

DOCUMENT RESUME

ED 288 903

TM 870 691

AUTHOR Posner, Michael I.  
TITLE Structures and Functions of Selective Attention.  
INSTITUTION Washington Univ., St. Louis, Mo.  
SPONS AGENCY Office of Naval Research, Arlington, Va. Personnel and Training Research Programs Office.  
REPORT NO ONR-87-5  
PUB DATE Jul 87  
CONTRACT N0014-86-K-0289  
NOTE 59p.; Paper presented at the Annual Meeting of the American Psychological Association (New York, NY, August 28-September 1, 1987).  
PUB TYPE Speeches/Conference Papers (150) -- Reports - Research/Technical (143)  
EDRS PRICE MF01/PC03 Plus Postage.  
DESCRIPTORS Attention; \*Attention Deficit Disorders; Medical Research; \*Neurological Impairments; \*Neurological Organization; Neurology; \*Receptive Language; Schizophrenia; \*Visual Perception  
IDENTIFIERS \*Brain Functions; Brain Research; Neuropsychology

ABSTRACT

While neuropsychology relates the neural structures damaged in traumatic brain injury with their cognitive functions in daily life, this report reviews evidence that elementary operations of cognition as defined by cognitive studies are the level at which the brain localizes its computations. Orienting of visual attention is used as a model task. The component facilitations and inhibitions in visual orienting are related to neural systems through the study of focal neurological lesions. Visual orienting is a part of a more general selective attention system that also involves orienting to language. Ability to be aware of and to act upon target events depends upon the connections of posterior orienting systems to anterior systems involved in target detection. These pathways have been examined in studies of focal changes in cerebral blood flow during performance of language tasks. Although there is not available a general analysis of the mental operations performed by these anterior systems, there is some evidence relating the dorsolateral prefrontal and areas of the medial surface to aspects of focal selection. To study the generality of the attentional system developed in this report, the putative deficits of attention in disorders such as schizophrenia are examined. (Author/MDE)

\*\*\*\*\*  
\* Reproductions supplied by EDRS are the best that can be made \*  
\* from the original document. \*  
\*\*\*\*\*

ED288903

**STRUCTURES AND FUNCTIONS OF SELECTIVE ATTENTION**

**Michael I. Posner  
Washington University, St. Louis**

**ONR 87-5**

**Research sponsored by:** Personnel and Training Research Program,  
Psychological Sciences Division,  
Office of Naval Research  
**Under Contract Number:** N0014-86-K-0289  
**Contract Authority Number:** NR-442a554

**Reproduction in whole or part is permitted for any  
purpose by the United States Government.**

**U.S. DEPARTMENT OF EDUCATION  
NATIONAL INSTITUTE OF EDUCATION  
EDUCATIONAL RESOURCES INFORMATION  
CENTER (ERIC)**

**+ This document has been reproduced as  
received from the person or organization  
originating it**

**( ) Minor changes have been made to improve  
reproduction quality**

**• Points of view or opinions stated in this docu-  
ment do not necessarily represent official NIE  
position or policy**

**BEST COPY AVAILABLE**

169 018 W  
M 870 691

REPORT DOCUMENTATION PAGE

1a. REPORT SECURITY CLASSIFICATION <b>Unclassified</b>		1b. RESTRICTIVE MARKINGS	
2a. SECURITY CLASSIFICATION AUTHORITY		3. DISTRIBUTION / AVAILABILITY OF REPORT <b>Approved for public release; Distribution unlimited.</b>	
2b. DECLASSIFICATION / DOWNGRADING SCHEDULE		4. PERFORMING ORGANIZATION REPORT NUMBER(S) <b>Technical Report #87-5</b>	
4. PERFORMING ORGANIZATION REPORT NUMBER(S) <b>Technical Report #87-5</b>		5. MONITORING ORGANIZATION REPORT NUMBER(S)	
6a. NAME OF PERFORMING ORGANIZATION <b>Washington University</b>	6b. OFFICE SYMBOL <i>(if applicable)</i>	7a. NAME OF MONITORING ORGANIZATION <b>Personnel &amp; Training Research Programs Office of Naval Research (Code 1142PT)</b>	
6c. ADDRESS (City, State, and ZIP Code) <b>Department of Neurology 660 S. Euclid, Box 8111 St. Louis, MO 63110</b>		7b. ADDRESS (City, State, and ZIP Code) <b>800 North Quincy Street Arlington, VA 22217</b>	
8a. NAME OF FUNDING / SPONSORING ORGANIZATION	8b. OFFICE SYMBOL <i>(if applicable)</i>	9. PROCUREMENT INSTRUMENT IDENTIFICATION NUMBER <b>N00014-96-KO289</b>	
8c. ADDRESS (City, State, and ZIP Code)		10. SOURCE OF FUNDING NUMBERS	
		PROGRAM ELEMENT NO <b>61153N</b>	PROJECT NO. <b>RR04206</b>
		TASK NO. <b>RR04206-OA</b>	WORK UNIT ACCESSION NO. <b>NR442a554</b>
11. TITLE (Include Security Classification) <b>Structures and Functions of Selective Attention</b>			
12. PERSONAL AUTHOR(S) <b>Posner, Michael I.</b>			
13a. TYPE OF REPORT <b>Technical</b>	13b. TIME COVERED FROM <b>01MAY87</b> TO <b>01MAY88</b>	14. DATE OF REPORT (Year, Month, Day) <b>July 20, 1987</b>	15. PAGE COUNT <b>46</b>
16. SUPPLEMENTARY NOTATION			
17. COSATI CODES		18. SUBJECT TERMS (Continue on reverse if necessary and identify by block number)	
FIELD <b>05</b>	GROUP <b>10</b>		
SUB-GROUP			
19. ABSTRACT (Continue on reverse if necessary and identify by block number)			
20. DISTRIBUTION / AVAILABILITY OF ABSTRACT <input checked="" type="checkbox"/> UNCLASSIFIED/UNLIMITED <input checked="" type="checkbox"/> SAME AS RPT <input type="checkbox"/> DTIC USERS		21. ABSTRACT SECURITY CLASSIFICATION <b>Unclassified</b>	
22a. NAME OF RESPONSIBLE INDIVIDUAL <b>Michael I. Posner</b>		22b. TELEPHONE (Include Area Code) <b>(314) 362-3317</b>	22c. OFFICE SYMBOL <b>ONR 1142PT</b>

# Structures and Functions of Selective Attention<sup>\*,1</sup>

Michael I. Posner

University of Oregon, Eugene and  
McDonnell Center for Higher Brain Function  
Washington University School of Medicine, St. Louis

## Abstract

A principle problem of neuropsychology is to relate the neural structures damaged in traumatic brain injury with their functions in the cognitive tasks of daily life. This lecture reviews evidence that elementary operations of cognition as defined by cognitive studies are the level at which the brain localizes its computations. Orienting of visual attention is used as a model task. The component facilitations and inhibitions in visual orienting are related to neural systems through the study of focal neurological lesions.

Visual orienting is a part of a more general selective attention system that also involves orienting to language. Our ability to be aware of and to act upon target events depends upon the connections of posterior orienting systems to anterior systems involved in target detection. We have examined these pathways in studies of focal changes in cerebral blood flow during performance of language tasks. Although we do not have a general analysis of the mental operations performed by these anterior systems, there is some evidence relating the dorsolateral prefrontal and areas of the medial surface to aspects of focal selection.

One way to study the generality of the attentional system developed in this lecture is to examine putative deficits of attention in disorders such as schizophrenia, depression and closed head injury where the organic basis for the deficit is largely unknown. Our preliminary studies of schizophrenia are used to support the utility of the joint functional and structural analysis proposed here.

\* This is a draft of a lecture to be given as the Master Lecture in the Neuropsychology of Attention at the American Psychological Association meeting, New York, August, 1987. To appear in a volume on Neuropsychology to be published by APA.

The central problem of neuropsychology is to understand the relationship between everyday life performance and the neurosystems that support it. On the one hand, clinical neuropsychologists are faced with neuroimages that provide a picture of the locations of lesions and on the other hand, they must discuss with patients, relatives and insurance companies, likely deficits in performance that will be seen in daily life.

Among the many deficits found in brain injury is the ability to maintain performance in the face of competing information. This requires selection of information among competing events. Selective attention is an old topic within experimental psychology (James, 1890; Titchener, 1908) and most frequently refers to performance when there are conflicts between signals. Attention has the role of selecting some signals for higher levels of processing, including conscious processing, while preventing access of other signals to those same high levels of processing. Selective attention plays an important role in most cognitive tasks including pattern recognition, reading and mental imagery (see Posner, 1982 for a historical review).

During the last dozen years it has been possible to work out some aspects of the neural structures involved in selective attention based upon work with humans (Posner, Walker, Friedrich and Rafal, 1984) and alert monkeys (Mountcastle, 1978; Wurtz, Goldberg & Robinson, 1980). The research has been accomplished by many different investigators, but studies have used similar tasks and have been constrained by our increased understanding of the anatomy of the visual system (Covey, 1985) and to some extent of the frontal lobes (Goldman-Rakic, in press). Thus, something of a common overall view has begun to emerge despite remaining conflicts and uncertainties. Edited volumes have summarized this work from anatomical, physiological, neuropsychological and cognitive perspectives (see Posner & Marin, 1985; Berluchi & Rizzolatti, 1987).

The work on spatial attention may serve as a useful model for understanding the way in which cognition is represented within the nervous system. It already has provided a basis for understanding some functions of selective attention such as visual pattern recognition (Treisman, 1987; Prinzmetal, Presti & Posner, 1986) including the recognition of visual words (Posner & Presti, 1987). Research on spatial attention provides a basis for understanding deficits of attention found in such diverse disorders as schizophrenia, depression and closed head injury.

This paper first traces a general framework for connecting cognitive and neural systems of selective attention. Next it reviews effects of unilateral brain lesions on the cognitive operations of visual spatial orienting. Studies discussed in this section show how damage to this system affects pattern recognition. Next evidence relating attention to language and attention to visual locations is reviewed in order to construct a general picture of the structure of the attention system. Finally, our knowledge about the structure and function of attention is applied to a condition whose organic basis is unknown - schizophrenia.

## I. Framework

It is useful to view the connection between cognitive systems and neurosystems in terms of a very general framework (Posner, 1986). This framework involves five levels of analysis shown in Fig. 1. At the highest

level are tasks of daily life. Cognitive scientists have developed a number of computational models for tasks such as visual imagery (Kosslyn, 1980), reading (Rumelhart & McClelland, 1981) and typewriting (Rumelhart & Norman, 1982). These tasks provide a view of the computations necessary for any electromechanical system to perform the cognitive tasks described. Some of these computational models consist of subroutines that operate on symbolic representation, labeled here as "elementary operations". They resemble the types of operations studied by cognitive experiments of the last twenty years (Posner, 1978). Each operation can be specified in terms of the input to the operation and its output. Sample operations include match, store, zoom, compare, engage and move. These operations sometime serve as labels on the box models of information flow that dominate textbooks of cognitive psychology.

### FIG. 1

In recent years a new form of computational model has arisen in several areas of psychology (see McClelland & Rumelhart, 1986 for a review). These parallel distributed or connectionist models do not discuss performance in terms of elementary operations directly but instead refer to facilitations and inhibitions between levels. Fortunately, as we have learned to measure elementary operations in chronometric experiments, we find that they can be specified in terms of component facilitations and inhibitions in performance. My own work has often attempted to describe mental operations in terms of these time-locked facilitations and inhibitions (Posner, 1978; Posner & Snyder, 1975) in reaction time. Methods for making such measurements have been described and have been widely applied (Jonides & Mack, 1984; Neely, 1986; Posner, 1978; Taylor, 1977). A great deal is known about how such measures can be taken and what pitfalls there are in using them (Jonides & Mack, 1984). The use of the words "facilitation" and "inhibition" in connectionist models and in the description of the components of elementary mental operations are biased to make one inquire as to whether such patterns are related to the activity of populations of nerve cells that might perform the computation. To what extent do our findings on facilitation and inhibition in the performance domain and in connectionist models relate to changes in populations of nerve cells? This relationship is a central question for the neuropsychology of cognition. If it is possible to move from the level of facilitation and inhibition in performance to the level of neurosystems, one can then see how it is possible to go from an understanding of lesions in an area of the nervous system to predictions about normal cognition.

The study of visual spatial attention has been extremely important to this enterprise. Visual spatial attention can be studied in people and in alert monkeys. The presence of an animal model provides opportunity to determine whether results obtained from performance studies of normal humans converge with those using single cell methodology. In so far as this link can be established, it is possible to move from the general study of neurosystems, as can be done in human beings by studies described here, to the study of individual nerve cells.

## II. Facilitation and Inhibition in Visual Spatial Attention

In attempting to work out a complex system like selective attention, it is important to study experimental situations or "model tasks" that define what we

mean by the phenomenon and allow us to study it in simple forms. An important aspect of selective attention is orienting to a source of visual signals. In this area, similar model tasks have been used in studies of animals, normal humans and patients (Posner & Marin, 1985; Berluchi & Rizzolatti, 1987). The importance of studying covert shifts of attention is the hope that the mechanisms involved in these shifts of attention will help us understand more general problems of selectivity in other modalities and in memory.

A very simple model task is illustrated in Fig. 2. The subject is cued to shift attention covertly to a visual location eccentric of where the eyes are currently fixed. In these experiments the cue may be an event that occurs at the location to which the subject is to attend (brightening of a box) or it may be a symbolic instruction (e.g. arrow at fixation) that informs the subject where to shift attention.

### Fig. 2

Many experiments have now been performed with both of these forms of cueing. The results for tasks illustrated in Fig. 2, uniformly show an advantage for the cued location over the uncued location that is closely time-locked to the occurrence of the cue. This relative facilitation has been measured in reaction time (Posner, 1980), probability of correct detections for near threshold stimuli (Bashinski & Bachrach, 1980), and increased electrical activity at the cued in comparison with the uncued location (Mangun, Hansen & Hillyard, 1986).

Two interrelated issues in interpretation of these findings remain in dispute. First, is the relative facilitation of the cued location a genuine improvement of information coming from the cued location or a reduction or inhibition of information coming from all other locations? Second, is the extent of the facilitated area. Most experiments seem to be consistent with the idea that facilitation occurs not only at the attended location but also in a gradient over a range of adjacent locations (Downing & Pinker, 1985, Rizzolatti, Riggio, Dascola, & Umiltà, 1987). The size of the area facilitated depends in part on the degree of eccentricity from the fovea and in part upon the complexity of the information in the visual field. How large a part of the visual field is represented by the focus of attention? This has been a widely disputed issue. For example, Hughes & Zimba (1985), have argued that attention acts simply by inhibiting the hemifield to which one is not attending. Others have found a facilitation localized to the neighborhood of the target and increasing with eccentricity from the fovea, with an inhibition stronger once one has crossed the midline (Downing & Pinker, 1985). These disputes indicate the complexity of the overlapping processes that accompany a shift of attention.

My basic approach to these complexities has been to develop a functional model that can both account for these findings and conforms to other properties associated with attention. According to this functional viewpoint (Posner & Cohen, 1984), three basic components are involved when attention is summoned by a cue located in the neighborhood of a likely target. These components combine to determine the net increase in efficiency at the cued location. First, the cue increases alertness because a target is now expected. It is known from previous work that alertness is not spatially selective (Posner, 1978) and

works to potentiate all targets following the cue. Second, the cue initiates a spatially selective movement of visual attention to the cued location. Such attention shifts are not fully automatic in sense of being unavoidable (Posner, Cohen, Choate, Hockey & Maylor, 1984), but they occur with little effort if the subject does nothing to avoid them (Jonides, 1981).

Third, the occurrence of a cue in the periphery initiates two forms of inhibition. The first, called "cost", is a consequence of orienting attention to the cue. Once attention is engaged at the cued location, all other locations will be handled less efficiently (inhibited) than if no such orienting had occurred because one must first disengage from the cued location before moving to targets at other locations. This form of inhibition is spatially selective only in the sense that it is not present within the focus of attention. A second form of inhibition also occurs. This is called "the inhibition of return" (Posner & Cohen, 1984). The inhibition of return depends upon the act of orienting to a spatial location (Maylor, 1985), but it is most clearly shown if one summons attention to a location and then returns it to a neutral location. The efficiency of the previously cued location is reduced with respect to comparable locations in the visual field for several seconds. The overlap between facilitation due to orienting of attention and the specific inhibition of a cued location helps to explain conflicts in the literature. Sometimes a cued location is handled more efficiently than other locations, sometimes less efficiently, depending upon the balance between the facilitation due to orienting of attention and the inhibition due to the reduction of efficiency of returning attention to an already cued location.

Are there any ecological advantages to this very complex constellation of internal events by a cue? Our theory rests on our finding that the relative facilitation obtained from a peripheral cue moves with the eyes as though it mapped in retinal coordinates (Posner & Cohen, 1984). This effect is not on the retina since it can be obtained in stereoscope. However, it preserves the coordinates of the retina as do many visual images at cortical levels. Inhibition of return on the other hand, does not move when the eyes do, it behaves as though it were dependent on the coordinates of the environment. When we move our eyes, the objects of the world appear to maintain their locations, thus many psychological phenomena maintain the coordinates of the environment as we move about it. It seemed to us of basic importance that one of our effects (facilitation) is retinotopic and the other, (inhibition of return) is environmental in this sense.

According to our view the facilitation effect serves to give priority to targets during a visual fixation. It allows us to give momentary priority to an object in the visual field as, for example, when we carefully examine the nose within a face. If the task demands high acuity, we are likely to move our eyes to the examined location and thus, produce a reorienting of attention back to the fovea. In reading, for example, the reduction of acuity with eccentricity may be the cause of the eye movement (Morrison, 1984). Attention allows a temporary emphasis outside of the fovea, and it is crucial as a guide to the oculomotor system to tell us where to move the eyes next.

We speculate that inhibition of return evolved to maximize sampling of novel areas within the visual fields. Once the eyes move away from target location, events occurring at that environmental location are inhibited and one is less likely to move the eyes back to them (Posner, Choate, Rafal & Vaughn, 1985). This reduces the effectiveness of an area of space in summoning



attention and serves as a bias for favoring fresh areas in which no previous targets have been presented. The long lasting nature of inhibition of return insures that two to three eye movements are biased against a return. The organization of facilitation and inhibition outlined above seems to represent an exquisite functional adaptation to the needs of the visual world.

We review the operations involved in our model task in Fig. 3. The top of the figure indicates the occurrence of a visual cue. The bottom indicates a set of partially sequential but overlapping mental operations induced by the cue. According to this diagram,

Fig. 3

the cue produces a non-spatially specific alerting effect which serves to interrupt ongoing performance. The cue also leads to calculation of its coordinates and in turn produces a disengagement of attention, a movement to the location of the cue and subsequent engagement of the target. If the subject's attention is withdrawn from the cue to another location, we can measure the inhibition at the target location that we call inhibition of return. Single cell recordings in monkeys and the study of patients with restricted neurological lesions can be used to examine the neurosystems that support each of these operations. The basic argument developed in the next section is that widely separated neurosystems are involved in the computation of these various mental operations.

### III. Deficits of Orienting from Focal Lesions

Several areas of the monkey brain have cells whose firing rates are enhanced selectively when the monkey's attention is directed to targets in their receptive fields (Wurtz, Goldberg & Robinson, 1980). In one of these areas, the superior colliculus, the selective enhancement occurs only when attention is directed overtly via eye movement. In a second area, the posterior parietal lobe, selective enhancement occurs when attention is directed overtly or when the monkey is required to maintain fixation while attending covertly to a peripheral stimulus. The third area of selective enhancement lies between the midbrain and cortical projections in the thalamic nuclei known as the pulvinar (Petersen, Robinson & Morris, 1987). Selective enhancement appears to be restricted to these three areas. The single cell results allow us to ask about the relationship between modulation of cellular activity and patterns of facilitation and inhibition found in our work with humans. In this sense they provide an opportunity to connect the last row of Figure 1 (cellular level) with the facilitatory and inhibitory performance changes described for visual spatial orienting. These connections are fundamental to our effort to see if the nervous system localizes the components of cognitive operations.

There is a long clinical history documenting the finding that lesions of the posterior part of one hemisphere can cause a severe deficit in reporting information on the side of space opposite the lesion (DeRenzi, 1982). Neglect of visual information contralateral to the lesion occurs most strongly when patients are confronted with simultaneous lateralized visual stimuli and stimuli contralateral to the lesion are frequently not reported (extinguished). The phenomena of neglect can arise from unilateral lesions of the midbrain and

thalamus as well as from a variety of cortical lesions. However, clinical observations seem to suggest parietal lesions on the right side as the most frequent area of damage leading to neglect and extinction (DeRenzi, 1982).

In recent years a number of these parietal patients have been studied in experiments using cues such as those described in the previous section (Baynes, Holtzman & Volpe, 1986; Morrow & Ratcliff, 1987; Nagel-Leiby, Buchtel & Welch, 1987; Posner, et al, 1982; Posner, et al, 1984; Posner, et al, 1987). The studies have been uniform in showing a particular type of deficit present in patients with right parietal lesions. These patients have a general advantage in reaction time for those targets that occur ipsilateral to the lesion in comparison to those that occur contralateral to the lesion. However, for many parietal patients there is little or no difference between the two types of targets if they follow a cue at the same location (Valid Trial). When attention is drawn to either side, these patients have nearly equal ability to detect the target at the cued location. Thus the ability to engage the target once attention is properly directed is not necessarily interrupted by parietal lesions although it is affected in many patients.

Striking results occur on trials when attention is cued to the side of the lesion and the target is presented to the side opposite the lesion. In some cases, targets show extinction, that is, targets are missed entirely by the subject (Posner, Cohen & Rafal, 1982). In other cases, targets are not completely excluded from consciousness, but show greatly delayed reaction time, sometimes two or three times the normal reaction time. The results suggest that this elevation in latency is simply a less severe form of complete exclusion from consciousness. Patients who miss signals completely when they remain present in the field only briefly will report them when they remain present but with greatly increased latency. The idea that a latency increase is a less severe form of difficulty than extinction fits with the account of covert orienting in normals discussed previously.

The pattern of increased reaction time to contralateral targets following miscues does not depend upon the miscue being ipsilateral to the lesion. Indeed, the increases in reaction time occur in both visual fields when the subject has to produce a covert movement in a contralesional direction from the cue to the target (LaDavas, 1987; Posner, et al, 1987). For patients with right parietal lesions, leftward movements from cue to target are longer than rightward movements to the same target. These findings suggest that the main deficit in parietal patients occurs in the disengage operation. It may be instructive to review the logic. On validly cued trials there is only a modest difference in reaction time on the two sides. Moreover, the improvement in reaction time following a valid cue appears to be about the same on the two sides of the field. We argue that this reduction is due to a shift of attention, thus many parietal patients (those with no difference between valid RTs between the two visual fields) are able to shift attention equally well to the two sides. However, once attention is engaged either at fixation or in either visual field, RTs to targets that lie in a contralesional direction are greatly elevated for even those patients with no differences on valid trials. Why should this be? Why should contralesional targets be at so great a disadvantage following a cue at another location? We reason that it must be because the parietal lesion has a special affect on disengaging attention.

This specific deficit in the disengage operation for contralateral targets found in parietal patients has been confirmed in a number of experiments

(Baynes, et al, 1986; Morrow & Ratcliff, 1987). There are several remaining complexities, that have not been successfully resolved. Using a central cue, Nagel-Leiby, et al, (1987) has found differences between males and females and that in some cases, frontal patients show more severe deficits than parietal patients. In addition, Morrow & Ratcliff (1987), who confirmed our basic result with right parietal patients, have found little deficit in left parietal patients and also found a similar pattern to the right parietal patients in one frontal lobe patient. The unique status of the parietal lobe that appeared clear in our earlier work seems somewhat in question. The issue may be partly resolved by the widespread effects that occur immediately following a lesion. For some months following an insult to the nervous system, there may be widespread changes in glucose utilization and blood flow over the entire hemisphere (Deuel & Collins, 1984). It is possible that some of the reports from other areas may have arisen because the patients were tested too early. The deficits we have reported persist even when patients are tested years after the stroke. Morrow & Ratcliff (1987) have traced these recovery effects for some months following lesions.

Another reason for finding these effects in frontal patients may be because the spatial attention system is not an isolated module that operates independently of other levels of control. Thus lesions of the frontal lobe may affect the spatial attention system along with a variety of other systems because it influences command systems necessary to allow for the disengagement process. This possibility will be discussed in more detail on page 000. It is not completely clear whether lesions of the left parietal lobe produce an identical pattern with the same strength as lesions of the right parietal lobe. These comparisons are, of course, always between subjects and thus can involve many sources of error not found in the within subject comparisons on which we have mostly relied.

### Forms of Neglect

Clinically neglect occurs after a wide variety of lesions. This may be in part because many of the reports of neglect are from studies of patients who are acutely ill and may have widespread metabolic problems following the initial insult. We have so far found only three groups of patients who show systematic deficits in visual spatial orienting even after relatively long periods of time after the lesions. These correspond to areas that give selective enhancement in single cell studies of alert monkeys. The reaction time patterns in these three forms of "neglect" are shown in Figure 4. It shows the reaction times to valid and invalid trials at short intervals between the cue and the target and divides them according to whether the target occurs in the field which is usually neglected (left panel) or the one which has no evidence of neglect (right panel). In the case of parietal lesions the neglected field involves the area of space contralateral to the lesion.

Fig. 4

A second group of patients have progressive supranuclear palsy, with lesions of the midbrain, including the superior colliculus and surrounding areas. These patients show an unique deficit in eye movements, having great difficulty making voluntary eye movements, particularly in the vertical direction. Impairment develops more slowly for horizontal movements. These

patients often come to the neurologist's attention because they neglect the lower part of the visual field. In the case of these patients we have systematically compared attention movements in the vertical direction with those in the horizontal direction (Posner, et al, 1985). The results for these patients are very striking and completely different for those found for parietal patients. As can be seen from figure 4, these midbrain patients have very long reaction times. The long reaction times may be due to the widespread reticular lesions along with the deficits that we have described. However, in the horizontal direction there is clear evidence of a validity effect. Even at short intervals, valid trials are systematically faster than invalid trials. Thus orienting to horizontal targets appear relatively normal. However, in the vertical direction, the validity effect does not emerge until much later. There is no evidence of a validity effect at the fast probe interval shown in Fig. 4 but usually by half a second a validity effect has emerged.

These data are very different from the parietal patients who show a greater than normal validity effect in the neglected field at the earliest intervals. Since the emergence of a validity effect is due to a shift of attention to the cued side, the findings from the midbrain patients suggest a specific delay in their ability to move attention to the target. Hence, if enough time is given following the cue, the vertical and horizontal directions both show validity effects. It appears that the deficit in the midbrain patients is in their ability to move attention covertly in the direction that has the largest eye movement deficit.

An additional finding with supranuclear palsy patients is that they lose inhibition of return in the vertical direction. Although they can move attention to a vertical cue if given sufficient time, they do not show the reduced tendency to return attention to a previously cued location (Posner, et al, 1985). This loss of inhibition of return was unique to these midbrain patients and is not found in control groups with cortical or other subcortical lesions. It fits quite well with the functional theory that identifies inhibition of return with the tendency to move the eyes to novel locations. Deficits in the move and inhibit operations provide more evidence in favor of the idea that specific neurosystems influence different aspects of the set of computations necessary to induce orienting of visual spatial attention.

A third form of "neglect" has been found following thalamic lesions that may involve the pulvinar (Rafal & Posner, 1987). As can be seen from Figure 4, these patients show another pattern of performance deficit, especially long reaction times for the invalid trials on the side opposite the lesion. This effect is similar to that found in parietal patients, although the deficit on invalid contralesional trials does not appear to last as long following the cue in the thalamic patients. Striking in the thalamic patients is that the increase in RT is also quite large for valid trials on the side contralateral to the lesion.

This constellation of deficits for both valid and invalid trials could be consistent with a purely visual defect. However, careful ophthalmologic testing of these patients, particularly in their six month follow-up, showed no evidence of ophthalmologic deficits. The second explanation would be a specific deficit in their ability to engage attention on the side contralesional to the target. This would suggest that these patients cannot use attention to make processing as efficient as it could be when targets that are contralesional. This supports the idea that thalamic lesions produce a

specific deficit in the engage operation and provide some support for a theory of the special role of thalamic areas in control of the attentional spotlight (Crick, 1984). In Crick's view, the thalamus is the area of the brain most likely to be involved in the search of the complex visual field for targets. A deficit in the engage operation would be consistent with this theoretical view.

Figure 4 summarizes three patterns that we have found present for posterior lesions related to aspects of poor RT performance to targets contralateral to the lesion. These include: parietal lesions and the disengage operation; thalamic lesions and the engage operation and finally, midbrain lesions and the move operation. The results do not show complete separation. For example, parietal patients frequently show engage deficits and thalamic lesions also produce disengage deficits as well. The known anatomy and close physiological connections of these areas would lead to the expectation that the three are in close contact. For covert orienting to occur all these operations must be performed. One assumes that the disengage operation begins the sequence, information is then sent to the midbrain to move attention to an already calculated location and when that is completed, it is possible for the system to work through thalamic sites to engage targets. An important point is that the thalamus (particularly the lateral pulvinar) represents an area allowing contact between parietal systems responsible for spatial attention and systems of the brain known to be responsible for pattern recognition. It is clear that patients with lesions of the parietal lobe do show deficits in pattern recognition process and we turn to evidence of this effect in the next section.

#### IV. Functions of Spatial Attention in Pattern Recognition

According to recent views of the neurophysiology of vision, there are two major systems extending from the primary visual cortex. The first extends from area V1 (striate cortex) to the inferotemporal cortex and is involved in the recognition of objects. The second extends from area V1 into the parietal lobe and is more responsible for localization of information and as we have discussed above, for visual spatial attention (Mishkin, Ungerleider, Macko, 1983). It is important to ask whether deficits in visual selective attention influence the pattern recognition process, and if so, in what way?

We have developed two different strategies to evaluate this issue. First, cueing in normals can be used to control orienting of attention, and can then explore the effects of such cues on pattern recognition. Second, patients with deficits in visual spatial attention due to specific lesions can be studied. It is possible to ask both whether the cueing known to be responsible for covert visual orienting influences pattern recognition and also whether the presence of lesions in areas related to visual spatial attention influences pattern recognition. The answer to both questions seems to be yes and provides us with information on the relationship of attention to pattern recognition.

According to one recent theory, visual spatial attention has the role of integrating visual features into conjunctions (Treisman, 1987). Individual features of objects such as color, orientation, or motion are to some extent registered in separate spatial maps in monkey cortex. This registration occurs in parallel across the entire visual field. If an object differs from its background by a single feature, it is possible for a person to respond to the presence of that feature rapidly and efficiently without attending to individual items in the field. If, however, the judgment requires the integration of features into a conjunction, such as looking for a red f in a

field of Ts and other red objects, spatial attention is needed and a more serial search is conducted. If normal subjects are cued to a location eccentric of the fovea, both feature and conjunction search are conducted more efficiently at the cued location. However, the effect on conjunction search is far stronger than for feature search (Prinzmetal, et al, 1986; Treisman, 1987). This suggests that although attention can effect the registration of features, it plays a more important role in the recognition of conjunctions.

Similarly, it is possible to study the effects of lesions of the visual spatial attention system on the visual search process. It is well known clinically that right parietal lesions produce a relative neglect of information on the side of space opposite the lesion. Experiments show that both right and left parietal lesions have clear effects on visual pattern recognition (Friedrich, Walker & Posner, 1985). Subjects were presented with two strings of letters, one above the other. The letter strings were identical half the time and half the time differed by a single letter. This difference could be in the beginning of the string, in the middle or at the end of the string. The subject's task was to press one key if the strings were identical and another if they were not. Subjects were free to move their eyes, and the letters remained present in the visual field until they responded. Left parietal patients showed extreme difficulty when the discrepant letter was at the end of the letter string. Reaction times were nearly 800 msec longer for differences found at the end than at the beginning. Moreover, the subjects frequently missed differences at the end. On the other hand, right parietal patients were slower and made more errors when the differences were at the beginning. This task is an attention demanding spatial search task and shows quite clearly the pattern recognition deficits in the parietal patients. The ability to organize and recognize differences on the side of space opposite the lesion is greatly impaired even when they can take the time to move their eyes and examine the stimulus in detail. These were patients far removed from the stroke and showed little evidence of clinical neglect or extinction. Despite the general recovery, the visual search task showed clear deficits.

An important distinction in the study of pattern recognition is between automatic and attended processes. By exploring automatic processes we can examine the operations for which attention is not needed. One process that has been a candidate for "automatic" is the ability of a visual word presented on the fovea to contact its visual, phonological and semantic representations in memory (LaBerge & Samuels, 1974; Marcel, 1983; Posner, 1978). The advantage of an integrated word, even in comparison to individual letters, has been an important theme in cognitive psychology and in recent connectionist models of visual word processing (McClelland & Rumelhart, 1986). It thus became of considerable interest when Sieroff & Michel (In Press) reported that patients who show profound extinction of individual short words when they are presented simultaneously to the right and left visual field showed no evidence of extinction with tachistoscopic presentation of a single word across the fovea, even when it covered the same visual angle as the word pair. Patients with both right and left parietal lesions showed clear evidence of extinction to simultaneous words but even right parietal patients showed little evidence of extinction to the single foveally presented word.

We compared the perception of single eight letter strings (Sieroff, Pollatsek & Posner, 1987) that either formed words or not. Tests of ten right parietal patients presented at bedside with 3 x 5 cards showed that the

patients missed the first few letters of nonwords, but not of words (see Figure 5). This result was confirmed by tachistoscopic testing of right and left parietal patients who were well past the lesion. The results showed that the recognition of the letters in nonwords appeared to depend upon an intact visual spatial attention system, but for words the lesion did not produce any spatially specific deficit. These results fit with the findings in cognitive psychology that word perception is superior to nonword perception. One reason given for the superiority of words is that recognition of visual words might have top down assistance from a visual lexical dictionary (McClelland & Rumelhart, 1986).

### Fig. 5

---

The cueing method can also be used to bias visual spatial attention in normals (Sieroff & Posner, 1987). Thus it should be possible to confirm our patient results by looking at the processing of normal subjects with attention drawn covertly either to the left or right end of strings of letters. To do this, we first presented a digit for 50 msec below the position in which would follow the first or last letter of a 100 msec exposure of a letter string. The results with the normal subjects were similar to those found with the patients. For words, biasing of attention to the beginning or end made little difference in the parts of the string correctly reported. For random letter strings, the subjects systematically missed information on the side of the word away from the cue. The more word-like the letter string, the less the effect of the cue on the subject's report.

These experiments show quite clearly that visual spatial attention deficits produced by parietal lesions can have very strong effects on pattern recognition. They also suggest that both formation of conjunctions and reports of stimuli making nonword strings are greatly affected by attention. However, for word strings, there is little or no effect of damage to the spatial attention system nor of shifts of spatial attention in normals. Foveal words appear to have automatic access at least to a visual lexicon.

Recent studies of normals using Positron Emission Tomography (PET) to study regional cerebral blood flow during visual language tasks, have provided additional evidence for the rapid packaging of individual letters into word forms (Petersen, Fox, Posner, Mintun & Raichle, 1986). In these studies subjects in separate blocks (a) watched passively while nouns were presented visually once each second, (b) pronounced the nouns, (c) generated uses (verbs) to the nouns. During 40 seconds of the task, regional blood flow was assayed by use of PET. A subtractive technique allowed examination of the neural systems active when either watching words passively or actively responding to them. The passive visual task activated areas of the prestriate cortex as far anterior as the occipital temporal boundary (see Fig. 6). This activation is very different from that found with auditory words (Fig. 6). When the subject was required to pronounce the words or to generate uses for the words, two parts of the anterior cortex (frontal lobe) were activated. One part was left lateralized and seemed specifically related to language (see Fig. 7,8). The repetition task appears to activate areas near and superior to the classic Broca's area. These areas appear to relate to the generation of the articulatory code of the visually presented word. The generate task activates areas more anterior (close to area 45) on the lateral surface that appear to be

related to the semantic operations in achieving the use of the presented word. The areas activated in the word generation task for visual and for auditory words are in close proximity but appear separate (Petersen, Fox, Posner & Raichle, 1987). The second set of anterior areas are on the medial surface and do not necessarily seem to be language related. These areas include the supplementary motor area and the cingulate cortex. We believe these areas may be parts of the anterior focal attention system that is discussed in the next section. Roland (1985) has reported several areas that seem to accompany almost all forms of cognitive activity. The areas to which he refers appear to be somewhat more anterior to the ones we have found active, but differences in our techniques may account for anatomical differences. In any case there do appear to be several candidate areas that may be involved in coordination of attention to visual spatial and language information.

Fig. 6,7,8

The fact that no posterior area other than in the occipital lobe was activated by visual words, whether the subject was passive or whether he was active, suggests that the visual analysis of words must take place within the occipital lobe. This result fits with several findings within the psychological literature. First, it fits well with the results described above in which subjects with right parietal lesions extinguished the left side of nonwords but not of word strings. The lesion result suggests that the word/nonword distinction must be made rather early in the nervous system. Second, models of interactive computations (McClelland & Rumelhart, 1986), require intimate feedback from higher levels to lower ones. The rich feedback available in the occipital lobe would make an ideal basis for this system.

Third, many cognitive studies with letters and words conducted in the late 1960s and early 1970s (see Posner, 1978 for a review) argued that visual codes of letters and words had access to output systems. When subjects were required to indicate whether a letter or word pair were physically identical they could do so independently of the names or semantics of the items shown. Nonetheless, words were responded to faster than nonwords. This would require the ability to route visual input to output mechanisms without having to go through phonetic, or semantic systems. As further support for this idea, we (Sandson & Posner, 1987) asked subjects to make lexical decisions about whether or not a string of letters made a word. They did the lexical decision task either alone or while also shadowing a verbal message. We found that priming of the target by an identical immediately prior string (identity priming) was not reduced by shadowing while all forms of semantic priming were reduced by shadowing. This result supports the idea that physical priming involves visual spatial pathways and their connection to output system (in this case manual), while semantic priming involves systems in which visual and auditory input is intermixed.

Although many previous anatomical theories of visual word reading had relied upon information reaching the angular gyrus or Wernicke's area, (Geschwind, 1965) current cognitive literature discusses the use of a purely visual code as a means of accessing semantic memory (See Carr & Pollatsek, 1985 for a review). These PET studies confirm the idea that visual spatial attention is not needed for pattern recognition of individual words outlined in this section, but also suggest the importance for anterior areas in higher levels of attentional control of language. It is to these higher levels of control that we now turn.



## V. Common Systems of Attention to Language and Visual Space

In this chapter we have been using two cognitive systems to examine control by attention. These are a visual spatial attention and one which processes language information. The two systems can be viewed in terms of a hierarchy of attentional control systems as shown in Figure 9. Visual spatial attention can be seen as part of a system involving orienting to sensory information. We know that parietal lesions can impair orienting to tactile and auditory information as well as to visual information. Moreover, impairments in different forms of sensory orienting are independent in the sense that auditory and visual extinction are not correlated among patients with parietal lesions (DeRenzi, Gentilini & Pattacini, 1984; Sieroff & Michel, 1987). Similarly, we found that a cue that draws attention to a spatial location was ineffective when the person did not also know the modality of the target (tactile or visual). These findings suggest separate neural systems within the parietal lobe responsible for attention to visual, tactile or auditory modalities.

Fig. 9

On the other hand, it is possible to compare the relative influence of modality (auditory vs. visual) with the influence of the type of cognitive system (spatial or language) in the control of attention. The two cognitive systems correspond to the two major branches of Figure 9. In a series of studies with normals (Posner & Henik, 1983) and patients (Walker, Friedrich & Posner, 1983) we have used a spatial version of the Stroop effect to study this issue.

In these experiments subjects are instructed to respond either to the visual words "left" or "right", to the location of these words on the screen, to visual symbols (arrows pointing to the left or right), or to auditory words ("left" or "right") that might be presented to the left or right ear. In different experiments manual or vocal responses have been used. In work with normals (Posner & Henik, 1983), we compared irrelevant dimensions using either the same cognitive system but a different modality than the attended event, with those in the same modality but a different cognitive system. When a person is to deal with a visual or auditory word the extent of facilitation or conflict in RT from words in the opposite modality is much greater than from spatial locations in the same modality. For example, the auditory word "right" interferes more with processing the visual word "left" than does the location of the visual word on the screen. Stimuli from the same cognitive system, even when they involve different sensory modalities, interact strongly. This motivates the common nodes for language and for space independent of modality (see Fig. 9).

Reading is one task that clearly involves both language and spatial attention since eye movements and higher level semantic codes are both involved. However, the choice of language and spatial attention was designed to allow for the possibility that above the spatial processing needed for foveating visual words, the control mechanisms for the two systems might be quite separate. So far we have shown that the visual spatial attention system includes the posterior parietal lobe, areas of the thalamus and midbrain. We now ask whether this visual spatial attention system is an independent module

that operates on its own or whether it operates in relation to a more complex attentional system that is also involved in the processing of, for example, auditory language.

One way to examine this task is to ask normal subjects and patients with parietal lesions to perform a language task and at the same time, to respond to cues and targets occurring at varying locations in the visual field (Posner, Inhoff, Friedrich & Cohen, 1987). For patients this required a very simple language task in which they listen to twenty words, one every second, and count the number of times the words contain a particular phoneme. While listening to these words, visual cues appeared at two locations in the field and we measured the speed of pressing a key to targets following those cues. The language task retarded the ability of a cue to draw the subject's attention to a location in visual space. These patients show large validity effects, (advantage of the cued location over the uncued one) by 100 msec when performing the spatial task alone, which under dual task conditions no validity effect was found until 500 msec. The same results can be obtained with normal subjects, however, they require a more complicated task than the very simple phoneme monitoring task used with patients. For example, a similar retardation of the cueing effect can be obtained if the subjects are required to count backwards by three (Posner, et al, 1984).

As expected the dual task increases reaction time to the visual spatial processing task. However, it does more than merely increase reaction time, it also retards the validity effect. This suggests that the ability to orient attention is retarded when the person is engaged in a language task. Language tasks interfere with some of the operations necessary to shift visual spatial attention to a cued location. Thus visual spatial attention is not an independent module but shares operation with a more general attention system also involved in the processing of language.

Can we say more about the interaction between visual attention and language processing? The use of patient populations does allow us to show that the interaction between visual spatial attention and language attention does not involve the parietal visual attention system. This conclusion stems from the finding that the parietal lobe lesion produces a deficit in the disengage operation. Patients would have to show a specific slowing on invalid contralateral targets when processing language, if language used the same parietal system. However, when engaged in the language task, patients show little difference in reaction time between targets that are ipsilateral versus contralateral to the lesion. Apparently the disengage deficit is local only to visual spatial attention and is not a general disengage deficit. The results of the PET scanning data support these findings since we find no common posterior areas that are involved in auditory and visual language processing. Thus if one seeks an area that deals with language processing (both in its visual and auditory form), and in visual spatial attention (see Fig. 8), one must move to anterior parts of the brain. Whatever system is involved in processing visual spatial and language information must lie in the frontal lobes and/or their related subcortical areas.

The frontal lobes are currently a very active area of research within neuropsychology. Good summaries of this work are available (Goldman-Rakic, in press). It is well known that lesions of this area can produce devastating effects on human thought and behavior that in one review has been likened to producing a person whose thought and behavior lacks coherence (Duncan, 1986).

One result of including frontal lobe function in the ability to allocate attention to visual space is to reconcile the existing conflicts in the literature. Even if the basic visual spatial attention system is posterior, as we have argued, its control system may lie within the frontal lobes and affect both language and spatial function. Thus findings that neglect can be obtained from frontal lesions may have to do with the command functions that act to allow the posterior areas to function (See Fig. 3). The common finding in experimental psychology that much of our attentive behavior is closely related to motor performance (Allport, 1980) fits with the idea that attentional systems lie in close proximity to symptoms controlling motor output.

Although I believe that midline systems that we have found activated in our PET scanning experiments (See Fig. 7,8) are likely to be part of the focal attention system of the frontal lobe, we do not yet have definitive studies that have localized the different arc computations that performed within the control structures found in the frontal areas. The relationship of computational models of executive function to the complex anatomy of the frontal lobes still remains in the future, although a beginning of this kind of thinking has arisen, particularly on the role of the dorsolateral prefrontal cortex in inhibiting conflicting responses (Diamond, 1987). One must keep in mind the lessons learned from the posterior attention system that such systems involve widely scattered cortical and subcortical sites. As we seek to understand the anterior attention control systems it is likely that we will discover many anatomically distinct areas to be involved.

## VI. Applications to Putative Disorders of Attention

The implication of the framework that has organized this lecture is that deficits in mental function must be described both in terms of the elementary operations impaired and of the neurosystems affected. To develop this theme we have been dependent upon cases in which the damaged anatomical area can be observed by neuroimaging. This is traditional neuropsychology. There are many putative disorders of attention, however, in which the underlying neural damage is unknown. These disorders are said to be attentional in the relatively loose sense that they seem to involve the ability of the person to concentrate, to interact appropriately with the environment, and do not seem to be simply due to sensory, motor or general cognitive damage. Four disorders in this category are depression, schizophrenia, closed head injury and attention deficit disorder. In each of these, the literature indicates a disorder of attention and while there are ideas about the organic basis of the disorder it is still unknown.

I would like to use schizophrenia as a model illustrating how the framework developed in this chapter may serve to guide research relating cognitive and neural systems. My interest in schizophrenia began with a study using Positron Emission Tomography in never medicated schizophrenics (Early, Reiman, Raichle & Spitznagel, 1987) showing a left basal ganglia abnormality. This anatomical result, together with the widely held belief that schizophrenia was a disorder of attention (Mirsky & Duncan-Johnson, 1986), led us (Posner, Early, Crippin & Reiman, 1987) to examine the operations of visual spatial attention among schizophrenics. The hypothesis was that there would be a right visual field deficit (because of the left hemisphere abnormality found in PET) that would occur under conditions in which attention had first been drawn to the left visual field (because of the attentional nature of the disorder). We used our

standard visual cueing method. Our initial results have confirmed this hypothesis (See Fig. 10).

Fig. 10

---

The advantage of using this simple task for the study of schizophrenia is that the attention deficit can be observed within a subject. The subject's performance in the left visual field and right visual field can be compared within the exact same task format. This result eliminates such explanations as lack of motivation, fatigue, and other general reasons that schizophrenics might differ from normals in task performance.

What might be the anatomical and psychological explanation underlying such a right visual field deficit of attention? One possibility is that a deficit of the parietal lobe accounts for the visual spatial abnormality shown in figure 10. In this case a separate anterior deficit would be needed to account for the problems with language processing that are found in the literature. There are known pathways that connect the posterior cortex and these tend to involve the basal ganglia as well (Alexander, DeLong and Strick, 1986). Another possibility is a deficit involving the anterior attention system common to spatial and language processing. One reason that a deficit in the common anterior attention systems seem likely is that schizophrenics who report auditory hallucinations appear to be somewhat more likely to show stronger visual spatial deficits. Moreover, Bick & Kinsbourne (1987) have shown that auditory hallucinations seem to be related to self generated voices by the patients. Our work with normals has suggested that it is possible to create a right visual field deficit somewhat similar to that found in schizophrenics by having them shadow auditory messages while responding to visual spatial cues.

It has been known for some time that schizophrenics have difficulty in selecting and holding a set (Weinberger, 1986). Weinberger (1986) has shown severe deficits in the Wisconsin Card Sorting Task. His work with PET shows that this task seems to be related to an area of the frontal lobe called the dorsolateral prefrontal cortex. This is an area of the brain that when lesioned in monkeys produces severe deficits in tasks involving conflicts between previously rewarded acts and current information (Goldman-Rakic, In Press). The mediation conflict between competing signals is of course a basic aspect of attentional control. To study this form of conflict we used the word-arrow version of the Stroop task described previously (page 28). We had previously shown that patients with right hemisphere lesions tend to respond well to the words but poorly to the arrows and the reverse for left hemisphere lesions. Unmedicated schizophrenics, like left hemisphere lesioned patients, show a very large preference for the arrow.

Both the word-arrow conflict results and Fig. 10 point to an anterior left hemisphere deficit that is attentional because of its strong interaction with cues. While the exact nature of the disorder of attention involved in schizophrenia remains a puzzling mystery, our results provide markers that seem to relate both to the laterality of the disorder and to its attentional nature. Within subjects markers for the schizophrenic syndrome provide us with new methods for investigating the nature of this disorder and perhaps, tying it to the underlying anatomy of the attention system. The ability to specify the

mental operations should open up new ways of linking human disorder to the underlying physiology. Even the preliminary results support the general framework of this chapter and may aid in the search of theory-driven hypotheses about the nature of other putative disorders of selective attention.

## VII. Summary

This chapter has attempted to lay out a very general empirical approach to the neuropsychology of normal attention and of its disorders. The approach uses both cognitive and anatomical data to develop a structural model of the neural systems involved in selecting an item for awareness. The major general conclusion is that the nervous system localizes cognitive operations in widely separated neural systems that are then orchestrated in performance.

To study disorders of attention one may seek links at the level of impairment of mental operations. For example, we find that schizophrenia impairs the ability to shift attention to an event in the right visual field and impairs the selection of a spatial cue. Or one may seek to link impairments in neural systems to individual operations, as in the assertion that right parietal lesions impair the ability to disengage attention to deal with a target located in a leftward direction. It is also possible to indicate the functional significance of an impairment, as for example when it is asserted that a parietal deficit impairs the ability to read.

Our analysis relates diverse methods such as cognitive experiments with normals, study of brain injury and mental disorders, and the use of neuroimaging techniques. Although our description of attention remains incomplete at both the computational and neural systems levels it already provides a basis for understanding some putative deficits in terms of their effects on the structures and functions of what we now regard as a cognitive system for the selection of information.

<sup>1</sup> This research was supported by ONR Contract N-0014-86-0289 and by the McDonnell Center for Higher Brain Function. I am grateful to Drs. Mary K. Rothbart, Steven E. Petersen and Jennifer Sandson for examination of earlier versions of this manuscript.

## Figure Captions

- Fig. 1. A general framework for levels of analysis in connecting cognitive tasks of daily life to neural systems.
- Fig. 2. A model task for the study of covert shifts of visual spatial attention.
- Fig. 3. The putative model operations that are set in motion by the presentation of a peripheral cue.
- Fig. 4. Three forms of neglect. The left panel shows performance when targets are in the non neglected visual areas. The right panel when they are in the neglected visual areas. Data are always from cue to target intervals of 100 msec or less.
- Fig. 5. Performance of ten right parietal patients on word and non word strings presented to them on cards shortly after their lesion.
- Fig. 6. Subtracted PET images of cerebral blood flow. The stimulated task involves visually presented words presented at fixation at the rate of one per second. The control condition is fixation alone. Both conditions involve an average over 40 seconds.
- Fig. 7. Subtracted PET images of cerebral blood flow. The stimulated task involves reading visually presented words. The control task is the passive reception condition described in Fig. 6.

Fig. 8. Subtracted images of images of cerebral blood flow averaged over ten subjects. The stimulated state is the generating a use for each presented noun stimulus. In the stimulated state each word is presented as described in Figure 8. (From Petersen, et al, 1987).

Fig. 9 A hierarchically distributed view of selective attention to spatial and language stimuli.

Fig. 10. Reaction times of never medicated schizophrenics, medicated schizophrenics and normals in the model cueing task for visual spatial attention. All data are from the 100 millisecc target to cue interval.



Figure 1

COGNITION AND NEURAL SYSTEMS

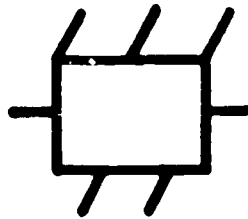
<u>LEVEL</u>	<u>GENERAL EXAMPLES</u>	<u>COVERT ORIENTING TASK EXAMPLES</u>
TASK	READING, SPEAKING IMAGERY	COVERT ORIENTING
ELEMENTARY OPERATIONS	NEXT, SCAN, NAME, ZOOM	DISENGAGE, MOVE, ENGAGE
COMPONENT ANALYSIS	FACILITATE PATHWAY INHIBIT PATHWAY	FACILITATE LOCATION
NEURAL SYSTEM	PROCESSING NEGATIVITY BLOOD FLOW LESIONS	MIDBRAIN (SUPERIOR COLLICULUS) PARIETAL LOBE
CELLULAR ACTIVITY	SELECTIVE ENHANCEMENT	LIGHT SENSITIVE CELLS

Figure 2

TIME 1



TIME 2



TIME 3

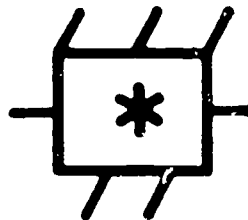


Figure 3

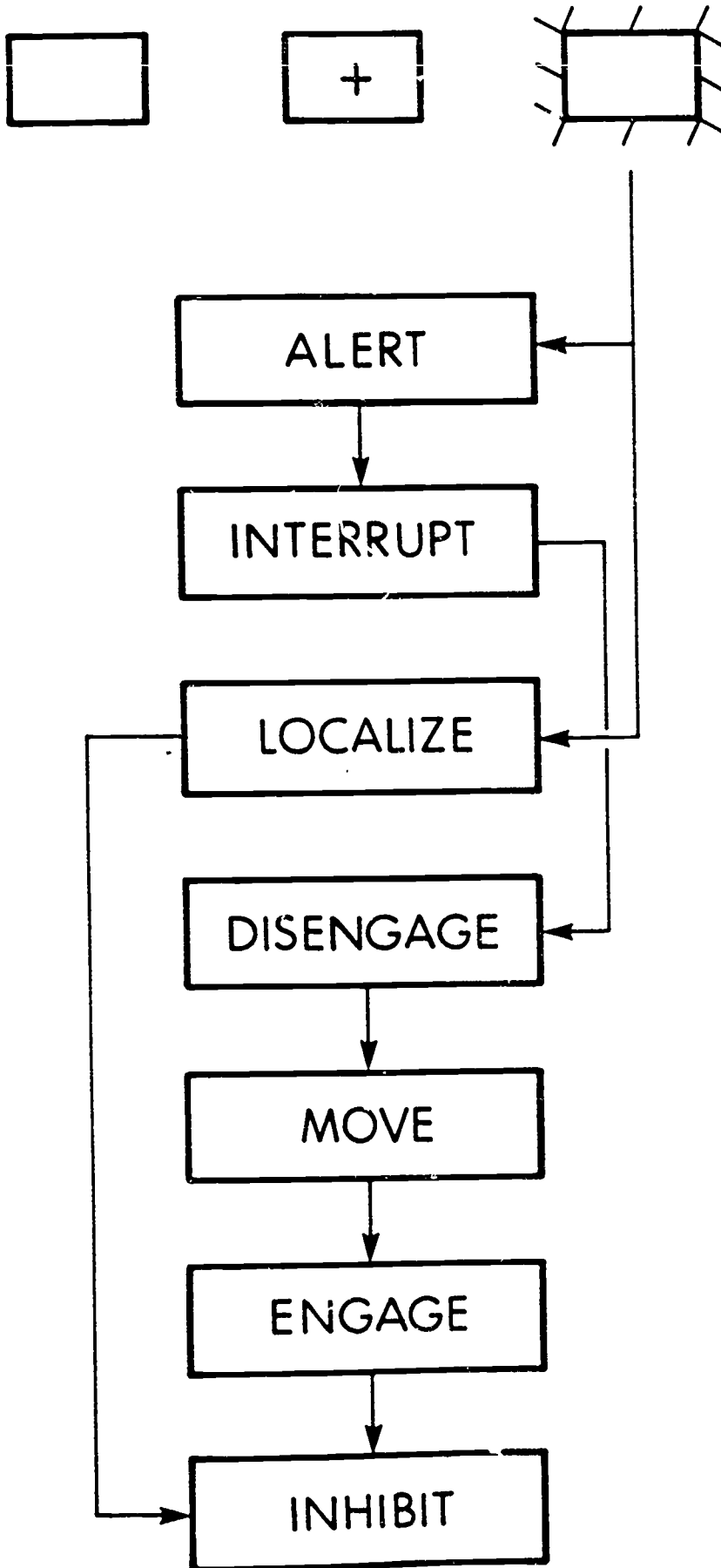


Figure 4

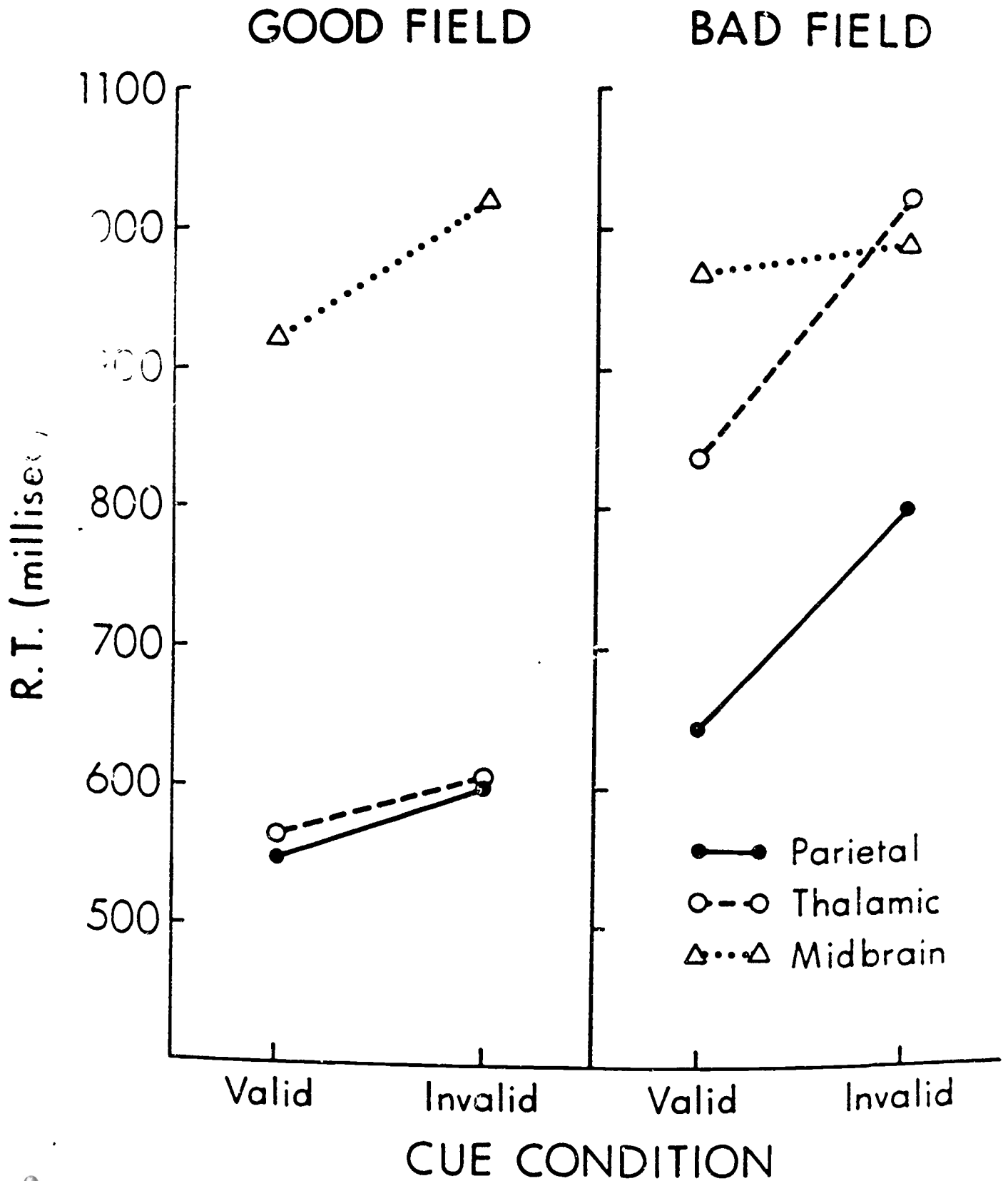
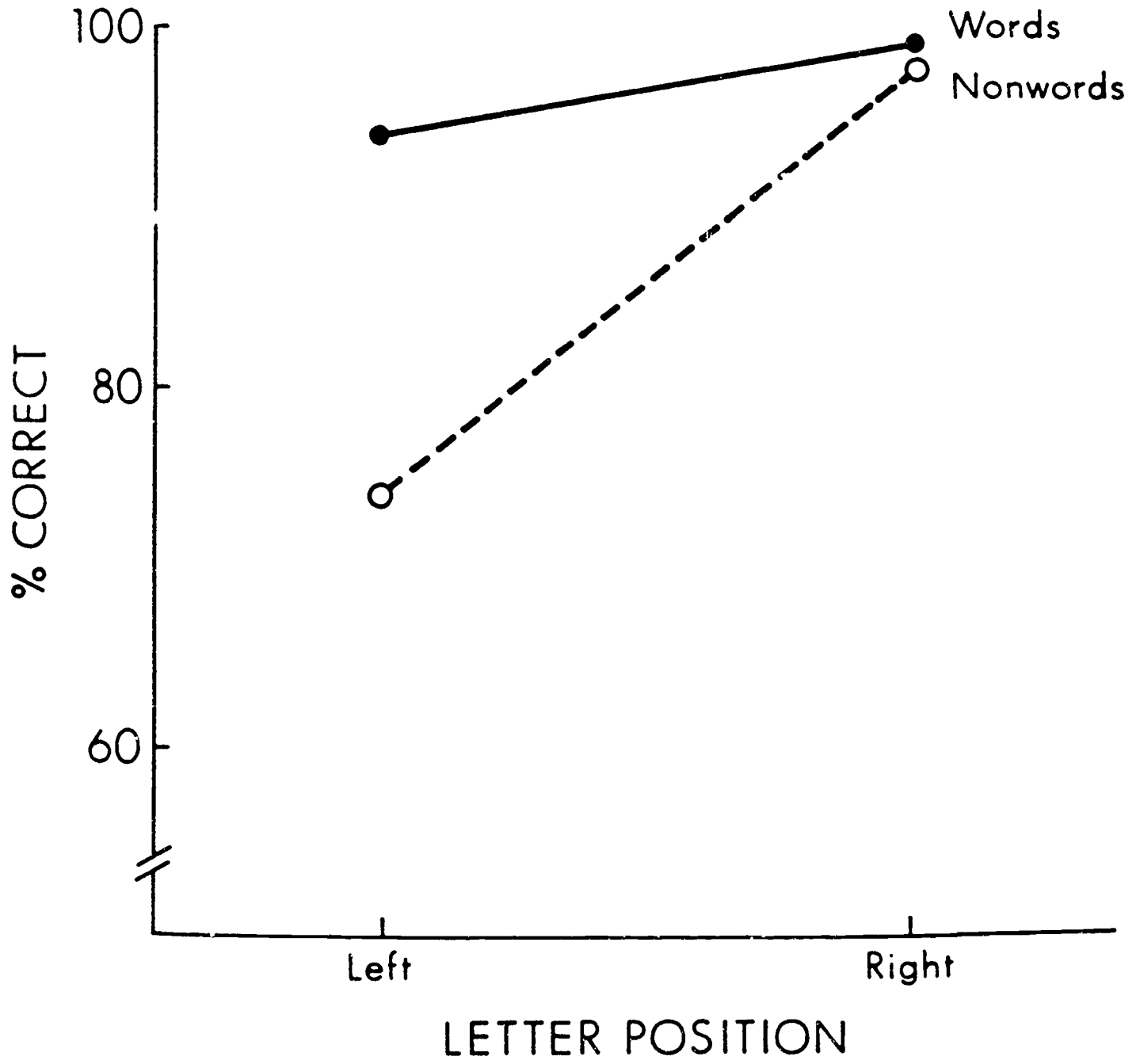


Figure 5





VISUAL STIM.



31.6

PASSIVE NOUNS &

FIX. PT. CTRL.

EXTRASTRIATE

VISUAL CTX.

4 CM. BELOW AC-PC

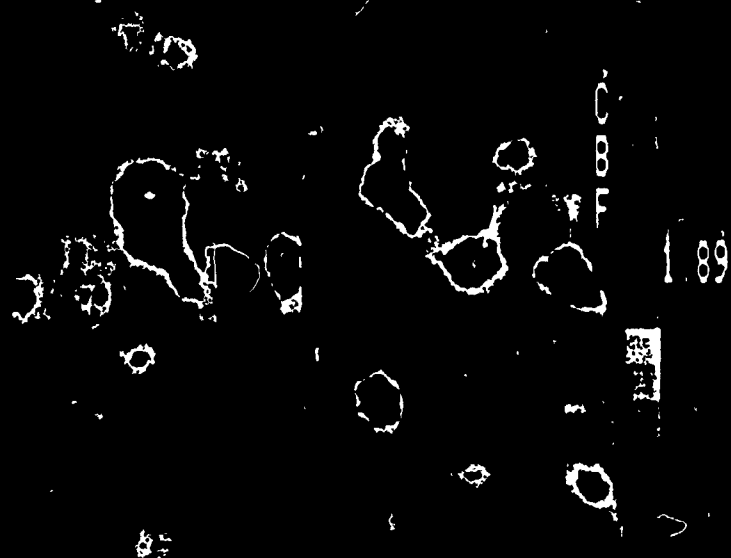
30.8

2.80

34.55

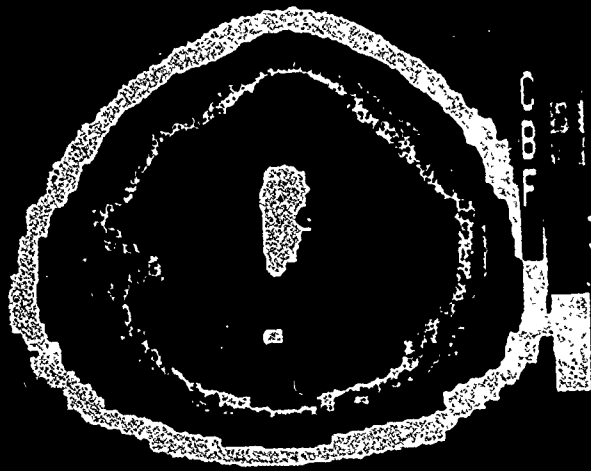
AUDITORY STIM. 3.78

2.28



1.89

Figure 6



REPEAT NOUNS -  
 PASSIVE NOUNS  
 SUPPLEMENTARY  
 MOTOR AREA  
 5.0 CM ABOVE AC-PC

VISUAL STIM.

AUDITORY STIM. 3.66

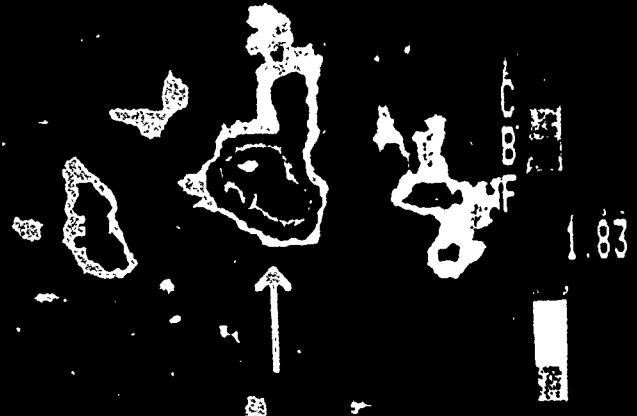


Figure 7

64.4

GENERATE USES -  
REPEAT NOUNS

LAT. ~ FRONTAL CTX.

2.0 CM ABOVE AC-PC



32.2

USUAL STIM.

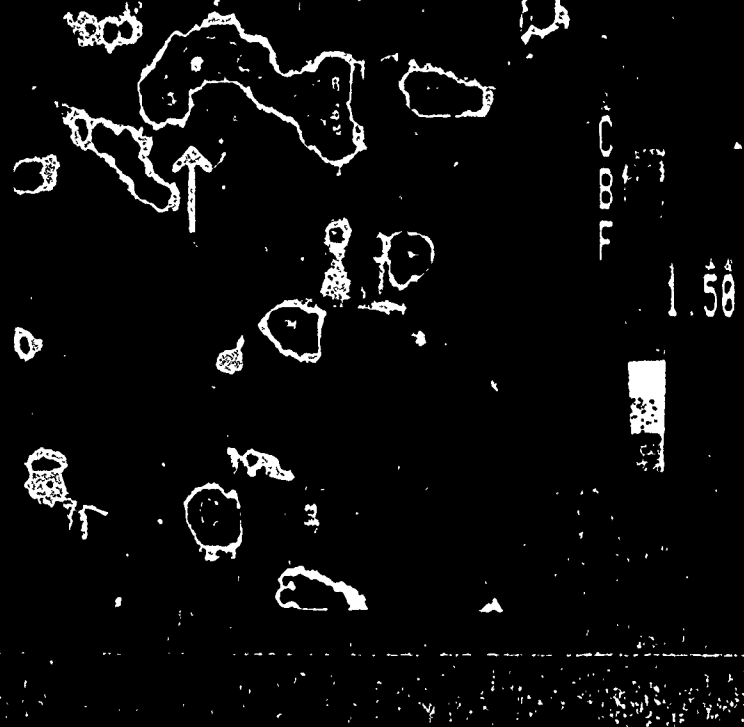
2.88

3.58



AUDITORY STIM.

3.88



1.79

1.58

Figure 8



Figure 9

## HIERARCHICAL SELECTIVE ATTENTION

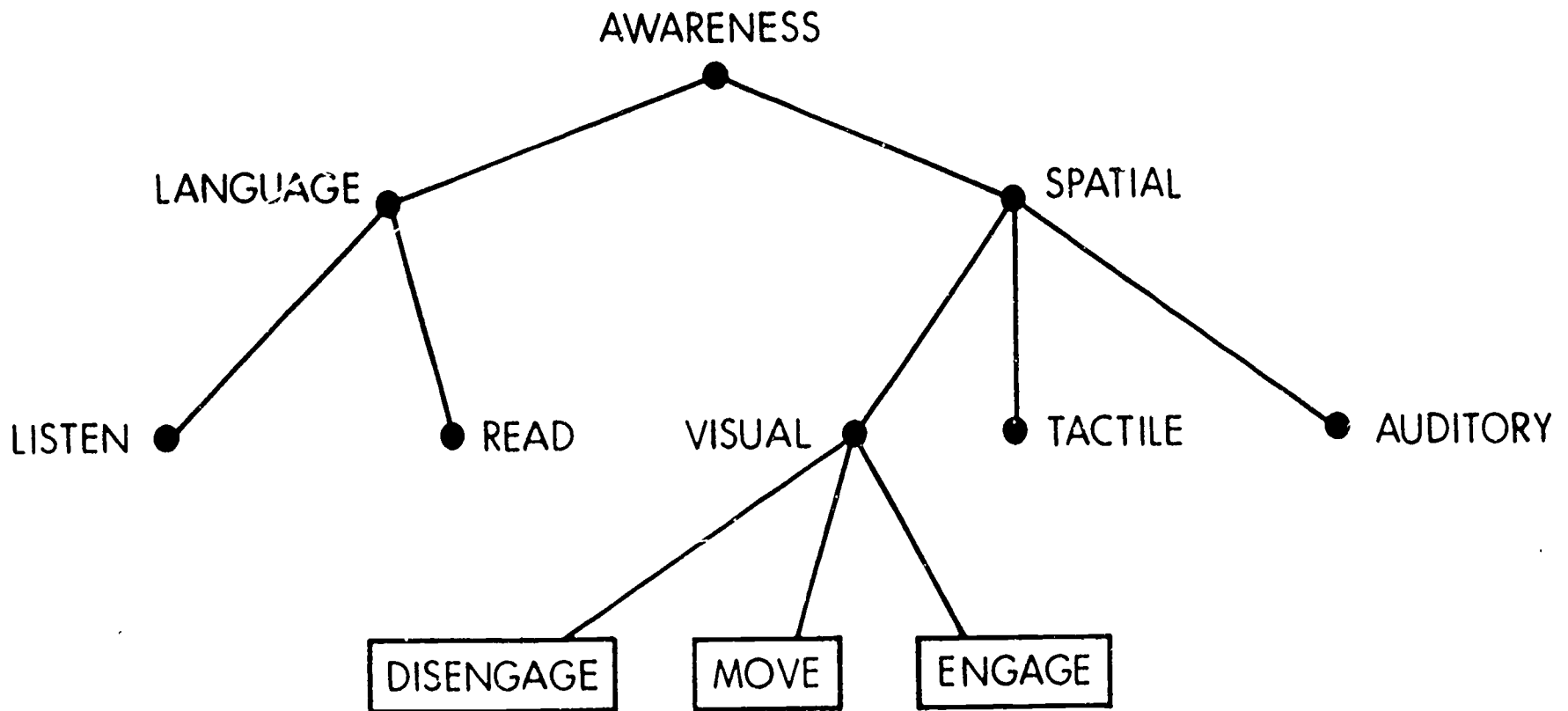
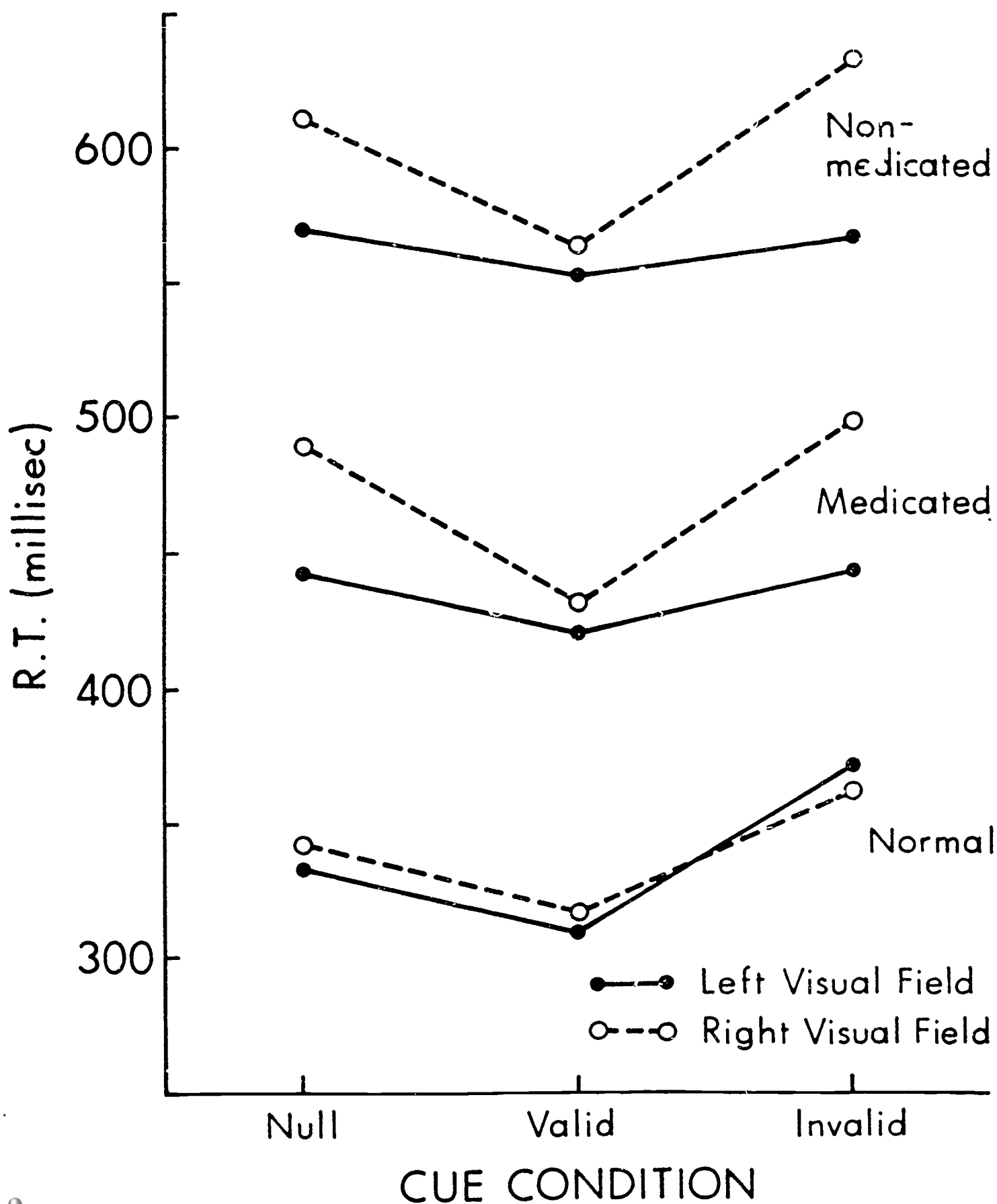


Figure 10



## References

- Alexander, G.E., DeLong, M.R., & Strick, P.L. Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual Review of Neuroscience*, 1985, 9, 357-381.
- Allport, D.A. (1980) in *Cognitive Psychology: New Directions* (Claxton, G., ed). pp. 112-153, Routledge and Keegan Paul.
- Bashinski, H.S. & Bachrach, V.R. (1980) Enhancement of perceptual sensitivity as the result of selectively attending to spatial locations. *Perception and Psychophysics* 28, 241-248.
- Baynes, K., Holtzman, J.D. & Volpe, B.T. (1986) Components of visual attention: alterations in response pattern to visual stimuli following parietal lobe infarction. *Brain* 109, 99-114.
- Berlucchi, G. & Rizzolatti, G. eds. (1987) *Selective visual attention*, *Neuropsychologia*, 25A.
- Bick, P.A. & Kinsbourne, M. (1987) Auditory hallucinations and subvocal speed in schizophrenic patients. *American Journal of Psychiatry*, 144:22-225
- Carr, T.H. & Pollack, A. (1985) Recognizing printed words: A look at current models. In D. Besner, T.G. Weller & G.E. MacKinnon (eds.) *Reading Research*, New York:Academic Press 2-73.
- Cowey, A. (1985) Aspects of cortical organization related to selective attention and selective impairments of visual perception: A tutorial review. In Posner, M.I. & Marin, O.S.M. (eds.) *Attention and Performance XI*. Hillsdale, N.J.: Erlbaum, 14-62.
- Crick, F. (1984) Function of the thalamic reticular complex: the search light hypothesis. *Proceedings of the National Academy* 81, 4586-4590.
- DeRenzi, E. (1982) *Disorders of Space Exploration and Cognition*. John Wiley, New York.
- DeRenzi, E., Gentilini, M. & Pattacini, F. Auditory extinction following hemisphere damage. *Neuropsychologia*, 1984, 22, 733-744.
- Deuel, R.M. & Collins, R.C. The functional anatomy of frontal lobe neglect in the monkey: behavioral and quantitative 2 DG studies. *Annals of Neurology*, 1984, 15, 521-529.
- Diamond, A. (1987) Development of progressive inhibitory control of action: retrieval of a contiguous object. Paper given to Society for Research in Child Development, Baltimore, MD, April 1987.
- Downing, C.J. & Pinker, S. (1985) in *Attention and Performance XI* (ed.) Posner, M.I. & Marin, O.S.M. Erlbaum: Hillsdale, N.J. 171-187.
- Duncan, J. (1986) Disorganization of behavior after frontal lobe damage. *Cognitive Neuropsychology* 3, 271-290.

- Early, T.S., Reiman, C.M., Raichle, M.E. & Spitznagel, E.L. (1987) Left globus pallidus abnormality in never-medicated patients with schizophrenia. *Proceedings of the National Academy* 84, 561-567.
- Friedrich, F.J., Walker, J. & Posner, M.I. (1985) Effects of parietal lesions on visual matching: implications for reading errors. *Cognitive Neuropsychology*, 1985, 2, 253-264.
- Geschwind, N. (1965) Disconnection syndrome in animals and man. *Brain*, 88:237-294.
- Goldman-Rakic, (In Press) Circuitry of primate prefrontal cortex and regulation of behavior by representational analysis. In Plum, F. & Mountcastle, V. (eds.) *Higher Cortical Function Amer. Physiological Society Handbook of Physiology*, 5, 373-417.
- Hughes, H.C. & Zimba, L.D. (1985) *Journal of Experimental Psychology: Human Perception and Performance* 11, 409-430.
- James, W. *Principles of psychology* (Vol. 1) New York:Holt, 1890.
- Jonides, J. (1981) in *Attention and Performance* (Vol. IX) (Long, J. and Baddeley, A., eds). pp. 87-207, Erlbaum.
- Jonides, J. & Mack, R. On the cost and benefit of cost and benefit. *Psychology Bulletin*, 1984, 96, 29-44.
- Kosslyn, S.M. (1980) *Image and Mind*. Harvard Press, Cambridge, MA.
- LaBerge, D.L. & Samuels, J. (1974) Toward a theory of automatic word processing in reading. *Cognitive Psychology*, 6, 293-323.
- LaDavas, E. (1987) Is hemispatial deficit produced by right parietal damage associated with retinal or cravitational coordinates. *Brain*. 110, 167-180.
- Mangun, G.R., Hansen, J.C. & Hillyard, S.A. The spatial orienting of attention: sensory facilitation or response bias? *ONR Technical Report SDEPL 001*, December 1986.
- Marcel, (1983) Conscious and unconscious perception. *Cognitive Psychology*, 15, 238-300.
- Maylor, E. A. (1985) in *Attention and Performance* (Vol. XI) (Posner, M.I. and Marin, O.S.M., eds), pp. 189-204, Erlbaum.
- McClelland, J.L. & Rumelhart, D.E. (1986) *Parallel distributed processing, explorations in the microstructures of cognition, Volume 1: Foundations*.
- Mirsky, A.F. & Duncan, C.C. Etiology and expression of schizophrenia: neurobiological and psychosocial factors. *Ann. Rev. Psych.* 1986, 37, 291-319.
- Mishkin, M., Ungerleider, L.G. & Macko, K.A. (1983) Object vision: Two cortical pathways. *Trends in Neuroscience*, 6:414-417.

- Morrison, R.E. (1984) Manipulation of stimulus onset delay in reading: Evidence for parallel programming of saccades. *Journal of Experimental Psychology: Human Perception and Performance*, Vol. 10, No. 5, 667-682.
- Morrow, L.A. & Ratcliff, G. (1987) Attentional mechanisms in clinical neglect. *Journal of Clinical and Experimental Neuropsychology*, Vol 9, Number 1, (Abstract).
- Mountcastle, V.B. (1978) Brain systems for directed attention. *Journal Royal Society of Medicine*, 71, 14-27.
- Nagel-Leiby, S., Buchtel, H. & Welch, K.M.A. (1987) Right frontal and parietal lobe contributions to the process of directed visual attention and orientation. *Journal of Clinical and Experimental Neuropsychology*, Vol. 9, Number 1, (Abstract).
- Neely, J., Keefe, D. & Ross, K. (1986) Retrospective postlexical processes produce the proportion effect in semantic priming. Paper presented at the meeting of the Psychonomics Society, New Orleans, Louisiana.
- Petersen, S.E. Robinson, D.L. & Morris, J.D. (1987) Contributions of the pulvinar to visual spatial attention. *Neuropsychologia*, 25, 97-105.
- Posner, M.I. (1978) *Chronometric Explorations of Mind*. Lawrence Erlbaum, Hillsdale, New Jersey.
- Posner, M.I. (1980) Orienting of attention. *Quarterly Journal of Experimental Psychology* 32, 3-25.
- Posner, M.I. (1982) Cumulative development of attentional theory. *American Psychologist*, 32:53-64.
- Posner, M.I. (1986) A framework for relating cognitive and neural systems. *EEG and Clinical Neurophysiology, Supplement 38*, 1986, 155-166.
- Posner, M.I., Choate, L.S., Rafal, R. D., & Vaughn, J. (1985) Inhibition of return: Neural mechanisms and function. *Cognitive Neuropsychology* 2, 211-228.
- Posner, M.I. & Cohen, Y. Components of attention. In H. Bouman and D. Bowhuis (eds.), *Attention and Performance X*. Hillsdale, N.J.:Lawrence Erlbaum, 1984, 55-66.
- Posner, M.I., Cohen, Y., Choate, L., Hockey, R. & Maylor, E. (1984) Sustained concentration: passive filtering or active orienting. In (Kornblum, S. and Requin, J., eds). *Preparatory States and Processes*, pp. 49-65, Erlbaum.
- Posner, M.I., Cohen Y. & Rafal, R.D. (1982) *Philosophical Transaction Royal Society of London Series B*. 2908, 187-198.
- Posner, M.I., Early, T., Cippin, P. & Reiman, E. Does Schizophrenia involve a left hemisphere deficit of attention? In preparation.

- Posner, M.I., Inhoff, A.W., Friedrich, F.J. & Cohen, A. (1987) Isolating Attentional Systems: A Cognitive-Anatomical Analysis. *Psychobiology*
- Posner, M.I., Walker, J.A., Friedrich, F.J. & Rafal, R.D. (1984) Effects of parietal lobe injury on covert orienting of visual attention. *Journal of Neuroscience* 4, 1863-1874.
- Posner, M.I. & Henik, A. (1983) Isolating representational systems. In J. Beck, B. Hope and A. Rosenfeld (Eds.), *Human and Machine Vision*, New York: Academic Press, 1983, 395-412.
- Posner, M.I. & Marin, O.S.M. (Eds.) (1985) *Attention and Performance XI: Mechanisms of Attention*. Hillsdale, N.J.: Lawrence Erlbaum
- Posner, M.I. & Presti, D. (1987) Selective attention and cognitive control. *Trends in Neuroscience*, 10, 12-17.
- Posner, M.I. & Snyder, C.R. (1975) Facilitation and inhibition in the processing of signals. *Attention and Performance V*. New York: Academic Press, 669-681.
- Prinzmetal, W., Presti, D. & Posner, M.I. (1986) Does attention affect feature integration? *Journal of Experimental Psychology: Human Perception and Performance* 12, 361-369.
- Rafal, R.D. & Inhoff, A.W. (1986) Midbrain mechanisms for orienting visual attention. In: *Program of the Eighth Annual Conference of Cognitive Science Society*, London: Lawrence Erlbaum, Abstract.
- Rafal, R.D. & Posner, M.I. (1987) Deficits in Visual Spatial Attention Following Thalamic Lesions, In preparation.
- Rizzolatti, G., Umiltà, C., Dascola, I. & Umiltà, C. Reorienting attention across the horizontal and vertical meridians: evidence in favor of a premotor theory of attention. *Neuropsychologia*, Volume 25, Number 1A.
- Roland, P.E. Cortical organization of voluntary behavior in man. *Human Neurobiology* (1985) 4:155-167.
- Rumelhart, J.L. & McClelland, D.E. (1981) An interactive activation model of context effects in letter perception: part 1. An account of basic findings. *Psychological Review*, 88, 375-407.
- Rumelhart, D. & Norman, D.A. Simulating a skilled typist. *Cognitive Science*, 1982, 6, 1-36.
- Sandson, J. & Posner, M.I. (1987) Effects of divided attention on identity and semantic priming. *ONR Technical Report*.
- Sieroff, E. & Michel, F. (1987) In right/left hemisphere patients and the problem of lexical access, *Neuropsychologia*.
- Sieroff, E., Pollatsek, A. & Posner, M.I. (1987) Recognition of visual letter strings following injury to the posterior visual spatial attention system, *Cognitive Neuropsychology*.

- Sieroff, E., & Posner, M.I. (1987) Cueing spatial attention during processing of words and letter strings in normals, *Cognitive Neuropsychology*.
- Taylor, D.A. (1977) Time course of context effects, *Journal of Experimental Psychology*, 106:404-426.
- Titchner, E.B. (1908) *Lectures on the elementary psychology of feeling and attention*. New York: Macmillan.
- Treisman, (1987) Features and objects in visual processing. *Scientific American*, 114-125.
- Walker, J.A., Friedrich, F.J., & Posner, M.I. (1983) Spatial conflict in parietal lesions. Paper presented to International Neuropsychology Society, San Diego.
- Weinberger, D.R (1986). The pathogenesis of schizophrenia. In Nasrallah, H.A. Weinberger, D.R. (eds.) *The Neurology of Schizophrenia*, 397-407.
- Wurtz, R.H., Goldberg, M.E. & Robinson, D.L. (1980) *Progress in Psychobiology and Physiological Psychology*, 9, 43-83.

Distribution List

Dr. Phillip L. Ackerman  
University of Minnesota  
Department of Psychology  
Minneapolis, MN 55455

Dr. Beth Adelson  
Department of Computer Science  
Tufts University  
Medford, MA 02155

Technical Director,  
Army Human Engineering Lab  
Aberdeen Proving Ground  
MD 21005

Dr. Robert Ahlers  
Code N711  
Human Factors Laboratory  
Naval Training Systems Center  
Orlando, FL 32813

Dr. Ed Aiken  
Navy Personnel R&D Center  
San Diego, CA 92152-6800

Dr. John Allen  
Department of Psychology  
George Mason University  
4400 University Drive  
Fairfax, VA 22030

Dr. Earl A. Alluisi  
HQ, AFHRL (AFSC)  
Brooks AFB, TX 78235

Dr. James Anderson  
Brown University  
Center for Neural Science  
Providence, RI 02912

Dr. Nancy S. Anderson  
Department of Psychology  
University of Maryland  
College Park, MD 20742

Technical Director, ARI  
5001 Eisenhower Avenue  
Alexandria, VA 22333

Dr. Gary Aston-Jones  
Department of Biology  
New York University  
1009 Main Bldg  
Washington Square  
New York, NY 10003

Dr. Alan Baddeley  
Medical Research Council  
Applied Psychology Unit  
15 Chaucer Road  
Cambridge CB2 2EF  
ENGLAND

Dr. James Ballas  
Georgetown University  
Department of Psychology  
Washington, DC 20057

Dr. Harold Bamford  
National Science Foundation  
1800 G Street, N.W.  
Washington, DC 20550

Dr. Isaac Bejar  
Educational Testing Service  
Princeton, NJ 08450

Dr. Alvah Bittner  
Naval Biodynamics Laboratory  
New Orleans, LA 70189

Dr. John Blaha  
Department of Psychology  
George Mason University  
4400 University Drive  
Fairfax, VA 22030

Dr. Sue Bogner  
Army Research Institute  
ATTN: rERI-SF  
5001 Eisenhower Avenue  
Alexandria, VA 22333-5600

Dr. Gordon H. Bower  
Department of Psychology  
Stanford University  
Stanford, CA 94306

Hr. Donald C. Burgy  
General Physics Corp.  
10650 Hickory Ridge Rd.  
Columbia, MD 21044

Dr. Gail Carpenter  
Northeastern University  
Department of Mathematics, 504LA  
360 Huntington Avenue  
Boston, MA 02115

Dr. Pat Carpenter  
Carnegie-Mellon University  
Department of Psychology  
Pittsburgh, PA 15213

Dr. Tyrone Cashman  
American Society of  
Cybernetics  
3428 Fremont Ave. South  
Minneapolis MN 55408

Dr. Alphonse Chapanis  
8415 Bellona Lane  
Suite 210  
Buxton Towers  
Baltimore, MD 21204

Dr. Paul R. Chatelier  
OUSDRE  
Pentagon  
Washington, DC 20350-2000

Mr. Raymond E. Christal  
AFHRL/HOE  
Brooks AFB, TX 78235

Dr. David E. Clement  
Department of Psychology  
University of South Carolina  
Columbia, SC 29208

Dr. Charles Clifton  
Tobin Hall  
Department of Psychology  
University of  
Massachusetts  
Amherst, MA 01003

Assistant Chief of Staff  
for Research, Development,  
Test, and Evaluation  
Naval Education and  
Training Command (N-5)  
NAS Pensacola, FL 32508

Dr. Michael Coles  
University of Illinois  
Department of Psychology  
Champaign, IL 61820

Dr. Allan M. Collins  
Bolt Beranek & Newman, Inc.  
50 Houlton Street  
Cambridge, MA 02138

Dr. Stanley Collyer  
Office of Naval Technology  
Code 222  
800 N. Quincy Street  
Arlington, VA 22217-5000

Dr. Leon Cooper  
Brown University  
Center for Neural Science  
Providence, RI 02912

Dr. Lynn A. Cooper  
Learning R&D Center  
University of Pittsburgh  
3939 O'Hara Street  
Pittsburgh, PA 15213

Phil Cunniff  
Commanding Officer, Code 7522  
Naval Undersea Warfare Engineering  
Keyport, WA 98345

Brian Dallman  
3400 TTM/TTGXS  
Lowry AFB, CO 80230-5000

LT John Deaton  
ONR Code 125  
800 N. Quincy Street  
Arlington, VA 22217-5000

Dr. Stanley Deutsch  
Committee on Human Factors  
National Academy of Sciences  
2101 Constitution Ave.  
Washington, DC 20418

Dr. R. K. Dismukes  
Associate Director for Life Sciences  
AFOSR  
Bolling AFB  
Washington, DC 20332

Distribution List



(Washington University/Posner) 1987/11/10)

Distribution List

Dr. Daniel Gopher  
Industrial Engineering  
& Management  
TECHNION  
Haifa 32000  
ISRAEL

Dr. Sherrie Gott  
AFMRL/MODJ  
Brooks AFB, TX 78235

Jordan Grafman, Ph.D.  
2021 Lyttonsville Road  
Silver Spring, MD 20910

Dr. Richard H. Granger  
Department of Computer Science  
University of California, Irvine  
Irvine, CA 92717

Dr. Steven Grant  
Department of Biology  
New York University  
1009 Main Bldg  
Washington Square  
New York, NY 10003

Dr. Wayne Gray  
Army Research Institute  
5001 Eisenhower Avenue  
Alexandria, VA 22333

Dr. Bert Green  
Johns Hopkins University  
Department of Psychology  
Charles & 34th Street  
Baltimore, MD 21218

Dr. James G. Greeno  
University of California  
Berkeley, CA 94720

Dr. William Greenough  
University of Illinois  
Department of Psychology  
Champaign, IL 61820

Dr. Stephen Grossberg  
Center for Adaptive Systems  
Room 244  
111 Cummington Street  
Boston University  
Boston, MA 02215

Dr. Muhammad K. Habib  
University of North Carolina  
Department of Biostatistics  
Chapel Hill, NC 27514

Prof. Edward Haertel  
School of Education  
Stanford University  
Stanford, CA 94305

Dr. Henry M. Halff  
Halff Resources, Inc.  
4918 33rd Road, North  
Arlington, VA 22207

Dr. Nancy F. Halff  
Halff Resources, Inc.  
4918 33rd Road, North  
Arlington, VA 22207

Dr. Ronald K. Hambleton  
Prof. of Education & Psychology  
University of Massachusetts  
at Amherst  
Hills House  
Amherst, MA 01003

Dr. Cheryl Hamel  
NTSC  
Orlando, FL 32813

Mr. William Hartung  
PEAM Product Manager  
Army Research Institute  
5001 Eisenhower Avenue  
Alexandria, VA 22333

Dr. Harold Hawkins  
Office of Naval Research  
Code 1142PT  
800 N. Quincy Street  
Arlington, VA 22217-5000

Prof. John R. Hayes  
Carnegie-Mellon University  
Department of Psychology  
Schenley Park  
Pittsburgh, PA 15213

Dr. Joan I. Heller  
505 Haddon Road  
Oakland, CA 94606

Distribution List

Dr. Stephanie Doan  
Code 6021  
Naval Air Development Center  
Warminster, PA 18974-5000

Dr. Emanuel Donchin  
University of Illinois  
Department of Psychology  
Champaign, IL 61820

Mr. Ralph Dusek  
ARD Corporation  
5457 Twins Knolls Road  
Suite 400  
Columbia, MD 21045

Dr. Ford Ebner  
Brown University  
Anatomy Department  
Medical School  
Providence, RI 02912

Dr. Jeffrey Elman  
University of California,  
San Diego  
Department of Linguistics, C-008  
La Jolla, CA 92093

Dr. William Epstein  
University of Wisconsin  
W. J. Brogden Psychology Bldg.  
1202 W. Johnson Street  
Madison, WI 53706

Dr. K. Anders Ericsson  
University of Colorado  
Department of Psychology  
Boulder, CO 80309

Dr. Jerome A. Feldman  
University of Rochester  
Computer Science Department  
Rochester, NY 14627

Dr. Paul Feltovich  
Southern Illinois University  
School of Medicine  
Medical Education Department  
P.O. Box 3926  
Springfield, IL 62708

Dr. Craig I. Fields  
ARPA  
1400 Wilson Blvd.  
Arlington, VA 22209

Dr. Gail R. Fleischaker  
Margulis Lab  
Biological Sci. Center  
2 Cummington Street  
Boston, MA 02215

Dr. Jane M. Flinn  
Department of Psychology  
George Mason University  
4400 University Drive  
Fairfax, VA 22030

Dr. Michaela Gallagher  
University of North Carolina  
Department of Psychology  
Chapel Hill, NC 27514

Dr. R. Edward Geiselman  
Department of Psychology  
University of California  
Los Angeles, CA 90024

Dr. Don Gentner  
Center for Human  
Information Processing  
University of California  
La Jolla, CA 92093

Dr. Lee Giles  
4FOSR  
Bolling AFB  
Washington, DC 20332

Dr. Eugene E. Gloye  
Office of Naval Research  
Detachment  
1030 E. Green Street  
Pasadena, CA 91106-2485

Dr. Joseph Goguen  
Computer Science Laboratory  
SRI International  
333 Ravenswood Avenue  
Menlo Park, CA 94025

Distribution List

Dr. Per Helmersen  
University of Oslo  
Department of Psychology  
Box 1094  
Oslo 3, NORWAY

Dr. Steven A. Millyard  
Department of Neurosciences  
University of California,  
San Diego  
La Jolla, CA 92033

Dr. Geoffrey Hinton  
Carnegie-Mellon University  
Computer Science Department  
Pittsburgh, PA 15213

Dr. J. M. Hollan  
Intelligent Systems Group  
Institute for  
Cognitive Science (C-015)  
UCSD  
La Jolla, CA 92093

Dr. John Holland  
University of Michigan  
2313 East Engineering  
Ann Arbor, MI 48109

Dr. Melissa Holland  
Army Research Institute for the  
Behavioral and Social Sciences  
5001 Eisenhower Avenue  
Alexandria, VA 22333

Dr. Keith Holyoak  
University of Michigan  
Human Performance Center  
330 Packard Road  
Ann Arbor, MI 48109

Dr. James Howard  
Dept. of Psychology  
Human Performance Laboratory  
Catholic University of  
America  
Washington, DC 20064

Dr. Lloyd Humphreys  
University of Illinois  
Department of Psychology  
603 East Daniel Street  
Champaign, IL 61820

Dr. Earl Hunt  
Department of Psychology  
University of Washington  
Seattle, WA 98105

Dr. Ed Hutchins  
Intelligent Systems Group  
Institute for  
Cognitive Science (C-015)  
UCSD  
La Jolla, CA 92093

Dr. Alice Isen  
Department of Psychology  
University of Maryland  
Catonsville, MD 21228

COL Dennis W. Jarvis  
Commander  
AFHRL  
Brooks AFB, TX 78235-5601

Dr. Joseph E. Johnson  
Assistant Dean for  
Graduate Studies  
College of Science and Mathematics  
University of South Carolina  
Columbia, SC 29208

CDR Tom Jones  
ONR Code 125  
800 N. Quincey Street  
Arlington, VA 22217-5000

Mr. Daniel B. Jones  
U.S. Nuclear Regulatory  
Commission  
Division of Human Factors Safety  
Washington, DC 20555

Dr. Douglas M. Jones  
Thatcher Jones Associates  
P.O. Box 6640  
10 Trafalgar Court  
Lawrenceville, NJ 08648

Dr. Jane Jorgensen  
University of Oslo  
Institute of Psychology  
Box 1094, Blindern  
Oslo, NORWAY

Dr. Marcel Just  
Carnegie-Mellon University  
Department of Psychology  
Schenley Park  
Pittsburgh, PA 15213

Dr. Daniel Kahneman  
The University of British Columbia  
Department of Psychology  
#154-2053 Main Hall  
Vancouver, British Columbia  
CANADA V6T 1Y7

Dr. Ruth Kanfer  
University of Minnesota  
Department of Psychology  
Elliott Hall  
75 E. River Road  
Minneapolis, MN 55455

Dr. Demetrios Karis  
Grumman Aerospace Corporation  
MS C04-14  
Bethpage, NY 11714

Dr. Milton S. Katz  
Army Research Institute  
5001 Eisenhower Avenue  
Alexandria, VA 22333

Dr. Steven W. Keele  
Department of Psychology  
University of Oregon  
Eugene, OR 97403

Dr. Wendy Kellogg  
IBM T. J. Watson Research Ctr.  
P.O. Box 218  
Yorktown Heights, NY 10598

Dr. Scott Kelso  
Haskins Laboratories,  
270 Crown Street  
New Haven, CT 06510

Dr. Dennis Kibler  
University of California  
Department of Information  
and Computer Science  
Irvine, CA 92717

Dr. David Kieras  
University of Michigan  
Technical Communication  
College of Engineering  
1223 E. Engineering Building  
Ann Arbor, MI 48109

Dr. David Klahr  
Carnegie-Mellon University  
Department of Psychology  
Schenley Park  
Pittsburgh, PA 15213

Dr. Ronald Knoll  
Bell Laboratories  
Murray Hill, NJ 07974

Dr. Sylvan Kornblum  
University of Michigan  
Mental Health Research Institute  
205 Washtenaw Place  
Ann Arbor, MI 48109

Dr. Stephen Kosslyn  
Harvard University  
1236 William James Hall  
33 Kirkland St.  
Cambridge, MA 02138

Dr. Kenneth Kotovsky  
Department of Psychology  
Community College of  
Allegheny County  
800 Allegheny Avenue  
Pittsburgh, PA 15233

Dr. David H. Krantz  
2 Washington Square Village  
Apt. # 15J  
New York, NY 10012

Dr. David R. Lambert  
Naval Ocean Systems Center  
Code 4417  
271 Catalina Boulevard  
San Diego, CA 92152-6800

Dr. Pat Langley  
University of California  
Department of Information  
and Computer Science  
Irvine, CA 92717

Distribution List

Distribution List

Dr. Marcy Lansman  
University of North Carolina  
The L. L. Thurstone Lab.  
Davie Hall 013A  
Chapel Hill, NC 27514

Dr. Jill Larkin  
Carnegie-Mellon University  
Department of Psychology  
Pittsburgh, PA 15213

Dr. Robert Lawler  
Information Sciences, FRL  
GTE Laboratories, Inc.  
40 Sylvan Road  
Waltham, MA 02254

Dr. Paul E. Lehner  
PAR Technology Corp.  
7926 Jones Branch Drive  
Suite 170  
McLean, VA 22102

Dr. Alan M. Lesgold  
Learning R&D Center  
University of Pittsburgh  
Pittsburgh, PA 15260

Dr. Jim Levin  
Department of  
Educational Psychology  
210 Education Building  
1310 South Sixth Street  
Champaign, IL 61820-6990

Dr. John Levine  
Learning R&D Center  
University of Pittsburgh  
Pittsburgh, PA 15260

Dr. Michael Levine  
Educational Psychology  
210 Education Bldg.  
University of Illinois  
Champaign, IL 61801

Dr. Clayton Lewis  
University of Colorado  
Department of Computer Science  
Campus Box #30  
Boulder, CO 80309

Dr. Bob Lloyd  
Dept. of Geography  
University of South Carolina  
Columbia, SC 29208

Dr. Frederic M. Lord  
Educational Testing Service  
Princeton, NJ 08541

Dr. Gary Lynch  
University of California  
Center for the Neurobiology of  
Learning and Memory  
Irvine, CA 92717

Dr. Don Lyon  
P. O. Box 44  
Higley, AZ 85236

Dr. William L. Maloy  
Chief of Naval Education  
and Training  
Naval Air Station  
Pensacola, FL 32508

Dr. Evans Mandes  
Department of Psychology  
George Mason University  
4400 University Drive  
Fairfax, VA 22030

Dr. Sandra P. Marshall  
Dept. of Psychology  
San Diego State University  
San Diego, CA 92182

Dr. Richard E. Mayer  
Department of Psychology  
University of California  
Santa Barbara, CA 93106

Dr. James McBride  
Psychological Corporation  
c/o Harcourt, Brace,  
Jovanovich Inc.  
1250 West 6th Street  
San Diego, CA 92101

Dr. Jay McClelland  
Department of Psychology  
Carnegie-Mellon University  
Pittsburgh, PA 15213

Dr. James L. McGaugh  
Center for the Neurobiology  
of Learning and Memory  
University of California, Irvine  
Irvine, CA 92717

Dr. Gail McKoon  
CAS/Psychology  
Northwestern University  
1859 Sheridan Road  
Kresge #230  
Evanston, IL 60201

Dr. Joe McLachlan  
Navy Personnel R&D Center  
San Diego, CA 92152-6800

Dr. James McMichael  
Assistant for HP Research,  
Development, and Studies  
OP 01B7  
Washington, DC 20370

Dr. Barbara Means  
Human Resources  
Research Organization  
1100 South Washington  
Alexandria, VA 22314

Dr. George A. Miller  
Department of Psychology  
Green Hall  
Princeton University  
Princeton, NJ 08540

Dr. Robert Mislevy  
Educational Testing Service  
Princeton, NJ 08541

Dr. William Montague  
NPRDC Code 13  
San Diego, CA 92152-6800

Mr. Melvin D. Montemerlo  
NASA Headquarters  
RTE-6  
Washington, DC 20546

Dr. Tom Moran  
Xerox PARC  
3333 Coyote Hill Road  
Palo Alto, CA 94304

Dr. Randy Mumaw  
Program Manager  
Training Research Division  
HumRRO  
1100 S. Washington  
Alexandria, VA 22314

Dr. Allen Munro  
Behavioral Technology  
Laboratories - USC  
1845 S. Elena Ave., 4th Floor  
Redondo Beach, CA 90277

Dr. Richard E. Nisbett  
University of Michigan  
Institute for Social Research  
Room 5261  
Ann Arbor, MI 48109

Dr. Mary Jo Nissen  
University of Minnesota  
#218 Elliott Hall  
Minneapolis, MN 55455

Deputy Technical Director  
NPRDC Code 01A  
San Diego, CA 92152-6800

Director, Training Laboratory,  
NPRDC (Code 05)  
San Diego, CA 92152-6800

Director, Manpower and Personnel  
Laboratory,  
NPRDC (Code 06)  
San Diego, CA 92152-6800

Director, Human Factors  
& Organizational Systems Lab,  
NPRDC (Code 07)  
San Diego, CA 92152-6800

Fleet Support Office,  
NPRDC (Code 301)  
San Diego, CA 92152-6800

Commanding Officer,  
Naval Research Laboratory  
Code 2627  
Washington, DC 20390

(Washington University/Posner) 1987/11/10

Distribution List

Distribution List

Dr. Harold F. O'Neill, Jr.  
School of Education - WRH 801  
Department of Educational  
Psychology & Technology  
University of Southern California  
Los Angeles, CA 90089-0031

Dr. Michael Oberlin  
Naval Training Systems Center  
Code 711  
Orlando, FL 32813-7100

Dr. Stellan Ohlsson  
Learning R & D Center  
University of Pittsburgh  
3939 O'Hara Street  
Pittsburgh, PA 15213

Director, Research Programs,  
Office Naval Research  
800 North Quincy Street  
Arlington, VA 22217-5000

Mathematics Group,  
Office of Naval Research  
Code 1111MA  
800 North Quincy Street  
Arlington, VA 22217-5000

Office of Naval Research,  
Code 1133  
800 N. Quincy Street  
Arlington, VA 22217-5000

Office of Naval Research,  
Code 1141NP  
800 N. Quincy Street  
Arlington, VA 22217-5000

Office of Naval Research,  
Code 1142  
800 N. Quincy St.  
Arlington, VA 22217-5000

Office of Naval Research,  
Code 142EP  
800 N. Quincy Street  
Arlington, VA 22217-5000

Office of Naval Research,  
Code 1142PT  
800 N. Quincy Street  
Arlington, VA 22217-5000  
(Copies)

Director, Technology Programs,  
Office of Naval Research  
Code 12  
800 North Quincy Street  
Arlington, VA 22217-5000

Special Assistant for Marine  
Corps Matters,  
ONR Code 00MC  
800 N. Quincy St.  
Arlington, VA 22217-5000

Dr. Judith Orasan  
Army Research Institute  
5001 Eisenhower Avenue  
Alexandria, VA 22333

Dr. Jesse Orlansky  
Institute for Defense Analyses  
1801 W. Beauregard St.  
Alexandria, VA 22311

Dr. Glenn Osge  
NOSC, Code 441  
San Diego, CA 92152-6800

Prof. Seymour Papert  
20C-109  
Massachusetts Institute  
of Technology  
Cambridge, MA 02139

Dr. Robert F. Pasnak  
Department of Psychology  
George Mason University  
4400 University Drive  
Fairfax, VA 22030

Daira Paulson  
Code 52 - Training Systems  
Navy Personnel R&D Center  
San Diego, CA 92152-6800

Dr. James Paulson  
Department of Psychology  
Portland State University  
P.O. Box 751  
Portland, OR 97207

Dr. James W. Pellgrino  
University of California,  
Santa Barbara  
Department of Psychology  
Santa Barbara, CA 93106

Dr. Nancy Pennington  
University of Chicago  
Graduate School of Business  
1101 E. 58th St.  
Chicago, IL 60637

Dr. Ray Perez  
ARI (PERI-11)  
5001 Eisenhower Avenue  
Alexandria, VA 22333

Dr. Steven Pinker  
Department of Psychology  
E10-018  
M.I.T.  
Cambridge, MA 02139

Dr. Hart J. Polson  
Department of Psychology  
Campus Box 346  
University of Colorado  
Boulder, CO 80309

Dr. Peter Polson  
University of Colorado  
Department of Psychology  
Boulder, CO 80309

Dr. Steven E. Pollock  
MCC  
9430 Research Blvd.  
Echelon Bldg #1  
Austin, TX 78759-6509

Dr. Michael I. Posner  
Department of Neurology  
Washington University  
Medical School  
St. Louis, MO 63110

Dr. Mary C. Potter  
Department of Psychology  
72 (E-10-032)  
Cambridge, MA 02139

Dr. Karl Pribram  
Stanford University  
Department of Psychology  
Bldg. 4201 -- Jordan Hall  
Stanford, CA 94305

Dr. Joseph Psotka  
ATTN: PERI-1C  
Army Research Institute  
5001 Eisenhower Ave.  
Alexandria, VA 22333

Dr. Mark D. Reckase  
ACT  
P. O. Box 168  
Iowa City, IA 52243

Dr. Lynne Reder  
Department of Psychology  
Carnegie-Mellon University  
Schenley Park  
Pittsburgh, PA 15213

Dr. James A. Reggia  
University of Maryland  
School of Medicine  
Department of Neurology  
11th Greene Street  
Baltimore, MD 21201

Dr. Fred Reif  
Physics Department  
University of California  
Berkeley, CA 94720

Dr. Daniel Reisberg  
Department of Psychology  
New School for Social Research  
65 Fifth Avenue  
New York, NY 10003

Dr. Lauren Resnick  
Learning R & D Center  
University of Pittsburgh  
3939 O'Hara Street  
Pittsburgh, PA 15213

Distribution List

Distribution List

Mr. Raymond C. Sidorsky  
Army Research Institute  
5001 Eisenhower Avenue  
Alexandria, VA 22733

Dr. Herbert A. Simon  
Department of Psychology  
Carnegie-Mellon University  
Schenley Park  
Pittsburgh, PA 15213

Dr. Zita M Simutis  
Instructional Technology  
Systems Area  
ARI  
5001 Eisenhower Avenue  
Alexandria, VA 22333

Dr. H. Wallace Sinalko  
Manpower Research  
and Advisory Services  
Smithsonian Institution  
801 North Pitt Street  
Alexandria, VA 22314

Dr. Derek Sleeman  
Stanford University  
School of Education  
Stanford, CA 94305

Dr. Edward E. Smith  
Bolt Beranek & Newman, Inc.  
50 Houlton Street  
Cambridge, MA 02138

Dr. Linda B. Smith  
Department of Psychology  
Indiana University  
Bloomington, IN 47405

Dr. Robert F. Smith  
Department of Psychology  
George Mason University  
4400 University Drive  
Fairfax, VA 22030

Dr. Alfred F. Smode  
Senior Scientist  
Code 07A  
Naval Training Systems Center  
Orlando, FL 32813

Dr. Richard E. Snow  
Department of Psychology  
Stanford University  
Stanford, CA 94306

Dr. Elliot Soloway  
Yale University  
Computer Science Department  
P.O. Box 2158  
New Haven, CT 06520

Dr. Kathryn T. Spoehr  
Brown University  
Department of Psychology  
Providence, RI 02912

James J. Staszewski  
Research Associate  
Carnegie-Mellon University  
Department of Psychology  
Schenley Park  
Pittsburgh, PA 15213

Dr. Ted Steinke  
Dept. of Geography  
University of South Carolina  
Columbia, SC 29208

Dr. Robert Sternberg  
Department of Psychology  
Yale University  
Box 11A, Yale Station  
New Haven, CT 06520

Dr. Saul Sternberg  
University of Pennsylvania  
Department of Psychology  
3815 Walnut Street  
Philadelphia, PA 19104

Dr. Albert Stevens  
Bolt Beranek & Newman, Inc.  
10 Houlton St.  
Cambridge, MA 02238

Dr. Paul J. Sticha  
Senior Staff Scientist  
Training Research Division  
HumRRO  
1100 S. Washington  
Alexandria, VA 22314

Dr. Gil Ricard  
Mail Stop C04-14  
Grumman Aerospace Corp.  
Bethpage, NY 11714

Dr. Mary S. Riley  
Program in Cognitive Science  
Center for Human Information  
Processing  
University of California  
La Jolla, CA 92093

Dr. Andrew M. Rose  
American Institutes  
for Research  
1055 Thomas Jefferson St., NW  
Washington, DC 20007

Dr. Ernst Z. Rothkopf  
AT&T Bell Laboratories  
Room 2D-456  
600 Mountain Avenue  
Murray Hill, NJ 07974

Dr. William B. Rouse  
Search Technology, Inc.  
25-b Technology Park/Atlanta  
Norcross, GA 30092

Dr. Donald Rubin  
Statistics Department  
Science Center, Room 608  
1 Oxford Street  
Harvard University  
Cambridge, MA 02138

Dr. David Rumelhart  
Center for Human  
Information Processing  
Univ. of California  
La Jolla, CA 92093

Dr. E. L. Saltzman  
Haskins Laboratories  
270 Crown Street  
New Haven, CT 06510

Dr. Fumiko Samejima  
Department of Psychology  
University of Tennessee  
Knoxville, TN 37916

Dr. Michael J. Samet  
Perceptronics, Inc  
6271 Variel Avenue  
Woodland Hills, CA 91364

Dr. Arthur Samuel  
Yale University  
Department of Psychology  
Box 11A, Yale Station  
New Haven, CT 06520

Dr. Roger Schank  
Yale University  
Computer Science Department  
P.O. Box 2158  
New Haven, CT 06520

Dr. Walter Schneider  
Learning R&D Center  
University of Pittsburgh  
3939 O'Hara Street  
Pittsburgh, PA 15260

Dr. Janet Schofield  
Learning R&D Center  
University of Pittsburgh  
Pittsburgh, PA 15260

Dr. Hans-Wilhelm Schroiff  
Institut fuer Psychologie  
der RWTH Aachen  
Jaegerstrasse zwischen 17 u. 19  
5100 Aachen  
WEST GERMANY

Dr. Robert J. Seldel  
US Army Research Institute  
5001 Eisenhower Ave.  
Alexandria, VA 22333

Dr. Michael G. Shafto  
ONR Code 1142PT  
800 N. Quincy Street  
Arlington, VA 22217-5000

Dr. T. B. Sheridan  
Dept. of Mechanical Engineering  
MIT  
Cambridge, MA 02139

(Washington University/Posner) 1987/11/10

Distribution List

Cdr Michael Suman, PD 303  
Naval Training Systems Center  
Code N51, Comptroller  
Orlando, FL 32813

Dr. Steve Suomi  
NIH Bldg. 31  
Room B2B-15  
Bethesda, MD 20205

Dr. Hariharan Swaminathan  
Laboratory of Psychometric and  
Evaluation Research  
School of Education  
University of Massachusetts  
Amherst, MA 01003

Mr. Brad Sympson  
Navy Personnel R&D Center  
San Diego, CA 92152-6800

Dr. John Tangney  
AFOSR/NL  
Bolling AFB, DC 20332

Dr. Kikumi Tatsuoka  
CERL  
252 Engineering Research  
Laboratory  
Urbana, IL 61801

Dr. Maurice Tatsuoka  
220 Education Bldg  
1310 S. Sixth St.  
Champaign, IL 61820

Dr. Richard F. Thompson  
Stanford University  
Department of Psychology  
Bldg. 4201 -- Jordan Hall  
Stanford, CA 94305

Dr. Martin A. Yalcott  
3001 Veazey Terr., N.W.  
Apt. 1617  
Washington, DC 20008

Dr. Douglas Towne  
Behavioral Technology Labs  
1845 S. Elena Ave.  
Redondo Beach, CA 90277

Dr. Robert Tsutakawa  
University of Missouri  
Department of Statistics  
222 Math. Sciences Bldg.  
Columbia, MO 65211

Dr. Michael T. Turvey  
Haskins Laboratories  
27D Crown Street  
New Haven, CT 06510

Dr. Amos Tversky  
Stanford University  
Dept. of Psychology  
Stanford, CA 94305

Dr. James Tweeddale  
Technical Director  
Navy Personnel R&D Center  
San Diego, CA 92152-6800

Dr. Zita E. Tyler  
Department of Psychology  
George Mason University  
4400 University Drive  
Fairfax, VA 22030

Headquarters, U. S. Marine Corps  
Code HPI-20  
Washington, DC 20380

Dr. David Vale  
Assessment Systems Corp.  
2233 University Avenue  
Suite 310  
St. Paul, MN 55114

Dr. Kurt Van Lehn  
Department of Psychology  
Carnegie-Mellon University  
Schenley Park  
Pittsburgh, PA 15213

Dr. Jerry Vogt  
Navy Personnel R&D Center  
Code 51  
San Diego, CA 92152-6800

Dr. Howard Wainer  
Division of Psychological Studies  
Educational Testing Service  
Princeton, NJ 08541

Distribution List

Dr. Beth Warren  
Bolt Beranek & Newman, Inc.  
50 Houlton Street  
Cambridge, MA 02138

Dr. Norman H. Weinberger  
University of California  
Center for the Neurobiology  
of Learning and Memory  
Irvine, CA 92717

Dr. David J. Weiss  
N660 Elliott Hall  
University of Minnesota  
75 E. River Road  
Minneapolis, MN 55455

Dr. Shih-Sung Wer.  
Jackson State University  
1325 J. R. Lynch Street  
Jackson, MS 39217

Dr. Keith T. Westcott  
FHC Corporation  
Central Engineering Labs  
1185 Coleman Ave., Box 580  
Santa Clara, CA 95052

Dr. Douglas Wetzel  
Code 12  
Navy Personnel R&D Center  
San Diego, CA 92152-6800

Dr. Barbara White  
Bolt Beranek & Newman, Inc.  
10 Houlton Street  
Cambridge, MA 02238

Dr. Barry Whitsel  
University of North Carolina  
Department of Physiology  
Medical School  
Chapel Hill, NC 27514

Dr. Christopher Wickens  
Department of Psychology  
University of Illinois  
Champaign, IL 61820

Dr. Heather Wild  
Naval Air Development  
Center  
Code 6021  
Warminster, PA 18974-5000

Dr. Robert A. Wisher  
U.S. Army Institute for the  
Behavioral and Social Sciences  
5001 Eisenhower Avenue  
Alexandria, VA 22333

Dr. Martin F. Wiskoff  
Navy Personnel R & D Center  
San Diego, CA 92152-6800

Mr. John H. Wolfe  
Navy Personnel R&D Center  
San Diego, CA 92152-6800

Dr. George Wong  
Biostatistics Laboratory  
Memorial Sloan-Kettering  
Cancer Center  
1275 York Avenue  
New York, NY 10021

Dr. Donald Woodward  
Office of Naval Research  
Code 1141NP  
800 North Quincy Street  
Arlington, VA 22217-5000

Dr. Wallace Wulfeck, III  
Navy Personnel R&D Center  
San Diego, CA 92152-6800

Dr. Joe Yasutake  
AFHRL/LRT  
Lowry AFB, CO 80230

Mr. Carl York  
System Development Foundation  
181 Lytton Avenue  
Suite 21D  
Palo Alto, CA 94301

Dr. Joseph L. Young  
Memory & Cognitive  
Processes  
National Science Foundation  
Washington, DC 20550

Distribution List (Washington University/Posner) 1987/11/10

AFOSR, Life Sciences Division  
University of Minnesota  
Department of Psychology  
Minneapolis, MN 55455

Dr. Jaime Carbonell  
Carnegie-Mellon University  
Department of Psychology  
Pittsburgh, PA 15213

Chief of Naval Education  
and Training  
Liaison Office  
Air Force Human Resource Lab.  
Operations Training Division  
Williams AFB, AZ 85224

Dr. John J. Collins  
Director, Field Research Office,  
Orland  
NPRDC Liaison Officer  
NTSC Orlando, FL 32813

Captain P. Michael Curran  
Office of Naval Research  
800 N. Quincy Street  
Code 125  
Arlington, VA 22217-5000

Dr. Joel Davis  
Office of Naval Research  
800 North Quincy Street  
Code 1141NP  
22217-5000

Defense Technical Information Ctr.  
Cameron Station, Bldg. 5  
Alexandria, VA 22314  
Attn: TC

ERIC Facility Acquisitions  
4833 Rugby Avenue  
Bethesda, MD 20014

Dr. Marshall J. Farr  
2520 North Vernon Street  
Arlington, VA 22207

J. D. Fletcher  
9931 Corsica Street  
Vienna, VA 22180

Dr. John R. Frederiksen  
Bolt Beranek & Newman  
50 Moulton Street  
Cambridge, MA 02138

Dr. David Navon  
Institute for Cognitive Science  
University of California  
La Jolla, CA 92093

Dr. Donald A. Norman  
Institute for Cognitive Science  
University of California  
La Jolla, CA 92093

Dr. Robert Sasmor  
Army Research Institute  
5001 Eisenhower Avenue  
Alexandria, VA 22333

Dr. Steven Zornetzer  
Office of Naval Research  
Code 1140  
800 N. Quincy St.  
Arlington, VA 22217-5000

Dr. Michael J. Zyda  
Naval Postgraduate School  
Code 52CK  
Monterey, CA 93943-5100