Neural Systems Control of Spatial Orienting

M. I. Posner; Y. Cohen; R. D. Rafal

Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences, Vol. 298, No. 1089, The Neuropsychology of Cognitive Function. (Jun. 25, 1982), pp. 187-198.

Stable URL:
http://links.jstor.org/sici?sici=0080-4622%2819820625%29298%3A1089%3C187%3ANSCOSO%3E2.0.CO%3B2-%23

Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences is currently published by The Royal Society.

Your use of the JSTOR archive indicates your acceptance of JSTOR's Terms and Conditions of Use, available at http://www.jstor.org/about/terms.html. JSTOR's Terms and Conditions of Use provides, in part, that unless you have obtained prior permission, you may not download an entire issue of a journal or multiple copies of articles, and you may use content in the JSTOR archive only for your personal, non-commercial use.

Please contact the publisher regarding any further use of this work. Publisher contact information may be obtained at http://www.jstor.org/journals/rsl.html.

Each copy of any part of a JSTOR transmission must contain the same copyright notice that appears on the screen or printed page of such transmission.

The JSTOR Archive is a trusted digital repository providing for long-term preservation and access to leading academic journals and scholarly literature from around the world. The Archive is supported by libraries, scholarly societies, publishers, and foundations. It is an initiative of JSTOR, a not-for-profit organization with a mission to help the scholarly community take advantage of advances in technology. For more information regarding JSTOR, please contact support@jstor.org.

http://www.jstor.org
Sun Jan 6 17:27:27 2008

Phil. Trans. R. Soc. Lond. B **298**, 187–198 (1982) [187]
Printed in Great Britain

Neural systems control of spatial orienting

M. I. Posner, Y. Cohen and R. D. Rafal
Department of Psychology, University of Oregon Eugene, Oregon 97403, *U.S.A.*
and Neurology Department, Good Samaritan Hospital, Portland, Oregon 97210, *U.S.A.*

A peripheral visual cue in an empty field (1) often summons head or eyes, or both, (2) improves efficiency at the cued position while attention is directed to it, even without overt movements, and (3) reduces processing efficiency at the cued position once attention is withdrawn. We have studied the time course and the effects of mid-brain and cortical damage on these components of orienting.

The facilitation arises from shifts in covert attention. In cases of mid-brain degeneration due to progressive supranuclear palsy, saccadic movements were abolished, while covert orienting still occurs. However, covert orienting was found to be delayed in directions in which eye movements were most affected, suggesting a role for mid-brain pathways in covert orienting. Parietal lesions can cause massive loss in detection contralateral to the lesion. This is especially true when attention has been directed to the opposite side. These findings relate aspects of covert orienting of attention to neural control systems.

INTRODUCTION

Perhaps the simplest of all cognitive acts involves the orienting of attention toward an event. We can direct attention to facilitate the processing of information presented at the sensory surface or of material stored in memory. Such acts are cognitive in the sense that they involve an active organism responding to environmental events in a way that is largely lost when the organism is asleep or anaesthetized. In the last several years we (Posner 1980; Posner & Cohen 1980*b*) have been studying the act of orienting by normal humans to visual stimuli occurring in a blank field in an effort to explore the component processes involved in spatial orienting. Our hope is that a fundamental grasp of these component processes will allow the establishment of useful links to underlying neural systems.

Current work in neuroscience (Mountcastle 1978; Wurtz *et al.* 1980) suggests that cells in the superior colliculus are of special importance in overt shifts of attention, while parietal lobe cells are involved more generally when attention is shifted to a visual object even without eye movements. In this paper we first outline an experimental method that has been used in normal human subjects to study the components of covert orienting and then examine the effects of midbrain and cortical damage by applying this method to populations of patients.

ATTENTION MOVEMENTS

In our experiments, the presentation of a central or peripheral cue indicating the likely location of a target produces a movement of attention in the direction of the cued location. We distinguish overt components of orienting such as postural changes or eye movements from a covert change of orienting that involves central attentional systems. Covert attention movements can be inferred by changes in the efficiency of target detection at the cued location in comparison with other locations in the visual field. Efficiency has been measured by changes in reaction time to report the presence of the target signal (Posner 1980), by the probability of detecting near-threshold stimulus events (Bashinski & Bachrach 1980; Remington 1980) or by changes in electrical activity induced by the target (Goldberg & Wurtz 1972). We have indexed these changes of efficiency in a variety of experimental situations that involve either simple detection of the presence of a luminance change or the recognition of the semantic category of the visual event (Posner 1978, 1980).

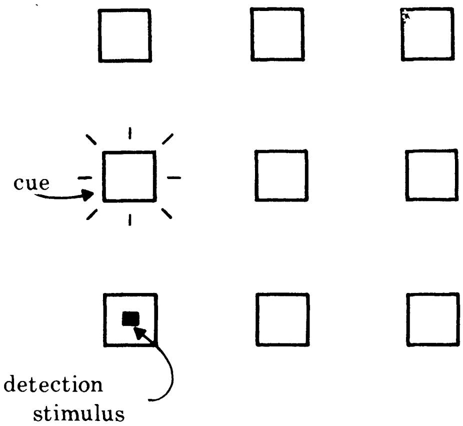

FIGURE 1. Paradigm for studying covert orienting in normal subjects. Three boxes are always present in the visual field. Subjects fixate on the centre box. At the start of the trial, one of the two peripheral boxes brightens (cue). After a specified interval a detection signal is presented in one of the boxes.

The changes in efficiency that we observe are sufficiently time-locked to cues that it seems reasonable to call them movements. For a peripheral visual cue 7° from the fovea we find that it takes approximately 50 ms for the changes in efficiency at the target location to begin and that there is a marked improvement in the efficiency at the target location over the first 150 ms after the cue.

This time-locking is shown quite exquisitely in a recent study. The basic experimental arrangement is shown in figure 1. Subjects fixated the central box (eye position was monitored by e.o.g.) and their task was to press a single key whenever a bright detection stimulus (target) appeared. Each trial began with a cue which was the brightening of one of the two peripheral boxes. In one condition the target appeared on the side that was brightened (cued side) 80 % of the time and on the side opposite to the cue 20 % of the time. In a second condition the target was on the side opposite to the cue 80 % of the time and the cued side 20 % of the time.

Results are shown in figure 2. The peripheral cue serves to summon attention in both conditions, leading to an initial advantage for the cued side. This occurs whether or not the target is likely to fall on the same side as the cue. However, in the condition where the target is unlikely to appear on cued side (triangles) the subject shifts attention to the side opposite the cue, showing an advantage there 300 ms after the cue. It might be suspected that the initial advantage to the cued side was due to brightness enhancement or some other purely sensory factor but we can show this is not so by using a peripheral cue involving dimming of the box or by the use of a purely central cue (Posner & Cohen 1980*b*).

The shifts of attention first toward the cue and then to the expected side are sufficiently time-locked that they can be observed over the 500 ms interval between cue and target. It has also been shown that probe events that occur with low probability at positions through which attention is passing become more efficient at a time intermediate between the cue and maximum facilitation at the end point for the attention movement (Shulman *et al.* 1979). This result suggests an analogue movement of facilitation from fixation toward the target position. A related finding has also suggested that attention moves between locations within a visual image in an analogue fashion (Pinker 1980). Figure 2 also illustrates that attention can be drawn from an external source or can be moved in accordance with internally generated expectations. Thus, the cue seems to draw attention toward a location even if there is a low probability of finding a target there, but the expectation of a target also has a powerful control over orienting.

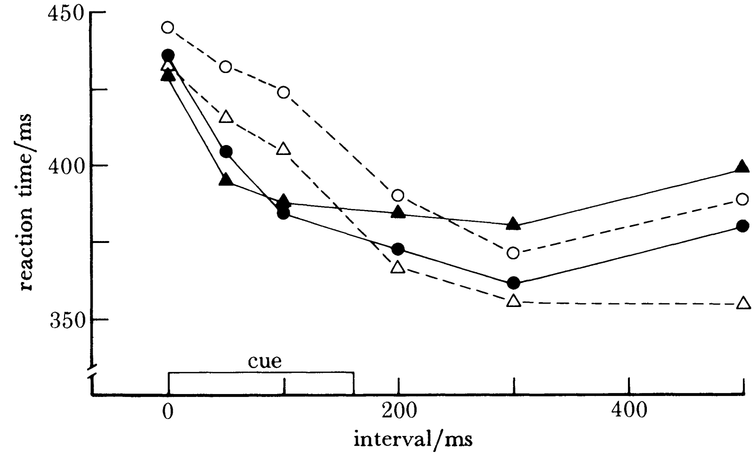

FIGURE 2. Reaction time for reporting the presence of the detection signal as a function of the interval (milliseconds) between cue and detection signal. The circles refer to a condition in which the detection stimulus is on the cued side with probability 0.8 and on the side opposite to the cue with probability 0.2. The triangles refer to a condition in which the detection stimulus is on the cued side with probability 0.2 and the side opposite to the cue with probability 0.8. Solid line, cued side; broken line, side opposite to the cue.

We have shown in separate experiments that attention will be drawn toward the cued side even when all locations are equally probable (Posner & Cohen 1980*b*). When an eye movement is summoned by a peripheral cue, attention moves to the cue approximately 200 ms before the eyes begin to move (Posner 1980). However, attention movements are not an inevitable consequence of eye movement. When the subjects can pre-programme changes of the eyes, they may do so without a shift of attention and indeed may move attention in the opposite direction of an eye movement (Posner 1980). This constellation of relations between covert orienting and eye movement provides evidence of a functional relation between the two systems, but one that can be decoupled. The systems can operate independently but, like hand and eye, usually tend to operate in close coordination. No doubt this learning occurs very early in life (Posner & Rothbart 1981) as attentional systems mature and come to control various peripheral devices such as hand and eye.

Recently, we have reported (Posner & Cohen 1980*b*) that a peripheral cue produces two opposed effects upon the information processing system. On the one hand, it summons a movement of attention that, as described previously, enhances efficiency of processing information at the target location. On the other hand, it also produces a temporary inhibition. This can be seen quite well in figure 2. At 500 ms the facilitation of a probable event on the side opposite to the cue is much greater than the facilitation of an equally probable event that occurs on the cued side. This appears to be due to the inhibition that arises when sensory information is used to summon attention.

Because the facilitation that we have described occurs from central as well as from peripheral cues, we call it attentional and see it as the result of orienting or aligning attention to a visual location. The inhibition effect, however, seems to depend upon the presentation of sensory information in the periphery because it does not arise when a central cue is presented. Because facilitation and inhibition have opposed influences upon the efficiency of information processing, tracing the time course of efficiency following a peripheral cue can only tell us about the net results of the orienting of attention and sensory inhibition. To observe a more purely inhibitory affect we summon attention away from the cued event at some time after the initial cue and then compare the efficiency of performance at the cued location with other locations of equal eccentricity in the visual field. Under these circumstances, it is clear that detection of the cued location is inhibited with respect to other locations in the visual field. There is a balance between the orienting of attention, on the one hand, and the sensory inhibition initiated by the peripheral cue, on the other. Perhaps this balance tends to prevent overcommitment of the subject's resources to a cued position and is in line with the general tendency of the nervous system to use opposed systems to maintain equilibrium.

The act of orienting attention to a location in visual space becomes more complex as we analyse its components experimentally. We are clearly not yet finished with the delineation of the components of orienting in normal subjects, even in the visual modality and less so in other sensory systems. Yet it seems important to attempt to link the results that we have so far to clinical and neuroscience observations. In particular, work in the alert monkey shows that cells in the superior colliculus, whose receptive field is in the neighbourhood of a target for an eye movement, are selectively enhanced well before the eyes begin to move (Goldberg & Wurtz 1972). Moreover, both clinical (Weinstein & Friedland 1977) and neuroscience observations (Mountcastle 1978; Robinson *et al.* 1980) argue that cells in the parietal lobe are closely related to orienting of visual attention, whether it be overt or covert. If these systems are indeed related to the mechanisms that we have been describing, it should be possible to observe their disruption in cases of insults to mid-brain and cortical structures. For the last year, we (Rafal *et al.* 1981; Walker *et al.* 1982) have been engaged in such an effort.

MID-BRAIN CONTROL

To further our understanding of the relation between movements of the eyes and covert facilitation, we have studied patients who suffer from a form of Parkinsonism called progressive supranuclear palsy (PSP) (Goodale 1980; Rafal & Grimm 1981; Rafal *et al.* 1981). In addition to the clinical and pathological abnormalities seen in Parkinson's disease, in PSP the superior colliculus, pretectum, periaqueductal grey and mesencephalic raphe degenerate, and clinically there is a distinctive opthalmoplegia with loss of voluntary saccades while vestibulo-ocular reflexes remain intact. Further, the oculomotor deficit is characteristically more severe in the vertical than in the horizontal plane; thus in some of these patients it is possible to compare performance in dimensions in which the eyes cannot be moved voluntarily with dimensions that show less severe impairment. To provide confidence that we could link any deficits found in covert orienting specifically to the mesencephalic lesions causing the opthalmoplegia (rather to some other clinical or pathologic abnormality that PSP shares with Parkinson's disease), we also studied four Parkinson's disease patients as controls, both off and on medication, in the same experiments.

In our first experiment (PSP no. 1) we studied three PSP patients. In this study we provided a long lasting visual cue (500 ms) followed by a target to which the subject has to respond. Cues were a 1° hexagon presented 8° above or below fixation in the vertical condition and 10° to the left or right of fixation in the horizontal condition. The target remained present for 1 s after presentation and we waited 5 s for a response before proceeding to the next trial. The target was presented on the cued side on 80 % of the trials (valid trials) and on the side opposite to the cue in 20 % of the trials (invalid trials). Details of the intervals between cue and probe are shown in figure 3. Each subject was run for several sessions of approximately 200 trials in each condition (horizontal and vertical).

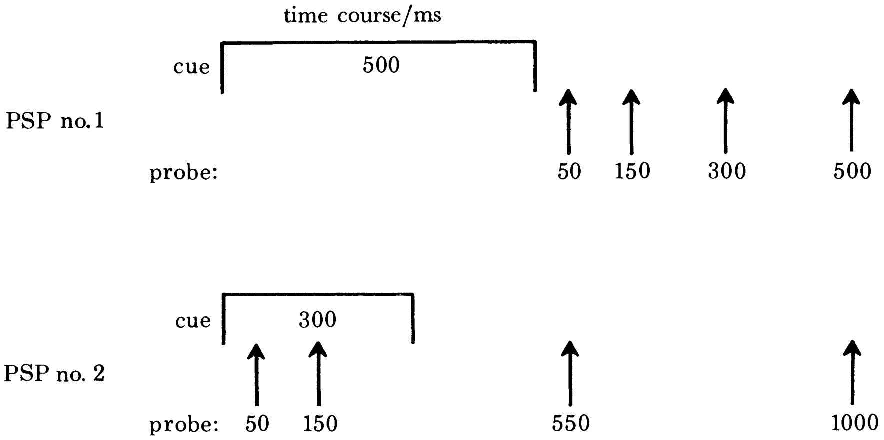

FIGURE 3. Paradigm used for the study of mid-brain and cortical patients. In PSP no. 1 the cue was left present in the field for 500 ms before presenting any detection stimuli (probes). In PSP no. 2 the detection stimuli (probes) are presented at short and long intervals following the cue onset.

The results of this study were simple and clearcut. Reaction times differed greatly between the three subjects ranging from speeds that would be roughly normal for this age range (500 ms) to reaction times more than twice normal. In general reaction times were slightly faster in the vertical than in the horizontal direction, but this result turned out to be accounted for by the differences in the eccentricities of the targets and appear to be equivalent when the same eccentricity is used. Each subject showed a clear advantage of the cued side over the side opposite to the cue at the earliest probe position studied that tended to decline over the interval (see figure 4). The initial advantage of the cued side was significant in each patient, and there was no overall interaction between direction (vertical against horizontal) and the magnitude of the advantage of the cued side. The results showed quite clearly that patients unable to make saccades in the vertical direction could still move covert attention in that direction. A more detailed analysis of the data of each patient suggested that there were longer reaction times when the cue was presented in the direction in which eye movements were relatively intact followed by a target in the direction in which eye movements were severely impaired. This suggested to us that there might be impairments in the latency of covert orienting in directions that showed the strongest eye movement problems.

For this reason we designed a second study (PSP no. 2) in which we presented a peripheral cue for 300 ms followed by targets occurring 50, 150, 550 and 1000 ms after the cue's onset (see figure 3). This allowed us to compare the speed of covert orienting in the horizontal and vertical direction. Otherwise the study was the same as outlined above. Four patients were studied, one of whom had been in the original experiment. The results of this study are somewhat more complex than in the previous study in that different patients had different patterns of eye movement and covert orienting deficits. The overall results (see figure 5) indicate that covert orienting was quite rapid in the horizontal direction. An advantage of the cued side is present at 50 ms and remains present over the entire interval. In the vertical direction there is no clear advantage of the cued side over the opposite side until 1000 ms after the cue.

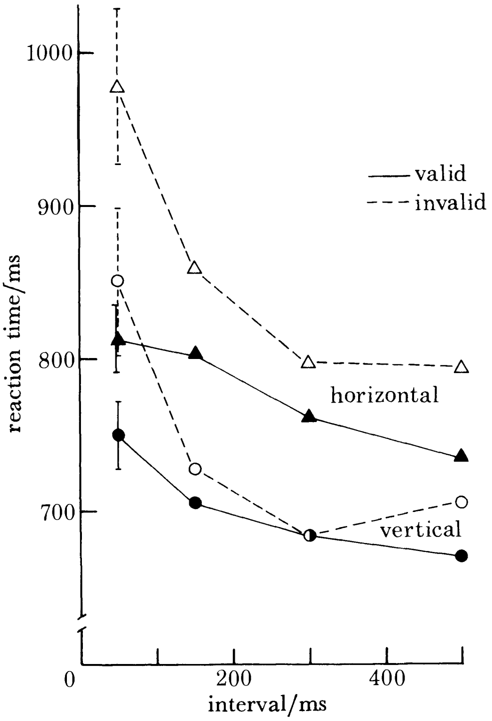

FIGURE 4. Reaction time as function of the interval between cue and detection stimulus (probe) presented in the vertical and horizontal dimensions. The valid side (solid lines and symbols) is the one that had the cue and on which 0.8 of the detection stimuli occur. Data from PSP no. 1. Bars indicate one standard error.

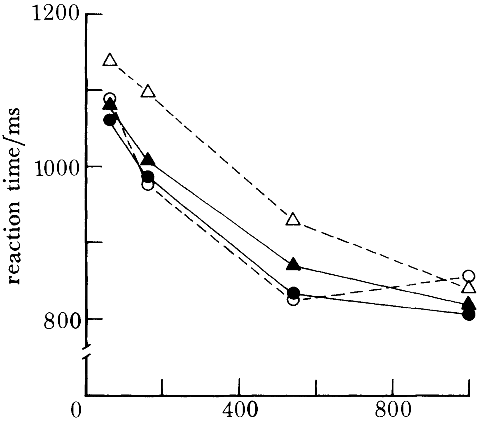

FIGURE 5. Reaction time as a function of the interval between cue and detection stimulus (probe) for stimulus presented in the vertical (circles) and horizontal (triangles) dimensions. The valid side (solid lines and symbols) is that the one that had the cue and on which 0.8 of the detection stimuli occur. Data are from PSP no. 2.

It is instructive to examine the one patient who had been in both studies and whose reaction times were quite short. She has a complete inability to move her eyes voluntarily in the vertical direction. She showed some slowing of rightward horizontal movement, but leftward movements were unimpaired as far as we could tell. Her reaction time results indicated a clear advantage of the cued side by 150 ms in the horizontal direction, but not until 550 ms in the vertical direction In the vertical direction she showed a disadvantage on the cued side at 50 and 150 ms. We also used this subject in an experiment with a central rather than a peripheral cue. Once again she showed much faster effects of the cue in the horizontal than in the vertical direction. In this patient there was no doubt that she could orient to cues in either direction, since she had demonstrated this in the first experiment. However, there were systematically longer times required to demonstrate covert orienting in the direction in which saccades were most affected.

It is useful to compare the data of the PSP patients with those obtained from four Parkinson patients (Walker *et al.* 1982) in the same task. The Parkinson patients had rather short reaction times generally and were similar in overall level of performance to the fastest PSP patient. However, the Parkinson patients show an advantage of the cued over the side opposite to the cue by 50 ms after input. The data of these four patients are quite similar to those found in normal young subjects. Neither the normals nor the Parkinson patients show any difference between covert orienting in the vertical and horizontal directions. Moreover, the Parkinson patients show no difference in the speed of covert orienting whether they are medicated or not. This finding suggests that the substantial impairment in mid-brain damage found in common between PSP and Parkinsonism does not contribute to the deficit in spatial orienting found in PSP patients. This result and the difference found in PSP patients between the two directions of orienting suggest that their problem is related to the unique part of the PSP syndrome that results in impairment of the eye movement system. Presumably this relates to the mesencephalic lesions that produce the distinctive ophthalmoplegia in the PSP patients.

Another result favouring a role for mid-brain mechanisms in the control of covert orienting in human beings has been reported by using somewhat similar techniques by Holtzman *et al.* (1981). Their patients had surgery that disconnected the two cerebral hemispheres. When either hemisphere was provided information about the place that a letter or digit would be presented, reaction time improved to the cued position and slowed for the uncued positions. This occurred irrespective of whether the target was given to the same hemisphere that had received the cue or to the opposite hemisphere. Since these studies used a long time between cue and target, we do not know how rapid the speed of covert orienting is in these patients.

We have summarized observations that implicate mid-brain pathways in the control of covert visual orienting. The data from PSP patients indicate that mid-brain damage specific to their ability to make saccades interferes with the latency of covert orienting. Despite the widespread character of the lesions in these patients the difference between the vertical and horizontal dimensions and the lack of interference with covert orienting in Parkinson's patients both suggest that the effects of covert orienting latency are from the specific systems that are involved with vertical eye movements. The role of mid-brain systems in aspects of orienting is supported by findings from patients with commissurotomy (Holtzman *et al.* 1981) and from blind sight (see Weiskrantz 1980).

However, even the extensive damage to voluntary eye movement found in PSP patients does not abolish their covert orienting ability even in the vertical direction. This fact suggests that other pathways are also important in the control of covert orienting. Such a conclusion also follows from important dissociations between eye movement control and the control of covert orienting that we have found in normals. For example, eye movements are more heavily influenced by temporal than by nasal visual input (Posner & Cohen 1980*a*), but the temporal field has no similar dominance over temporal order judgements (Posner & Cohen 1980*a*) or the direction of covert orienting (Shulman 1979). These findings suggest important dissociations between eye movement systems and those controlling covert orienting and conscious awareness of the stimulus event.

Cortical control

It has long been known that parietal lesions will sometimes lead to an inability to be aware of a stimulus opposite the lesion if it is presented at the same time as an event ipsilateral to the lesion (extinction) (Weinstein & Friedland 1977). Studies on alert monkeys have suggested that cells in area 7 are enhanced when the monkey attends to a stimulus even if he does not move his eyes toward it (Robinson *et al.* 1980). These studies have led to the idea that area 7 may be related to important aspects of spatial attention (Mountcastle 1978; Wurtz *et al.* 1980).

We have examined six patients suffering from insult to the right parietal lobe due either to vascular or to tumour processes. Five of these six patients were diagnosed clinically as having extinction and visual neglect without hemianopsia. We have attempted to determine the relation between the lesions and these clinical observations.

Our experiments were conducted in the same way as described before (see figure 3). Our results are preliminary but they do suggest very close relationships between the clinical syndrome and the attentional orienting mechanisms that we have been discussing in this paper. Since valid trials occurred 80 % of the time, the results for these trials are most complete. For five patients, information going directly to the undamaged hemisphere is faster at all intervals studied than information going directly to the damaged hemisphere. The one exception was the patient who was not diagnosed as having visual neglect.

For two of our patients, the data are complete enough to compare valid and invalid trials. Consider the first patient, R.S., who suffered from a tumour to the right parietal lobe which on computed tomography scan appeared to involve most of the right parietal lobe. The optic radiations and thalamic area were spared.

His data are shown in figure 6. For valid trials (solid lines) this patient showed a small but consistent advantage to targets occurring on the side ipsilateral to the lesion over those occurring contralateral to the lesion. The time between the cue and the target did not affect this relation strongly suggesting that the results were not mediated by overt eye movements. We cannot be certain of this, however, because it is very difficult to determine the degree to which the eyes are correctly fixated in these patients.

We can first examine the relation between valid and invalid trials where the target occurs ipsilateral to the lesion. There is a clear validity effect that emerges 550 ms after the cue. This is relatively slow compared with a validity effect in normal subjects. The validity effect is of a size that approximates normal.

The results are stirkingly different when targets are presented on the side contralateral to the damaged hemisphere. After a valid cue, reaction times are only somewhat longer than those after a valid cue to the undamaged side, but when attention is to the non-neglected field and a target occurs on the neglected side, there is massive interference with the processing efficiency of the target information. At short intervals, the interference is so great that the patient usually does not detect the target within the 5 s allowed. This represents a confirmation of the classical extinction phenomena where on double simultaneous stimulation the subject is unable to report information contralateral to the lesion. However, even with intervals of 1000 ms, so that the cue has been gone from the field for more than 700 ms, there is still a massive interference with reaction time.,R.S. generally detects the presence of the target, but he is extremely slow in doing so.

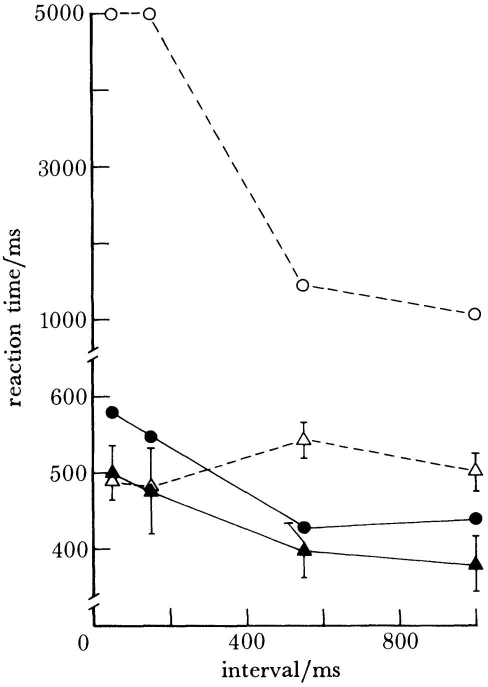

FIGURE 6. Median reaction time for valid (solid lines) and invalid (broken lines) trials for one right parietal patient (R.S.). Bars indicate one standard error from the median. The *Y* axis is broken between 600 and 1000 ms to allow data to be shown conveniently. A median reaction time of 5000 ms indicates that most targets were not responded to within the allowed interval. ●, ○, Left target; ▲, △, right target.

Let us now examine the other patient, not diagnosed as suffering from visual neglect but who had a form of extinction on double simultaneous stimulation in the tactile domain. Valid trials for this patient showed no difference between the damaged and undamaged side in reaction time. However, like R.S., an invalid trial in which the target is presented to the side opposite the lesion produces a very powerful interference effect. When the interval is short, many targets are missed and the interference shows a very gradual reduction with this interval but still remains present to the longest time studied.

Of particular interest is a finding that we made in patient R.S. that has not yet been documented in other patients. In this condition, we used a central cue that was an arrow at fixation extending less than $\frac{1}{4}$° across the mid-line with the arrowhead pointing either to the right or left of fixation. The patient is instructed to orient attention in the direction of the arrow because 0.8 of the targets occurred on the cued site. For valid trials we get similar results to those obtained with the peripheral cue. He was faster on the side ipsilateral to the lesion that contralateral to the lesion. For invalid trials, we also get the same result as obtained with the peripheral cue. Thus, when the physical event is a central arrow that instructs orienting rather than a stimulus on the contralateral side, one still obtains evidence for extinction. This result suggests that extinction does not depend upon the physical cue but on the way in which it directs central attention.

These results are still fragmentary but they do suggest important relations between damage at the parietal lobe and covert attention as we have measured it. According to the theory that has been developed from the study of normals, the delay in processing an event on the uncued side is due to a slowing in the time for that event to enter a general attentional system responsible for detecting the event. The fact that under conditions where the uncued event is slowest one can find a complete inability to be aware of the event in some parietal patients provides support for the idea that such latency shifts are a sign of systems responsible for awareness in normal persons. They also confirm many findings documented in the clinical literature but add quantitative details to them and provide a better basis for comparison with mid-brain patients and normal persons than has been available through clinical observation.

Conclusions

These studies have attempted to dissect the simple act of cognitive orienting into a set of time-locked facilitatory and inhibitory components. We have been able to show that some of these components are affected by brain injury at mid-brain and cortical levels. The details of mid-brain and cortical effects are different. At the level of the mid-brain, only the latency of covert orienting seems to be affected. Targets are uniformly detected and there is no evidence of extinction from a misdirection of attention. Patients are able to orient attention in directions to which they cannot move their eyes voluntarily. None the less, the latency changes we found are accompanied by certain clinically observable changes in the natural orienting behaviour of these patients (Rafal & Grimm 1981). For example, they often fail to turn spontaneously toward individuals who approach them, even though they may be quite able to do so on command.

In contrast, the parietal lesions produce a massive loss of information from the field opposite the lesions in cases where the subject's attention is directed away from the target. Extinction to double simultaneous stimulation may be seen as a limiting condition in which attentional shifts occur in close temporal proximity to the target event. It seems to be the attentional shift and not the simultaneous occurrence of the cue and target that produces extinction.

Experiments have shown quite clearly that the orienting mechanisms described in this paper are important in high-level cognitive activity. For example, Chang (1981) was able to show that such covert orienting mechanisms are intimately involved in increasing the sensitivity of the visual field to the right of fixation during reading normal English and to the left when English is read upside-down. Treisman & Gelade (1980) have argued for the importance of such covert mechanisms in tying together features of visual objects. Pinker (1980) believes similar covert orienting effects to be involved in our attending to parts of the three-dimensional visual field and to memory images of such fields. Thus, from the cognitive point of view, attentional orienting has been shown to be an important feature of complex human activity.

The experiments that we describe here seem to be capable of linking such orienting mechanisms to underlying neural systems at both mid-brain and cortical levels. Our results suggest that both mid-brain and cortical sites are important in the efficient execution of covert orienting. Taking our results together with those of Goldberg & Wurtz (1972), it appears that when tasks involve eye movement, attention produces facilitation at the level of the superior colliculus.

Although Goldberg & Wurtz are unable to find evidence for selective enhancement when the task does not involve eye movements, it is still possible that mid-brain systems affect the ability of higher centres to shift attention to a cue. Our findings and those of Holtzman *et al.* (1981) argue that they do. Similar issues arise at the level of the parietal lobe. Our results suggest that attention vastly improves the efficiency of the damaged parietal lobe. Some neural system is producing the enhanced efficiency due to a cue. It seems unlikely that the strong enhancement is due to the damaged parietal lobe.

It might be argued that attention to spatial information involves an entirely different attention system than is used for language information. In this view, attention in the spatial domain might be seen as an independent module. Our results argue against this view. When peripheral cues are presented in tasks when the subject's attention is occupied by a central language task, (i.e. counting backward by threes), the cues do not produce normal facilitation (G. R. J. Hockey & M. I. Posner 1980, unpublished). This result shows that spatial attention draws upon at least some of the internal systems that are involved in attention to language and thus argue against interpreting spatial attention experiments as an entirely separate domain of attention. We suggest from this that attention itself is probably not a strictly parietal function, but is mediated by the parietal system when it involves certain tasks like spatial orienting. Perhaps a close examination of the ability of the subject to execute commands to shift attention in the visual field will provide an indication of the system that serves as the source of the attentional effects that we have observed.

In any case, both the time-locking of spatial attention to cues and the ability to show effects of lesions at various levels of the nervous system suggest that the method that we have been using provides a tool useful in obtaining a more complete understanding of the internal mechanisms underlying at least one important cognitive function.

This research was supported in part by a grant from the National Science Foundation, grant no. BNS 79-23527, to the University of Oregon and in part by a grant from the Murdock Foundation to the Neurological Sciences Center, Good Samaritan Hospital, Portland, Oregon. The authors are grateful to Dr O. S. M. Marin for his support and assistance in this research.

REFERENCES (Posner *et al.*)

Bashinski, H. S. & Bachrach, V. R. 1980 Enhancement of perceptual sensitivity as the result of selectively attending to spatial locations. *Percept. Psychophys.* **28**, 241–248.

Chang, F. R. 1981 Distribution of attention within a single fixation in reading: studies of the perceptual span. Ph.D. thesis, University of Oregon.

Goodale, M. A. 1980 Visuomotor deficits in patients with progressive supranuclear palsy. Paper delivered to the International Neuropsychological Society, San Francisco.

Goldberg, M. E. & Wurtz, R. H. 1972 Activity of superior colliculus in behaving monkeys. II. Effect of attention on neuronal responses. *J. Neurophysiol.* **35**, 560–574.

Holtzman, J. D., Sidtis, J. J., Volpe, B. T., Wilson, D. H. & Gazzaniga, M. S. 1981 Dissociation of spatial information for stimulus localization and control of attention. *Brain*, **104**, 861–872.

Mountcastle, V. B. 1978 Brain mechanisms for directed attention. *Jl R. Soc. Med.* **71**, 14–27.

Pinker, S. 1980 Mental imagery and the third dimension. *J. exp. Psychol.: gen.* **109**, 354–371.

Posner, M. I. 1978 *Chronometric explorations of mind: the third Paul M. Fitts lectures.* Hillside, N.J.: Lawrence Erlbaum Associates.

Posner, M. I. 1980 Orienting of attention. *Q. Jl exp. Psychol.* **32**, 3–25.

Posner, M. I. & Cohen, Y. 1980*a* Attention and the control of movements. In *Tutorials in motor behavior* (ed. G. E. Stelmach & J. Requin), pp. 243–258. Amsterdam: North-Holland.

Posner, M. I. & Cohen, Y. 1980*b* Consequences of visual orienting. Paper presented to the Psychonomics Society, November 1980.

Posner, M. I. & Rothbart, M. 1981 The development of attentional mechanisms. In *Nebraska Symposium on Motivation* (ed. J. H. Flowers), pp. 1–52. Lincoln, Nebraska: University of Nebraska Press.

Rafal, R. D. & Grimm, R. J. 1981 Progressive supranuclear palsy: functional analysis of the response to methysergide and anti-Parkinson agents. *Neurology*. **31**, 1507–1518.

Rafal, R. D., Posner, M. I. & Walker, J. A. 1981 Spatial orienting and visually guided behavior: clinical and experimental observations in patients with progressive supranuclear palsy. Paper presented to the International Neuropsychology Society, Atlanta.

Remington, R. W. 1980 Attention and saccadic eye movements. *J. exp. Psychol.: hum. Percept. Perform.* **6**, 726–744.

Robinson, D. L., Goldberg, M. E. & Stanton, G. B. 1980 Parietal association cortex in the primate: sensory mechanisms and behavioral modulations. *J. Neurophys.* **41**, 910–932.

Shulman, G. L. 1979 Spatial determinants of attention allocation. Ph.D. thesis, University of Oregon.

Shulman, G. L., Remington, R. W. & McLean, J. P. 1979 Moving attention through visual space. *J. exp. Psychol.: hum. Percept. Perform.* **5**, 522–526.

Treisman, A. & Gelade, G. 1980 A feature integration theory of attention. *Cogn. Psychol.* **12**, 97–136.

Walker, J. A., Posner, M. I. & Rafal, R. D. 1982 Separation of effects on cognition versus motor performance due to the basal ganglia dysfunction of Parkinson patients. Presented at International Neuropsychological Society, Pittsburg.

Weinstein, E. A. & Friedland, R. P. 1977 *Hemi-inattention and hemispheric specialization.* New York: Raven Press.

Weiskrantz, L. 1980 Varieties of residual experience. *Q. Jl exp. Psychol.* **32**, 365–386.

Wurtz, R. H., Goldberg, M. E. & Robinson, E. L. 1980 Behavioral modulation of visual responses in the monkey: stimulus selection for attention and movement. *Prog. Psychobiol. physiol. Psychol.* **9**, 43–83.