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# The effect of previous experience upon operant performance following cerebellar lesions in the rat

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Lesions of the fastigial nuclei and cerebellar vermis, but not lesions of the dentate nuclei, were found to produce marked performance deficits on a differential reinforcement of low rates (DRL) schedule of reinforcement. This deficit was characterized by an abnormal number and distribution of responses within the schedule interval. Lesions, however, did not produce a deficit following preoperative training or when subjects were tested on a fixed-interval (FI) schedule. In addition, when DRL and FI performance was contrasted, all subjects were responsive to schedule contingencies. Results suggest that the DRL deficit following cerebellar lesions is due to a tendency to perseverate in response strategies, and is not related to a global disruption of timing or a pervasive inability to suppress responding.

The involvement of cerebellar structures in the regulation and coordination of motoric functions is well documented and is clearly evident in the clinical consequences of cerebellar insult. Such consequences often include dysmetrias and asynergias related in large part to an inability to inhibit motor movements. (Dow, 1961; Dow & Moruzzi, 1958; Holmes, 1917, 1939). More recently, the role of the cerebellum in motoric functioning has been suggested to include the neural encoding and storage of well-learned motoric sequences. Such theories postulate that the cerebellum plays a critical role in the establishment and execution of learned motor sequences in a manner similar to that of cerebellar involvement in postural and reflex mechanisms. It is postulated that as motoric sequences become well practiced, the cerebellum develops a means of facilitating the smooth execution of movements within the sequence (Eccles, Ito, & Szentagothai, 1967; Fujita, 1982; Gilbert, 1974; Ito, 1974; Marr, 1969). In addition, there is a growing body of data indicating that cerebellar structures may play an important role in the control and elaboration of complex motivated behaviors (Berntson & Micco, 1976; Berntson & Torello, 1982; Dow, 1974; Lavond, McCormick, & Thompson, 1984; Watson, 1978b). A number of highly organized behaviors, including grooming, eating, and attack, may be elicited with electrical stimulation of the anterior cerebellum and rostral fastigial nuclei. These behaviors are not merely motoric automata resulting from the elicitation of complex reflexive behaviors, but evidence serial organization, goal direction, and sensitivity to the stimulus features of the goal object (Berntson, Potolicchio, & Miller, 1973; Berntson & Paulucci, 1979; Watson, 1978a). That such stimulation has motivational consequences is evidenced in the self-administration of stimulation at many cerebellar loci from which these behaviors can be elicited (Ball, Micco, & Berntson, 1974). Additionally, lesions of the paleocerebellum may result in a reduction or disruption of exploratory behavior, social interactions, and defensive responses, in the absence of any overt motoric deficits (Berman, Berman, & Prescott, 1974; Berntson & Schumacher, 1980; Berntson & Torello, 1982; Peters & Monjan, 1971; Watson, 1978b). Related to this suggestion is the finding that cerebellar injuries following establishment of the conditioned association eliminate the classically conditioned nictitating membrane response in the rabbit without impairing the unconditioned response (Lavond et al., 1984; McCormick et al., 1981; McCormick & Thompson, 1984). These data indicate that cerebellar injury may profoundly compromise learned behaviors without overtly disrupting their motoric basis.

Common to these cerebellar influences may be their control over sequential integration of behavioral functions at all levels of organization, ranging from relatively simple reflex acts to complex behavioral processes. Thus, cerebellar injury may disrupt learned behavioral sequences when such injuries involve tissue that may serve to facilitate the rapid and smooth execution of behaviors but not be essential for the expression of individual behavioral components. In addition to these findings, further studies into the consequences of cerebellar injury have demonstrated pronounced perseverative deficits that appear to be unrelated to any specific loss of memorial functioning

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or motoric ability. Mazes that require sequential alternations of left and right turns present tremendous difficulties for rats with paleocerebellar lesions (Pellegrino & Altman, 1979), and more extensive injuries have been shown to impair performance in less complex mazes that do not require such alternations (Lashley & McCarthy, 1926; Thompson, 1974). Similar deficits have been demonstrated with two-choice visual discrimination tasks (Buchtel, 1970; Davis, Watkins, Angermeier, & Rubia, 1970). These deficits appear to result from the animal's inability to inhibit responding or to switch response strategies. Such behavioral sequelae are reminiscent of motor deficits seen following cerebellar injury; dysmetria, dysdiadochokinesis, and the decomposition of movement. Moreover, previous investigation in this laboratory (Kirk, Berntson, & Hothersall; 1982) has demonstrated that subjects with paleocerebellar lesions exhibit a pronounced performance deficit when required to specifically withhold a previously established operant response in a differential reinforcement of low rates (DRL) schedule. This deficit, however, was overcome when an overt "collateral" behavior was made available. It is plausible, in light of these findings, that the DRL deficit resulted from an inability to organize or sequence behaviors, rather than from a loss of timing ability or motoric dysfunction per se. Similarly, such schedule performance may result either from an inability to withhold responding or from the perseverative use of a response strategy that results in consistent mistiming of the schedule interval. The present studies were designed to explore this issue and to further characterize the nature of operant deficits following cerebellar injuries.

## EXPERIMENT 1

The cerebellum has vast anatomical and functional connections with virtually every level of the neuraxis. The anterior cerebellar vermis projects primarily to the fastigial nuclei, which, in turn, provide ascending outputs to multiple sites within the midbrain reticular formation, midbrain central gray, nuclei of the extrapyramidal motor system, and more diffuse projections to thalamus, hypothalamus, and diverse limbic areas (Anand, Malhotra, Singh, & Dua, 1959; Angaut & Bowsher, 1970; Dietrichs, 1984; Harper & Heath, 1973; Heath & Harper, 1974; Snider, 1975), as well as descending projections to the vestibular nuclei, brainstem reticular formation, and spinal gray matter (Andrezik, Dormer, Foreman, & Person, 1984; Brodal, 1981; Martin, King, & Dom, 1974; Snider, Maiti, & Snider, 1976). The dentate nuclei, however, provide the major rostral outflow of the cerebellum, via the superior cerebellar peduncle, to principally extrapyramidal structures, such as the red nucleus, basal ganglia, and to the ventral lateral nucleus of the thalamus, from which influences are radiated to widespread cortical areas (Brodal, 1981; Dow, 1961, 1974; Dow & Moruzzi, 1958; Modianos & Pfaff, 1976; Sprague & Chambers, 1959; Snider, 1967).

In general, deficits in species-characteristic behaviors have been reported following lesions of the anterior cerebellar vermis or the fastigial nuclei within what has been classically termed the paleocerebellum (Larsell, 1934; 1937). In contrast, the dentate nuclei, associated with the neocerebellum, have been recently implicated in a form of associative learning (Fish, Baisden, & Woodruff, 1979; Lavond et al., 1984; McCormick & Thompson, 1984). In a previous study, Kirk et al. (1982) reported a marked DRL performance deficit following injuries within the paleocerebellum. To replicate and more fully clarify the cerebellar systems involved in this deficit, both paleocerebellar and neocerebellar injuries were examined.

### Method

**Subjects.** The subjects were 72 male albino rats (90-120 days of age) obtained from the colony at Charles River or bred in the laboratory from the same strain of animals. The subjects were group-housed and maintained under a 12-h light/dark cycle with ad-lib food (Purina Lab Chow) and water.

**Surgery.** Surgery was performed under sodium pentobarbital anesthesia (55 mg/kg ip) following pretreatment with atropine sulfate (.12 mg ip). Once fully anesthetized, each subject was secured in a Kopf stereotaxic instrument, and the skull was exposed. Electrode coordinates (fastigial, AP −11.5 mm, ML ±0.8 mm from the midline, DV −7.7 mm below the skull; dentate, AP −9.6 mm, ML ±3.5 mm, DV −4.5 mm) were derived from the atlas of Fifkova and Marsala (1967). Trephine holes were then drilled, and a monopolar electrode, insulated except for .5 mm at the tip, was lowered to the appropriate sites. Bilateral electrolytic lesions were then induced (1.5-mA anodal dc current for 10 sec), the electrode was withdrawn, and the scale incision was sutured. Control animals were anesthetized and mounted in the stereotaxic instrument, but received no further surgical manipulation. Following surgery, the animals were administered a broad-spectrum antibiotic (Duracillin, 200,000 units) and returned to individual home cages.

**Apparatus.** The apparatus consisted of eight conventional operant chambers, each with a single bar, food well, and houselight on the front wall. The chambers were isolated within individual sound-attenuating chests, and white noise was used to mask extraneous sounds. Reinforcement schedules were programmed and response measures recorded by an Apple microcomputer interface located in a room adjacent to the testing chambers.

**Procedure.** After 21 days of postoperative recovery, the subjects were reduced to 85% of normal body weight and were maintained at this level throughout the remainder of behavioral testing. Training and test sessions were 1 h in length and were conducted 6 days a week, between 10:00 a.m. and 7:00 p.m., during the light portion of the light/dark cycle. Using conventional operant techniques (Anger, 1956; Innis, Reberg, Mann, Jacobson, & Turton, 1983; Innis, Simmelhag Grant, & Staddon, 1983; Slonaker & Hothersall, 1972), the subjects were trained to barpress for appetitive reinforceforcement (45-mg Noyes pellet). After acquiring the operant response and earning 100 reinforcers, the subjects were shifted to a DRL 5-sec schedule. Thereafter, when subjects earned 10 reinforcers, the schedule interval was progressively increased by 5 sec until a DRL 20-sec schedule was attained. Behavioral testing continued for 24 sessions. The total number of responses emitted, the number of reinforcers earned, and the individual interresponse times (IRTs) were recorded for each session.

**Histology.** After the completion of all behavioral testing, the subjects were sacrificed, by an overdose of sodium pentobarbital, and perfused intracardially with normal saline followed by 10% formalin. After the brains were removed and frozen with dry ice, 50-$\mu$ sections were cut with a Reichert microtome. Every fifth section through the lesion was slide-mounted and stained with cresyl vio-

let. The locations and extents of the lesions were then plotted by direct projection onto diagrams of Fifkova and Marsala (1967) (B&L Tri-simplex microprojector). To minimize error in the estimated lesion size arising from shrinkage or distortion of the tissue over the long survival time employed, care was taken to draw lesion boundaries on the basis of remaining tissue rather than acellular areas. Lesions were evaluated and then classified (fastigial, dentate, vermal) by a judge who was unaware of the behavioral data.

## Results

**Histological results.** Histological examination revealed that lesions either were limited to the dentate nuclei (neocerebellum) or were confined to what is generally termed the anterior paleocerebellum, including portions of the anterior vermis and fastigial nuclei (Figure 1).

To evaluate any behavioral differences that might be due to variance in either size or location of the lesions, estimates of lesion area were obtained through planimetric analysis (K&E 620015 Compensating Polar Planimeter) of the standard lesion reconstructions. In addition, the dorsal-ventral, rostral-caudal, and medial-lateral centers of the lesions were determined. Consistent with previous investigation (Kirk et al., 1982), analysis of these data failed to reveal any ubiquitous pattern in the performance of animals with lesions of the anterior vermis, or its projection site, the fastigial nucleus. Furthermore, analysis of the performance of subjects with such lesions in the present group again failed to show any differences between lesions of the fastigial nuclei and lesions restricted to the anterior paleocerebellum [$t(22) = .603$, $p > 5$]. Accordingly, subjects with vermal and fastigial lesions were pooled for subsequent analysis.

**Behavioral results.** All subjects with cerebellar lesions demonstrated marked motor impairments following the lesioning; this included tremor and ataxia, especially of the hindlimbs. Consistent with previous reports (Berntson & Schumacher, 1980; Fish et al., 1979; Modianos & Pfaff, 1976), these overt motoric impairments diminished rapidly, and by the commencement of behavioral testing, 30 days after surgery, lesioned animals were virtually indistinguishable from normal animals.

Cerebellar lesions did not appear to impair subjects' ability to acquire the CRF barpress response for appetitive reinforcement. Lesioned subjects and sham-operated controls required an average of three test sessions to acquire the barpress response and earn 100 reinforcers on the CRF schedule. When switched to the DRL task, however, differences between lesioned subjects and controls became apparent. As illustrated in Figure 2, subjects with vermal/fastigial lesions showed an impaired acquisition of the DRL task, characterized by reduced efficiency and an elevation in response rate, especially within the early phases of the schedule interval. Subjects with lesions of the dentate nuclei, however, showed essentially normal acquisition of the DRL task and only small increases in response rate (see Figure 3). Analyses of variance revealed that although all groups showed a reduction in the number of barpresses with training (see Figure 3) emitted [$F(3,207) = 40.072$, $p > .001$], the subjects differed in the number of responses emitted [$F(2,69) = 6.163$, $p = .003$]. Moreover, although all groups showed improvement in the efficiency ratio (ER = reinforcers/responses; Kramer & Rilling, 1970) [$F(3,207) = 58.123$, $p < .001$], there were group differences in this measure as well [$F(2,69) = 3.755$, $p = .027$].

Interresponse-time data (see Figure 4) revealed that animals with paleocerebellar lesions evidenced maximal responding at intervals too short to satisfy schedule requirements. In contrast, animals with dentate lesions showed a normal IRT distribution. A two-way ANOVA on the IRT distributions revealed that, with continued training, all groups were altering their response tendencies to fit the temporal contingencies of the schedule [$F(9,585) = 37.3ll$, $p < .001$]. However, there were again lesion-related differences in the IRT distributions [$F(3,65) = 2.947$, $p = .038$]. Furthermore, there was a strong interaction between the surgical and time factors [$F(27,585) = 2.699$, $p < .00l$], reflecting the failure of animals with fastigial/vermal lesions to suppress responses during the early phases of the DRL interval (see Figure 4).

Analysis of the standard lesion reconstructions failed to reveal any consistent relationship between lesion size and DRL performance in either the dentate or the vermal/fastigial lesion group. Moreover, subsequent regression analysis between lesion size and terminal efficiency on the DRL schedule confirmed this result (dentate, $R^2 = .08$; vermal/fastigial, $R^2 = .02$).

## Discussion

The overall pattern of results presented in this experiment is consistent with the report that paleocerebellar lesions result in a postoperative DRL deficit (Kirk et al., 1982). The present findings, however, also demonstrate that such deficits are related to destruction of the anterior vermis and/or fastigial nuclei, but are not apparent after lesions of the dentate nuclei. The performance deficit is characterized by overresponding early in the schedule interval, together with a peak shift toward IRTs of shorter duration. Three possible explanations for the failure of lesioned subjects to redistribute their responses toward longer IRTs are that they are unable to appropriately time the schedule interval, are unable to inhibit responding, or are simply slower to acquire the schedule constraints.

# EXPERIMENT 2

It has been argued that the development of "collateral behaviors" may serve to mediate timing of the schedule interval and thus improve timing performance (Hothersall, Alexander, & Slonaker, 1972; Laties, Weiss, Clark, & Reynolds, 1965; Laties, Weiss, & Weiss, 1969; Slonaker & Hothersall, 1972). In this regard, the explicit provision for a collateral behavior, through the introduction of a chewing block, has been shown to alleviate DRL deficits following cerebellar injury. The rapid improve-

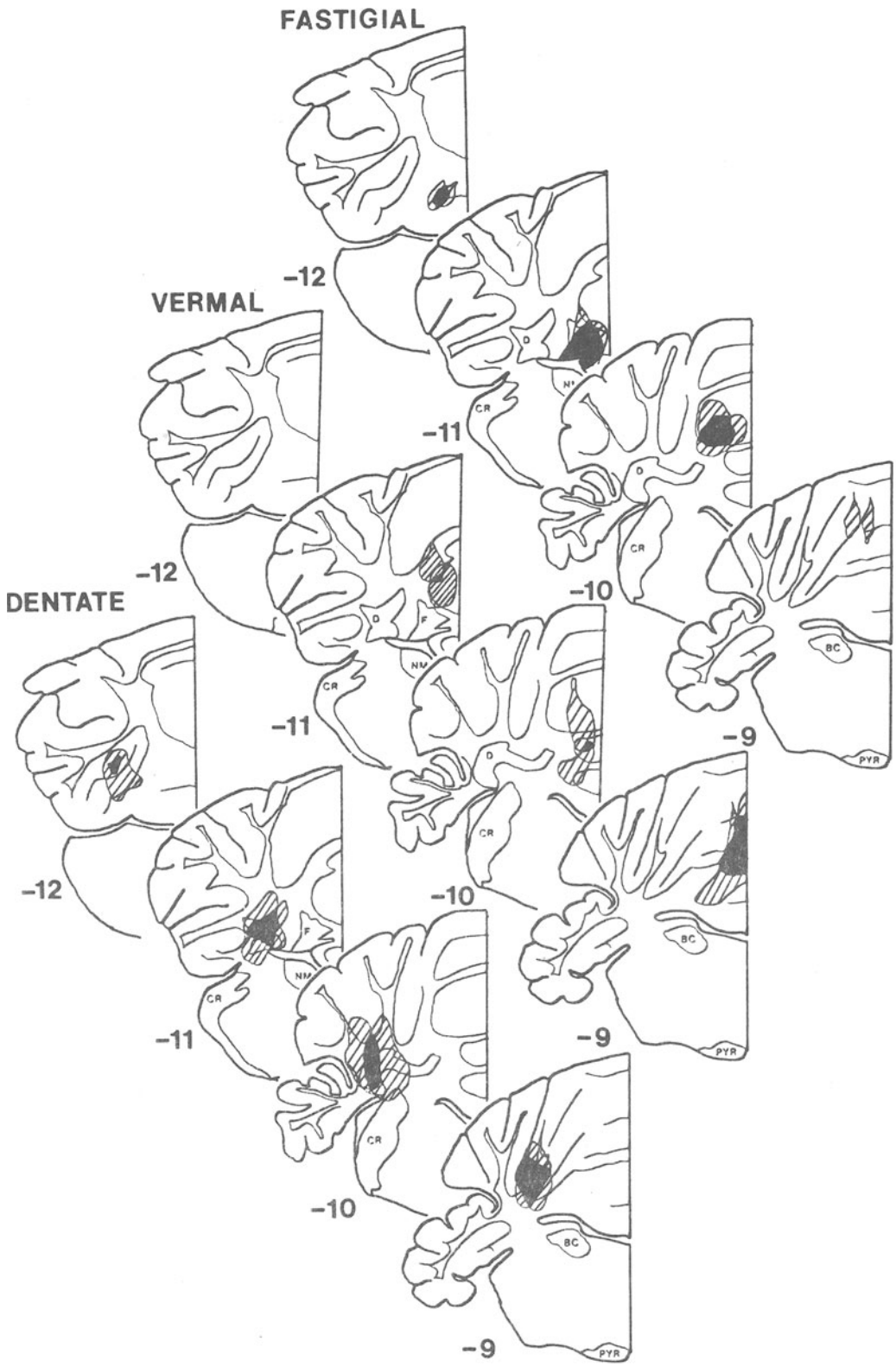


**Figure 1. Representative dentate, vermal, and fastigial lesions. Areas of unilateral injury are hatched, and areas of bilateral injury are blackened.**

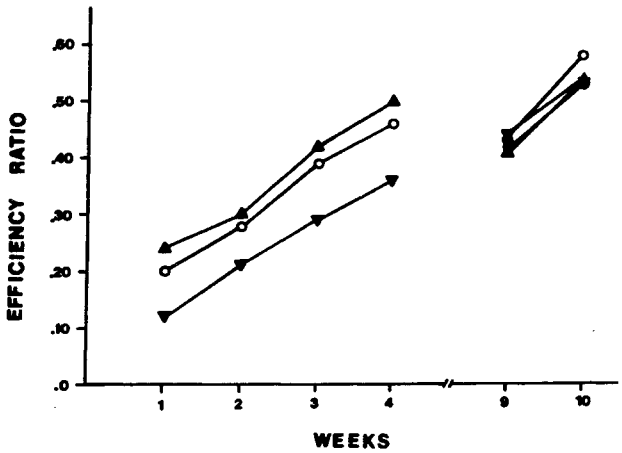


**Figure 2. Efficiency ratio (reinforcers/responses) performance measures obtained during acquisition (Weeks 1-4) and testing following a 30-day break in training (Weeks 9 and 10), for subjects with vermal/fastigial lesions (inverted and closed triangles) and dentate lesions (closed triangles), and for sham-operated controls (open circles). In addition, data for subjects receiving vermal/fastigial lesions following 4 weeks of behavioral training (closed circles) are included for Weeks 9 and 10.**

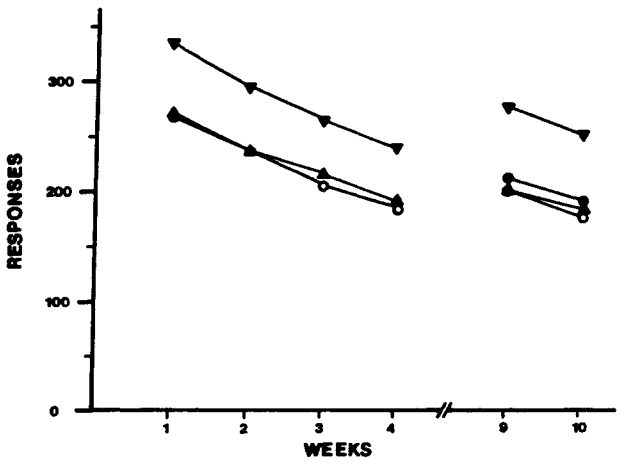


**Figure 3. Response measures obtained during acquisition (Weeks 1-4) and testing following a 30-day break in training (Weeks 9 and 10), for subjects with vermal/fastigial lesions (inverted and closed triangles) and dentate lesions (closed triangles), and for sham-operated controls (open circles). In addition, for subjects receiving vermal/fastigial lesions following 4 weeks of behavioral training (closed circles) are included for Weeks 9 and 10.**

ment seen in these animals following introduction of the block suggests that their performance deficit is not simply the result of a learning deficit. Although this improvement may be due to an enhancement of timing ability by the collateral activity, it is also possible that the collateral activity may provide a response competitor which serves to disrupt perseverative barpressing. According to this view, the DRL deficit may be due to an inability to inhibit responding. A related possibility is that the DRL deficit is due to a perseveration of response set or strategy, carried over from original CRF training. This latter argument suggests that subjects with cerebellar injury may be capable of performing well on the DRL schedule, but would acquire the schedule more slowly than controls on transfer from a CRF schedule

The latter hypothesis may suggest that cerebellar lesions would have nominal effects in subjects that were well trained on the task prior to receiving their injuries. To test this hypothesis directly, control subjects from Experiment 1 were subsequently given paleocerebellar (fastigial) lesions and then retested on the DRL task. In addition, previously lesioned animals were again tested on the DRL task to assess the effects of long recovery times and extended training.

## Method

Upon completion of behavioral testing, the 37 control subjects from Experiment 1 were paired on the basis of previous performance. Fifteen subjects were given paleocerebellar lesions; the remaining subjects were sham-operated according to the procedures described in Experiment 1. After 21 days of postoperative recovery, the subjects were reduced to 85% of normal body weight and were maintained at this level for the remainder of behavioral testing. The subjects were then given 12 additional test sessions on the DRL task using the procedures and apparatus described in Experiment 1.

Upon completion of all behavioral testing, the experimental subjects were sacrificed and prepared for histological examination using the procedures outlined in Experiment 1.

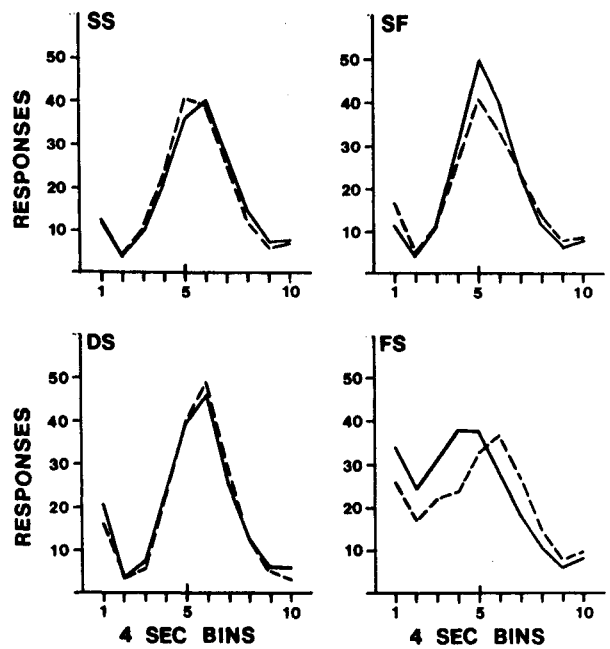


**Figure 4. Distributions of interresponse times (IRTs) for Weeks 4 (solid lines) and 10 (dashed lines) of DRL training, for sham-operated controls (SS) and for subjects with dentate lesions (DS), for subjects with fastigial lesions (FS), and for subjects that received fastigial lesions after 4 weeks of operant training (SF).**

## Results

**Histological results.** Histological examination revealed that the anterior paleocerebellar lesions the experimental subjects received were comparable in size and loci to those of the vermal and fastigial groups described in Experiment 1 and illustrated in Figure 1. Consequently, these lesions are not illustrated here.

**Behavioral results.** Upon reintroduction to the test chambers, all subjects attained efficiency ratios equivalent to those reported at the end of Experiment 1, but showed an increase in response rate [$F(1,63) = 9.194$, $p = .003$; see Figure 3]. Consistent with the findings in Experiment 1, lesion-related differences in response levels persisted [$F(3,63) = 5.796$, $p = .001$], due to the high response levels of subjects that received paleocerebellar lesions prior to operant training (Newman-Keuls test on differences between all pairs of means $p > .05$). As illustrated in Figures 2 and 3, however, all subjects continued to show increases in efficiency [$F(1,63) = 59.275$, $p < .001$] with concomitant reductions in responses [$F(1,63) = 18.177$, $p < .001$]. With additional testing, significant group differences in efficiency disappeared [$F(3,63) = .103$, $p > .05$]. Although efficiency ratios of lesioned animals ultimately approached those of control subjects, inspection of the IRT distributions for the 6th week of DRL training (see Figure 4) revealed that the subjects that received cerebellar injuries prior to DRL training continued to show abnormal IRT distributions and to emit more responses overall (see Figure 3).

In contrast to these results, the subjects that received cerebellar lesions following DRL training did not differ from sham-operated controls in efficiency [$F(1,35) = .344$, $p > .05$], response rate [$F(1,35) = .228$, $p > .05$], or IRT distribution [$F(1,35) = .628$, $p > .05$; see Figures 2 and 3]. These data suggest that DRL performance after cerebellar lesions is partially recoverable, and that preoperative training may offer some protection against the effects of subsequent cerebellar lesions.

## Discussion

The results of this experiment indicate that preoperative training greatly reduces the effects of subsequent paleocerebellar lesions. Moreover, subjects that have received cerebellar lesions prior to training do improve in efficiency following a protracted break and additional testing. However, although efficiency ratios improve with extended training, animals without preoperative training continued to show elevated response rates and abnormal IRT patterns after extended operant training. Thus, their improvement appears to reflect a uniform decrease in responding rather than selective inhibition of responses early within the schedule interval, which is characteristic of intact subjects.

The high response rates shown by subjects with cerebellar lesions do not appear to reflect a global deficit in inhibition of motor responses. If it did, one would expect a comparable deficit in subjects given preoperative training. Rather, the present results are more consistent with the hypothesis that cerebellar lesions result in a deficiency in the ability to alter a response set or strategy. Consequently, animals with cerebellar lesions continue to respond in a manner inappropriate to the DRL schedule.

# EXPERIMENT 3

Results from the previous experiments suggest that the DRL deficit seen following paleocerebellar lesions results from a perseverative increase in responding, especially within the early phases of the schedule interval. Preoperative DRL training permits animals with paleocerebellar lesions to perform normally on a DRL task, withholding responses within the early phases of the schedule interval. In addition, these results suggest that this deficit is not reflective of a global disruption of the ability to suppress responding, since animals trained on the DRL task prior to cerebellar injury perform normally. It is not clear, however, whether this deficit results from a timing deficiency or from the perseverative use of a response strategy acquired during CRF pretraining.

To investigate these possibilities directly, animals with paleocerebellar (fastigial) lesions and sham-operated controls were trained upon either a DRL or a fixed-interval (FI) schedule. Both of these tasks permit a test of timing ability, but each requires a different response strategy for optimal performance. Thus, if lesioned subjects suffered timing deficits, they would demonstrate not only poor DRL performance, but impairment on the FI task as well. In addition, shifting subjects from one schedule to the other would permit an assessment of potential perseveration of response strategies.

## Method

**Subjects.** The subjects were 24 male albino rats (90-120 days of age) obtained from Charles River or bred in the laboratory from the same strain of animals. The subjects were group-housed and maintained under a 12-h light/dark cycle with ad-lib food and water.

**Procedure.** Twelve animals were given cerebellar lesions, and the remaining subjects were sham-operated according to the procedures outlined in Experiment 1. The subjects were reduced to 85% of normal body weight following 21 days of postoperative recovery, and were maintained at this level for the remainder of behavioral testing. The training and test sessions were 1 h in length. The subjects were trained to barpress for appetitive reinforcement using the apparatus and according to the procedures described in Experiment 1. After the subjects had acquired the operant response and earned 100 reinforcers on a CRF schedule, they were shifted to either a DRL or a FI 5-sec schedule. Both schedules provide a test of a subject's ability to accurately time a specified interval; the DRL schedule, however, specifically requires that subjects withhold responding for the duration of the schedule interval. After the subjects earned 10 reinforcers, the schedule interval was progressively increased until either a DRL or a FI 20-sec schedule was attained. Behavioral testing continued for 24 additional test sessions (4 weeks), after which the subjects were again permitted ad-lib access to food. To permit direct comparisons between subjects in the present experiment and those in the previous experiments, all subjects were sham-operated according to the procedures outlined in Experiment 1. Following 21 days of postoperative recovery, the subjects were again reduced to 85% of their ad-lib body weight. They were then reintroduced to the testing chambers and given 12 test ses-

sions on the alternate schedule. Total responses emitted, reinforcers earned, and interresponse times were recorded for each test session.

Following the completion of all behavioral testing, the subjects were sacrificed and prepared for histological examination according to the procedures described in Experiment 1.

## Results

**Histological results.** Experimental subjects were found to have received lesions of the anterior paleocerebellum that were indistinguishable from those of the vermal/fastigial group described in Experiment 1 and illustrated in Figure 1. Consequently, these lesions are not illustrated here.

**Behavioral results.** All subjects readily acquired the barpress response for appetitive reinforcement, requiring an average of three test sessions to learn the operant and earn 100 reinforcers on a CRF schedule. Inspection of Figure 5 reveals several striking differences between the schedules and the order in which they are experienced. In general, both lesioned and control subjects responded at much higher rates on an FI schedule than on a DRL schedule. It is interesting to note that although lesioned animals, regardless of schedule order, made more responses than did the controls on the DRL schedule [$t(22) = 1.727$, $p < .05$], they tended to respond less than intact subjects on the FI schedule. Moreover, although previous FI experience does not obviously affect subsequent levels of responding upon DRL, the reverse does not appear to be the case. Lesioned animals continue to emit low rates of responding when shifted from DRL to FI. These data are further confirmation of the results obtained in Experiment 1; when lesioned animals received initial training on the DRL schedule, they performed poorly. In contrast, when animals with such lesions were initially trained on FI and then switched to the DRL schedule, their performance was similar to that of intact animals with the same operant history.

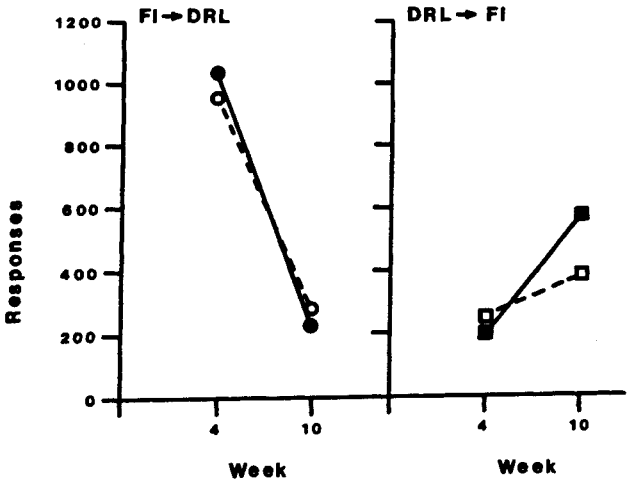


**Figure 5. Response measures for subjects with fastigial lesions (dashed lines) and for sham-operated controls (solid lines) on fixed-interval (FI) and differential reinforcement of low rates (DRL) schedules. Symbols denote order of schedule presentation: FI to DRL (circles; left panel) and DRL to FI (squares; right panel).**

It was postulated above that the poor performance of animals with paleocerebellar lesions might be due to a deficit in timing ability. The timing ability of subjects with cerebellar lesions on FI, however, appears good. Lesioned animals did not differ from normals in either median response time (17.6 sec for lesioned animals vs. 17.5 sec for controls) or in the dispersion of responses as indicated by the kurtosis of the response distributions (3.44 vs. 3.88). A three-way ANOVA (surgery × order × schedule) on median response times confirmed this observation [$F(1,20) = 3.414$, $p > .05$]. A significant interaction between surgical and schedule factors [$F(1,20) = 4.421$, $p > .046$], however, indicates that the operant deficits of animals with paleocerebellar lesions were restricted to the DRL schedule (14.6 sec for lesioned subjects vs. 19 sec for controls). These findings suggest that the DRL deficit following cerebellar lesions is not due to a global deficit in timing ability per se.

As suggested above, it is possible that perseveration might account for lesion-related differences in DRL performance. The results of the present study support the conclusion from Experiment 2 that this perseveration does not result from a general deficit in motor inhibition. If such perseveration were due to a global motoric deficit, one would expect animals with cerebellar injuries to consistently emit more responses than controls. A three-way ANOVA on responses, however, failed to reveal any such surgical effect [$F(1,20) = .347$, $p > .05$]. Moreover, as is apparent in Figure 5, subjects with cerebellar injuries showed lower response rates on the FI schedule than did normal animals. Furthermore, lesioned animals that were initially trained on the DRL task emitted fewer responses on the subsequent FI task than did either normal animals or lesioned animals initially trained on the FI schedule. Although the efficiency ratio is not conventionally employed for measuring FI performance, it does provide a means of estimating the effects of the punishment contingency upon response rate. A three-way ANOVA on this measure confirmed that previous DRL experience resulted in more efficient FI performance [$F(1,20) = 7.893$, $p = .01$]. These findings support the view that DRL deficits following cerebellar injuries are due to perseveration of response strategies rather than to a global deficit in response inhibition.

The ability of animals with cerebellar lesions to perform well on a FI but not a DRL schedule is clearly reflected in the distribution of responses within the schedule interval (see Figure 6). Furthermore, the sharp and appropriately timed response peaks evident in these distributions for the FI schedule argue against the hypothesis that cerebellar injuries result in timing deficits. In view of the strong order effect revealed above, initial analyses on the response distribution data were performed separately. A two-way ANOVA performed upon these data for subjects that had received initial FI training confirmed that they responded differentially during sched-

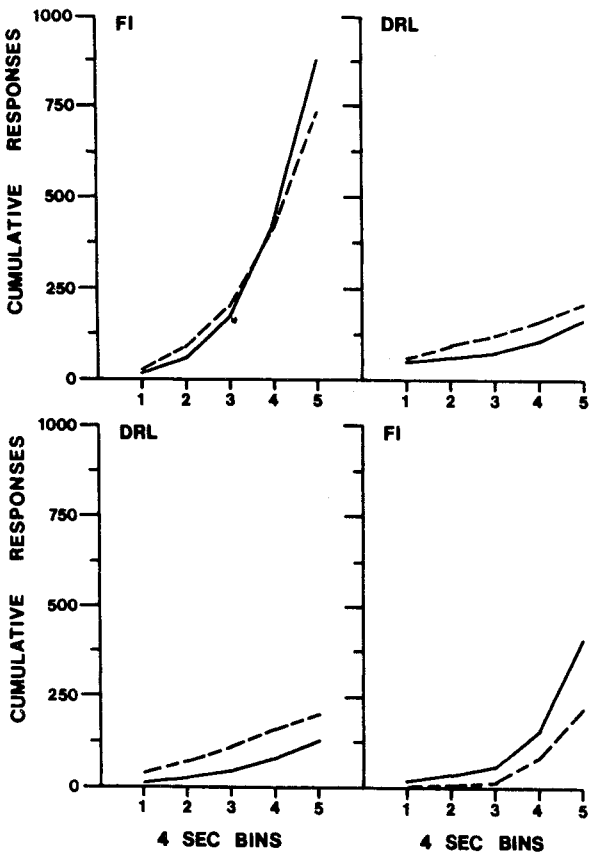


**Figure 6. Cumulative distributions of responses within the schedule interval for subjects with fastigial lesions (dashed lines) and for sham-operated controls (solid lines) for Weeks 4 and 10 of behavioral testing on fixed-interval (FI) and differential reinforcement of low rates (DRL) schedules.**

ule intervals [$F(9,108) = 32.203$, $df = 9,108$, $p < .001$]; there were significant differences between distributions for the schedules [$F(1,12) = 30.103$, $p < .001$] and a strong interaction between schedule and the temporal distribution of responses [$F(9,108) = 26.672$, $p < .001$]. Again, a similar pattern was found for subjects with initial training on the DRL schedule. The subjects distributed their responses in accordance with the temporal dynamics of the schedules [$F(9,108) = 19,275$, $p < .001$] and again responded differently on the two schedules [$F(1,12) = 9.015$, $p = .016$]. Once again, there was a strong interaction between the schedule and temporal factors [$F(9,108) = 10.084$, $p < .001$].

To more fully contrast the effects of order and schedule, response distributions were transformed to cumulative responses and a suppression index, designed to provide a quantitative measure of departure from a uniform response rate throughout the schedule interval, was calculated (Fry, Kelleher, & Cook, 1960). This transformation of the data tends to reduce the effects of higher response rates within the earliest portion of the schedule interval, permitting a more direct comparison of selective response suppression within the interval across schedules. A three-way ANOVA on the suppression indices revealed that there were significant differences between the response patterns of intact and lesioned subjects [$F(1,20) = 4.353$, $p = .047$] and confirmed differences between the schedules [$F(1,20) = 95.003$, $p < .001$]. Moreover, as shown above, the order of schedule presentation [$F(1,20) = 8.647$, $p = .008$] was found to affect response distributions, reflecting the only modest increase in responding late in the interval on the FI schedule following DRL training. Furthermore, an interaction between these effects [$F(1,80) = 4.29$, $p = .049$] suggests that lesioned animals may not switch schedules as readily as the overall response measures indicate.

### Discussion

It has been postulated that the DRL deficit seen following cerebellar injury may result from poor timing ability or an inability to inhibit overresponding, possibly reflecting some underlying motoric dysfunction. The performance of lesioned animals on a FI schedule clearly demonstrates that they are capable of accurately judging the schedule interval. If cerebellar injuries resulted in a timing deficit, one would expect either a shift in the response distribution toward shorter intervals or a flattening of the peak in the response distribution. The results in the present study failed to reveal any differences in FI performance of the distribution of responses between lesioned and control subjects when they were initially trained on this task.

One of the characteristic features of the DRL deficit is an increased number of responses. The absence of overresponding on the FI schedule, however, indicates that the DRL deficit is not reflective merely of a global deficit in response inhibition.

## GENERAL DISCUSSION

Lesions of the rostral vermis and/or fastigial nuclei produced a marked performance deficit when subjects were subsequently tested on a DRL 20-sec schedule. This deficit was characterized by an increase in response rate sufficient to preclude effective performance. Furthermore, lesioned animals not only emitted more responses than intact subjects, but also demonstrated abnormalities in the temporal patterning of their responses. This finding confirms the previous report of such deficits following cerebellar injuries (Kirk et al., 1982), and is consistent with a wider body of data indicative of cerebellar involvement in the elaboration and organization of behavior (Berntson & Torello, 1982; Watson, 1978b). In contrast, lesions of the dentate nuclei did not produce any appreciable alterations in performance. Such findings may be reflective of the rostral projections of the fastigial nucleus, including connections to a variety of limbic system and forebrain structures: amygdala, hypothalamus, septal area, hippocampus, and thalamus (Anand et al., 1959; Angaut & Bowsher, 1970; Harper & Heath, 1973; Heath, Dempsey, Fontana, & Meyers, 1978; Heath & Harper,

1974; Whiteside & Snider, 1953), or of less direct connections via the ventral tegmental area to divergent basal forebrain structures (Crutcher & Humbertson, 1978; Jacobowitz & MacLean, 1978; Snider, 1975; Snider & Maiti, 1976; Snider et al., 1976).

Paleocerebellar lesions did not prevent the ultimate development of efficient DRL performance. With extended training on the DRL task, lesioned animals were able to reduce their excessive response rates sufficiently to perform at levels approximating normal performance. In spite of these improvements in performance, there remained a characteristic residual disturbance in number and distribution of responses within the schedule interval.

In contrast, operant performance on a FI schedule was unimpaired, and lesioned animals showed a greater efficiency than did normals with a similar operant history on this schedule. The lower number of responses emitted by lesioned animals on the FI task, together with the accuracy of their timing performance, indicates that cerebellar lesions do not produce a global deficit in timing ability or a pervasive inability to inhibit responding. Following extensive FI training, control subjects emitted responses on the DRL task at a level similar to that of subjects with fastigial lesions with only brief exposure to CRF and the progressive DRL training schedule. These data suggest that paleocerebellar injuries may affect the ability to alter response strategies, resulting in the perseverative intrusion of a response set developed during prior training (i.e., CRF). This suggestion is consistent with the finding that previous experiences on the DRL task provides some protection against the DRL deficit seen following cerebellar lesions. Moreover, when subjects with fastigial lesions were shifted from the DRL to the FI schedule, they behaved as if the more restrictive DRL schedule was still operative. Thus, previous experience with a schedule that specifically punishes high response rates results in a continued lower rate of responding. This effect is most apparent in the almost complete suppression of responses within the early phases of the FI schedule (see Figure 6) by lesioned animals following initial DRL training. At present, the most plausible explanation of the DRL deficit appears to be based on an inability to adequately suppress responses within the early phases of the schedule interval, related in part to an impaired ability to switch response strategies. Preoperative training would permit subjects to acquire an appropriate response strategy prior to cerebellar injury. These animals need only to emit previously learned behaviors in order to perform well on the schedule.

The pattern of results presented here is consistent with that found in previous reports describing deficits on a number of behavioral tasks related to lesions of the cerebellum (Berntson & Torello, 1982; Watson, 1978b). Moreover, a reexamination of these results in light of the present findings suggests that perseveration of response strategies may account for many of these deficits. Pellegrino and Altman (1979) reported a deficit in maze learning when subjects were required to alternate left and right turns. Although both experimental and control subjects showed good acquisition of an initial maze task, lesioned animals were demonstrably impaired when required to shift response strategies to perform a subsequent alternation task. Furthermore, perseveration of response strategy is consistent with reports that animals with cerebellar lesions demonstrate impaired extinction of a visual discrimination task (Rubia, Angermeier, Davis, & Watkins, 1969; Davis et al., 1970).

In summary, the behavioral data presented support a growing recognition in the literature that the concept of cerebellar functioning should be expanded to include the elaboration and sequential organization not only of motor acts, but also of more complex behaviors as well.

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