver and P. C. Bucy, Preliminary analysis of functions of the temporal lobes in monkeys, *Arch. neurol. Psychiat.*, 42, 1939, 979-1000.

(439, 441, 443, 447) had received an equivalent sham operation.[5] The body-weights ranged from 4.3 to 6.6 lb., with no significant difference between groups.

Procedure. All tests were carried out in a sound-insulated Skinner box which is described in the report previously cited. *S* had access in the test-chamber to a lever and a food cup. Reinforcements for bar-pressing were made available in accordance with a 2-min. 'fixed-interval' schedule. Each reinforcement consisted of a ½ gm. lab food pellet (P. J. Noyes and Co.). The test-sessions were of 60-min. duration, spaced on alternate days so as to occur three times weekly; a two-day interval between sessions overlapped the weekends. The *S*s were maintained on a daily diet of 8 to 10 Purina Lab Chow pellets and one quarter of an orange, supplemented by four unshelled peanuts on non-test days. They were fed from 2 to 3 hr. after each test and 24 hr. before the succeeding test. As a check on the maintenance-regimen, body-weights were measured before each session.

The test-conditions described above furnished a behavioral base line for the variables under study. Following the preliminary training, each *S* received a total of nine such control-sessions preoperatively and, after a two-week recovery period, the same number of sessions postoperatively. Two *S*s in each group received an additional month of tests with different amounts of reinforcement.[6] Their control-levels were reëstablished before proceeding with the present experiment. This difference in experience, which was equivalent for the two groups, did not seem to affect the results.

In the experiment proper, the *S*s were switched from a 24-hr. cycle of deprivation to one which alternated *ad libitum* feeding with prolonged deprivation of food. The *S*s were tested after 70 hr. of *ad libitum* feeding, defining 'satiation'-conditions, and after a corresponding period of food-deprivation. This meant that test-sessions were now spaced every third day. The satiation- and deprivation-conditions were alternated until five tests had been carried out under each of them, the sequence always beginning with the satiation.

During the satiation-periods, which began one hour after the preceding test, the food pans in the home cages were filled with fresh Purina Chow pellets and were replenished three times daily. The amount of food made available far exceeded the animals' eating capacity. In addition, a point was made of presenting fresh chow at least one-half hour before each satiation-test. One hour after the satiation-tests, the pans and disposal trays were cleared of all food, and the *S*s then deprived for 70 hr. until the next test. It was not, however, possible to control for copraphagia, evident in two of the amygdalectomized monkeys.

Results. Fig. 1 plots the changes in performance following prolonged deprivation of food, using the three preceding control-sessions as a base line; the values in the legend refer to the range of responses in the control-sessions. It is quite clear from these data that the amygdalectomized monkeys were far less responsive than the normal monkeys to the deprivation, although they were by no means insensitive to it. The over-all group difference, averaging across sessions, is significant by analysis of variance at well beyond the 5% level ($F = 7.25$ for 1 and 6 *df.*). The apparent interaction

[5] J. S. Schwartzbaum, Changes in reinforcing properties of stimuli following ablation of the amygdaloid complex in monkeys, *J. comp. physiol. Psychol.*, 53, 1960, 388-395.

[6] Schwartzbaum, *ibid.*, 390.

between groups and repeated test-sessions could not be verified statistically ($F < 1$ for 4 and 24 *df.*). All of the amygdalectomized monkeys showed an increase in bar-pressing, but it was not systematically related to the repeated tests.

These effects of the lesion were associated with differences in predeprivation body-weight as measured at the end of each *ad libitum* feeding. The amygdalectomized monkeys exceeded the normals in weight-gain during the initial *ad libitum* feeding period and, thereafter, maintained a higher

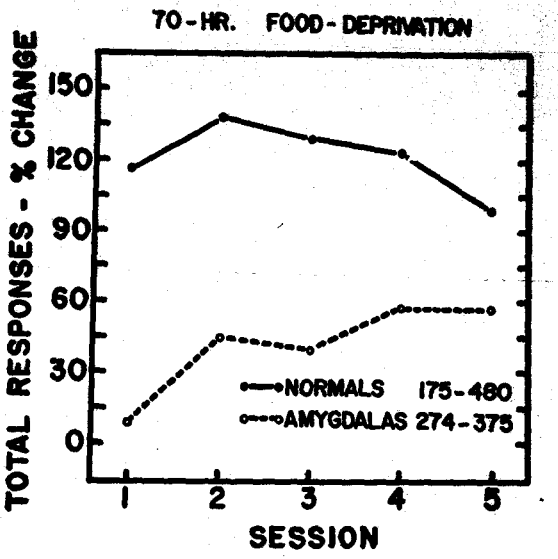

FIG. 1. MEAN PERCENTAGE-CHANGES FOLLOWING PROLONGED DEPRIVATION OF FOOD

level of body-weight both before and after deprivation. Average increases of 12% and 5% over control-levels were obtained respectively for the two groups after the *ad libitum* feedings ($p < 0.05$ by a two-tailed Mann-Whitney U test). This difference in weight-gain reflects the amount of food consumed and not the energy expended. There were no gross differences between the groups in cage activity that could account for such findings. The results, in effect, provide evidence of hyperphagic tendencies in the amygdalectomized animals.

Bar-pressing under satiation-conditions was not affected by the lesion. Both groups reduced their output by about 50% of control-levels, but variability among individual *Ss* was high.

EXPERIMENT II

In this experiment, the deprivation- and satiation-tests were separated from one another to control body-weights during predeprivation and to minimize possible interactions between the two conditions.

Subjects. A new group of five amygdalectomized monkeys (*AM* 351, 352, 395, 400, 541) and four normal monkeys (344, 390, 509, 502) were used. Each group included one cynomolgus monkey, together with rhesus monkeys. The performance of the two species was quite similar. The lesions, as reconstructed anatomically, were equivalent to those in the first experiment. The rhesus monkeys had extensive experience with discriminative problems and with bar-pressing. Body-weights of the two groups, which were not distinguishable, ranged from 5.2 to 10.0 lb.

Procedure. Test-conditions were maintained as before, except that the sessions were run daily, 6 days a week. Control-levels of performance were established under approximately 21 hr. of food-deprivation. After 10 to 12 control-sessions, the *Ss* received two consecutive 70-hr. deprivation-tests with two-day intervals in the testing. Each deprivation-test was followed by a triple ration of food so as to maintain body-weights at control-levels.

A separate series of four satiation-tests was carried out after performance had been restabilized for 9 to 12 control-sessions. The *Ss* were allowed to eat *ad libitum* for four consecutive days, and were tested once each day. The initial test came 22 hr. after the start of feeding. The food pans were replenished with fresh chow pellets several times a day, including once before each session. An attempt was made to obtain additional information on responsiveness to prolonged deprivation immediately after the *ad libitum* feedings. Both groups, however, showed little change in bar-pressing with respect to presatiational control-levels. Presumably, the repeated satiation-tests were responsible for this suppression, since single satiation-tests, as shown in Experiment I, did not have such a marked effect.

Results. Fig. 2 shows the intra-session patterns of performance of the two groups under control- and deprivation-conditions. It is clear from the changes in bar-pressing that amygdalectomized monkeys were again not as responsive as the normals to the increase in deprivation. An analysis was performed on the percentage-changes in total output. On the initial deprivation-test, the normal group increased its total output by 95%, in contrast to a 20% increase for the lesion group ($t = 2.48$ for 7 $df.$; $p < 0.05$). The same trend was evident on the second deprivation-test with increases of 68 and 20%, respectively, but variability among *Ss* was much greater and t was not significant.

The results depicted in Fig. 2 distinguish between the effects of the lesion and increased hunger as produced by extension of the deprivation-period. Examination of the control-data shows that the lesion acted primarily to stabilize operant activity, reducing the slope of the decrement in performance. Thus, in agreement with previous findings,[7] four out of

[7] Schwartzbaum, *ibid.*, 391.

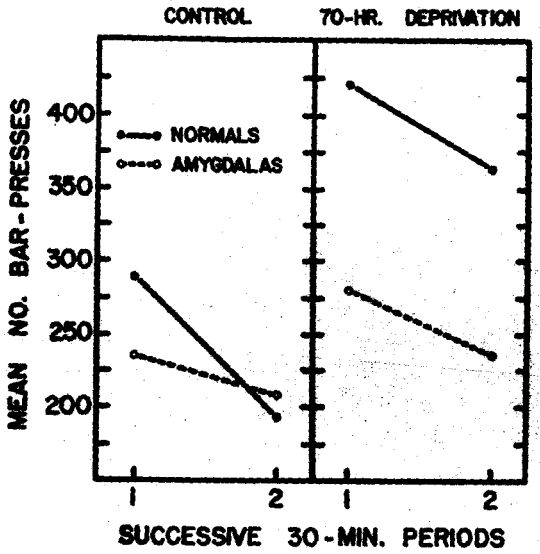

FIG. 2. MEAN NUMBER OF RESPONSES DURING SUCCESSIVE 30-MIN. PERIODS FOLLOWING PROLONGED DEPRIVATION OF FOOD

the five amygdalectomized *Ss* continued to make more responses percentage-wise in the last half of the control-tests with respect to the first half than did the normal *Ss*. Mean values of 93.8 and 67.8% were obtained respectively for the two groups. The group-differences in bar-presses in the first

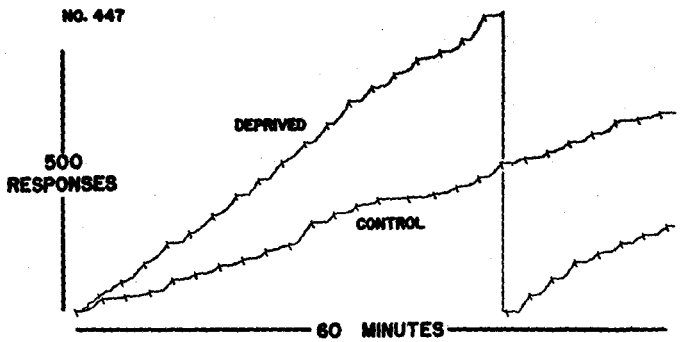

FIG. 3. CUMULATIVE RECORDS UNDER CONTROL CONDITIONS AND AFTER PROLONGED DEPRIVATION OF FOOD

half of the sessions were not significant statistically. Prolonged deprivation, on the other hand, increased sharply the initial or peak-rate of responding by each of the normal *Ss*. It was quite clear from the individual cumulative records of performance, as illustrated in Fig. 3, that this increase occurred within the 2-min. intervals between reinforcements. Hence, while

the lesion affected the persistence of responding, the increased hunger affected the peak-rate of responding.

Bar-pressing activity after satiation, in agreement with the previous findings, was not altered by the lesion. Both groups reduced their output within the first session by approximately 68%. These results are more conclusive in the sense that the variability in performance among *Ss* was relatively low. Nevertheless, much of the bar-pressing did not seem to be motivated by the food as a 'primary reward.' About 60% of the pellets delivered were recovered after the test-sessions, with somewhat greater variability among the animals with lesions. The satiation-tests were the only ones in which this occurred. The groups again differed in weight-gain, an average increase of 15% being recorded for the amygdalectomized animals and 10% for the normal animals ($p < 0.05$ by the *t*-test). Virtually all of this increase appeared within the first 24 hr. of *ad libitum* feeding, and, again, the difference may be taken as evidence of hyperphagia.

Discussion

The results indicate that amygalectomy does not increase hunger or the drive for food. First, an increase in hunger produced by deprivation influenced performance in a manner that differed from that of the lesion. Secondly, the brain-damaged animals were far less, instead of more, responsive than normal animals to prolonged deprivation of food.

A substantial increase in hunger, incident to a change in deprivation, affected the peak-rate of response within the 2-min. intervals between reinforcements. It had no consistent effect upon the persistence of responding within sessions in terms of the percentage-decrement in performance. Such changes are not specific to the conditions imposed. They vary systematically over a wide range with the degree of deprivation.[8] Thus, it is fair to assume that an effect of the lesion upon hunger, commensurate to its hyperphagic consequences, would be expressed by an increase in peak-rate of response.

Amygdalectomy did not have this effect. Although the lesion attenuated the decrement in performance within sessions, it did not alter the peak-rate or, for that matter, the total output; however, the latter might be a function of the limited duration of test-session.[9] These negative findings

[8] B. F. Skinner, *The Behavior of Organisms*, 1938, 341-405; H. F. Harlow, Primate learning, in C. P. Stone (ed.), *Comparative Psychology*, 3rd ed., 1951, 211.

[9] It should be apparent from these findings that the processes which control the peak-rate of response within the intervals between reinforcements can be isolated to a certain extent from the processes which maintain these rates across intervals. This differentiation provided by a fixed-interval schedule contrasts with that of a fixed-ratio schedule (*cf.*, Murray Sidman and W. C. Stebbins, Satiation effects under

agree with pre- and postoperative comparisons of rate of performance for food, and with other operant data obtained several months after the surgery[10] More persuasive, however, is the decreased responsiveness of amygdalectomized monkeys to prolonged deprivation, since an increase in hunger would imply exactly the opposite result. Indeed, the consistently lower level of performance with prolonged deprivation suggests that under some conditions amygdalectomized monkeys may have a *lower* than normal drive for food. This effect was not likely due to some initially higher level of drive, since it occured after *ad libitum* feeding, as well as after restricted amounts of feeding. Nor can the effect be attributed to some form of debilitation or reduced level of activity. The control-data argue against this possibility, as do also the results of tests made on locomotor activity in amygdalectomized monkeys.[11]

There are two ways of interpreting the more persistent pattern of responding by the amygdalectomized monkeys. First, it can be attributed to an impairment in satiety mechanisms specific to hunger. This assumes that the normal decrement in performance related to the reinforcing events in the situation, rather than to other consequences of bar-pressing. Consistent with this reasoning, increased amounts of reward, which enhance the decrement, have been found to accentuate the stabilizing effects of the lesion.[12] The amygdalectomized monkeys showed little change in performance. Similarly, controlled amounts of prefeeding have less of a depressing effect upon the operant activity of amygdalectomized monkeys than of control monkeys.[13] The animals with lesions are not, however, insensitive to deprivation-conditions. When allowed to approach a point of satiation, as after the *ad libitum* feedings, their output fell within normal bounds. They may simply require an excess amount of food to attain this state.

Secondly, the more sustained response may be symptomatic of a general disturbance in habituation-processes, extending to other classes of stimuli beside food. Thus, amygdalectomized animals are reported to be generally more responsive, as well as persistent in responding, to objects in their

fixed-ratio schedules of reinforcement, *J. comp. physiol. Psychol.*, 47, 1954, 114-116). Still other processes are responsible for the temporal patterning of responses within the fixed intervals. Neither the enhanced deprivation nor the lesion altered the temporal distributions. Total output as an indicator of performance obviously confounds all of these measures.

[10] Lawrence Weiskrantz, Behavioral changes associated with ablation of the amygdaloid complex in monkeys, *J. comp. physiol. Psychol.*, 49, 1956, 386; Schwartzbaum, Response to changes in reinforcing conditions of bar-pressing after ablation of the amygdaloid complex in monkeys, *Psychol. Rep.*, 6, 1960, 215-221.

[11] J. S. Schwartzbaum, W. A. Wilson, Jr., and Rolande Morrissette, Effects of amygdalectomy on locomotor activity in monkeys, *J. comp. physiol. Psychol.*, in press.

[12] Schwartzbaum, *op. cit.*, *J. comp. physiol. Psychol.*, 391.

[13] Weiskrantz, Behavioral changes associated with ablation of the amygdaloid complex in monkeys, Unpublished Doctoral dissertation, Harvard University, 1953.

environment, the so-called "hypermetamorphotic" effect described by Klüver and Bucy.[14] They also show much less of a locomotor reaction-decrement with repeated tests in a novel situation.[15] These findings suggest a broader context for the present results. General factors of habituation are presumably involved in satiation, insofar as the satiation arises from repeated exteroceptive sensory consequences of the food aside from post-ingestional consequences.

Amygdaloid hyperphagia would, therefore, not appear to be associated with an increase in hunger-drive, but rather with some form of defect in satiation or habituation specific or not specific to food-consumption. Whether or not such a defect is a sufficient condition of the hyperphagia cannot be stated. The evidence of hyperphagic tendencies in the amygdalectomized monkeys indicates that both sets of effects were at least present concurrently.[16]

SUMMARY

Two experiments were performed in an effort to determine whether the hyperphagic effects of amygdalectomy reflect an increase in drive for food. Groups of monkeys that had received either bilateral ablation of the amygdaloid complex or an equivalent sham operation were tested in a bar-pressing situation under different conditions of deprivation and satiation. The bar-presses were reinforced with food in accordance with a fixed-interval schedule.

The two major findings were, first, that amygdalectomy decreased responsiveness to prolonged deprivation of food and, second, that it attenuated satiation-like decrements in performance within test-sessions. With prolonged *ad libitum* feeding, the amygdalectomized animals gave evidence of hyperphagia, but then performed normally for food.

It would appear that amygdaloid hyperphagia is not associated with an increase in drive for food, but rather with a defect in satiation or habituation that is either specific or not specific to food stimuli.

[14] Pribram and Bagshaw, *op. cit.*, 356; Klüver, and Bucy, *op. cit.*, 987.

[15] Schwartzbaum, Wilson, and Morrisette, *op. cit.*, in press.

[16] It may be noted that amygdaloid hyperphagia resembles in certain respects hypothalamic hyperphagia. Both appear to involve disturbances in satiation in the absence of any concomitant increase in drive for food, and, indeed, may be associated with a reduced level of drive (Philip Teitelbaum, Random and food-directed activity in hyperphagic and normal rats, *J. comp. physiol. Psychol.*, 50, 1957, 486-490; Philip Teitelbaum and B. A. Campbell, Ingestion patterns in hyperphagic and normal rats, *J. comp. physiol. Psychol.*, 51, 1958, 135-141). The large anatomical projection from the amygdala to the ventromedial region of the hypothalamus (W. R. Adey and M. Meyer, Hippocampal and hypothalamic connections of the temporal lobe in the monkey, *Brain*, 75, 1952, 358-384) further suggests a close functional relationship. But in view of the complexities of satiation, the apparent quantitative differences in the hyperphagia (Morgane and Kosman, *op. cit.*, 599), and the lack of any direct comparisons between the lesion-effects, any specific conclusions would be premature.