Journal of Comparative and Physiological Psychology
1970, Vol. 72, No. 2, 296-305

VISUAL-LEARNING DEFICITS FOLLOWING CEREBELLAR DAMAGE IN RATS[1]

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Experiments with rats suggest that: (*a*) cerebellar damage hinders learning but not performance in some visual-learning tasks under conditions of high motivation; and (*b*) the location and size of the cerebellar lesions are important variables for showing this effect. It is speculated that the intact cerebellum may act to modulate "motor arousal" and prevent overfacilitation of inappropriate responses during learning.

Visual and auditory projections to the cerebellum were first studied by Snider and Stowell in the cat (1942a, 1942b; Stowell & Snider, 1942) and have been subsequently found in fish and reptiles (Gusel'nikov & Ivanov, 1958), birds (Whitlock, 1952), rats,[3] dogs (Gastaut, Naquet, Badier, & Roger, 1951) and monkeys (Snider & Eldred, reported by Munson, 1965).

Visually evoked impulses come to the cerebellum by two routes, the first from the optic nerve via tectal and pontine relays, and the second from the primary visual cortex of the cerebrum (see Fadiga & Pupilli, 1964). The cerebellar projection area for visual stimuli is most evident in the medial portions of several lobules posterior to fissura prima: Larsell's lobules VI, VII, and VIII. With intense stimuli, lateral portions of the cerebellar hemispheres may be activated as well (Fadiga, Pupilli, & von Berger, 1956). Auditory stimuli may have access to a wider area (Bonnet & Bremer, 1951; Marsh & Warden, 1964).

No clear topographical relationship has yet been found between the retinal fields and the cerebellar projection area, nor has the functional significance of this area been discovered. Infrequent instances of visual disturbances following cerebellar damage have provided only equivocal evidence. In some cases, an apparent modification of sensation may be explained by the loss of motor expression (Chambers & Sprague, 1955a, 1955b; Sprague & Chambers, 1959); in others, damage to additional brain stem structures and possible involvement of the colliculi necessitate cautious interpretation (Goldstein, 1927; Mills, Frazier, de Schweinitz, Weisenburg, & Lodholz, 1905). The latter studies, observations in man, have not been replicated by modern clinicians and their validity has been questioned (Holmes, 1939). One study with dogs (Fanarjyan, 1961) suggests that cerebellar damage hinders the extinction of a well-learned response to visual cues—his experiment will be described in more detail with the discussion of Experiments 2 and 3.

On the basis of electrophysiological findings, Snider (1950) proposed that the cerebellum may modulate cerebral excitability and thus influence sensory (or other) thresholds. However, attempts to find modifications of auditory (Munson & Monjan, 1967) or visual[4] thresholds after cerebellar damage have been unsuccessful. Nor were

[1] This report is based on a dissertation submitted to McGill University in partial fulfillment of the requirements for the PhD degree. The author wishes to thank R. B. Malmo and Elise Buchtel for their helpful comments and critical reading of the manuscript. The work was supported in part by a research fellowship to the author from the National Institute of Mental Health, and in part by grants to R. B. Malmo from the Defence Research Board, National Research Council of Canada, and from the National Institute of Mental Health.

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[3] Unpublished observations by the author; C. Bourassa, personal communication, November 1966.

[4] C. Bourassa, personal communication, November 1965.

auditory frequency discriminations affected by cerebellar lesions (Meyer & Woolsey, 1952).

In general, neither positive findings of sensory losses following cerebellar damage nor the numerous negative instances have helped in assessing the function of visual projections to the cerebellum: The behavioral techniques, experimental subjects, and lesions employed in these different experiments have been too varied to allow meaningful generalizations. The present series of experiments was designed to overcome some of the deficiencies of earlier studies by examining the effects of a single discrete cerebellar lesion (in most cases) using two levels of aversive stimulation (shock) and two levels of appetitive motivation (food).

Experiment 1

Method

Subjects. Forty-six male hooded rats of the Royal Victoria strain were divided into two groups as follows: 21 rats with discrete cerebellar lesions; 25 rats with sham operations. All animals were housed individually with food and water available ad lib.

Apparatus. A modification of the two-choice discrimination box described by Thompson and Bryant (1955) was used (Figure 1). It was constructed of varnished plywood except for the floor of the goal area (Masonite board) and the grid area (⅛-in. stainless-steel rods spaced 1.5 cm. apart). The entire apparatus was covered with a hinged ¼-in.-thick plate of Plexiglas. The discrimination stimuli consisted of: (*a*) vertical vs. horizontal white stripes on black background; and (*b*) an upright vs. inverted white triangle on black background. The stimulus cards were centered on 10 × 11 cm. swinging doors with their bottom edges 1 cm. above the grid. Areas 1, 2, 3, and 4

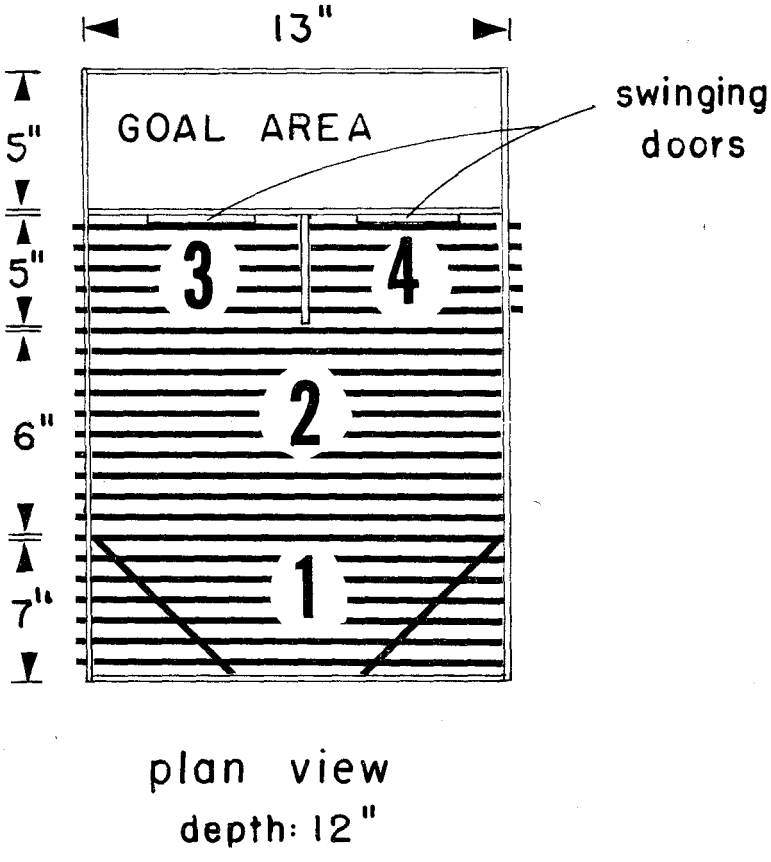

Fig. 1. Schematic view from above of apparatus for shock-motivated simultaneous discrimination experiments.

were independently electrified by a Grason-Stadler shock scrambler set at either .2 ma. (mild shock) or .8 ma. (strong shock).

Surgical and histological procedure. Subjects weighed 270–350 gm. at the time of surgery. Following anesthesia by a 6% solution of pentobarbital sodium (60 mg/kg), the subject was placed in a Stoelting stereotaxic instrument and its head shaved. The bone over the cerebellum (Os Interparietale) was exposed and removed by dental drill. If the animal was to be part of the sham-operation group, Gelfoam was inserted into the bone opening, the soft tissues and skin reclosed, and the wound sutured with 3-0 silk thread. If the animal was to be part of the treatment group, a narrow suction tip fashioned from glass tubing was used to aspirate the medial cortex and underlying cerebellar tissue of Lobules VI (Bolk's "simplex") and VII (Ingvar's "medius medianus"), with some damage to Lobule V (Bolk's "culmen;" see Zeman & Innes, 1963). The resulting space was filled with moist Gelfoam and the wound was closed as in the sham-operation condition. For all animals, Achromycin powder was sprinkled on the closed wound, 200,000 units of penicillin were injected im, and a .5% solution of Megamide (5 mg/kg) was injected ip.

At the completion of testing, all animals were sacrificed with ether and perfused intracardially with saline followed by 10% formol-saline. The brains were removed, photographed (in some cases), and sliced on a freezing microtome. Coronal sections of 40 μ thickness were taken at intervals of 120 μ or 400 μ in the region of the lesion. Mounted sections were stained with .1% luxol fast blue and counterstained with 1.0% neutral red (technique after Pearse, 1956). The lesions employed in Experiments 1 and 2 were slightly larger and extended more anteriorly than the large lesions shown in Figure 3A.

Behavioral procedure. Testing began at least 3 wk. after surgery. Animals with discrete cerebellar lesions seldom showed outward signs of motor dysfunction after the first week. All subjects were given a code number and were tested "blind" to reduce the possibility of experimenter bias. On the first day of pretraining, individual subjects were allowed to explore the apparatus with the doors open for 2 min. The rat was then placed in Area 1 facing away from the goal area and was given brief shocks if it did not leave Area 1 within 5 sec. or Area 2 within 25 sec. When the rat entered the goal area, a 30-sec. intertrial interval was begun; such an interval was maintained throughout the experiment. After the subject made three consecutive runs to the goal area without any shocks, the last entrance chosen was blocked and the shock turned on in front of it. Animals were always allowed to retrace after encountering a locked door. After the subject ran once through the unblocked opening without any shocks, that door was closed and locked, the other one unlocked, and the shock shifted to the new side. Pretraining was complete when the subject entered the goal area once more without receiving shock. Each animal learned only one problem under one level of shock. The number of control and treatment-group animals, respectively, in each condition was as follows: stripe discrimination, mild shock: 8 and 7; stripes, strong shock: 9 and 9; triangles, strong shock: 8 and 5.

On the second day, discrimination training began. Metal-backed stimulus card holders were held by magnets imbedded in the doors and the position of the positive stimulus was changed according to a modified Gellermann (1933) series during the intertrial interval. On trials when the discriminanda were not shifted, the experimenter went through the usual motions of changing them in order not to give the subject any unintentional cues about the position of the positive stimulus on the following trial. Since the same stimulus card in the stripe discrimination was either positive or negative throughout training for each individual subject, it is possible that olfactory cues may have facilitated learning (Phillips, 1968). However, since errors were recorded when the subject touched the charged grid area extending more than 12 cm. in front of the locked door, the use of olfactory information is unlikely. The triangle stimuli were "shifted" from one side of the choice point to the other by a 180° rotation of the cards.

All animals received 20 training trials each day until they reached a criterion of 18 correct out of any 20 consecutive trials. The numbers of correct and incorrect choices and the time taken to go from Area 1 to the goal area were recorded. The animal was transferred from its home cage to the apparatus on a wet sponge and the grid surface was cleaned and polished before each new subject was tested in order to retain as nearly as possible the same shock characteristics for each animal. Five of the treatment group animals which had achieved learning criterion for the stripe discrimination with .2-ma. shock were required to perform the same discrimination after receiving enforced .8-ma. shock (½ sec.) in Area 1 at the beginning of each trial for a session of 20 trials. Four animals from each group which had learned the triangle discrimination were subsequently given a free choice (no shock; both doors unlocked) on 10 transfer trials with the same triangle contours but with the black and white areas interchanged (for details, see Buchtel, 1969).

Results

Stripe discrimination. With mild shock, the mean trials to criterion for the control group ($n = 8$; $\bar{X} = 52.1$) and the treatment group ($n = 7$; $\bar{X} = 54.7$) were not significantly different. With strong shock, the mean trials to criterion for the control group ($n = 9$) and the treatment group ($n = 9$) were 51.0 and 87.6, respectively.

This difference is statistically significant ($t = 2.90$, $df = 16$, $p < .01$).

The mean times taken to go from Area 1 to the goal area were not significantly different for the two groups at either shock level. Treatment group animals which had learned under conditions of mild shock and which were given .8-ma. shocks in Area 1 at the beginning of each trial were able to perform the discrimination at or above criterion level.

Triangle discrimination. The control group ($n = 8$) and the treatment group ($n = 5$) learned the triangle discrimination in a mean of 101.9 trials and 108.8 trials, respectively. This difference is not statistically significant. On transfer trials, all rats chose the opposite form on at least 7 of the 10 trials and half of the rats chose it every time. This suggests that the animals made the original discrimination on the basis of relative distribution of light rather than on the basis of contours (Buchtel, 1969).

Experiment 2

Method

Subjects. Thirty-two male hooded rats were divided into two groups as follows: 14 rats with damage to cerebellar cortex as described in Experiment 1; 18 rats with sham operations.

Apparatus. The essential components of the apparatus included an operant-conditioning box with a lever, food dish, and calibrated drinking tube. The conditioning box was housed in a sound-proof chamber containing a food dispenser, a 6-v. house light, and a speaker for masking noise. Food pellets were introduced into the food cup by remote control.

Surgical and histological procedure. Discrete cerebellar lesions and histological examinations were identical to those described for Experiment 1.

Behavioral procedure. Subjects were placed on one of two regimens of restricted food intake (see Collier, Levitsky, & Squibb, 1967) to lower their body weight to 95–100% of free-feeding weight (low-motivation condition) or to 85–90% of free-feeding weight (high-motivation condition). Pretraining began 3 days later. On the first 2 days of pretraining, randomly paired rats were placed in the apparatus for 20 min. and each lever press was reinforced by a Noyes 45-mg. food pellet. On subsequent days, the rats were tested individually to assure that each would make 50 lever presses within 20 min., and discrimination training was begun on the day after this criterion was met.

Each of six daily discrimination sessions consisted of 65 trials but only the last 50 trials of each session were analysed. The onset of the house light signalled the beginning of a trial and the light remained on for 20 sec. or until the animal responded with a lever press which was reinforced by a food pellet. Lever presses during the 10-sec. average intertrial interval—when the house light was off—were not reinforced and were counted as errors. Measures taken included the number of trials, the number of unrewarded lever presses (except those during the ½ sec. after the presentation of food), and the sum of response latencies to the onset of the light. On Day 7 of training the house light was on continuously to determine its control over lever pressing.

Results

Figure 2 shows the results for treatment and control groups under the two conditions of motivation. Scores were derived by multiplying the individual error scores by 100 and dividing by the number of errors committed on the first day by that animal. This transformation controls for possible differences between individuals in baseline response rates. The right half of Figure 2 shows that under conditions of low motivation, the treatment and control-group rats learned the discrimination at the same rate ($F = .30$, $df = 1/14$). Errors for both groups decreased over sessions ($F = 20.71$, $df = 4/56$, $p < .01$), indicating learning. Raw numbers of errors did not differ for the two groups. The left half of Figure 2 shows that with high motivation, the treatment group did not learn the discrimination as quickly as the control group ($F = 25.98$, $df = 1/14$, $p < .01$). Errors for both groups decreased over sessions ($F = 26.45$, $df = 4/56$, $p < .01$). An analysis of the raw numbers of errors also shows slower learning by the treatment group ($F = 7.70$, $df = 1/14$, $p < .05$). Groups did not differ in latency of response to the onset of the light. On Day 7 all animals increased the number of unreinforced presses during the intertrial interval when the light was on continuously, indicating that the light was being used as a discriminative cue.

Experiment 3

The previous two experiments indicated that under conditions of high motivation, the learning of some visual cues is slower

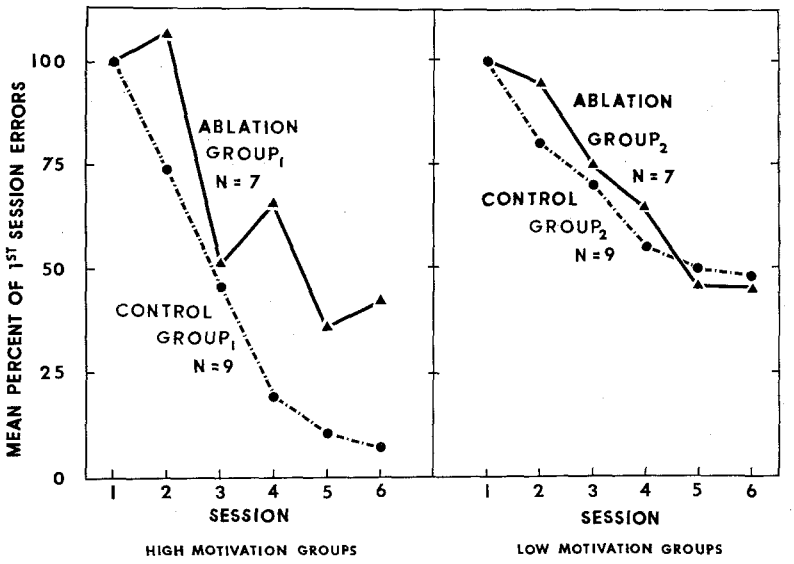

FIG. 2. Learning scores for light-on, light-off discrimination under the two levels of food motivation in Experiment 2.

than normal following damage to midline cerebellar cortex. Experiment 3 was designed to examine the effects of lesions in different parts of the cerebellum under conditions where cerebellar damage had been shown to retard learning, namely, in learning to use light as a cue for food reward under conditions of high motivation.

Method

Subjects. Sixty-five male hooded rats were divided into six groups as follows: 11 rats with small lesions in Midline Lobule VI (medial simplex group); 10 rats with damage to both medial and lateral portions of Lobule VI (medial plus lateral simplex group); 11 rats with damage to lateral portions of Lobule VI, sparing the medial portions (lateral simplex group); 10 rats with damage to Lobule V (culmen group); 11 rats with damage to Lobules VII, VIII, and IX (posterior group); and 12 rats with sham operations.

Apparatus and behavioral procedure. The apparatus and procedures were the same as described in Experiment 2 except that the number of sessions was increased from 6 to 8, and the effect of continuous light on pressing rate was not tested.

Surgical and histological procedure. Suction ablations were carried out in the manner described in Experiment 1. After completion of testing, an independent judge classified diagrams of the lesions into five treatment groups according to the superficial placement of the lesion (see Figure 3). Histological examination differed from that of Experiment 1 only in that saggital sections at intervals of 300 μ were taken instead of coronal sections, and the staining was by thionin (technique after Davenport, 1960). Because of the irregularity of the ablations, the volume was determined by measuring the amount of tissue missing from each section taken through the lesion and multiplying by .3 mm.

Results

Variance in learning speed was unusually large and the data of four rats were dropped from analysis because both the Day 8 raw number of errors and the Day 8 percentage error scores were more than two SDs above the means of their respective groups (these included two rats from the medial simplex group, one rat from the lateral simplex group, and one rat from the sham group); the data of five rats were not considered because the size or placement of their lesions was judged to be unlike those of the other rats; and four animals were classified by the judge as belonging to groups other than those expected on the basis of the surgical protocols. This left 56 rats divided among the following groups: 10 medial simplex; 7 lateral simplex; 7 medial plus lateral simplex; 9 culmen; 12 posterior; and 11 sham.

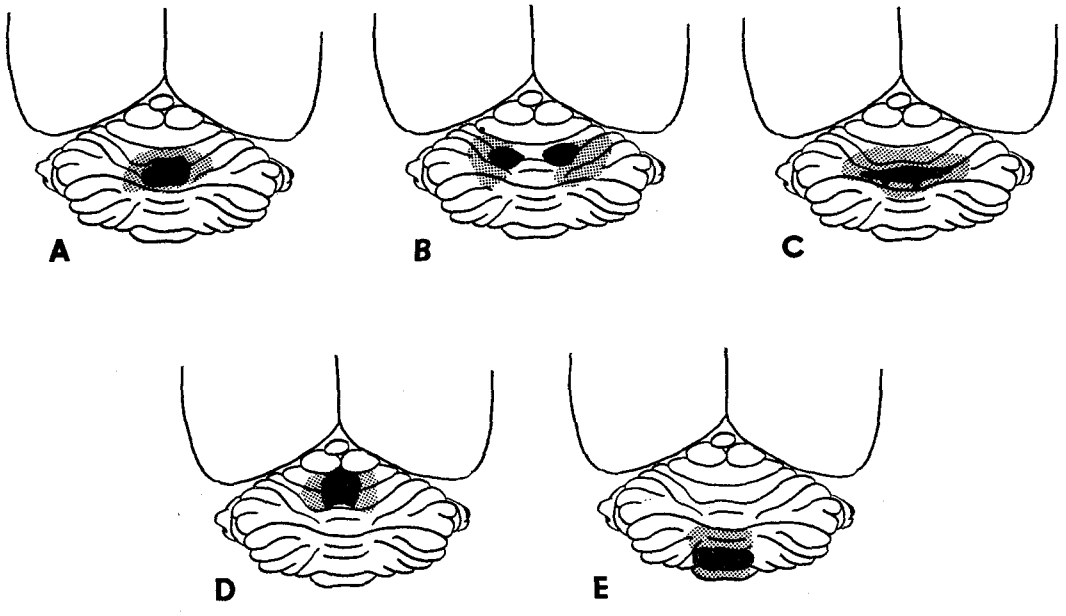

FIG. 3. Schematic view from above of smallest (black area) and largest (shaded area) ablations in the treatment groups of Experiment 3. (A, medial simplex; B, lateral simplex; C, medial plus lateral simplex; D, culmen; and E, posterior. Black and shaded areas represent deep parts of ablation; superficial cortical damage occasionally extended beyond these boundaries.)

The largest and smallest lesions in each group are diagrammed in Figure 3, and saggital sections of representative ablations are shown in Figure 4.

An overall two-way analysis of variance indicates a significant difference in percentage error scores for the six groups ($F = 2.83$, $df = 5/50$, $p < .05$). Figure 5 shows the learning curves for the control group and the two groups which differed significantly from the control: medial plus lateral simplex group ($F = 5.12$, $df = 1/16$, $p < .05$) and culmen group ($F = 4.74$, $df = 1/18$, $p < .05$).

Variance within groups was partly due to variation in lesion size, but in an unexpected way. Considering all 45 treatment-group rats, there was a negative correlation between volume of the ablation and the Day-8 percentage-error score (Spearman $r_s = -.25$, $t = 1.69$, $df = 43$, $p < .05$, one-tailed). All treatment groups showed this negative correlation as reflected in Figure 6 which shows the groups divided according to whether the lesion was smaller or larger than the average for the group. If the data for the 12 subjects with lesions smaller than the average for their respective groups are taken from the three groups which showed the largest negative correlations (lateral simplex, medial plus lateral simplex, and posterior), and are compared as a group with the control group, the degree of learning deficit is striking ($F = 4.39$, $df = 1/21$, $p < .05$) and there is a significant interaction of groups and days ($F = 2.51$, $df = 6/126$, $p < .01$).

DISCUSSION

The results of Experiments 1 and 2 suggest that damage to the rat cerebellum causes a deficiency in learning some visual cues under conditions of high motivation. These lesions were located in an area which would be expected to receive visual projections. Experiment 1 also shows that the learning of some stimuli was normal in these cerebellar damaged animals: Triangle discriminanda were learned at the same rate by control and treatment groups. However, an earlier study (Buchtel, 1965) showed that a simultaneous discrimination based on brightness dif-

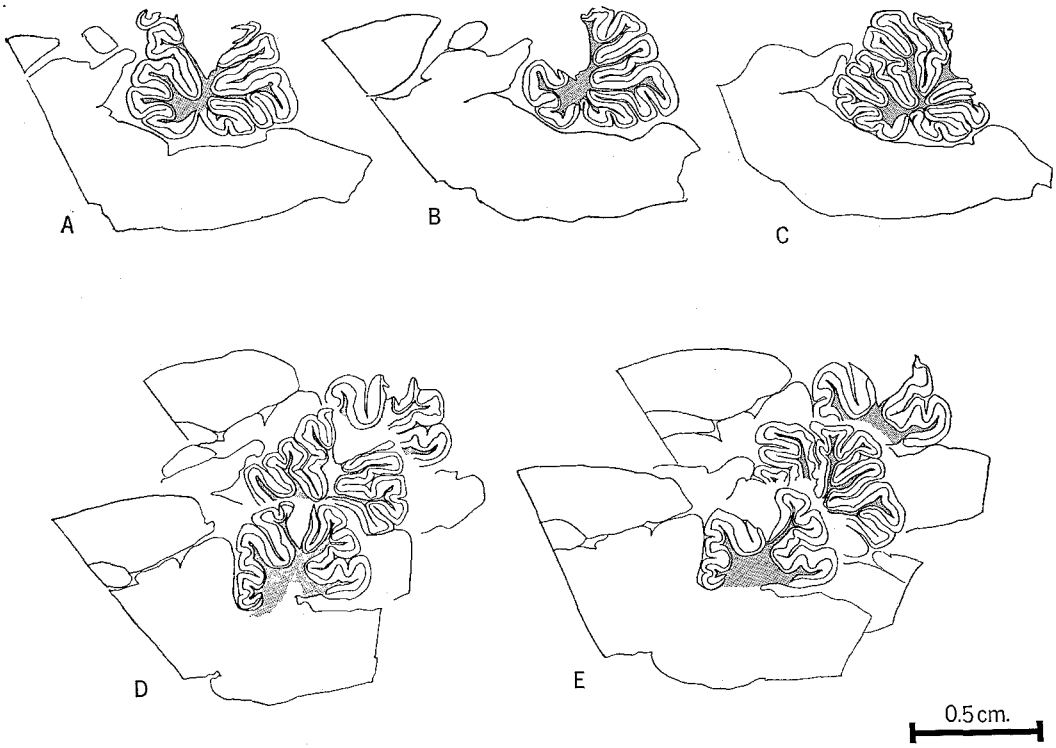

FIG. 4. Drawings of brain sections with representative lesions used in Experiment 3. (A, medial plus lateral simplex; B, culmen; C, posterior; D, medial plus lateral simplex; E, lateral simplex. A, B, and C show midsagittal sections; D and E show midsagittal sections and sections taken 3 mm. to each side.)

ferences (black vs. white) was less hindered by cerebellar damage than a discrimination based on pattern (vertical vs. horizontal stripes). Thus it is significant that performance on the triangle transfer tests indicates that the subjects were probably discriminating on the basis of flux cues within the figures rather than on the basis of contours (see Buchtel, 1969).

The results of Experiment 1 also suggest that cerebellar damage hinders learning but not performance under conditions of high motivation: Strong preshocks before each trial after the completion of learning did not differentially affect the performance of the stripe discrimination by the treatment-group rats. This finding makes it unlikely that the treatment-group animals under conditions of strong shock learned slowly because of motor dysfunction per se, since such a dysfunction should interfere with performance both before and after learning the significance of the visual cues. Of course, performance of very fine coordinated movements under such conditions of motivation would probably be affected by cerebellar damage (Lipton, 1966).

The results of Experiment 3 show that both the placement and the size of the lesion determine the effect of cerebellar damage on learning speed. Learning deficits were greatest with small lesions in Lobule V (culmen) and small lesions in Lobule VI (simplex) except when in the latter case they involved medial cortex alone. One might infer from this observation that for lobulus simplex, at least, the more lateral cerebellar nuclei (dentate and interpositus) and associated cortex are more important for efficient visual learning under these experimental conditions than the medial nuclei (fastigial) and associated cortex. The paradoxical effect of lesion size is difficult to explain. Perhaps the removal of a whole system or subsys-

tem causes a shift of function to healthy tissue, while tissue only partly damaged continues to perform its functions, but poorly. This finding may account for the lack of positive results following total cerebellectomy.

The task in Experiments 2 and 3 was similar to that in Fanarjyan's (1961) experiment with dogs. His subjects first learned to make a leg flexion when visual auditory stimuli (CSs for shock) were presented. Following this initial training, a slightly different stimulus (e.g., a brighter light) was presented interspersed with the shock trials but was never itself followed by shock. Control animals stopped responding to this neutral stimulus sooner than did animals with cerebellar damage.

During the preparation of this manuscript, a study was published showing an effect of cerebellar damage on the initial phases of learning a light-on, light-off discrimination by cats (Rubia, Angermeier, Davis, & Watkins, 1969). These authors presented suggestive evidence for slower learning following discrete lesions of lateral cerebellar cortex. Unfortunately, the small number of subjects ($n = 2$) and the lack of an adequate control group require caution in interpreting their findings

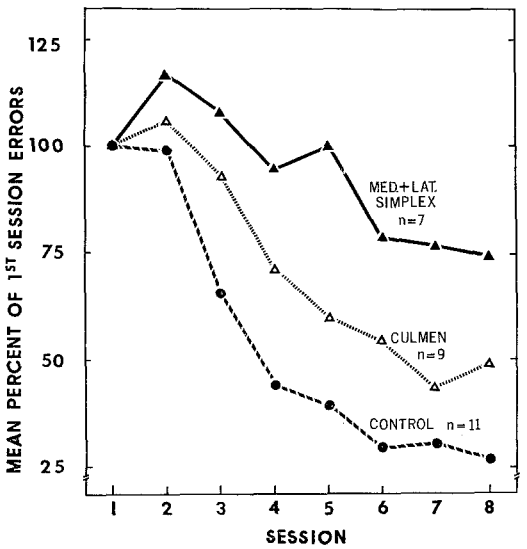

FIG. 5. Percentage error scores in Experiment 3 for the control group and groups which learned significantly slower than the control group.

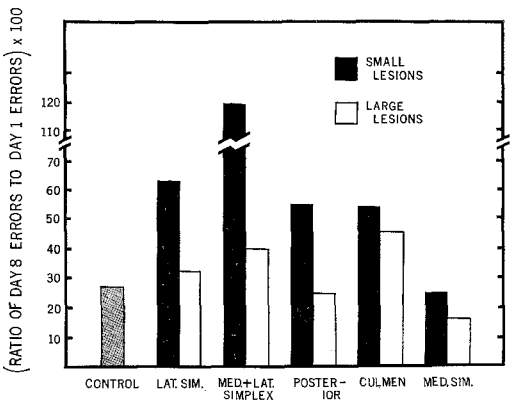

FIG. 6. Comparison of mean percentage error scores on Day 8 of discrimination training in Experiment 3 as a function of lesion size.

as definitive proof of the conclusion reached here. However, it is interesting to note that their subjects were maintained at 80% of free-feeding weight, which may have produced a level of motivation equivalent to that used in Experiment 2.

A factor common to some of the studies which show behavioral effects of cerebellar damage is the necessity of learning to inhibit a well-learned response. In the case of Experiment 2, the subjects had to learn not to make level presses during the intertrial interval when the house light was off. Even the simultaneous stripe discrimination in Experiment 1 may be seen as a problem of "not responding" since rats tend to choose horizontal stripe patterns over vertical stripe patterns (Lashley, 1938; but see also Dodwell, 1961). Thus it is interesting that under conditions of strong shock, the treatment-group rats which took longest to learn tended to be those for which the positive stimulus was the vertical rather than the horizontal stripe pattern ($p = .083$, Mann-Whitney U). No such relationship was found for the control-group animals or for the treatment-group animals under conditions of mild shock. This finding provides further evidence that the slower learning was not due simply to poor coordination since impaired motor behavior should hinder learning irrespective of which stimulus is positive.

The results of these experiments do not give an unequivocal answer to the question of how the cerebellum contributes to visual learning or indeed whether learning deficits following cerebellar damage are limited to visually guided behavior. On the contrary, the early studies of Stowell and Snider (1942) suggest that auditory discriminations should also be influenced by cerebellar damage. Regardless of which sensory modalities are ultimately implicated, a working hypothesis is suggested by the present finding of an interaction between cerebellar damage and level of motivation; that is, the cerebellum acts to reduce disruption of learning during periods of high motivation by preventing overfacilitation of motor responses, especially in tasks where inappropriate responses already have a high probability of expression.

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(Received November 6, 1969)