Exp Brain Res (1982) 48: 449–454

Experimental Brain Research

Research Note

Localization and Detection of Visual Stimuli in Monkeys with Pulvinar Lesions*

C.C. Leiby III[1], D.B. Bender[2], and C.M. Butter[1]

[1] Dept. of Psychology and Neuroscience Laboratory, University of Michigan, Ann Arbor, MI 48109, USA
[2] Division of Neurobiology, Physiology Dept., State University of New York, 4234 Ridge Lea Rd., Buffalo, NY 14226, USA

Summary. Since the pulvinar receives a major ascending projection of the superior colliculus, pulvinar lesions might produce behavioral impairments resembling those that follow colliculus lesions. To test this possibility, we examined the effect of pulvinar lesions in monkeys on the localization and detection of brief light flashes, a task in which monkeys with colliculus lesions are severely impaired. Some of the pulvinar-lesioned monkeys showed localization impairments similar to those in monkeys with colliculus lesions. However, histological analyses of the lesions suggested that these deficits were related not to the pulvinar damage per se, but rather to interruption of corticotectal fibers that pass through the pulvinar. We conclude that the pulvinar is not critical for the ability to locate and detect brief visual stimuli.

Key words: Monkeys – Visual localization – Pulvinar lesions – Superior colliculus

Physiological and anatomical evidence clearly implicates the primate pulvinar in vision. Both the inferior and lateral pulvinar are visually responsive and retinotopically organized (Bender 1981b, 1982), and have reciprocal connections with visual cortical areas (Benevento and Davis 1977; Benevento and Rezak 1976; Campos-Ortega and Hayhow 1972; Ogren and Hendrickson 1976; Ungerleider et al. 1980; Whitlock and Nauta 1956). In view of the extensive connections between pulvinar and visual cortex, one might have thought that animals with pulvinar lesions would show behavioral impairments similar to those which follow lesions of prestriate or inferior temporal cortex. However, this is not the case; monkeys with pulvinar lesions fail to show deficits in a variety of visual discrimination tasks (Chow 1954; Mishkin 1972; Ungerleider et al. 1977). In only one investigation with adequately reported histology have pulvinar lesions been accompanied by a discrimination deficit (Chalupa et al. 1976).

A different view suggests that the visual functions of the pulvinar depend on the input it receives from the superior colliculus; the major ascending outflow from the superficial layers of the superior colliculus terminates within the inferior ("tectorecipient") pulvinar (Benevento and Fallon 1975; Partlow et al. 1977). Thus, according to this view, monkeys with pulvinar lesions might show behavioral impairments similar to those which follow superior colliculus lesions. To investigate this possibility, we studied the effects of inferior pulvinar lesions on spatial localization of brief light flashes, a task in which monkeys with colliculus lesions are severely impaired (Butter et al. 1978).

The subjects were seven experimentally naive rhesus monkeys (*Macaca mulatta*), six male and one female, weighing 3.6–7.3 kg. The apparatus and behavioral procedures were identical to those described previously (Butter et al. 1978). The monkey faced a horizontal array of stimulus-response panels located on a curved metal perimeter. It could initiate a trial with a "start press", i.e., pressing the "fixation" panel in the center of the perimeter after a small, dim light spot on the fixation panel changed color. On half the trials, the start press was followed by a 50 ms light flash on one of the side panels; a press on that panel produced a liquid reward. On the remaining trials, the start press was not followed by a light flash; the animal was required to press a "no light panel" located above the fixation panel to

* Supported by research grants MH 26489 from the National Institute of Mental Health and EY02254 from the National Eye Institute

Offprint requests to: Dr. D.B. Bender (address see above)

0014–4819/82/0048/0449/$ 1.20

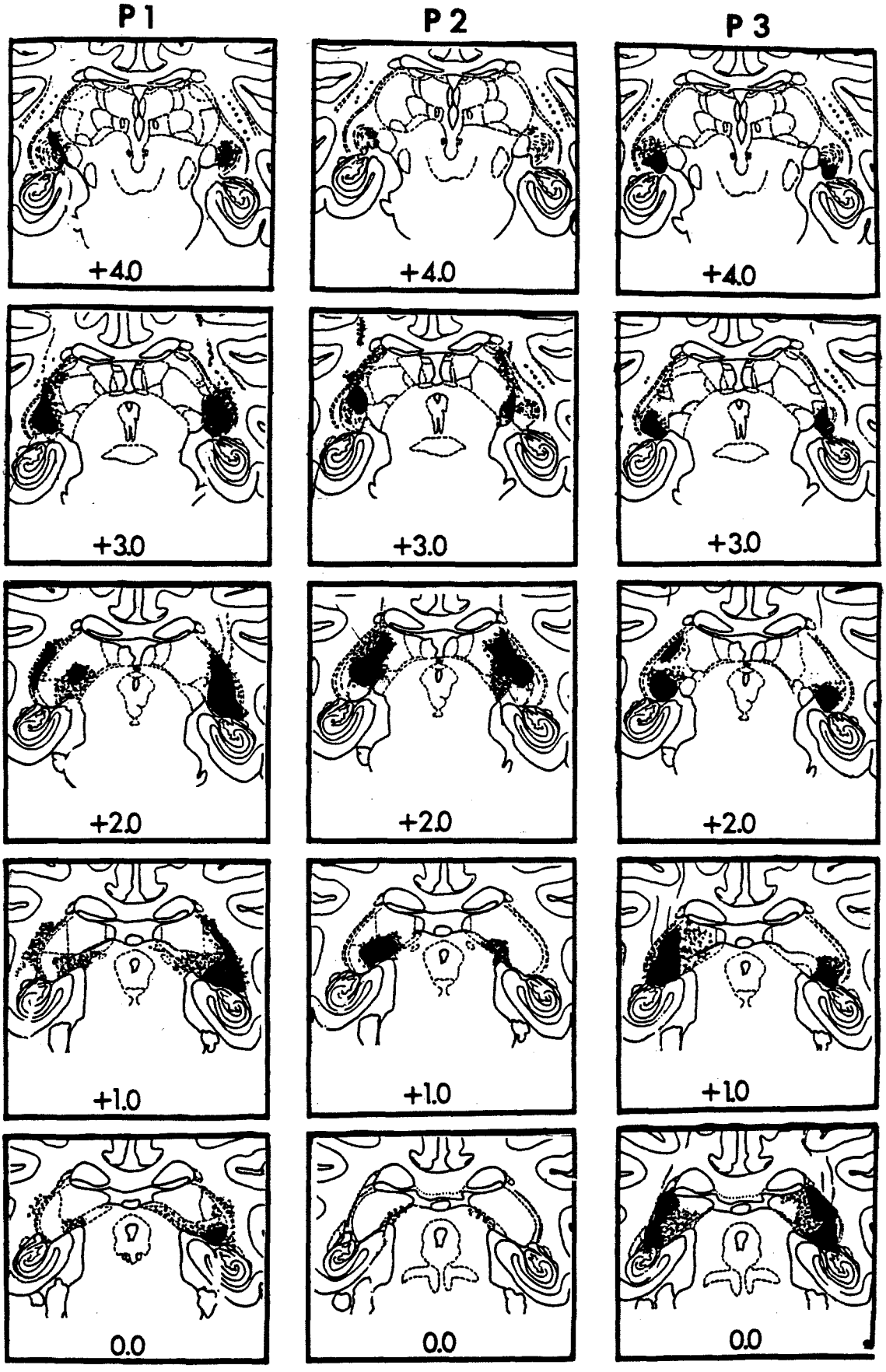

Fig. 1A

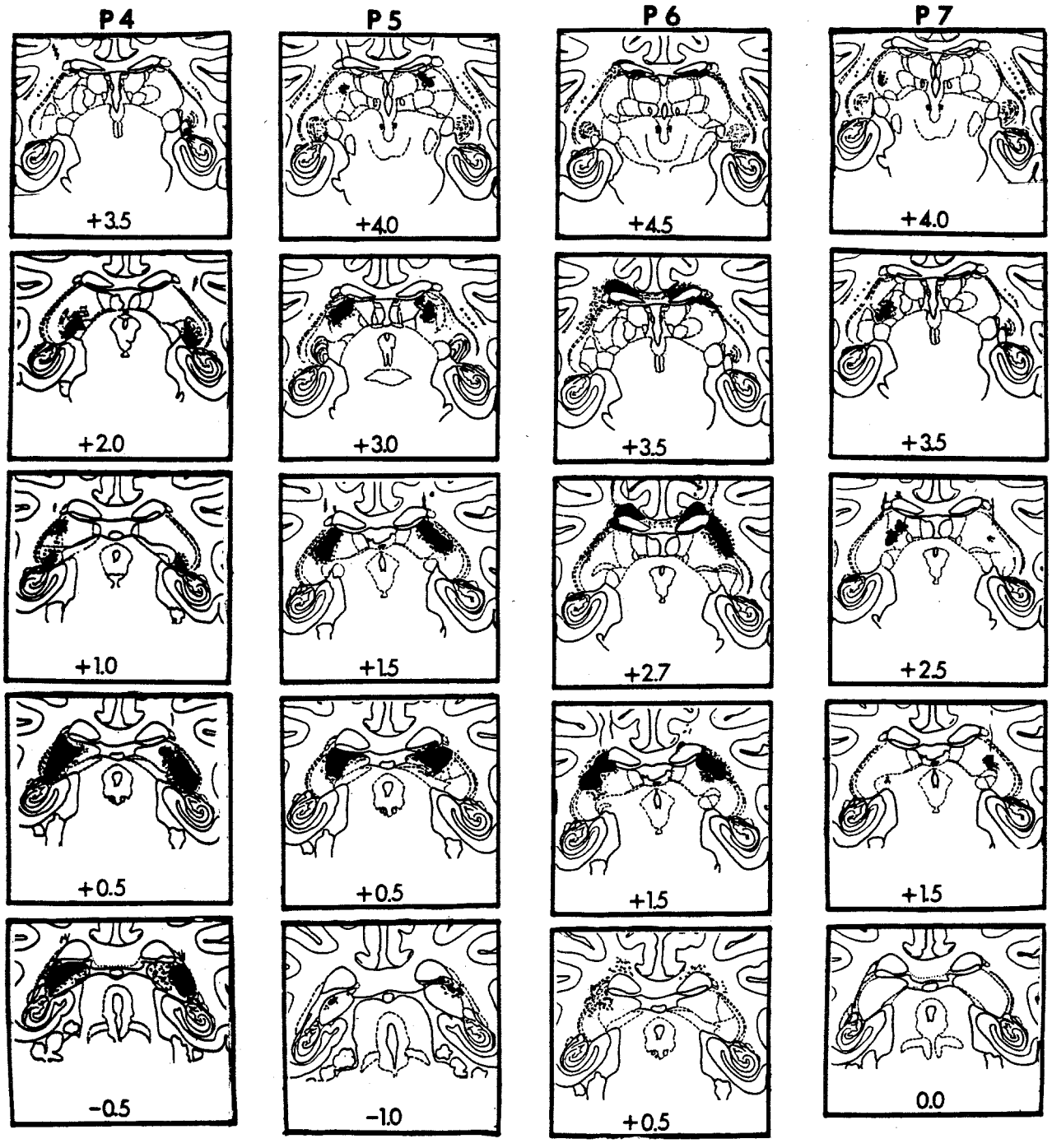

Fig. 1A, B. Representative cross sections through pulvinar lesions. *Black areas:* total cell loss. *Stippled areas:* partial cell loss and/or gliosis. Numbers below each section refer to stereotaxic levels

obtain reward. An error was followed by a "time-out" period (7 s), during which a trial could not be initiated. The monkeys were trained on the task until they responded correctly on at least 90% of both light and no-light trials in each of eight consecutive sessions. Two weeks later they were tested for an additional five sessions by the same procedures; all showed good retention of the task. Ten to 14 days later, they underwent surgery[1]. Four animals (P1, P2,

1 The lesions were made by passing sufficient radio frequency current through a thermistor-tipped electrode to raise its tip-temperature to 73–80° C for 1 min. The electrode was guided to four sites in each thalamus by microelectrode recordings of units in the caudal pole of the lateral geniculate nucleus, adjacent to the inferior pulvinar. Details of the surgical procedures may be found elsewhere (Butter et al. 1978; Bender 1981b)

P3, P4) received lesions intended to bilaterally destroy the tectorecipient zone of the pulvinar. Three animals (P5, P6, P7) served as controls: Their lesions were intended to destroy the dorsal part of the lateral and medial pulvinar, thus sparing the tectorecipient zone. Approximately 3 weeks following surgery, the monkeys were tested by the same procedures used before surgery for a variable number of sessions, depending on whether and to what extent they were impaired. In addition, they were tested in every sixth session with 1 s light flashes presented on the side panels. After completing behavioral testing, the animals were killed and their brains prepared for histological examination by procedures reported previously (Butter et al. 1978).

Analysis of the histological material[2], showed that the lesions were largely as intended (see Fig. 1). Thus, the tectorecipient zone was extensively damaged in P1, P2, P3 and P4 (although in P2 there was sparing of its ventral half). In the remaining animals (P5, P6, P7) the lesions damaged the dorsal parts of the lateral and medial pulvinar, sparing the tectorecipient zone. It is important to note, however, that the tectorecipient zone lesions also damaged two fiber systems traversing the pulvinar. First, in P1, P2 and P3 (but not in P4), extensive bilateral damage to the ventral half of the lateral pulvinar apparently disrupted many corticotectal fibers which pass through this region on their way to the brachium of the superior colliculus: The brachium was smaller in these three animals (P1: 7.4 mm, P2: 4.5 mm, P3: 5.6 mm) than it was in either the remaining four operated animals (range: 7.9–8.4 mm), or a group of three controls from another study (range: 8.8–10.2 mm). Second, all four tectorecipient zone lesions damaged the ventromedial base of the inferior and lateral pulvinar bilaterally, especially in P3 and P4. The lesions, therefore, may have injured retinotectal fibers since they pass through this area (Hendrickson et al. 1970; Tokunaga et al. 1981). Finally, the posterior tip of the lateral geniculate nucleus (involving the central 4° of the lower visual field) was damaged bilaterally in P1 and only very slightly in P2 and P3.

Of the four animals with tectorecipient zone lesions, three (P1, P2, P3) showed consistent and lasting post-operative impairments (see Fig. 2). The fourth (P4) was not impaired, nor were the three control animals (P5, P6, P7). P1 and P2 showed the most severe deficits and were impaired in responding to peripheral flashes throughout post-operative testing (5 months). P3 showed increased errors to peripheral flashes during the first 2 months of post-operative testing; its performance then improved in the left periphery but deteriorated in the center. With further testing, however, this monkey reattained pre-operative performance levels by session 60.

The three impaired animals showed increases in localization errors (pressing a side panel other than the one on which a flash appeared), detection errors (pressing the "no light" panel), and response omissions; no particular error type predominated. Nor did these monkeys mislocalize in one particular direction. When the flash duration was lengthened to 1 s, these animals performed at normal control levels, a result also found in colliculus-lesioned animals (Butter et al. 1978). None of the seven monkeys was impaired in responding on no-light trials.

At first glance, the results suggest that lesions of the pulvinar's tectorecipient zone impair localization of visual targets in the same way that tectal lesions do. Three of the four animals with inferior pulvinar damage were impaired, whereas no animal with an intact inferior pulvinar was impaired. Furthermore, like monkeys with tectal lesions (Butter et al. 1978), the impaired animals, P1, P2, and P3, showed long-lasting and severe deficits in localizing and detecting brief flashes primarily in the periphery of the visual field. They differed from monkeys with superior colliculus lesions only in that they did not mislocalize in one particular direction.

The histological results, however, suggest that damage to inferior pulvinar neurons may not be the cause of the deficit. P4, like the impaired monkeys, had a massive tectorecipient zone lesion, yet showed no deficit. Furthermore, the extent of tectorecipient zone damage was unrelated to the severity of the deficit in the impaired animals: The lesion in P3, the least impaired of the three impaired monkeys, was larger than P2's and at least as large as P1's. There was also no consistent relation between the presence or degree of damage to the structures outside the tectorecipient zone of the pulvinar.

2 For each animal, the severity of damage to the different regions of the pulvinar was judged by: (a) Plotting the pulvinar lesion on tracings of the brain sections and then estimating the percent overlap between these plots and each pulvinar region, (b) weighting each estimate by the size of the given region on the cross section, and (c) averaging these weighted estimates over all cross sections. Since in some animals there was shrinkage in the brachium of the superior colliculus, brachial widths were estimated in all animals as follows. The dorsoventral width of tracings of the brachia in cross sections at AP +1.0 and AP 0.0 was measured at three points along the medial to lateral extent of the brachium; each measurement was then divided by the width of the brainstem in each section to take account of variations in brain size. The relative brachial widths were then averaged for each monkey

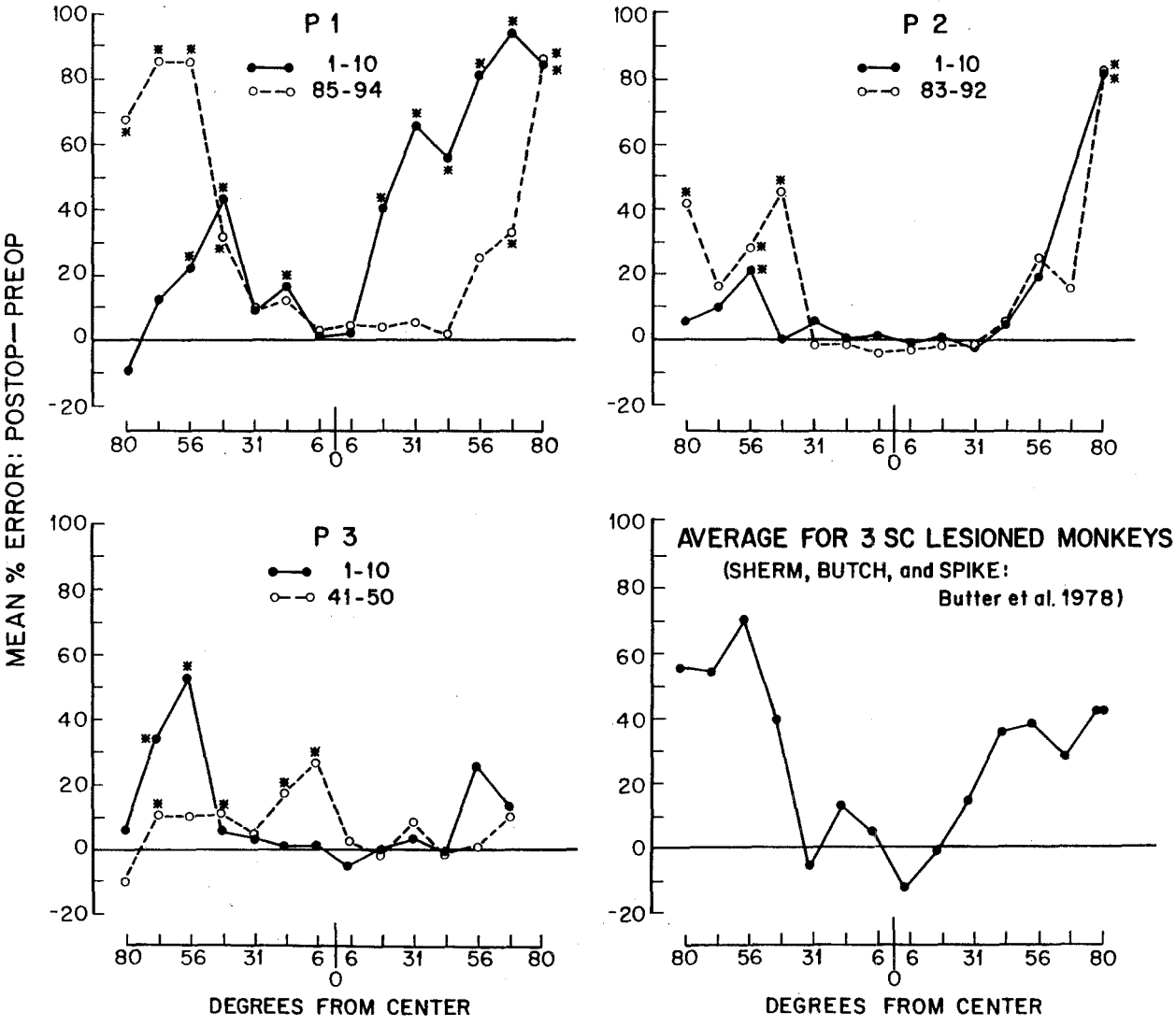

Fig. 2. Difference between postoperative and preoperative error rates for light flashes on each side panel. *Solid lines:* data from the first block of ten postoperative sessions. *Dashed lines:* data from the last ten postoperative sessions in which a monkey was impaired. Each point represents an error difference score that was computed as follows. Postoperative testing was divided into consecutive blocks of ten 250-trial sessions. The percentage total errors in responding to light flashes in each block of sessions was calculated. From these postoperative error scores, the percent total errors made to flashes presented on the corresponding panel during the preoperative criterion and retention test sessions were subtracted. *Asterisks* indicate statistically significant increases in errors: difference scores greater than +10% were converted to *t* values, using a pooled variance estimate based on both pre- and postoperative data. A monkey was considered to be impaired in responding to flashes on a particular side panel during a particular postoperative block if the one-tailed t-value was significant at or beyond the 0.01 level. The difference scores for P3 at the panel 80° to the right are omitted owing to the high preoperative error rate at that site

Discussion

We believe that the deficits resulted from damage to fibers of cortical origin passing through the lateral pulvinar, adjacent to the tectorecipient zone, on their way to the brachium of the superior colliculus; many of these fibers terminate within the tectum (Campos-Ortega and Hayhow 1972; Benevento and Fallon 1975; Whitlock and Nauta 1956). These corticotectal fibers appeared to be most severely damaged in the three impaired animals; their brachia were smaller than either those of the unimpaired animals or those of normal control animals. Furthermore, the one unimpaired animal with a massive lesion of the tectorecipient zone, P4, had only slight damage to this fiber system. Damage to retinotectal fibers may also have contributed to the deficit, since all tectorecipient-zone lesions encroached on these tectal afferents as they pass along the ventromedial surface of the inferior pulvinar before collecting in the

brachium to enter the colliculus (Tokunaga et al. 1981; Hendrickson et al. 1970). This possibility seems unlikely, however, since of the two animals (P3 and P4) with the most severe damage to the ventromedial surface, one was least impaired and the other unimpaired.

Although we think the behavioral impairments resulted from damage to corticotectal fibers rather than from pulvinar damage per se, we cannot rule out the possibility that damage to both the tectorecipient zone and to the corticotectal and retinotectal fibers is critical. The respective contributions to localization performance of the pulvinar and the tectal afferents traversing it could be evaluated by injecting kainic acid into the pulvinar. For example, we have recently shown (Nagel-Leiby S, Bender DB, Butter CM, unpubl. observ.) that electrolytic but not kainic acid lesions of the pulvinar impair the performance of monkeys in another task sensitive to superior colliculus lesions – color discriminations with stimulus-response separation (Butter 1974).

Our results suggest that the pathway from the tectum to the inferior pulvinar is not critical for the ability to localize or detect brief stimuli, a conclusion supported by the recent finding that tectal lesions do not markedly affect the visual responses of inferior pulvinar neurons (Bender 1981a). Our results do suggest, however, that disruption of corticotectal fibers impairs localization of visual stimuli as severely as colliculus lesions themselves. According to this view, while cortical input may not be crucial to the visual activation of colliculus cells in the monkey (Schiller et al. 1974), it may indeed be critical for behavior mediated by the colliculus.

Acknowledgements. The authors thank Judith Lehman, Marylin Hoy and Susan Wolf for their editorial assistance in preparing the manuscript.

References

Bender DB (1981a) Effects of superior colliculus and striate cortex lesions on the visual response properties of inferior pulvinar neurons in the monkey. Soc Neurosci Abstr 7: 760

Bender DB (1981b) Retinotopic organization of macaque pulvinar. J Neurophysiol 46: 672–693

Bender DB (1982) Receptive field properties of neurons in the macaque inferior pulvinar. J Neurophysiol 48: 1–17

Benevento LA, Davis B (1977) Topographical projections of the prestriate cortex to the pulvinar nuclei in the macaque monkey: An autoradiographic study. Exp Brain Res 30: 405–424

Benevento LA, Fallon J (1975) The ascending projections of the superior colliculus in the rhesus monkey. J Comp Neurol 160: 339–362

Benevento LA, Rezak M (1976) The cortical projections of the inferior pulvinar and adjacent lateral pulvinar in the rhesus monkey (*Macaca mulatta*): An autoradiographic study. Brain Res 108: 1–24

Butter CM (1974) Effect of superior colliculus, striate and prestriate lesions on visual sampling in rhesus monkeys. J Comp Physiol Psychol 87: 905–917

Butter CM, Weinstein C, Bender D, Gross CG (1978) Localization and detection of visual stimuli following superior colliculus lesions in rhesus monkeys. Brain Res 156: 33–49

Campos-Ortega J, Hayhow W (1972) On the organization of the visual cortical projection to the pulvinar in *Macaca mulatta.* Brain Behav Evol 6: 394–423

Chalupa LM, Coyle RS, Lindsley DB (1976) Effect of pulvinar lesions on visual pattern discrimination in monkeys. J Neurophysiol 39: 354–369

Chow K-L (1954) Lack of behavioral effects following destruction of some thalamic association nuclei in monkey. AMA Arch Neurol Psychiat 71: 762–771

Hendrickson A, Wilson ME, Toyne MJ (1970) The distribution of optic nerve fibers in *Macaca mulatta.* Brain Res 23: 425–427

Mishkin M (1972) Cortical visual areas and their interactions. In: Karczmar AG, Eccles JC (eds) Brain and human behavior. Springer, Berlin Heidelberg New York, pp 187–208

Ogren MP, Hendrickson AE (1976) Pathways between striate cortex and subcortical regions in *Macaca mulatta* and *Saimiri sciureus:* Evidence for a reciprocal pulvinar connection. Exp Neurol 53: 780–800

Partlow GD, Colonnier M, Szabo J (1977) Thalamic projections of the superior colliculus in the rhesus monkey, *Macaca mulatta:* A light and electron microscopic study. J Comp Neurol 171: 285–318

Schiller PH, Stryker M, Cynader M, Berman N (1974) Response characteristics of single cells in the monkey superior colliculus following ablation or cooling of visual cortex. J Neurophysiol 37: 181–194

Tokunaga A, Akert K, Garey LJ, Otani K (1981) Primary and secondary subcortical projections of the monkey visual system. An autoradiographic study. Brain Res 214: 137–143

Ungerleider LG, Galkin TW, Mishkin M (1980) Visuotopic organization of striate projections to inferior and lateral pulvinar in rhesus monkey. Soc Neurosci Abstr 6: 481

Ungerleider LG, Ganz L, Pribram KH (1977) Size constancy in rhesus monkeys: Effects of pulvinar, prestriate and inferotemporal lesions. Exp Brain Res 27: 251–269

Whitlock PG, Nauta WJH (1956) Subcortical projections from the temporal neocortex in *Macaca mulatta.* J Comp Neurol 106: 183–212

Received January 4, 1982 / Accepted September 14, 1982