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EFFECTS OF SPATIAL AND NONSPATIAL DISTRACTORS ON PERFORMANCE LATENCY OF MONKEYS WITH FRONTAL LESIONS[1]

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Rhesus monkeys with lesions of the dorsolateral frontal cortex were tested in a situation where a behavioral task was occasionally interrupted by the presentation of visual or auditory distractors. The increase in response latency due to the distractor was greater for the subjects with frontal lesions than for normal controls. The increased distractibility is due to an increase in the duration of distraction-evoked behavior and, in the case of the spatial distractors, also to an increase in the probability that the distracting input will be sampled. "Behavioral habituation" (decrease in distraction duration) took place at the same rate in both groups, although the asymptotic levels eventually reached might differ under appropriate conditions. The subjects with frontal lesions, even more than their controls, were found to be more sensitive to variations in the spatial location of the distractor than to changes in the visual aspect of the cues.

Evidence has accumulated to show that frontal lesions affect the orienting reaction in monkey and man. Specifically, the GSR component of orienting is depressed while behavioral orienting responses (Luria, Pribram, & Homskaya, 1964) and EEG arousal (Grueninger, Kimble, Grueninger, & Levine, 1965) continue to occur. These findings led Kimble, Bagshaw, and Pribram (1965) to suggest that habituation depends on the evocation of the autonomic components of the reaction, a suggestion supported by a more extensive analysis (Bagshaw & Benzies, 1968). Thus, a plausible and testable explanation for the frontal primate's heightened responsiveness to novelty (Pribram, 1960), i.e., its distractibility, is provided.

Recently another issue concerning frontal lobe function has come to a head. Monkeys with frontal lesions are able to perform a nonspatial object alternation somewhat better than they are able to perform the classical spatial-alternation task, though by no means as well as do control *Ss* (Mishkin & Pribram, 1955). These findings have led to the conclusion that frontal monkeys are deficient in processing spatial cues. Objections can be leveled against this conclusion, however. For example, one study (Mishkin, Vest, Waxler, & Rosvold, 1966) comes to the spatial interpretation on the basis of results practically identical to those obtained in an earlier study (Pribram, 1961) which reached the opposite conclusion.

While the present authors were puzzling about a way to resolve this discrepancy in interpretation, a technique was devised and utilized in the laboratory to investigate the distractibility of hippocampectomized monkeys (Douglas & Pribram, 1969). An interesting result of this study was the fact that operated *Ss* reacted differently to spatial and to nonspatial distractors. The authors immediately realized that this observation provided a technique that might possibly help to decide whether monkeys with dorsolateral frontal lesions could indeed process spatial cues and whether this processing was different in any respect from that of the normal primate. An experiment was therefore designed which would provide quantitative information concerning the role of the primate frontal cortex in distractibility, behavioral habituation, and the processing of spatial cues.

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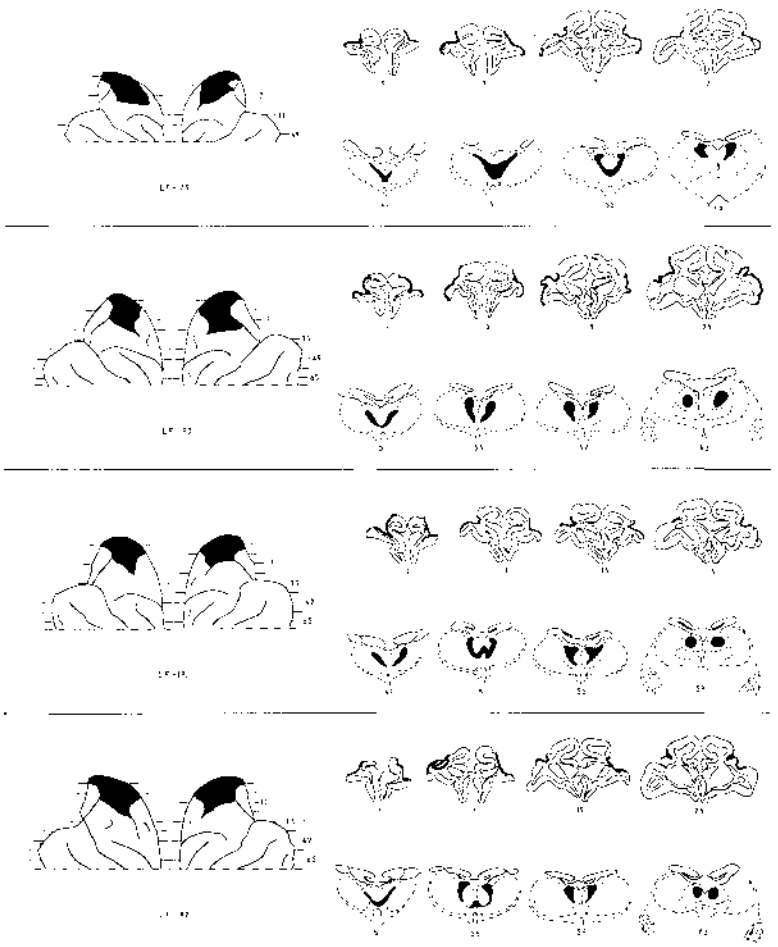

FIG. 1. Reconstruction and cross sections of frontal lesions and of resulting thalamic degeneration.

METHOD

Subjects, Surgery, and Histology

Subjects were nine preadolescent rhesus monkeys. Five of these had been subjected to bilateral subpial resection of the dorsolateral frontal cortex some 3 yr. earlier; the other four served as controls. All had been tested on a variety of problem-solving tasks and had also been subjects in studies measuring autonomic indicators of orienting and habituation. Histological analyses were performed according to the technique of Sherer and Pribram (1962). Essentially these show bilaterally symmetrical lesions of the dorsolateral frontal cortex extending forward from the anterior bank of the arculate sulcus, excising the pole and involving the lip of the frontal lobe. Analysis of thalamic degeneration suggests that fibers from the orbital portion of the frontal lobe were also interrupted, since a good deal of retrograde gliosis and depletion of neurons is seen in the midline and medial intralaminar nuclei (Chow & Pribram, 1956).

Apparatus

The apparatus used for testing was a modification of that described by Pribram, Gardner, Pressman, and Bagshaw (1962) differing mainly in that stimulus presentation and recording were accomplished by means of a PDP-8 rather than a special-purpose computer. The display consisted of a 4 × 4 array of 16 depressable panels upon which stimuli could be projected from the rear. A food cup was located below the display. For purposes of programming each panel was assigned a letter, as can be seen below.

A B C D
E F G H
I J K L
M N O P

The panels were on one of the walls of the testing enclosure, the top two rows at eye level. Illumination was provided by an overhead incandescent light. Subjects were watched by *E* through a one-way glass which made up another wall of the enclosure.

Procedure

At each session a few minutes were allowed for "adjustment," following which the testing program began. A trial was initiated by the illumination of Panel P. Pressure on the panel resulted in the darkening of Panel P and the immediate illumination of Panel A. In turn, a press on this panel turned it off, released a banana pellet, and terminated the trial. After a lapse of 10 sec. a new trial would begin, an identical procedure being repeated on all trials. Latencies (to .001 sec.) between the presentation of the first stimulus and the first response, and also between responses to the first and second stimuli, were automatically recorded. Only the latter, the interresponse latencies, are considered in this paper. All *S*s were originally trained on this response sequence (with no distractors presented) until they met the criterion of a day's run of 50 trials completed within less than 10 min. (5–8 days of training).

Similar procedures were used in all distraction tests. In every case *S*s were given daily sessions of 50 total trials each, with 4 distraction trials intermixed among the regular trials. The distraction trials were presented in a pseudo-random fashion such that they were separated by at least 5, but not more than 10, regular trials, and no distraction trial occurred earlier than the eleventh trial of a day's run. The procedure used on a distraction trial was similar to that occurring on regular trials except that the press of Panel P resulted in the appearance of a distracting stimulus *simultaneously* with the appearance of the stimulus on Panel A. When the distractor was a symbol, *S* could depress the panel upon which it was displayed and these responses were recorded. However, a response to the distractor did not result in a reward nor in any change in the situation. A trial was terminated only when Panel A was pressed, at which time both it and the distracting stimulus disappeared and reward was delivered. The distraction conditions were given in the order in which they are described.

Condition 1 (Stimulus Varied, Location Constant)

This task was designed to test *S*'s sensitivity to variation in a stimulus pattern presented at a specific spatial location and *S*'s ability to habituate to distraction in this location despite the variations in stimulus patterns. Eight abstract patterns were used as distractors. On each of the four distraction trials of a day's run one of these patterns appeared in Panel Location F. No pattern was repeated until all had been used. The sequence was shuffled so that no pattern appeared twice on the same or on successive days. Subjects were tested under this condition for 4 successive days.

Condition 2 (Stimulus Constant, Location Varied)

This task was designed to test *S*'s sensitivity to the distractor's location in space and to test *S*'s ability to habituate to or "gate out" a specific stimulus regardless of its location. In this condition, one hitherto unused pattern was presented in each of eight spatial locations. The location used in Condition 1 was omitted. The same panel was never used twice on the same or on 2 successive days. All eight locations occurred twice in the 4-day series.

Condition 2′ (Stimulus Constant, Location Constant)

To ascertain whether the results in Condition 2 might have been the result of some unusual property of the stimulus used, rather than of the spatial variable under investigation, the same pattern used in Condition 2 was presented on four successive distraction trials of the same day in Panel Location B.

Condition S (Buzzer)

A buzzer was used as a distractor instead of the patterns projected on panels. Otherwise, the procedure was the same as in Conditions 1 and 2 above.

Results and Discussion

"Distraction duration" was considered to be the difference between the interresponse latency on a distraction trial and *S*'s median interresponse latency on nondistraction trials for that day. The first 10 trials of each day were omitted to decrease warm-up effect, and the median was chosen instead of the mean to avoid undue influence from occasional unintentional distractions (e.g., *S*'s dropping of a pellet). The group mean distraction durations recorded under Conditions 1, 2, and 3 are presented graphically in Figure 2.

As a check, the raw interresponse latencies on distraction trials were also analyzed. The findings were essentially the same as those presented. The overall average interresponse latencies on nondistraction trials were .537, .463, .441, .857, and .583 sec. for the five *S*s with frontal lesions and .565, .975, .528, and .366 sec. for the four normal controls. Thus, the median interresponse latencies of the two groups did not differ in the absence of distractors ($U = 10$, maximum possible overlap for groups of this size).

The Group × Day × Subject (pseudo three-way) analyses of variance indicated that the mean distraction durations recorded from the frontally ablated groups were significantly higher than those of the

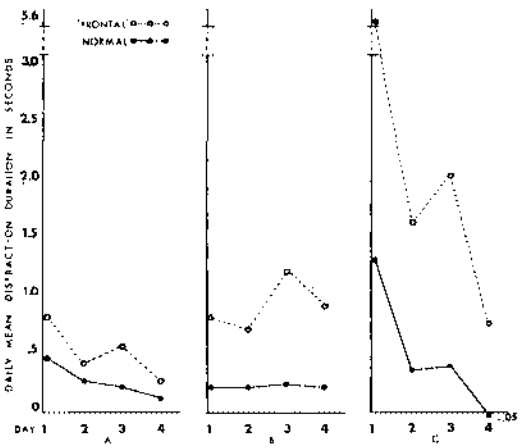

FIG. 2. Daily mean distraction duration (mean distraction trial latency minus median latency) for (*a*) Condition 1—stimulus varied, location constant; (*b*) Condition 2—location varied, stimulus constant; and (*c*) Condition 3—buzzer.

normal group under all experimental conditions ($F = 8.3$, $df = 1/7$, $p < .05$; $F = 5.9$, $df = 1/7$, $p < .05$; $F = 47.5$, $df = 1/6$, $p < .001$; for Conditions 1, 2, and 3, respectively). When *Ss*' mean distraction durations for each condition were ranked, there was only a single overlap, which occurred in Condition 1. The only other significant effect in these analyses was that of days (behavioral habituation) in Conditions 1 and 3 ($F = 4.5$, $df = 3/21$, $p < .05$; $F = 6.7$, $df = 3/18$, $p < .05$, respectively). The Group × Day interaction for the three conditions gave F values approximating unity.

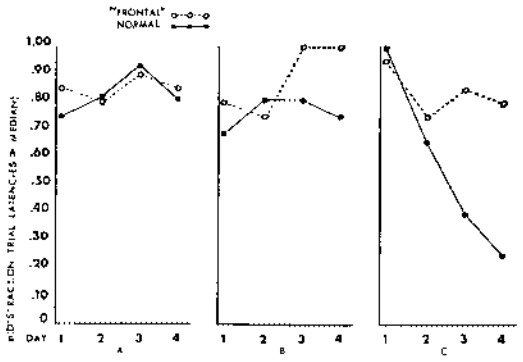

FIG. 3. Proportion of distraction trial latencies greater than *S*'s median latency for (*a*) Condition 1—stimulus varied, location constant; (*b*) Condition 2—location varied, stimulus constant; and (*c*) Condition 3—buzzer.

The results are further analyzed as they specifically apply to four views of the deficit produced by this lesion, i.e., increased distractibility (Pribram, 1961), failure to habituate (Luria et al., 1964), difficulty in responding to spatial cues (Mishkin et al., 1966), and inability to inhibit responses (Brutkowski, Mishkin, & Rosvold, 1963). In the interests of clarity, the results will be discussed as they are presented.

Distractibility

The increase in response time which "mean distraction duration" represents could have been brought about by either or both of two underlying factors. The *Ss* with lesions may have been distracted by a greater proportion of the distractors, or they may have recovered more slowly whenever they were distracted. The proportion of latencies on the distraction trials which were greater than *S*'s median normal trial latency provides some insight into this problem. If the distractors had no effect whatever, one would expect this statistic to have an average value of .50, while any delay the distractor might produce should bias this indicator toward 1.00. Through examination of this proportion (Figure 3A), one finds that under Condition 1 the two groups are essentially identical in the consistency of their distraction. A Mann-Whitney U test for overall group difference yields a U value of 9.5, where 10 is the value for the maximum theoretically possible overlapping of the two sets of scores. This means that under this condition all of the difference observed in the mean distraction durations must have come from a slower recovery on the part of the frontally ablated animals. The results from the first 2 days of Condition 2 (Figure 3B) confirm this finding ($U = 10$). In the third condition (Figure 3C) a different situation prevailed. The loud buzzer placed at the back of the cage proved a very strong distractor. However, with experience, the control animals, at least, were no longer "distracted" so much as "galvanized" by the occurrence of this stimulus. The frontal *Ss* seemed more prone to orient toward the back of the cage when the buzzer sounded, whereas the control ani-

mals often ducked or lunged more vigorously than usual at the second task stimulus. The end effect on the normal group, at least, would appear somewhat equivalent to "stimulus-intensity dynamism." For this group the stimulus still evoked a response, but the response was directed toward the rewarded task instead of the distractor.

Behavioral Habituation

Analysis of variance confirms the apparent decrease in distraction durations through the course of Conditions 1 and 3. Since the Group × Day interaction in the two conditions was negligible, it is apparent that the frontally ablated group showed behavioral habituation at the same rate as the normal animals. Of course, the higher initial values of the frontally ablated animals would result in their requiring a greater number of days for the mean distraction duration to reach some specified level.

A comparison of the graphs of distraction duration and of the proportion of distraction trials greater than *S*'s median latency for Condition 1 (stimulus varied, location constant) proved interesting. It shows that the apparent "habituation" observed in *both* groups is not due to a decrease in the probability that a given distractor will produce an effect. The habituation would seem to be the result of faster recovery rather than gating out or increasing insensitivity to the distractors. This increase in behavioral efficiency, as opposed to desensitization or gating of the input, was very evident in Condition 3 (buzzer). In this condition both groups showed a significant decrease in distraction duration. The normal *Ss*' distraction duration and proportion of distraction trial latencies greater than median both sank below the level expected if no distractor were present. With respect to the latter statistic, Figure 3C clearly shows the sharp contrast between the trends of the two groups ($U = 0$, $p = .036$). The absence of a consistent trend in the scores of the frontally ablated monkeys strongly suggests that these *Ss* would never show the paradoxical "negative distraction" observed in the control subjects in the presence of this strong distractor.

Spatial Deficit

It was predicted that if the frontally ablated animals were deficient in registering spatial information, they would have much greater difficulty habituating under the first condition (stimulus varied, location constant) than would the normal controls. This should have been the case since the distractors differed widely in pattern, but were all consistent in location. However, the rate of behavioral habituation proved to be the same for both groups. If the animals with lesions were less able to process and record the location of the distractor, they might also have shown relatively rapid habituation under Condition 2 (stimulus constant, location varied). On the contrary, the frontally operated monkeys were even more disrupted throughout this condition than in the first. Ranking *Ss* on the basis of the difference between their distraction durations under Conditions 1 and 2 showed no overlap between groups ($U = 0$, $p = .016$). Although neither group habituated under Condition 2, Figure 3B shows that repetition actually heightened the distractor's effectiveness upon *Ss* with lesions. Under Condition 2′, where the stimulus used in Condition 2 was presented only in Location B, the mean distraction duration of the frontal operates approximated the low level of the normal group by the fourth presentation. Thus, although *Ss* with dorsolateral-frontal lesions proved capable of learning to minimize the disruptive effect of varied patterns appearing in one specific spatial location, the distraction produced by a single pattern could be maintained for the animals by simply shifting its spatial location. It might be said that with regard to distraction, the frontally ablated animals were more, not less, sensitive to spatial location than were the normal controls.

In order to examine the present results in terms of the spatial aspects of the "cognitive field," the results of Condition 2 were analyzed with respect to location instead of experimental day. The mean distraction durations obtained for the various locations

TABLE 1

CONDITION 2: MEAN DISTRACTION DURATION IN SECONDS AS A FUNCTION OF DISTRACTOR LOCATION IN PANEL MATRIX

Task Stimulus 2	.595 .217	1.524 0.207	.345 .179
.976 .315		.500 .116	.745 .203
	1.497 0.318	1.209 0.290	
			Task Stimulus 1

Note.—Boldface = frontal; lightface = normal.

used are presented in Table 1. The location effect was significant ($F = 2.4$, $df = 7/49$, $p < .05$), as might be expected, but the value of the F ratio for the Group × Location interaction was only 1.351. Thus, there would appear to be no difference between the two groups with respect to which locations proved most effective. The frontally operated monkeys were not at all immune to the distractors which were at greater distances from the task stimuli.

In summary, under the present experimental conditions, the frontal lesion increased, not decreased, the sensitivity of the animal to the distractor's location. This was true regardless of where the distractor appeared.

"Inhibitory" Deficit

Although the pressing of the distractor panel was never rewarded, a record was kept of all such presses. With four distraction trials per day *Ss* with frontal lesions averaged 1.6, .2, 16, and 0 presses for the 4 days of Condition 1, and .8, 1.2, 1.8, and .8 presses for the 4 days of Condition 2. The only press recorded from a normal subject occurred on Day 1 of Condition 1, making the group average for that day .25 presses. In contrast, if we combine both conditions we find that no frontally ablated *S* pressed less than three distractors. This yields a U of 0 and $p = .016$.

The analysis might suggest that the increased distraction durations recorded for the frontally ablated animals were the result of time wasted in pressing of unrewarded stimuli. However, this is not the case. When *Ss'* overall averages were calculated, omitting all trials on which a press occurred, there was still only one overlap between the two groups ($U = 1$, $p = .032$). Therefore, not all of the frontally ablated animals' increase in distraction duration can be attributed to the time wasted by actually pressing unrewarded panels.

In Condition 3 it was observed that the increased distraction duration of the frontally ablated group seemed largely due to their persistent orientation toward the buzzer when it sounded. In contrast, normal *Ss* eliminated such disruptive responses and actually showed an increased response speed under the influence of the "distractor."

DISCUSSION

The results of these experiments provide quantitative confirmation of increased distractibility in *Ss* with frontal lesions. The increased distractability observed is due to an increase in the duration of their distractor-evoked behavior and, in the case of the spatial distractors, also to an increase in the probability that the distracting input will be sampled. In addition, the results indicate that monkeys with dorsolateral frontal cortex resections do orient and behaviorally habituate to varied visual stimuli appearing in a specific spatial location. Such *Ss* also react strongly to changes in the spatial aspects of distracting stimuli. Further, the results indicate that for sophisticated monkeys, dorsolateral frontal ablations produce an increased sensitivity to distraction produced by changes in the spatial location of cues when compared (*a*) with control *Ss* and (*b*) with visual distractors which do not vary in location. The question is raised, therefore, whether the impairments shown by monkeys with dorsolateral frontal lesions on spatial tasks are due to the marked dis-

tracting effect that spatial novelty has for these *Ss*.

The same question is raised by the results in Condition 3 (buzzer) with regard to those experiments which indicate the possibility of an auditory deficit in frontally ablated *Ss*. In such experiments care would have to be taken to assure that stimulus-oriented behavior would not handicap discrimination performance.

These results can be explained by the assumption that various response tendencies or plans (Miller, Galanter, & Pribram, 1960) are constantly competing with each other for control of behavior, and that only one such plan can control behavior at any time. If activity of the frontal cortex maintains the competitive advantage of any plan already in the process of execution until it reaches completion, the behavioral results observed in the present experiments and in others would follow. In the present experiments the correct response was uniquely specified by the task environment. Were the correct response which follows the distraction ambiguously specified (i.e., variable over time) as in delayed alternation or in delayed response (Pribram, 1961), one would expect a breakdown in performance.

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