

Caudate Nucleus Lesions: Behavioral Effects in the Rat

Abstract. *Bilateral lesions within the caudate nucleus led to a marked impairment on acquisition and retention of a spatial alternation habit but produced no deficit in Hebb-Williams maze performance. Conversely, subtotal bilateral lesions of posterior cortex produced no significant deficit on the alternation habit but markedly impaired maze performance.*

In the monkey, lesions involving either frontal cortex or caudate nucleus lead to specific and lasting impairment on the classical forms of delayed response and delayed alternation tasks (1). Similar impairments of delayed alternation performance appear after frontal lesions in the dog and cat (2). In the chimpanzee, however, frontal cortical lesions produce smaller deficits on these tasks (3), and corresponding effects of frontal lesions in man may be even less specific and less enduring (4). Thus there appear to be phylogenetic differences in the extent to which anterior forebrain lesions affect performance of these tasks.

We developed a form of alternation task for use with the rat, in order to study the effects of caudate nucleus lesions in this mammal. To control for specificity of effects of the lesion, we also examined the effect of posterior cortical lesions on performance of this task, and tested the effects of both lesions on performance in a maze.

Our alternation task utilized a box with two levers (5). Each lever press that followed a response to the opposite lever resulted in delivery of a 45-mg food pellet to a food well located between the levers. Whenever an error

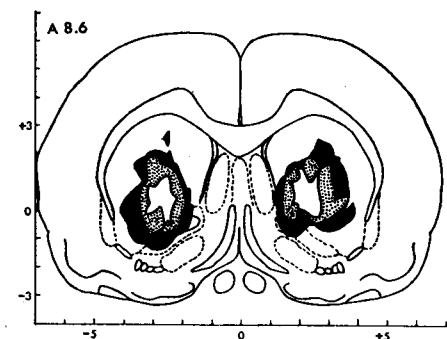


Fig. 1. Composite of lesions of caudate group ($N = 17$) superimposed on coronal section at level A 8.6, from De Groot (see 6). All or part of the central white area was damaged in 11 or more rats, the stippled area in 6 to 10 rats, and the black area in 3 to 5 rats. One scale division = 1mm.

was made (that is, whenever the same lever was depressed twice in succession), a 5-second time-out was introduced. During this interval the lights within the box were extinguished and all responses were without effect. At the end of the time-out period (as at the start of each daily test session), the first response was neither rewarded nor punished; it merely served to program the succeeding alternation sequence. Each daily session lasted until 100 responses (excluding responses during time-out) had been made. Accuracy of performance was measured by dividing the number of rewards by the number of responses (excluding the first response of the session, all responses during time-out, and the first response after each time-out period). Training was continued until a criterion of 80 percent correct responses was achieved on four out of five consecutive days, or until 13,000 trials had been run, whichever was less.

Forty-seven male hooded rats of the Long-Evans strain were used. They were approximately 120 days old at the beginning of the experiment. During training periods, the animals were maintained at 80 percent of the body weights established when they were allowed free access to food. There were two main groups. The first, or "retention," group learned the alternation task prior to operation and was retested postoperatively to determine the effect of the lesions upon retention of the habit. The second, or "acquisition," group was operated upon after learning to press the bars to obtain food reward but before being trained on the alternation task. In the retention group, ten animals received partial bilateral lesions of the caudate nucleus (Fig. 1), six received partial bilateral lesions of posterior cortex (Fig. 2), and four served as unoperated controls. In the acquisition group, seven animals received partial caudate nucleus lesions, five received partial posterior cortical lesions, and 35 served as unoperated controls. The cortical lesions were made by suction, the caudate ones electrolytically (6).

In the retention experiment (Fig. 3, top) the caudate operates required many more trials to reach criterion than either the cortical operates or the unoperated controls ($p < .002$) and showed much poorer savings than either group ($p < .002$) (7). Furthermore, three of the caudate operates (Nos. 31, 61, and 63) did not attain the criterion within 13,000 postoperative trials, while one other animal (No.

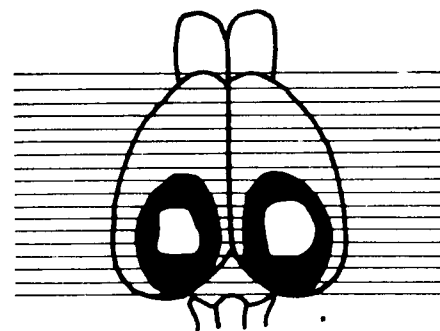


Fig. 2. Maximum (black) and minimum (central white) extents of cortical lesions in posterior cortex group. One scale division = 1 mm.

23) only pressed the lever a few times during many weeks of postoperative testing. The performance of the cortex lesion group and the unoperated group did not differ.

In the acquisition experiment (Fig. 3, bottom) the animals with caudate lesions were markedly inferior to unoperated controls in learning the alternation task ($p < .001$). The group with cortical lesions acquired the task somewhat more slowly than did the unoperated animals, but neither the difference between the normal ani-

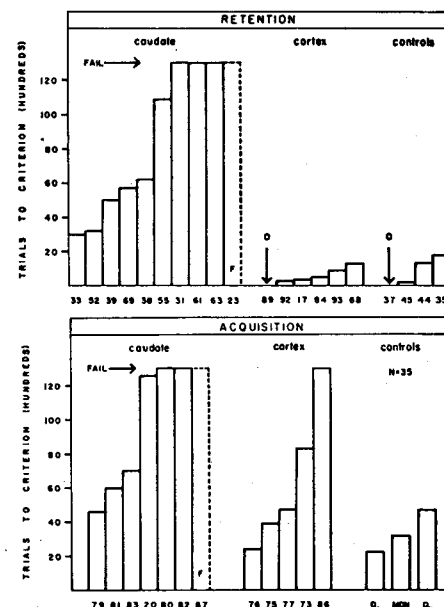


Fig. 3. Errors made by individual animals during (top) retention and (bottom) acquisition tests on the alternation task. (The right-hand portion of the bottom, however, shows quartile and median performance scores of 35 normal control animals.) 0 = perfect retention without additional retraining. F = failure to attain criterion in 13,000 trials. The dotted bars for animals with scores of F indicate that they had extensive opportunity to press the levers, although they did not do so.

mals and those with cortical lesions nor the difference between the caudate operates and cortex operates reached significance ($p = .13$ and $.27$, respectively).

Thus both acquisition and retention of the alternation habit were severely impaired by lesions of the caudate nucleus but not by lesions of posterior cortex. In order to exclude the possibility that the alternation deficit reflected either a general learning impairment or a motivational change, the present animals and others with similar lesions and experience were tested in a Hebb-Williams maze according to the procedure outlined by Rabinovich and Rosvold (8). On the 12 test problems the caudate group ($N = 13$) made a mean of 190.5 errors, the unoperated group ($N = 9$) made a mean of 152.7 errors, and the cortex group ($N = 9$) made a mean of 320.9 errors. The performance of the normal and caudate groups did not differ significantly, whereas both made significantly fewer errors than the cortex group ($p < .002$ in both cases). Since animals with caudate lesions were unimpaired on the maze task but failed the alternation test, it seems reasonable to assert that they showed neither a generalized learning deficit nor a marked disruption of motivation to work for food.

In conclusion, the caudate nucleus in the rat, as in the monkey, is necessary for adequate alternation performance. Further experiments are required to determine whether caudate lesions in rat and monkey impair alternation performance by equivalent mechanisms (9; 10).

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References and Notes

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4. S. L. Chorover, M. Cole, G. Ettlinger, unpublished data.
5. Grason-Stadler Co., Inc., model E3125B, with the addition of a light within the food well which operated for one-half second when food was presented.
6. A Grass model LM-2 lesion maker was used to produce the caudate lesions through a formvar-coated 10-mil wire with 1.0 mm of the insulation removed at the tip. The unit scale was set at 85 and current was passed for 30 seconds between the wire and a large electrode plug inserted in the anus. Location of the electrode in the brain was determined in accordance with stereotaxic coordinates from J. De Groot, *The Rat Forebrain in Stereotaxic Coordinates* (Noord-Hollandsche Uitgevers Maatschappij, Amsterdam, 1959) (Verhandelingen der koninklijke nederlandse akademie van wetenschappen, afd. natuurkunde, Tweede Reeks, Deel LII, No. 4), pp. 1-40. The caudate placement was aimed at L 2.5; A 8.6; V + 1.5.
7. The statistical values presented in this report are based upon comparisons made by means of two-tailed Mann-Whitney U tests [S. Seigel, *Non-Parametric Statistics for the Behavioral Sciences* (McGraw-Hill, New York, 1956)].
8. M. S. Rabinovich and H. E. Rosvold, *Can. J. Psychol.* 5, 122 (1951).
9. It should be noted that our apparatus did not include a screen interposed between the animal and the levers. When monkeys with anterior forebrain lesions are tested in lever-pressing apparatus without such screens, their deficiency on delayed alternation may be reduced (J. Stamm, in K. Akert and J. M. Warren, *Frontal Granular Cortex and Behavior*, in press) or even absent (K. Battig, H. E. Rosvold, M. Mishkin, *J. Comp. Physiol. Psychol.* 53, 400 (1960)).
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