Effects of Posterior Parietal and Frontal Neocortical Lesions in the Squirrel Monkey

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Brightness discrimination, three forms of a spatial pattern discrimination in which the essential cue and site of reinforcement were separated (SSP), delayed response, form discrimination, and three forms of a spatial pattern discrimination in which the essential cue and site of reinforcement were identical (ISP) were given, in the order mentioned, to two groups of squirrel monkeys with frontal or parietal cortical lesions and to an unoperated control group. Monkeys with frontal lesions were impaired on delayed response, and those with parietal lesions were impaired on form and SSP discriminations. Neither group was impaired on brightness discrimination. The results confirm and extend previous findings by other investigators that the posterior parietal cortex of nonhuman primates is critically involved in visually guided spatial discriminations when the primary cue and the site of reinforcement are separated.

Behavioral studies designed to investigate the function of the posterior parietal cortex of nonhuman primates have tended to emphasize the appearance of tactile discrimination deficits after lesions in this region (Blum, 1951; Ettlinger & Kalsbeck, 1962; Ettlinger, Morton, & Moffett, 1966; Moffett & Ettlinger, 1970). On the other hand, deficits in visual discrimination have typically been associated with lesions of the inferotemporal cortex (Blum, Chow & Pribram, 1950; Ettlinger et al., 1966; Pribram & Barry, 1956; Wilson, 1957).

The results of these studies seem to suggest some disparity in functional neuroanatomy between man and other primates. Although parietal lesions in man are reported to result in tactile deficits (e.g., astereognosis, increased threshold for two-point discrimination, decreased position sense, extinction upon bilateral simultaneous stimulation), many of the deficits that are commonly attributed to parietal dysfunction in man appear to require, at least in part, the integration of visual stimuli. Examples of such deficits are constructional apraxia, spatial disorientation or topographical agnosia, performance on perceptual intelligence tests, and judgment of distance (Bar-

low, 1970; Critchley, 1953; Piercy & Smyth, 1962; Reitan, 1966; Whitty & Newcombe, 1965).

One possible reason for the relative lack of deficits on visually mediated tests in monkeys, as opposed to man, after posterior parietal lesions is the lack of comparability of the tasks employed. Typically, the visual tasks used to test parietal monkeys have involved either two-dimensional (pattern) or three-dimensional (object) discriminations. A review of the reported effects of parietal lesions in man does not suggest that deficits on such tasks would occur in human patients. To the contrary, it has been reported (Ettlinger, Warrington, & Zangwill, 1957) that in a sample of parietal patients who were examined tachistoscopically, none evidenced any gross deficits in the perception of visual form or pattern. If one examines the nature of the deficits that occur after parietal lesions in man, it appears that many involve some type of "visual–spatial" or "spatial" impairments. In fact, deficits characteristic of parietal damage in man are frequently described as impairments of "spatial orientation" or "spatial judgment" or as "spatial agnosia," "visual–spatial difficulties" or "disorders of spatial thought."

On the basis of these data, we hypothesized that a spatial factor might be a crucial element in visual tests designed to elicit deficits in parietal animals. This hypothesis was

reinforced by two earlier studies, both of which found discrimination deficits after posterior parietal lesions in *Macaca mulatta* on tasks in which a separation between the stimulus or discriminative cue and the site of reinforcement was introduced (Bates & Ettlinger, 1960; Ettlinger & Wegener, 1958). More recently, a similar finding emerged from a study by Pohl (1973). In Pohl's work, deficits were most pronounced on spatial reversal trials or on trials in which the stimulus (landmark cue) was discontiguous with the manipulandum. The purposes of the present study were to develop tasks that might help clarify the nature of the visual–spatial deficit seen in earlier investigations and to study their effect when administered to another primate, the squirrel monkey (*Saimiri sciureus*), after lesions of the posterior parietal cortex. Monkeys with lesions of the dorsolateral prefrontal cortex were included, primarily as operated controls, but also to add to the knowledge about this much-studied neocortical area (Konorski, Teuber, & Zernicke, 1972; Warren & Akert, 1964).

METHOD

Subjects

Twelve adult male squirrel monkeys (*Saimiri sciureus*) with varying degrees of Wisconsin General Test Apparatus (WGTA) experience served as subjects. None had previous experience on the tasks used in this work. Assignment to the three groups was done in a random fashion. The groups consisted of posterior parietals (P), frontals (F), and unoperated controls (C). During the course of the experiment the monkeys were housed in individual cages and fed ad lib after each testing session. They were given fresh fruit approximately five times each week; water was always available.

Surgery

Subjects were anesthetized with sodium pentobarbital (18 mg/kg of body weight); atropine (1.8 mg/kg) was given in the same injection. With the use of a small circular saw with a guard to control the depth of the cut, it was possible to remove a bone flap unilaterally over the intended lesion site. During the operation, this bone flap was kept moist in a gauze pad soaked in sterile saline. During the sawing, care was taken to prevent overheating the underlying tissue by keeping the surface beneath the saw well saturated with sterile saline. Once the bone flap was removed, the dura was cut on three sides and retracted. The smaller blood vessels traversing the area of the exposed cortex were cauterized along the boundaries of the bone removal. Major blood vessels in the area were left intact. The exposed cortex was aspirated within the designated limits of the intended lesion. Attempts were made to remove as much cortex as possible around the areas of the intact blood vessels. After aspiration, all bleeding was arrested, the dura and bone flap were replaced, and the scalp was sutured. Furacin was applied topically to the wound. The subject was then given Mikedimide (with dosage dependent upon the depth of anesthesia at the time) and Bicillin (.25 cc). Two weeks after the initial operation, the same procedure was used to make a similar lesion in the contralateral hemisphere. Half the subjects received lesions of the left hemisphere in the first-stage operation.

Posterior parietal lesions. Since the cortex of the squirrel monkey has not been completely mapped, the lesion site was determined by a subtractive method. Benjamin and Welker (1957) have mapped the somesthetic cortex of the squirrel monkey. According to their results, the somesthetic representation on the cortical surface remains anterior to the lateral and intraparietal sulci. Thus, by selecting the level of the dorsal termination of the superior temporal sulcus as the anterior limit of our lesion, we could be relatively assured of not encroaching upon the primary somatosensory projection area.

The posterior limit of the parietal lesion was determined on the basis of electrophysiological findings (Cowey, 1964), which indicated that a small indentation on the dorsolateral convexity of the hemisphere several millimeters posterior to the intraparietal sulcus marks the rostral limit of the prestriate cortex. Thus, the posterior extent of our lesion was defined by a line between (a) a point near the midline just dorsal to the small indentation mentioned above and (b) a point on the lateral surface 2.5 mm posterior to a vertical line passing through this indentation and level with the ventral limit of the intended lesion (see Figure 1).
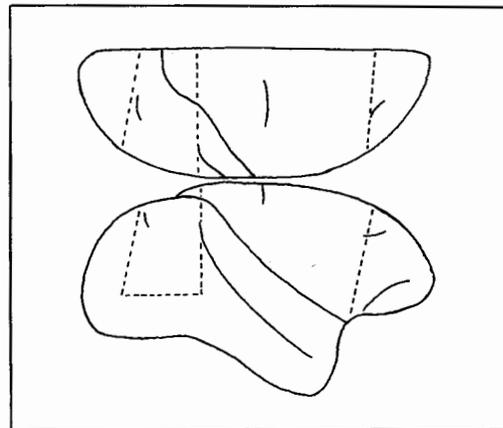

FIGURE 1. Projected sites for parietal and frontal lesions.

By the selection of these coordinates for the posterior extent of the lesion, any encroachment on Area 19 will be the result of the most liberal estimates of its location, and Area 18 should be only minimally included, if at all. It may be noted that the majority of studies dealing with posterior parietal function in rhesus monkeys have used the anterior bank of the lunate sulcus as the posterior boundary for the lesion and, hence, have probably included a large portion of Area 18 in the "parietal" ablations (see Crosby, Humphrey, & Lauer, 1962, p. 505).

Finally, the intended lesion extended dorsally nearly to the midline (i.e., as close as possible without risk of damaging the sagittal sinus) and ventrally to within approximately 8 mm of the base of the brain.

Frontal lesions. The description provided by Akert (1964) served as a guideline for the frontal lesions. Generally, the lesions encompassed the entire dorsolateral surface of the hemisphere anterior to a line between (a) a point near the midline immediately dorsal to the center of the dorsalmost of the two "grooves" (which Akert suggests "may be homologous to either sulcus arcuatus or sulcus principalis," p. 377) and (b) the point at which the lateral sulcus begins on the lateral surface of the hemisphere (see Figure 1).

Histology

After the conclusion of testing, all operated animals were sacrificed with a lethal dose of sodium pentobarbital and profused through the heart with physiological saline followed by a solution of 10% formalin. The brain was then removed, and a gross determination of the extent of the lesion was made. The brains were embedded in celloidin and sectioned coronally at 40 μm. Every fifth section was stained with thionin.

Behavioral Procedures

General behavioral assessment. By means of a rating scale developed for this purpose (available upon request), gross estimates of the subject's visual capacity, reaching accuracy, general sensorimotor ability, and level of fear were assessed on Days 3, 6, and 9 after the second operation. The subject's ability to scan his environment was assessed by a method similar to that used by Chow (1951), i.e., by having the animal retrieve one, two, or three currants presented on a board placed directly in front of the subject. The subject's scanning ability was defined in terms of (a) the latency to retrieve the first currant and (b) the total time required to retrieve all currants when more than one was presented. This measure also served as a gross index of the normality of the subject's visual fields. Another test of the subject's visual functions involved discriminating among edible and inedible objects (e.g., a currant, a metal nut, a small piece of wood, and a section of black electrical cable).

The subject's reaching and grasping abilities were assessed by presenting a currant on the end of a thin metal rod and allowing the subject to retrieve it. Ratings were also made of the subject's ability to handle its food, locomotion in its home cage, and the degree of withdrawal responses to various stimuli (e.g., the examiner and a large leather glove).

Pretraining. Pretraining and testing were done in a modified WGTA and begun 2 wk after the second-stage operation. Pretraining consisted of allowing the subject to respond consecutively to currants placed in uncovered food wells, food wells baited and covered with a grey balsa block, and finally, food wells baited and covered while hidden behind an opaque screen.

Brightness discrimination. The first training task was a brightness discrimination problem. This was one of two control tasks, i.e., tasks with which none of the groups was expected to experience abnormal difficulty. The discriminanda consisted of two plastic toy cups (4 cm in height) placed 22.3 cm apart on a gray stimulus tray. A white cup served as the positive stimulus, and a black cup as the negative. In the first two sessions (40 trials each) of this task, a correction procedure was used, but in subsequent sessions a noncorrection paradigm was followed.

The criterion for this and the remainder of the discrimination tasks was 80% correct in 40 consecutive trials. In the event of perseveration (defined as 10 consecutive responses to the same position), the opposite position was baited until two consecutive responses were made to that side. Except for this modification, Gellermann's (1933) sequences were used to determine the site of reinforcement for this and all subsequent discrimination problems. No individual problem was continued for more than 1,000 trials (25 sessions). Subjects were tested on 6 days of each week.

Spatial pattern discrimination-I. This task was conceived, in part, on the basis of the suggestion of Ettlinger et al. (1957) that "patients with visual-spatial disorders are severely limited in their ability to scan and integrate an extended situation" (p. 357). As noted earlier, the spatial separation of the stimulus cue and the site of reinforcement might be a crucial variable in obtaining certain discrimination deficits with parietal monkeys (Bates & Ettlinger, 1960; Ettlinger & Wegener, 1958; Pohl, 1973). This problem made use of wooden balls that created a discontiguous spatial array or pattern (see Figures 2 and 3). Although it could be considered that the entire pattern provided the stimulus cue, the relative position of only one element in the array was a sufficient cue for the solution of the problem, since the remaining elements were the same in the two arrays presented at a given time. This arrangement had the advantage of enabling the experimenter to present the problem in two modes: (a) one that will hereafter be identified as a separated spatial pattern (SSP), in which there was a separation between the essential stimulus cue and the site of reinforcement (see Figure 2) and (b) another that will be referred to as the identical spatial pattern (ISP), in which the essential cue
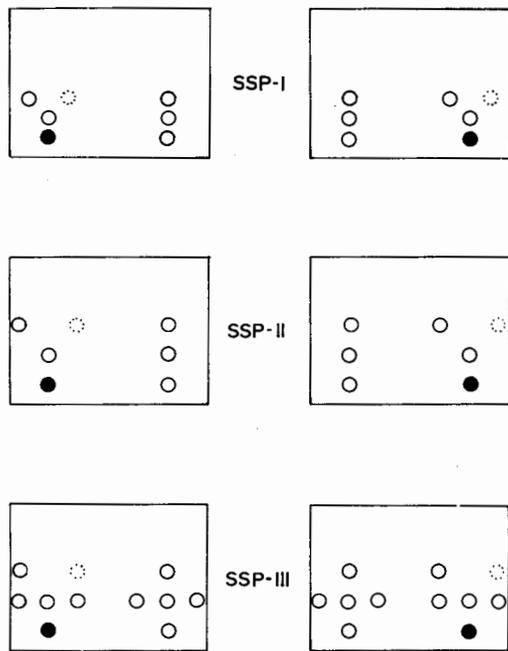
FIGURE 2. Schematic representation of the three forms of the separated spatial pattern task (SSP). (Dark circle indicates site of reinforcement.)

and the site of reinforcement were identical (see Figure 3). Deficits obtained on SSP but not on ISP would suggest that the critical variable was the separation of the essential stimulus cue and the site of reinforcement. Deficits on both SSP and ISP would indicate that the difficulties are in the spatial pattern discrimination per se.

The second task consisted of only the SSP presentation; the ISP presentation was given as the fifth task. As indicated in Figure 2, the spatial pattern problem was divided into three forms, representing three levels of difficulty as determined from pilot work. Small yellow balls, approximately 2.5 cm in diameter and flattened on each end, were used to construct the patterns. The problems were presented on a gray stimulus tray with food wells that were 22.3 cm apart.

The first form of the problem (SSP-I) consisted of two groups of three balls, each forming the two patterns seen in Figure 2. The horizontal distance between the balls was 3.5 cm center to center, with the critical ball also being displaced 3.5 cm from midline. Right/left placement of the reinforced pattern on the tray and the right/left placement of the critical cue relative to midline were based on Gellermann's (1933) schedules. The first two sessions of SSP-I used a correction procedure; the remaining sessions followed a noncorrection paradigm.

Provided the subject reached criterion within 1,000 trials on SSP-I, he was given the SSP-II task.

This was similar to SSP-I except that the horizontal distance between the balls was increased to 5 cm. The third form of the problem (SSP-III), presented after the successful completion of SSP-II, used 10 balls in the arrangement shown in Figure 2. The distance between the balls in this case was also 5 cm. A noncorrection procedure was used throughout the SSP-II and SSP-III tasks.

Delayed response. The third task, a delayed response (DR) problem, was included to dissociate the effects of frontal and parietal lesions. The frontal animals were expected to show selective impairment on this task. The procedure in this problem was based on that employed by Miles and Blomquist (1960). The test objects used to cover the food wells were two identical, white, hexagonal-shaped containers, 2.2 cm in height, 2.6 cm in diameter, spaced 22.3 cm apart. After the stimulus tray was placed just out of the subject's reach, the experimenter placed a currant in the center between the two food wells, allowing it to remain there for 3 sec. The currant was then moved to one of the food wells, and both food wells were then covered by the test objects. To maximize the probability that the subject was attending to the placement of the reinforcement, the experimenter held the currant in position over the positive food well until it appeared that the subject had attended to the placement. After the designated delay interval, the tray was advanced, and the subject was allowed to respond. It should be emphasized that at
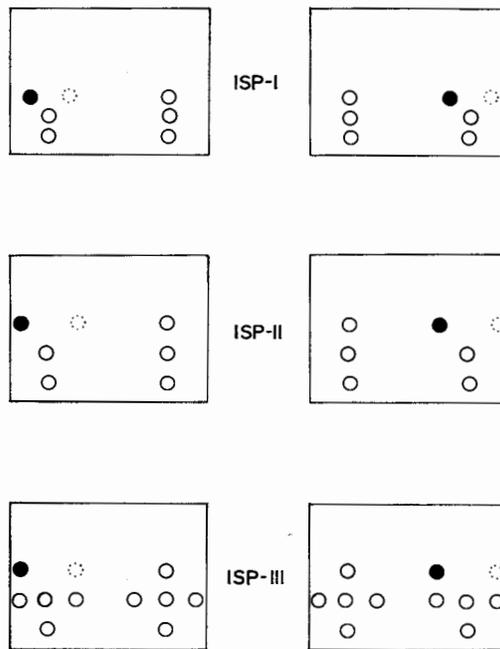

FIGURE 3. Schematic representation of the three forms of the identical spatial pattern task (ISP). (Dark circle indicates site of reinforcement.)

no time was an opaque screen interspersed between the animal and the stimuli. A noncorrection procedure was used throughout.

Each subject was given a series of pretraining sessions, in which the test tray was advanced as soon as the test objects were placed over the food wells (producing approximately a 1-sec delay). After the subject achieved an 80% success rate on 40 consecutive trials, the remaining sessions consisted of randomly selected 3-, 6-, or 9-sec delays. Again, criterion was set at 80% of 40 consecutive responses.

Form discrimination. This task, like the brightness discrimination described above, was intended as a "control" problem in the sense that major deficits were not expected from the operated groups. The discriminanda were two gray plywood squares (6.2 cm²) presented on a black stimulus tray, 22.3 cm apart. One of the discriminanda was presented oriented as a square (reinforced cue), and the other was rotated so as to be presented as a diamond (nonreinforced cue). The first two sessions followed a correction procedure; the remaining, a noncorrection paradigm.

Spatial pattern discrimination-II. This task was similar to the second (SSP) except that here the ISP forms of the spatial discrimination task were used, in which the site of reinforcement and the critical cue were identical (see Figure 3). The training procedures used were the same as those for the SSP task.

RESULTS

Anatomical Data

Reconstructions of the cortical lesions are presented in Figure 4. The lesions sustained by the parietal and frontal animals were within the predefined boundaries, but tended to be somewhat smaller in surface extent than originally planned. Conversely, some lesions (particularly in Subjects P1 and P2)
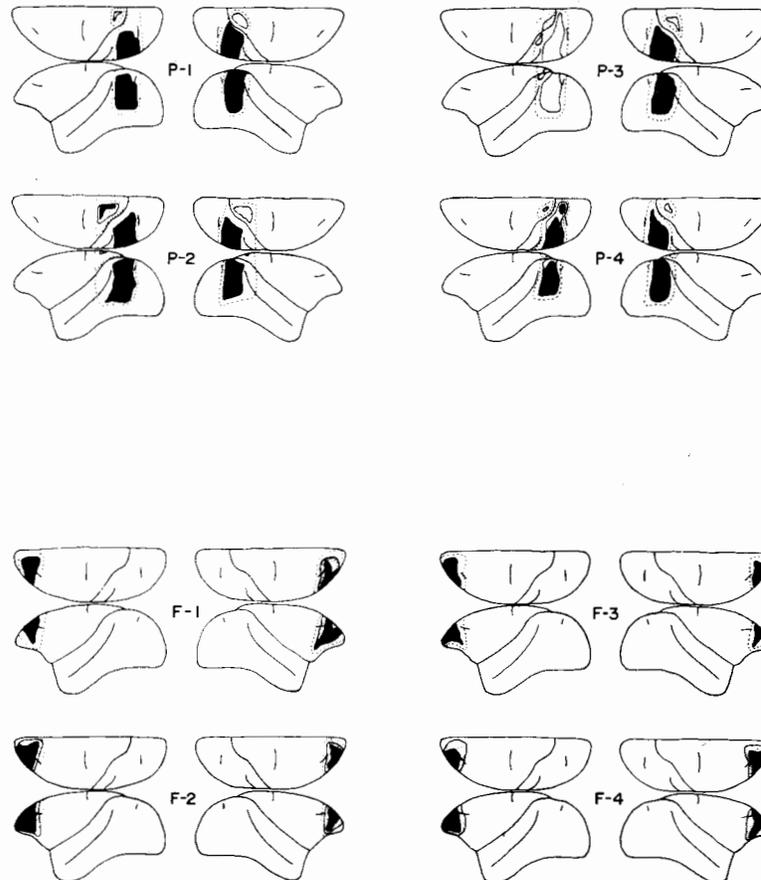

FIGURE 4. Reconstructions of dorsal and lateral views of parietal (P) and frontal (F) lesions. (Dark portions represent areas of greatest destruction in each animal. Areas set off by dotted lines indicate very slight damage, and stippled areas reflect moderate destruction.)

were deeper than planned, extending well into the underlying fiber tracts. When the parietal lesions are compared with Benjamin and Welker's (1957) map of the somatosensory cortex, there is an indication that this latter region was essentially spared. Histological examination also revealed that the lesions failed to encroach upon the striate cortex, which can be identified by the presence of the stripe of Gennari, by the enlargement of cortical layer III, and by the shift of layer IV toward the gray matter (Cowey, 1964; Spatz, Tigges, & Tigges, 1970).

Parts of the prestriate cortex, however, were involved in the parietal lesions, although the exact extent of the involvement is difficult to determine. As Zeki (1969) noted, the difficulties stem from the discrepancies in cytoarchitectural labeling of the subdivisions of the prestriate cortex in the

maps of Brodmann (1905) and von Bonin and Bailey (1947). Furthermore, there is still some question as to the exact location of the prestriate cortex in the squirrel monkey (Spatz et al., 1970; Zeki, 1971).

As can be seen in Figure 5, most thalamic degeneration in the parietal group occurred in the dorsal portion of the inferior pulvinar and in the lateral pulvinar. Some degeneration was typically found in the dorsal portions of the lateral geniculate nuclei. The lateral posterior nuclei also seemed to sustain mild degenerative changes in some cases. There appeared to be no degenerative changes in the pretectal area, superior colliculi, or striate cortex of any animal.

With the exception of the lateral geniculates, the pattern of thalamic degeneration after the parietal lesions was generally consistent with earlier findings regarding the thalamocortical connections of the posterior
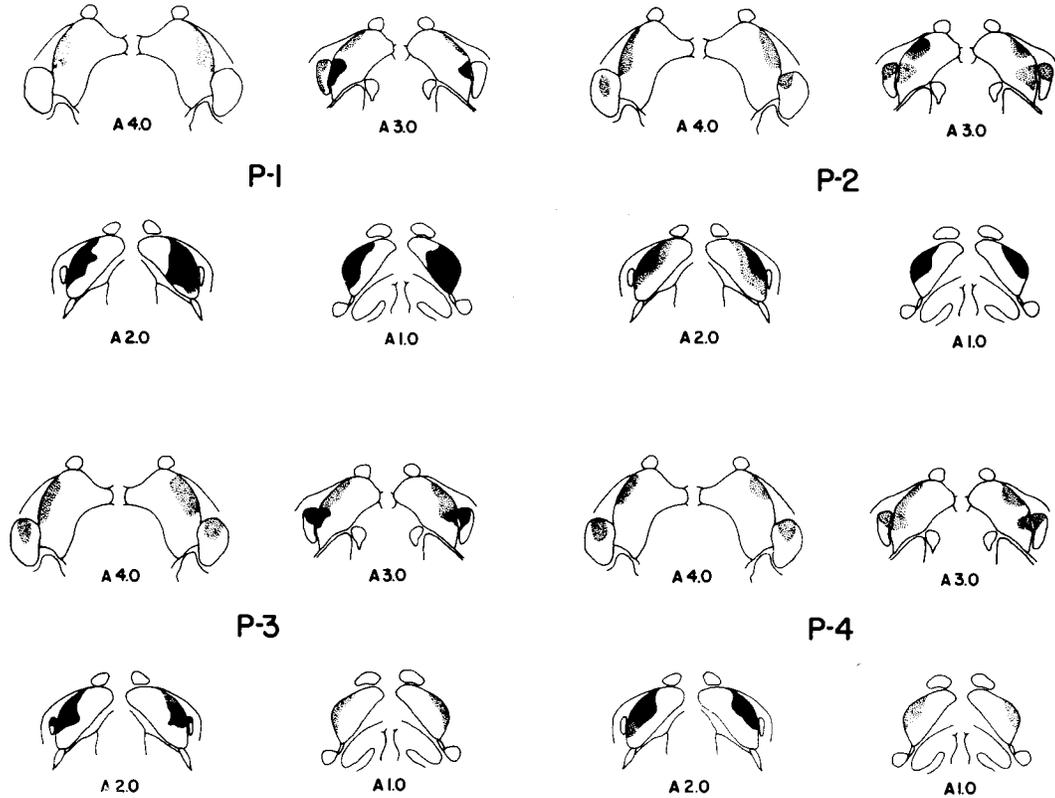

FIGURE 5. Schematic representation of thalamic degeneration in four parietal animals. (Darker portions indicate areas of more intense cell loss and gliosis. The A–P sections represent those of Emmers & Akert, 1963.)
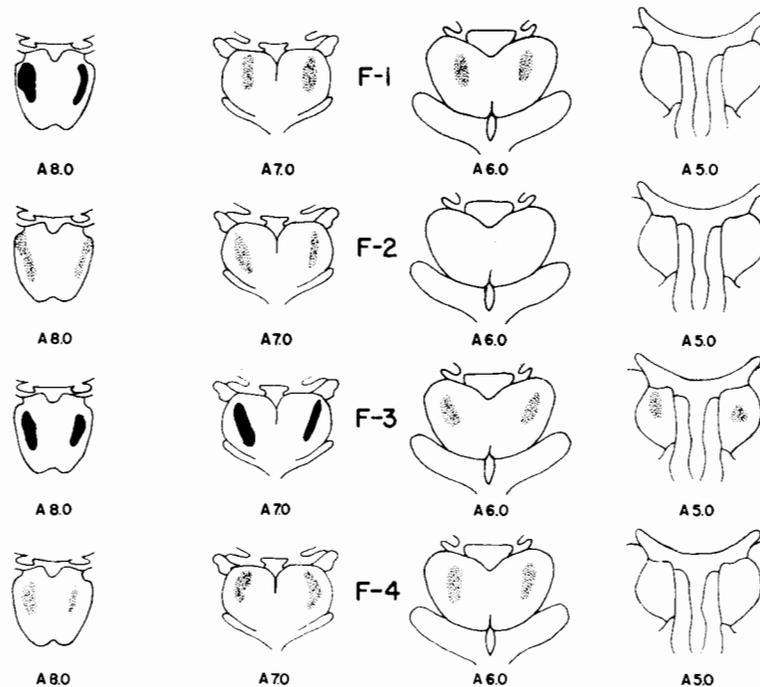
FIGURE 6. Schematic representation of thalamic (dorsal medial nucleus) degeneration in four frontal animals. (Darker portions indicate areas of more intense cell loss and gliosis. The A–P sections represent those of Emmers & Akert, 1963.)

parietal areas (e.g., Chow, 1950; Crosby et al., 1962; Petras, 1971; Walker, 1938). However, it is noted that degeneration in the lateral geniculate nuclei is frequently reported after lesions of the posterior parietal cortex (e.g., Bates & Ettlinger, 1960; Blum, 1951; Blum et al., 1950; Chow, 1951, 1952; Ettlinger & Kalsbeck, 1962; Ettlinger & Wegener, 1958; Moffett & Ettlinger, 1970). Since the geniculocortical radiations apparently terminate exclusively in the striate cortex in both the squirrel monkey (Spatz et al., 1970) and the rhesus monkey (Hubel & Wiesel, 1969), the changes seen in this nucleus are probably not the result of the cortical destruction but rather of interruption of the visual radiations underlying the posterior parietal and prestriate areas. The lesions in the present study did typically encroach on the white matter beneath the aspirated cortex. In the frontal animals, the thalamic degeneration was generally confined to the parvocellular portion of the dorsal medial nucleus (see Figure 6).

General Behavior

With the exception of early, minor locomotor difficulties in Subjects P2 and P4, there was no indication of any prolonged deficits on the general behavioral measures used in the present study. However, most of the animals in the operated groups seemed to show varying degrees of increased fearfulness during the early postoperative recovery period. This fear was most evident when the animals were placed in the WGTA. The response rates of a number of animals during attempts to administer the behavioral rating tasks were either very low or entirely absent, especially on Days 3 and 6 postoperatively; consequently, the reliability of these ratings is questionable. Nevertheless, this fearfulness seems to have abated in all animals by the time formal testing was begun, since they were able to respond appropriately in the WGTA at that time.

It may be noted that postoperatively all four frontals gave indications of poor self-grooming. Their coats had a matted and dirty

appearance, a condition that persisted until they were sacrificed. This effect was most striking in Subjects F1 and F2. One wonders whether these observations are related to the deterioration in personal hygiene and grooming habits that have been described as sequelae to frontal lesions in man (Grinker & Sahs, 1966).

Behavior Test Results

Trials to criterion for each animal in the brightness discrimination task may be seen in Table 1. One animal, P1, received, instead, a color discrimination task (yellow–reinforced vs. green–nonreinforced), before it was deemed best to avoid the possible complications of including hue and saturation as stimulus variables. However, there were no significant differences in the number of trials to criterion among the three groups whether this animal was included in the analysis of variance, F $(2,9) = 2.64$, $p >$ .05, or not, F $(2,8) = 1.27$, $p > .05$.

The performance of the groups on the SSP task is shown in Table 2. All parietals and two of the frontals failed to reach criterion within 1,000 trials. Because of their failure

TABLE 1

TRIALS TO CRITERION ON BRIGHTNESS
DISCRIMINATION (BD), DELAYED
RESPONSE (DR), AND FORM
DISCRIMINATION (FD) TASKS
IN MONKEYS

Subject	BD	DR (1-sec)	DR (3, 6 and 9 sec)	FD
C1	67	38	81	99
C2	39	40	645	196
C3	38	102	309	119
C4	56	74	36	79
F1	32	71	388	182
F2	34	88	1,000[a]	142
F3	38	36	1,000[a]	92
F4	66	35	1,000[a]	70
P1	176	115	244	326
P2	138	167	40	247
P3	39	35	501	243
P4	55	36	238	286

Note. Identification of subjects: C = control; F = frontal lesions; P = posterior parietal cortical lesions.

[a] Failure to reach criterion.

TABLE 2

TRIALS TO CRITERION FOR TASKS IN WHICH CUE
AND SITE OF REINFORCEMENT WERE
SEPARATED (SSP)

Subject	SSP-I	SSP-II	SSP-III	SSP-I retest[a]
C1	705	40	364	—
C2	555	243	435	—
C3	583	200	69	136
C4	407	388	319	85
F1	1,000[b]	—	—	—
F2	723	133	81	66
F3	1,000[b]	—	—	159
F4	550	91	150	40
				—
P1	1,000[b]	—	—	
P2	1,000[b]	—	—	400[b]
P3	1,000[b]	—	—	482[b]
P4	1,000[b]	—	—	458[b]

Note. Identification of subjects: C = control; F = frontal lesions; P = posterior parietal cortical lesions. Where there are no entries, the animals were not tested.

[a] Retested 2 days after completion of a series of tasks (in which cue and site of reinforcement were identical) in order to assess possible practice effects.

[b] Failure to reach criterion.

to reach criterion in 1,000 trials on the SSP-I task, the parietals and the two frontals were not tested on the remaining forms of this task. The differences among the groups in trials to criterion (using 1,000 as the score for animals that failed to reach criterion) on SSP-I was significant, F $(2,9) = 9.05$, $p <$ .01. A Newman-Keuls test on the group means revealed that the unoperated controls required significantly fewer trials to reach criterion than either the frontals $(p < .05)$ or the parietals $(p < .01)$. There was no significant difference between the two operated groups on this measure.

Whereas all animals in the unoperated and parietal groups were able to reach criterion within 1,000 trials on the delayed response task, three of the four frontals were unable to do so (see Table 1). Analysis of variance of the trials to criterion measure showed a significant difference among the groups, F $(2,9) = 6.6$, $p < .05$. There was no significant difference between the unoperated and parietal groups, but the frontals were significantly different from both of

TABLE 3

TRIALS TO CRITERION FOR TASKS IN WHICH CUE
AND SITE OF REINFORCEMENT WERE
IDENTICAL (ISP)

Subjects	ISP-I	ISP-II	ISP-III
C1	112	82	417
C2	40	217	519
C3	64	82	150
C4	134	61	143
F1	46	70	285
F2	78	38	98
F3	84	87	381
F4	36	70	77
P1	419	99	472
P2	199	41	192
P3	241	41	198
P4	229	233	634

Note. Identification of subjects: C = control;
F = frontal lesions; P = posterior parietal cortical
lesions.

these groups ($p < .05$). There was no signifi-
cant difference among the various delay in-
tervals, either within or among the three
groups. During pretraining on delayed re-
sponse, which involved an approximate 1-sec
delay, the frontals performed comparably
with the other groups.

On the form discrimination task (see Ta-
ble 1), the unoperated and frontal groups
averaged 123 and 121 trials to criterion, re-
spectively. The parietals required 275 trials,
on the average, to reach criterion. The dif-
ference among groups was significant, F
$(2,9) = 14.1, p < .01$.

Although its performance was substan-
tially improved over that on the SSP-I task
(see Table 3), the parietal group still re-
quired significantly more trials to reach cri-
terion on the ISP-I task than did either the
unoperated controls or frontal animals, F
$(2,9) = 12.86, p < .01$. There were no sig-
nificant differences among the three groups
on ISP-II, $F (2,9) = .5$, or ISP-III, $F (2,9)$
$= .8$.

DISCUSSION

Parietal Deficit

The main results of the present work are
those concerned with spatial pattern dis-
criminations. Two distinct factors will be
discussed. The first, seen in both the SSP
and the ISP series of tasks, involves spatial
pattern discriminations per se. The second,
seen only in the SSP tasks, involves the sep-
aration of the primary cue within a pattern
from the site of reinforcement. Spatial pat-
tern discrimination, as studied in the pres-
ent work, does not appear to be disrupted by
posterior parietal lesions. After only slight
difficulty on ISP-I, the simplest and easiest
of the series, parietals performed as well as
unoperated and frontal monkeys on ISP-II
and ISP-III. The slight deficiency in per-
formance seen in the parietals on ISP-I
can be attributed to the relative lack of
transfer from the SSP tasks, since the ISP
series followed the SSP series in the experi-
ment. Parietals performed at chance levels
after 1,000 trials on the SSP-I task and were
not given the SSP-II and SSP-III problems.
All unoperated controls reached criterion on
all SSP tasks, as did two frontals. A third
frontal was so near criterion at the planned
maximum of 1,000 trials that it was allowed
to reach criterion (in 1,003); however, it was
not given the SSP-II and SSP-III problems.
Although the fourth frontal did not reach
criterion on SSP-I, it had a higher rate of
successful responses over the last 100 trials
than did any of the parietals.

The only difference between the SSP series
of problems and the ISP series was that in
the former the primary cue and the site of
reinforcement were separated, whereas in
the latter series, the cue and site of rein-
forcement were identical. As noted above,
the parietals failed totally to make the SSP
discrimination, but were quite successful in
the ISP discriminations. Others have re-
ported deficits following parietal lesions on
tasks in which there was separation of the
primary cue and the site of reinforcement
(Bates & Ettlinger, 1960; Ettlinger & Wege-
ner, 1958; Pohl, 1973). In the case of the
first two studies, however, the nature of the
possible relationship of this variable to pa-
rietal deficits was not discussed. Our inter-
pretation of this data would differ from that
of Pohl (1973), who concluded that parietal
lesions yield an inability to mediate allocen-
tric spatial orientation, which was defined as

spatial orientation depending on external cues. Pohl's landmark reversal task appears to have both the allocentric cue variable and the separation of cue and reinforcement variable operating. If, as suggested here, both the ISP and SSP tasks require discrimination on the basis of allocentric cues, then the successful performance of the parietals on the ISP task seems to rule out failure to use allocentric cues as the explanation for parietal deficits. Rather, the total failure of the parietals to acquire the SSP discrimination indicates the separation of cue and reinforcement site as the basis for the parietal deficits. Unfortunately, Pohl's task, as described, does not allow one to separate the two variables experimentally.

Since the ISP tasks, mastered by all groups including the parietals, came after the SSP tasks, it was decided a posteriori to retest the available animals on SSP-I in order to assess possible practice effects. Two unoperated monkeys, three frontals, and three parietals were available for retest. Two days after completion of the ISP series, retraining on SSP-I was begun. Each monkey was given a minimum of 400 trials or twice the number taken by that animal to reach criterion on ISP-I. These results may be seen in Table 2. The unoperated and frontal animals performed well, but the parietals again failed to reach criterion on SSP-I. This finding adds further support to the conclusion that parietals are impaired in spatial discriminations when the primary cue and the site of reinforcement are separated.

A possible explanation for the difficulty that monkeys with parietal lesions have when the cue and site of reinforcement are separated may involve attentional mechanisms. If one analyzes the design of the SSP and ISP tasks (Figures 2 and 3), it can be seen that the primary cue (the ball that is not fixed) or the primary cue complex (this would include the ball(s) adjacent to the primary cue and in relation to which the latter varies) is the same for both tasks. However, the predicted primary focus of attention (the locus of reward) is different on the two tests, both absolutely and in relation to the primary cue. Since the parietals were able to reach criterion on the ISP-I task and

performed comparably with the unoperated monkeys on the ISP-II and ISP-III tasks, it may be assumed that these animals were able to "perceive" and "interpret" the primary cue (i.e., make a judgment of "reward" or "no reward", given the relative position of the primary cue in the ISP array). Success on the SSP task might require a shift in the focus of attention from the site of reinforcement to the primary cue or primary cue complex. The deficit of the parietal animals is hypothesized to lie in their relative inability to accomplish this shift in focus of attention and at the same time manage to maintain the perceptual whole. Thus, the basis of the parietal deficit seen here may be similar to that proposed by Ettlinger et al. (1957) to account for the visual–spatial deficits found in human subjects. They suggested that while the normal individual

in surveying an extended space, can shift his attention freely from one object to the next without loss of the implicit system of spatial relationships upon which orderly perception depends ... patients with visual–spatial disorders are severely limited in their ability to scan and integrate an extended situation. (p. 357)

It may be recalled that compared with unoperated controls, the monkeys with frontal lesions were deficient on the SSP-I task, although three of them reached criterion. It is not clear whether this deficiency of the frontal animals was related to the mechanism of competing responses hypothesized by French (1962) to account for the deficits experienced by animals with frontal lesions on tasks involving either spatial or temporal discontiguities or whether it reflected difficulties comparable with those hypothesized to affect the parietals' performance. Nevertheless, it appears obvious that the frontals were relatively less affected on this type of task than were the animals with parietal lesions.

A somewhat unexpected finding in the present study was the deficit exhibited by the monkeys with parietal lesions on the form discrimination task. Visual–object or visual–pattern discrimination deficits have usually been associated with inferotemporal lesions, and such deficits generally do not occur with parietal ablations. In reassessing

the literature in light of the present findings, it appears that previous studies have frequently used visual form (pattern or object) discriminations in which the discriminanda may have been somewhat more distinguishable than those used in the present work. Examples from other studies include a diamond vs. black and white stripes (Blum et al., 1950; Chow, 1951, 1952), a circle vs. a rectangle or triangle (Chow, 1951, 1952; Pasik, Pasik, Battersby, & Bender, 1958; Wegener, 1968), a cross vs. a square (Iwai & Mishkin, 1969), and other apparently highly distinguishable stimuli (Bates & Ettlinger, 1960; Pribram & Barry, 1956; Wilson, 1957).

The visual form discrimination problem in the present study consisted of identical forms (squares), one of which was rotated 45°. Cowey and Gross (1970) and Wegener (1968) found that animals with foveal prestriate and parieto-temporo-preoccipital lesions, respectively, were impaired on visual discriminations involving similar forms, differing in orientation. However, their results on this point are inconclusive in that deficits were also found on tasks in which dissimilar discriminanda were used. Wilson (1957) failed to find deficits in parietal animals on a discrimination consisting of a left-facing L-shaped object vs. one that was rotated 180°. However, Wilson intended this to be an "easy" discrimination, and even the inferotemporal animals failed to show significant deficits on this task. On the other hand, Ades and Raab (1949) and Riopelle and Ades (1953) found evidence suggesting that discrimination tasks devised by rotating or producing "mirror images" of the stimulus objects were disruptive to animals with lesions of neocortical Areas 18 and 19.

The findings of the present study suggest that certain visual form discriminations, which consist of similar figures differing only in orientation, may provide a more sensitive test of the effects of parietal lesions in nonhuman primates than discriminations in which dissimilar figures are used. It is noted that Ettlinger et al. (1957) suggested that deficits in spatial judgment in human subjects may be evidenced, in part, by their "failure to perceive the correct orientation of geometrical forms" (p. 358). It is noteworthy that this deficit may also be interpreted as the same type of "spatial integration" difficulty presumed to impair performance on the SSP task. Orientation is a spatial phenomenon and derives its meaning from one thing (organism or object) in relation to another. If external referents or background cues (e.g., the stimulus tray) are relevant in the form discrimination task employed in the present study, then apparently some integration of the spatial relationship between the object (stimulus cue) and these external referents must take place if the cue is to be correctly interpreted.

It must be emphasized that the deficits experienced by the parietal group on the SSP-I, ISP-I, and form discrimination tasks do not appear to be attributable to a generalized performance decrement as a result of the lesions. The parietal group did not differ significantly from the unoperated controls on the brightness discrimination, delayed response, or the ISP-II and ISP-III tasks.

An important question remains as to whether the deficits observed in the present study are, at least in part, the result of the degeneration seen in the lateral geniculate nuclei. Evidence to the contrary would be that the degeneration found in these nuclei was generally quite moderate (actually very slight in Subject P4) and that these animals were able to perform adequately on several visual tasks, viz., the differentiation of edible objects, the brightness discrimination task, and the ISP-II and ISP-III tasks. However, one must still acknowledge that in the studies of Pohl (1973), Bates and Ettlinger (1960), Ettlinger and Wegener (1958) as well as in the present study, the lateral geniculate nuclei did appear to undergo mild to moderate retrograde degeneration. These appear to be the only studies involving animals with parietal lesions in which deficits were found on tasks involving the separation of stimulus cue and site of reinforcement; relevance of the degeneration in the lateral geniculate nuclei to the deficits observed in the present study merits further investigation.

Frontal Deficit

As expected, the frontal group experienced the greatest difficulty on the delayed response task (three of four animals failing to reach criterion in 1,000 trials). As in the Miles (1964) study, the delayed response deficit in frontals occurred even when the stimuli remained in view of the subject during the delay interval. In addition, increasing the delay beyond 3 sec did not exacerbate the deficit. Neither the frequently invoked interpretations of hyperactivity nor perseveration would appear to account for the delayed-response deficit in the present work. There appeared to be no correlation between either intertrial or intratrial behavior and test performance for any of these animals. The incidence of perseveration was about equally distributed among the three groups.

Summary

In conclusion, the evidence suggests that bilateral lesions of the posterior parietal cortex in both rhesus and squirrel monkeys produce deficits in spatial discrimination when the primary stimulus cue and the site of reinforcement are separated. This deficit appears to occur in both the tactual (Bates & Ettlinger, 1960; Ettlinger & Wegener, 1958) and visual (Bates & Ettlinger, 1960; Ettlinger & Wegener, 1958; Pohl, 1973; present study) modalities. A useful direction for future study is to attempt a clear delineation of the exact anatomical structures or areas that must be ablated in order to produce this particular deficit. Mishkin (1972) has suggested that possible disruptions of extrafoveal vision as a result of damage to the prestriate areas may be critical in eliciting deficits after posterior parietal lesions. On the other hand, cortical Areas 5 and 7 have apparently been the principal sites of the lesions in studies so far reported. The present study has provided an alternative hypothesis, namely, that posterior parietal lesions possibly cause the disruption of attentional mechanisms that might be critical for perceptual integration of spatially discontiguous stimuli.

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