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Visual-spatial selective attention and reading ability in children:
A study using event-related potentials and behavioral measures

Anllo-Vento, María Lourdes, Ph.D.

The University of North Carolina at Greensboro, 1991

VISUAL-SPATIAL SELECTIVE ATTENTION AND READING ABILITY

IN CHILDREN: A STUDY USING EVENT-RELATED POTENTIALS
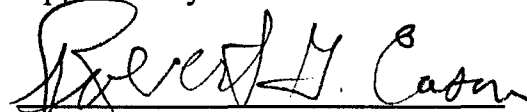
AND BEHAVIORAL MEASURES

by

Marla Lourdes Anllo-Vento

A Dissertation Submitted to
the Faculty of the Graduate School at
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of the Requirements for the Degree
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APPROVAL PAGE

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Reading-disabled subjects have shown a pattern of visual-perceptual processing which is consistent with a deficit in the pathway that encodes transient visual information. Given that this processing stream appears to mediate spatial information, it was hypothesized that children with poor reading skills would also be relatively deficient in attentional spatial-cueing tasks.

Here, the paradigm included two successive stimuli: a central cue and a peripheral target. The cue was either directional (a right or left arrow), or neutral (a circle). The target, a white square, appeared 600 ms later and was randomly flashed 8 degrees in the periphery of the right or left hemifield. Subjects were instructed to respond with their right index finger every time the target was validly cued by the preceding cue. Invalidly and neutrally cued trials did not require a response.

Eighteen children, 9.75 years-old on average, volunteered to participate in the study. The subjects were a subset of a sample of 83 children which were selected in kindergarten as being at risk of developing a reading disability. At the time of testing, all subjects were attending the 4th grade. The group in this study had average general intelligence and reading ability. Subjects were assigned to a high or a low reading level by means of a median split of their 3rd-grade Woodcock-Johnson Reading Cluster scores.

Scalp potentials evoked by the cue revealed differences between the brain's response to the right and left arrows, starting approximately 240 ms after cue onset. But it was not until about 320 ms after the cue that the responses of poor and good readers started to diverge. Differences in brain activity as a function of arrow direction were present in good, but not poor, readers. The differences

between the two reading groups increased as the target's onset neared. Once the target appeared, differences in brain activity between validly and invalidly cued stimuli also distinguished the poor from the good readers in that the good readers showed greater validity effects. In addition, behavioral responses were related to both prior brain activity and the subject's reading ability.

These findings are interpreted as supporting the idea that the voluntary directing of attention to a cued location results in enhanced activity in those areas of the brain that will process the ensuing stimulus. The outcome of the experiment also suggests that visual-spatial selective attention may be related to reading ability, and that both cognitive processes could be mediated, in part, by the same neurobiological system.

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This project is, in part, an inheritance: I set out to continue and extend the work of my advisor, the late Dr. M. Russell Harter. His work guided and inspired me, though often I wished he were there. I was most fortunate, however, to count on Dr. Robert Eason, who trusted me and provided me with the warmest of supports. He helped me make the transition from student to colleague, while remaining a friend.

I am particularly indebted to Dr. Walter Salinger, who always challenged my thinking, and taught me most of what I know about the brain and its development. All other members of my committee, Drs. Frank Wood, Reed Hunt, Frederick Morrison and John Lepri, also contributed to my intellectual development, and made me give the best I had.

I could not have reached this goal without the love and encouragement of my parents, who were willing to sacrifice our closeness so that I would grow intellectually. But, most of all, I am grateful to my best friend and companion, Ken Snowden, who was always there to remind me that the grass was green and luscious on the other side of the pain.

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CHAPTER I

INTRODUCTION

The survival of an organism often depends on its ability to select what is relevant from all information in its environment. Increased environmental complexity demands greater refinement and flexibility in selection. In humans, successful goal-oriented behavior implies selective attention: attention that distinguishes what is pertinent from what is not, and helps to establish a course of action.

Selective attention needs to be differentiated from two other related, yet separate, phenomena. Attention may trigger, and generally does, an overt movement or action. But orientation towards a source of information should not be equated to the observable response that usually follows. In fact, covert selection and shifts in orientation can be separated from overt movement in both humans (Posner, 1978, 1980) and animals (Fuster, 1990; Robinson & Petersen, 1986; Wurtz, Goldberg & Robinson, 1980). Selective attention also does not mean generalized, widespread enhancement in alertness or arousal. Increases in alertness involve overall changes in responsiveness without selective implications (Robinson & Petersen, 1986).

Selective attention, then, can be operationally defined as the capability of an organism to favor the processing of information that is behaviorally relevant. "It implies withdrawal from some things to deal effectively with others." (James, 1890).

This definition implies that organisms are unable to process all available information. Though evolution might have "selected" some design features to

capture pertinent aspects of the organism's environment, such as the range of the electromagnetic spectrum that can be perceived, such structural attributes do not constitute the focus of this investigation. The studies reported here presume the existence of a voluntary capability to selectively enhance or suppress processing of information that can be encoded and transmitted. The presumption of such selective capability rests on a vast body of literature, which demonstrates that stimulus processing can be modulated by attention, behaviorally as well as neurophysiologically (for reviews, see Hirst, 1984; Johnston & Dark, 1986; Robinson & Petersen, 1986).

In the 1960s, following a predominantly behavioristic period, attention reclaimed its place as a legitimate subject of psychological inquiry with the advent of cognitive psychology. At that time researchers embarked on a long-lasting debate concerning the way in which selection mechanisms operate. Some argued that information was filtered very early in the processing sequence, according to the physical features of the stimulus (Broadbent, 1958; Treisman, 1964). Others showed that semantic characteristics of the stimulus could be processed very early on, and countered that selection occurred only after all information had been fully processed (Deutsch & Deutsch, 1963; Norman, 1968). The conflict between "early" and "late" theories of selection was not resolved at the behavioral level, and the discussion moved on to other theoretical alternatives, such as Kahneman's (1973) view of attention as resource allocation.

During the last decade there has been renewed interest in selective attention. Recent behavioral evidence has established that part of the selection process is preattentive, unconscious, and based on fast, mostly parallel input (Julesz, 1984; Treisman, Cavanagh, Fischer, Ramachandran & von der Heydt,

1990). But, as features take on behavioral relevance or need to be integrated into objects, stimulus processing slows down, reflecting a shift from the parallel to the serial mode (Kahneman & Treisman, 1984; Koch & Ullman, 1985; Neisser, 1976). It appears, therefore, that focused or selective attention is a prerequisite for stimulus recognition, even when based on a single feature (Sagi & Julesz, 1985; Saarinen & Julesz, 1991; Treisman & Gormican, 1988). A more precise understanding of the brain mechanisms of sensation has revealed similar events at the physiological level. As subjects switch from passive gathering of stimulus information to active selection of relevant targets, a concomitant transition occurs in brain activity: from the posterior, sensory areas of the brain towards the frontal cingulate cortex (Posner, Petersen, Fox & Raichle, 1988). Thus, both human and animal behavioral and neurophysiological studies concur in emphasizing the inherently selective nature of attention (Harter & Aine, 1984; Robinson & Petersen, 1986; Wurtz et al., 1980).

This study examines the temporal and topographical patterns of brain activity that accompany the performance of a visual-spatial cueing task. One basic hypothesis is that changes in cortical potentials and behavioral responses are associated with shifts of attention in visual space. The other is that this relationship varies with the subject's reading ability. The remainder of this section reviews the literature on each of these topics in turn.

Neurophysiological correlates of selective spatial attention

The methodology used here constitutes a profitable approach to the study of selective attention. Because the temporal resolution of the event-related potential (ERP) technique is in the order of milliseconds, it affords a real-time chronometrical analysis of neural activity which would not be attained with

reaction times and other behavioral indices. In addition, ERPs recorded in conjunction with behavioral measures provide a first approximation to the physiological correlates of the behavior under study.

It has been shown repeatedly over the last twenty years that scalp potentials evoked by attended stimuli are larger in amplitude than those evoked by unattended stimuli (Eason, Harter & White, 1969; Harter, Aine & Schroeder, 1982; Näätänen, 1982; Rugg, Milner, Lines & Phalp, 1987; Van Voorhis & Hillyard, 1977). The phenomenon has been observed not only in the visual system, but also in other sensory modalities (for reviews, see Hillyard & Picton, 1987; Regan, 1989). Enhancement of the brain's response under conditions of focused attention has been demonstrated even when the relevant event is the omission of a stimulus, rather than its occurrence (Simson, Vaughan & Ritter, 1976). Such selective activation of scalp potentials parallels changes in the firing patterns of some cells in the visual cortex of the monkey (Galambos & Hillyard, 1981).

Harter and his colleagues, using ERP recordings, demonstrated that the point at which different stimulus features were selected depended on their behavioral relevance (reviewed in Harter & Aine, 1984). In these studies, brain potentials were recorded over the surface of the scalp in response to various features of visual stimuli. The averaged potential evoked by the unattended stimulus feature was subtracted from the brain's averaged response to the attended feature. The resulting negative deflection, termed *selection negativity*, varied in onset latency as a function of the type of feature, and indicated that stimulus characteristics were serially selected. Selection of the relevant spatial frequency preceded selection of the relevant orientation, but both were selected only after

the appropriate location had been determined. Finally, conjunctions of features, such as spatial frequency and orientation, required yet more processing time (Harter & Aine, 1984, 1986; Harter & Guido, 1980; Previc & Harter, 1982).

A series of simultaneous behavioral studies demonstrated that when attention was directed to a given spatial location, stimuli were processed more efficiently at that location than at any other (Posner, Snyder & Davidson, 1980). Attention was selectively summoned to a given point in space by means of an antecedent warning cue which indicated the most likely location of the subsequent target. The demonstration that cueing resulted in improved performance led to the analogy of attention as a spotlight or zoom-lens that the subject could shift voluntarily from one location to another (Posner et al., 1980; Eriksen & Yeh, 1985).

The notion of spatial location as a basic feature of visual processing is corroborated by neurophysiological studies that show that the image's spatial layout is preserved along the visual pathway, starting at the receptor surface (Kandel & Schwartz, 1985; Van Essen, 1985). In addition, recent evidence from neuroanatomical (Ungerleider & Mishkin, 1982), neurophysiological (Harter et al., 1982; Haxby et al., 1991; Maunsell & Newsome, 1987), and clinical neuropsychological (Levine, Warach & Farah, 1985; Lynch, 1980; Morrow & Ratcliff, 1988) research strongly suggests that a stream within the visual pathway specializes in the processing of the spatial characteristics of the stimulus. For instance, lesions of the posterior parietal cortex in humans result in a tendency to neglect information that originates in the contralateral hemifield (DeRenzi, 1982; Lynch, 1980). These patients also respond more slowly to targets in the visual field contralateral to the lesioned hemisphere when they are preceded by cues in the

ipsilateral hemifield (Posner, Walker, Friedrich & Rafal, 1984, Petersen & Robinson, 1989). It has been inferred from the available evidence that posterior parietal cortex participates in the processing of spatial location, attentional control, and preparation for action in the immediate environment (Mesulam, 1981, 1990; Posner et al., 1988; Rizzolatti, Gentilucci & Matelli, 1985).

The standard paradigm used in electrophysiological studies of visual-spatial attention requires the subject to focus or direct attention towards one visual hemifield while ignoring the other. Stimuli are then randomly presented in the same or the opposite hemifield as the subject maintains fixation and sustains attention to the relevant visual location. ERPs evoked by stimuli in the attended and ignored visual fields are recorded separately and later compared (Eason, 1981; Harter et al., 1982; Hillyard, Munte & Neville, 1985; Rugg et al., 1987). Differences obtained between the potentials evoked by the same stimulus under these two attention conditions are thought to reflect the activity of those brain mechanisms responsible for allocating attention in visual space.

A modified version of this task directs subjects to shift attention towards a cued location on a trial-by-trial basis. The instructive cue is a central arrow pointing randomly to one or the other hemifield. The arrow is followed by the imperative stimulus, a peripheral flash of light requiring a motor response only when presented in the cued visual field. As already mentioned, paradigms similar to this one have been used in reaction-time and detection studies, and have resulted in behavioral improvements at the cued location attributed to selective attention (Mangun & Hillyard, in press; Posner, 1978, 1980; Remington, 1980).

The averaged evoked wave form, that is the ERP recorded under these conditions, is characterized by a series of deflections whose amplitudes are

modulated by changes in attentional focus (Harter, 1991; Harter, Anllo-Vento & Wood, 1989a; Harter, Miller, Price, LaLonde & Keyes, 1989b; Hillyard et al., 1985; Mangun & Hillyard, 1987). The response of adults to the peripheral target is larger when the preceding arrow pointed in its direction rather than in the opposite direction (Harter & Anllo-Vento, in press; Mangun & Hillyard, 1987, 1990). A similar phenomenon has been reported in young children (Harter, 1991; Harter et al., 1989b).

A cueing paradigm similar to the one just described enables the recording of brain activity evoked by the cue and preceding the appearance of the target. It is during this critical interval that the subject should be covertly shifting attention towards the cued location and getting ready to respond. Brain potentials evoked by the cue during this interval reveal changes in amplitude across hemispheres as a function of cue direction (Harter et al., 1989b; Harter & Anllo-Vento, in press). Such differences presumably reflect the subject's selective directing of attention towards the cued hemifield in anticipation of the incoming target.

Cueing paradigms fall into the general category of paired-stimulus paradigms. The cue or warning signal (S1) is the first stimulus, while the target constitutes the second stimulus of the pair (S2). The characteristic potential obtained in this task in response to the warning signal consists of a sensory visual evoked potential (VEP) to the cue stimulus, followed in turn by a broad positivity and a slow negative deflection. The slow cortical positivity peaks about 300-400 ms after the cue, and probably belongs to the family of *late positive deflections* (LPD) also referred to as P3 (Verleger, 1988). A slow negative potential, termed the *contingent negative variation* (CNV), is seen increasing in amplitude as the time for the contingent motor response approaches (Cohen, 1969). While this negative

potential tends to maintain a symmetrical hemispherical distribution (Deecke, Kornhuber, Lang, Lang & Schreiber, 1985), it also has been shown to respond to the type of material involved in the task (Uhl, Lang, Lang, Kornhuber & Deecke, 1988). Similarly, unlike the symmetrical cortical potential evoked by a non-directional warning stimulus, a directional arrow cue results in an asymmetrical brain response (Harter et al., 1989b). Even before the CNV commences, the slow positivity preceding it already indicates that each hemisphere responds selectively to the direction of the cue (Harter & Anllo-Vento, in press; Harter et al., 1989b). The onset latency of this asymmetrical response indicates that it is not the simple consequence of the marginal physical differences between a right- and a left-pointing arrow, which, if real, would appear earlier as part of the sensory VEP.

The first question in this study concerns the brain's response to cue directionality. To date, no electrophysiological visual-spatial study has compared directional and non-directional cues. The prediction is that brain activity will vary with the type of cue. Differences between directional and non-directional cues could be attributed to attentional or response requirements, or both. The neutral, non-directional cue entails neither an attentional shift nor a motor response, while directional arrows share response requirements but differ in spatial allocation. Timing of the point at which brain activity signals the differential processing of cue directionality will reveal whether attentional requirements are present concurrently with response requirements, thus suggesting that both types of processing are engaged concomitantly. If the directional cue elicits no attentional shift, and acts only as a warning signal, no differences would be found between the potentials evoked by directional and non-directional cues. The second question is whether the direction of the cue is selectively processed as well. If so, the brain

potentials evoked by a right and a left arrow should not be the same. Even if a directional cue served both as an alerting signal and a spatially selective cue, each separate contribution could be assessed by comparing directional and non-directional cues first, then testing for differences in brain activity between right and left arrows.

Previous ERP studies of visual-spatial cueing have focused solely on the effect of prior cueing on the brain's response to the target (Mangun & Hillyard, 1987, 1988, 1990). This study also will investigate the impact of cueing on the brain's processing of the target. Though less innovative than the processing of the cue, this aspect deserves examination, if for no other reason than it will place the present experiment in the context of previous empirical work. In addition, it will help establish that subjects selectively direct attention to the cued hemifield.

It is hypothesized that post-target effects in this experiment will replicate previous findings by Harter and his collaborators (Harter & Anllo-Vento, in press; Harter et al., 1989a, 1989b). Validly cued targets should evoke larger ERP amplitudes than would invalidly cued targets. Additionally, the invalid condition will be compared to the valid and neutral conditions separately. Both the valid and invalid conditions entail a discrimination of the target location; subjects cannot determine whether a response is appropriate until they process the location of the target. Once the discrimination is made, however, the brain's response in the invalid and neutral conditions should be similar, since in both cases a response is not required.

Some remarks are in order before the end of this section. They concern three design features of this study that differentiate it from previous ones. First, the probability of the target being validly or invalidly cued is the same. Most

behavioral and electrophysiological cueing studies have manipulated target probability, such that valid cueing is considerably more likely than invalid cueing (Anllo-Vento & Harter, 1988; Downing & Pinker, 1985; Mangun & Hillyard, 1987, 1990; Posner, 1980). It could be argued, therefore, that validity effects are due to stimulus control rather than to voluntary shifts of attention. In fact, motor potentials following the CNV vary as a function of whether the movement is triggered by a stimulus or is self-initiated (Kurtzberg & Vaughan, 1982). It is unlikely, however, that the magnitude of a target-evoked brain response is a function of probability alone. Though reduced in amplitude, ERP enhancements are observed as a function of cue validity, even when target probability is not manipulated (Harter & Anllo-Vento, in press; Harter et al., 1989a, 1989b). In this study, the target appeared equally often in the valid and invalid hemifields, so that the directional cue had no informational value.

A second consideration relates to the task demands of most behavioral studies of selective attention. In order to observe reaction-time differences between validly and invalidly cued stimuli, responses have to be recorded in both situations. Instructing the subject to respond to all stimuli is tantamount to encouraging the deployment of attention to the supposedly unattended location. To avoid this bias, subjects were instructed to respond only when the target stimulus appeared in the cued visual field.

Finally, it should be noted that this study placed no memory demands on the subject, since the cue stayed on throughout the trial. The relevant hemifield was signalled at all times in each trial, so there was no need for the subject to retain cue type or direction until the target's appearance.

Selective spatial attention and reading ability

This study predicts that the brain activity evoked by a spatial cueing task will vary as a function of reading ability. It is proposed that some components of the brain network involved in visual-spatial covert orienting are involved in reading as well. This section summarizes experimental evidence in support of this notion.

The presumed neurobiological basis of learning disabilities has received much attention in recent years (for reviews, see Geschwind & Galaburda, 1985; Ollo & Squires, 1986; Hynd & Semrud-Clikeman, 1989). This area of research has concentrated on understanding the etiology, diversity and educational implications of these disorders, but the behavioral and neurobiological alterations observed in learning disabilities could also be used as an experimental tool (Picton & Stuss, 1984). Contrasting the behavioral impact of various language pathologies could serve as a means of extending our present knowledge of language neurobiology. Moreover, if some learning disabilities are due to anomalous neurobiological development, as suggested by Geschwind and Galaburda (1985), their investigation could provide data on the behavioral correlates of abnormal brain organization that might result from such developmental alterations (e.g., Harter et al., 1989a; Neville & Lawson, 1987).

Two types of studies have attempted to relate abnormal brain morphology and learning disabilities: postmortem anatomical examination of brains of patients with a known history of the disorder (Drake, 1968; Galaburda & Kemper, 1979; Galaburda & Eidelberg, 1982; Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985), and studies which employ brain imaging techniques such as computer tomography (CT) and magnetic resonance imaging (MRI) (Haslam,

Dalby, Johns & Rademaker, 1981; Rumsey, Dorwart, Vermess, Denkla, Kruesi & Rapoport, 1986; Duara et al., 1991). Unfortunately, methodological flaws and inconsistencies have led to inconclusive findings from these two areas of research (Hynd & Semrud-Clikeman, 1989).

Neuropathological studies are scarce; only six cases are found in the literature (Drake, 1968; Galaburda & Kemper, 1979; Galaburda et al., 1985). Let us consider three general problems before we summarize their findings. First, postmortem cases usually are characterized by limited psychometric evaluations of reading achievement or language delay. Secondly, case studies often do not provide concurrent data on random normal brains that can be used as controls. Thirdly, most evaluations are confined to macroscopic neuroanatomical features that might be secondary to the hypothesized neurodevelopmental abnormalities. Nevertheless, there are indications that the brains of reading-disabled (RD) individuals, more often than those of normals, tend to be symmetrical in the area of the planum temporale, which is traditionally assigned to Wernicke's area and related to language processing. In addition, focal neuroanatomical abnormalities (i.e., dysplasias and ectopias) are present in RD brains, and are significantly concentrated in left frontal, left temporal and right frontal cortices.

These findings led Geschwind and Galaburda (1985) to postulate that abnormal neural development of the perisylvian areas of both hemispheres was a determining factor in RD. They proposed a neuropathological model including a constellation of factors such as left handedness, hormonal and immune disorders. Studies have substantiated a greater incidence of left-handedness in the RD population, but evidence tying the co-occurrence of left-handedness and RD to other factors included in their model is still insufficient (Hynd &

Semrud-Clikeman, 1989; Hynd, Semrud-Clikeman, Lorys, Novey & Eliopulos, 1990).

Prompted by claims for a neurobiological basis of reading disability, investigators have recently applied new imaging techniques to the study of the morphological characteristics that differentiate normal from RD brains. In their critical review of the evidence from CT and MRI studies, Hynd and Semrud-Clikeman (1989) analyzed 8 reports containing data on 224 RD and 581 normal subjects. These studies often failed to provide consistent diagnostic criteria and adequate assessment, and lacked control of other significant variables such as handedness and coexisting neurological or psychiatric disorders. Although the reported findings were somewhat inconsistent, there was some evidence of a relationship between RD and a departure from the normal pattern of asymmetry of the perisylvian and parieto-occipital brain regions.

More recently, Hynd et al. (1990) have investigated the anatomical differences found between normal, RD, and hyperactive children as assessed from MRI scans. While both RD and hyperactive children showed a narrower right anterior region than normals, only the RD children displayed a higher incidence of planum temporale symmetry or reversed asymmetry. Left-handedness also was more frequent among RD subjects. These results were interpreted as supporting the existence of aberrant patterns of lateralization in severe RD subjects, especially involving Wernicke's area, and possibly due to alterations in corticogenesis. But Duara et al. (1991) reported contrary evidence on the neuroanatomical MRI differences between RD and normal adults. RD subjects were characterized by reversed symmetry in a midposterior brain region corresponding to the angular gyrus, accompanied by a larger splenium of the

corpus callosum. Their neuropsychological index of RD severity was positively correlated with the area of the right posterior pole, thus corroborating the functional significance of this anatomical finding. No group differences were found, however, in the brain region encompassing the planum temporale. Inconsistencies between these two reports (Duara et al., 1991; Hynd et al., 1990) could be due to developmental differences (adults vs. children), criteria used in subject selection (admitting or not a co-diagnosis of hyperactivity), or measurement technique (areal vs. linear measurements, respectively). Both studies are sound, and each replicates prior findings; their incompatibilities reflect the difficulties inherent in characterizing a complex behavioral deficit in neuroanatomical terms alone.

The etiology of RD may involve aberrant brain morphology, but its probable developmental nature and the microscopic alterations (e.g., abnormal neural connectivity) that it is likely to involve suggest the need for analyses beyond those conducted to date. It seems that certain non-invasive functional measures may be better suited to discover the malfunctioning processes underlying RD. Unfortunately, some functional brain-imaging techniques, such as positron-emission tomography (PET) cannot be applied to the study of developmental RD since they would expose very young subjects to radiation. Magnetoencephalography, on the other hand, offers great spatial and temporal resolution, but has not resolved difficulties in its application to relatively complex behavioral paradigms. Event-related potentials do not involve radiation risks nor logistical obstacles and, consequently, seem well-suited to investigate differences in brain function between normal children and children with learning disabilities. This research area, however, has suffered from flaws resulting in contradictory or

ambiguous findings. As in the case of neuropathological investigations, methodological inconsistency has been the norm rather than the exception. In addition, many studies have used passive stimulation, but made inferences about cognitive processes whose engagement depends on active, demanding tasks. Subject classification and assessment have been inconsistent (Ollo & Squires, 1986), and some studies have included subjects suffering from both RD and attention deficit, two disorders that appear to involve independent behavioral and electrophysiological correlates (see below; also Felton, Wood, Brown, Campbell & Harter, 1987; Harter, Diering & Wood, 1988a; Harter, Anllo-Vento, Wood & Schroeder, 1988b; Holcomb, Ackerman & Dykman, 1985), despite their frequent co-occurrence. Finally, until recently the lack of developmental norms for ERP data constituted a particularly detrimental problem in the study of a presumedly developmental disorder.

Nonetheless, some consistent differences in ERP patterns between normal and RD subjects have emerged in recent years (for reviews, see Harter, 1991; Ollo & Squires, 1986; Otto et al., 1984; Picton & Stuss, 1984). Probably the most consistent result has been the presence of differences in hemispheric activation between disabled and normal readers. The amplitude of various late ERP measures in RD subjects is symmetrically distributed or shows a pattern of lateralization contrary to that of normal readers (Conners, Blouin, Winglee, Lougee, O'Donnell & Smith, 1984; Harter et al., 1988b, 1989a; Johnstone, Galin, Fein, Yingling, Herron & Marcus, 1984; Preston, Guthrie, Kirsch, Gertman & Childs, 1977; Shucard, Cummins, Gay, Lairsmith & Welanko, 1984; Symann-Louett et al., 1977). Group differences in hemispheric activation, when found, have been often localized about the parietal region, particularly over the left

hemisphere (Conners et al., 1984; Harter et al., 1988b, 1989a; Preston et al., 1977; Symann-Louett et al., 1977). In addition, evidence for distinct brain activity in normal and reading-impaired subjects has arisen most often from tasks requiring the engagement of cognitive processes (Harter et al., 1988a, 1988b, 1989a, 1989b; Johnstone et al., 1984; Shucard et al., 1984), although group differences are also found in strictly sensory tasks (Conners, 1970; Livingstone, Rosen, Drislane & Galaburda, in press; May, Lovegrove, Martin & Nelson, 1991). The available results, taken together, may represent a functional correlate of some of the anatomical findings cited above.

A few reports have made more explicit contributions to the general trends summarized above. The amplitude of various late deflections of the ERP wave form, particularly the P3 or LPD seems to be reduced in RD subjects (Dainer, Klorman, Salzman, Hess, Davidson & Michael, 1981; Harter et al., 1988a, 1988b, 1989a; Holcomb et al., 1985; Loiselle, Stamm, Maitinsky & Whipple, 1980). The significance of this finding is unclear, given the presence of a similar pattern in a variety of psychopathologies (Otto et al., 1984; Regan, 1989). It might reflect, in part, the propensity of ERP studies to quantify this robust positive potential almost to the exclusion of all others.

Some investigators, however, have analyzed slow potentials other than P3, and also revealed differences between normal and disabled readers. Cohen (1980) detected a symmetrical decrease of CNV amplitude in RD subjects, in spite of their normal reaction-times. He concluded that such a finding could not be explained in terms of an attentional deficit, since there were no differences in performance between poor and normal readers. Jones and Michie (1986) used a paired-stimulus paradigm, similar to the one in this study, with a constant inter-

stimulus interval of one second. Again, normal and RD subjects did not differ behaviorally when matching word-like stimulus patterns, but their brain activity varied in two significant ways. First, RDs were characterized by right occipital preponderance in the amplitude of an early transient negativity (N230), while normal controls displayed symmetrical activation. Second, CNV morphology differed between the two groups: it reached its asymptote approximately 200 ms before the second stimulus in RD subjects, while it continued to increase up to 100 ms after target presentation in the control group.

Dainer et al. (1981) recorded ERPs from normal and learning-disabled (LD) subjects as they were engaged in the continuous performance task (CPT). In a variation of this task, the subjects were instructed to respond to the target letter only when preceded by an 'E'. Then, a preparatory CNV developed after the warning stimulus (i.e., the letter 'E') and before any subsequent letter. The amplitude of the CNV changed as a function of age, but did not differ between LD children and normal controls. This finding should be evaluated with caution, however, since diagnosis was mis-specified in their statistical model as a within-subject variable.

Harter and his collaborators have recently described several differences between normal children and children with RD or poor readers (Harter, 1991; Harter et al., 1988a, 1988b, 1989a). They reported a reduction of the task-relevant modulation of P3 over the central region of the left hemisphere of RD boys, but not normals (Harter et al., 1988b, 1989a). As a consequence, brain activity was symmetrical in RD subjects but not in normal readers. Reduced lateralization of P3 amplitude in disabled readers was present not only in verbal tasks such as letter

discrimination (Harter et al., 1988b), but also in visual-spatial tasks (Harter et al., 1989a).

Harter et al. (1988a, 1989a) found earlier differences in brain activity between RDs and controls, in addition to the pattern of decreased P3 asymmetry in RD boys. A positive deflection peaking at approximately 240 ms post-stimulus was reduced in amplitude over the left central hemisphere of the RD children, when discriminating between letters and non-letter patterns (Harter et al., 1988a). The same trend was observed in adults that had been diagnosed as reading impaired when they were children (Naylor, Wood & Flowers, 1990). In contrast, the task-relevant enhancement of the N1 component in a visual-spatial task was greater for children with RD than normals (Harter et al., 1989a). The finding of preserved ability to direct attention in visual space, as reflected by the task-relevant N1 increase in RD boys, corroborated the previously hypothesized lack of an attentional deficit in RD. Indeed, Holcomb et al. (1985) demonstrated that RD and attentional deficit disorder (ADD) were associated with different ERP patterns. ADD children showed less attentional enhancement of brain potentials than either normals or RD children, while the reading impaired had diminished P3 amplitudes over the parieto-occipital midline that did not change with attentional manipulations. Unfortunately, these investigators did not record ERPs at lateral electrode sites, thus preventing the comparison of RD, ADD and normal subjects in their patterns of lateralization.

A longitudinal study of brain potentials in visual-spatial tasks recently reported some interesting, albeit preliminary, trends in normal and RD boys (Anllo-Vento, Miller & Harter, 1990). Two similar cueing paradigms were used across the five-year period separating the two testing sessions. The most

conspicuous finding was the remarkably diminished amplitude of the CNV preceding the target in impaired readers. In contrast, RDs and controls showed comparable enhancement of the potentials evoked by a peripherally presented, validly cued target. This latter result supported the claim of no deficits in selective attention in impaired readers, but failed to replicate the greater modulation of visual-spatial attention which had been previously reported in the RD subjects (Harter et al., 1989a). Finally, RD boys were characterized by a shallower posterior-anterior gradient of brain activation than normal controls, suggesting less cortical localization in poor than in good readers.

Harter (1991) reviewed the differences in brain activity observed between poor and good readers, and proposed a functional interpretation of various components of the ERP wave form. He hypothesized that P240 and P3 reflected the selection of features of central stimuli, designated *type-selection*. In contrast, he associated P1 and N1 with the allocation of attention in visual space, termed *location-selection*. He argued that a reading impairment entailed deficient type-selection with a concurrent absence or even compensatory development of location-selection. As articulated, his hypothesis implied the presence of a selective impairment in the visual pathway carrying information about the form or shape of objects, and opposed the notion of reading disability as an exclusively verbal deficit.

With his theoretical proposition, Harter (1991) incorporated into the study of learning disabilities a neuroanatomical and neurophysiological distinction (Ungerleider & Mishkin, 1982) that had marshalled increasing experimental support in recent years (Corbetta, Miezin, Dobmeyer, Shulman & Petersen, 1990; Haxby et al., 1991). In this model, visual information is processed by two relatively

segregated neural systems: the *where* pathway, which has its target in posterior parietal cortex and specializes in information about motion and the location of objects in visual space; and the *what* pathway, which courses through the inferior-temporal cortical region and appears to mediate information about fine detail and the shape of objects. At the microscopic level, this distinction is approximated by functional differences between various cell classes present in the visual system. The two most salient types, Y and X cells, constitute parallel channels in charge of processing time and motion information, on the one hand, and color and fine detail, on the other (Lennie, 1980; Maunsell & Newsome, 1987). The relative ratio of these cell types varies as a function of retinal location, X cells being more numerous in the foveal and parafoveal regions while Y cells are relatively more abundant towards the periphery of the retina. Neuroanatomical tracings in rhesus monkeys suggest a remarkable degree of segregation between these two streams (Baizer, Ungerleider & Desimone, 1991). Cells projecting to parietal cortex tend to originate from the far peripheral representation of prestriate visual cortex, while neurons ending in temporal regions arise from foveal or central representations.

The relative segregation of the *what* and *where* pathways and the functional characteristics of cells that contribute to each one of them, have led some researchers to infer that reading impairment constitutes a failure of the *what* system to develop appropriately, and a concurrent sparing of the *where* system (Geiger & Lettvin, 1987; Conners, 1990; Harter, 1991). Various lines of evidence endorse this idea: the fact that reading involves resolution of the fine detail or high spatial frequencies present in fine print; the common finding of cortical abnormalities in the temporal lobe of dyslexic brains; the facilitation of peripheral visual perception in impaired readers (Geiger & Lettvin, 1987); the left-

hemisphere impairment often observed in reading disability, and the left hemisphere specialization in analytical, rather than global, sensory processing (Lamb, Robertson & Knight, 1989; Jonsson & Hellige, 1986; Posner & Petersen, 1990).

The alternative point of view claims that reading disability is characterized by abnormal functioning of the *where* stream. This stance has gained recent experimental support from psychophysical (DiLollo, Hanson & McIntyre, 1983; Lovegrove, Bowling, Badcock & Blackwood, 1980; Williams, LeCluyse & Bologna, 1990), electrophysiological (Livingstone et al., in press; May et al., 1991) and neuropathological studies (Livingstone et al., in press). Livingstone et al. (in press), for instance, analyzed cell-body size in the lateral geniculate nucleus (LGN) of dyslexic and normal brains. The human LGN, like the monkey's, is subdivided into a series of layers. The dorsal, or parvocellular, layers contain small X-like neurons, while the ventral, or magnocellular layers, are innervated by large Y-like cells. Magnocellular and parvocellular neurons are segregated, and remain so even beyond primary cortex and possibly up to higher cortical association areas (Baizer et al., 1991; Hubel & Livingstone, 1987; Maunsell, Nealey & DePriest, 1990). Livingstone et al. (in press) reasoned that if the magnocellular or *where* pathway were damaged in RD, as suggested by psychophysical evidence, it might be possible to find a neuroanatomical correlate in the segregated layers of the LGN. Indeed, the size of the neurons in the magnocellular layers of the LGN was about 27% smaller in dyslexic than in normal brains.

There is a functional correlate of this neuroanatomical finding (May et al., 1991; Livingstone et al., in press). Logically, a selective magnocellular deficit should translate into greater difficulty in the processing of fast, transient

information by RD subjects. One way to test this hypothesis is to record VEPs in response to stimuli that vary along such physical dimension, and presumably engage the magnocellular pathway. May et al. (1991) conducted one such study, and reported lower VEP amplitudes and significantly shorter response durations to low spatial frequencies in poor readers. Likewise, Livingstone et al. (in press) found RD subjects to have diminished VEPs for rapid, low-contrast stimuli, but normal responses to slow or high-contrast stimuli. Taken together, these two studies support the idea of abnormal visual processing in the magnocellular pathway of the reading impaired. Since similar fast and slow parallel streams are found in other systems (e.g., somatosensory, see Dykes, 1983), it is conceivable that RD subjects have a pervasive malfunctioning of their fast sensory subdivisions.

There are also a number of psychophysical and behavioral studies that might be explained by defective magnocellular functioning in RD subjects. To date, impaired readers have shown diminished contrast sensitivity at stimulus durations of more than 150 ms (Lovegrove et al., 1980), visual persistence about 100 ms longer than normals (DiLollo et al., 1983; Lovegrove et al., 1986), slower flicker fusion at low spatial frequencies (Martin & Lovegrove, 1987), poor temporal-order or sequencing judgments (May, Williams & Dunlap, 1988); slower target detection with dynamic, but not static, visual displays (Casco, Dellantonio & Lupi, 1990); greater spatial location discrepancies than normals (Solman & May, 1990), and more prolonged foveal masking in conjunction with enhancement effects in the periphery (Williams, LeCluyse & Bologna, 1990).

Yet one more source of evidence favoring an impairment of the magnocellular or *where* pathway comes from research showing abnormal patterns of eye movements during reading in RD children (Pavlidis, 1986; Rayner, 1986).

There is a close anatomical and neurophysiological connection between the oculomotor system and the visual pathway that processes spatial and motion information (Newsome & Wurtz, 1988). It could be argued, therefore, that any oculomotor abnormality observed in RD children is more likely related to magnocellular than parvocellular malfunctioning.

The two positions outlined above, of parvocellular versus magnocellular deficits, rest on the assumption of complete segregation between the magnocellular and parvocellular streams. But there is additional evidence that these two pathways interact, in spite of their relative structural and functional independence (Breitmeyer, 1980; Breitmeyer & Valberg, 1979). It is possible that their mutual relationships constitute the problem in RD. For instance, Breitmeyer (1980), and Breitmeyer and Ganz (1976) have suggested that in normal reading transient activity elicited by eye movement, that is activity involving the magnocellular pathway, serves to interrupt the persistence of the visual image mediated by the sustained (i.e., parvocellular) channels. This process facilitates the perception of high spatial frequencies, or detailed information, which constitute a vital element of reading.

The suggestion that RD involves the impairment of a visual processing-stream, whichever it might be, implicitly acknowledges a major contribution of sensory factors in RD etiology. A sensory or perceptual deficit has been contested by cognitive researchers (Vellutino & Scanlon, 1982) on the basis of the verbal nature of most differences found between normal and RD children. It should be noted, however, that much of the experimental evidence on the non-linguistic nature of RD has utilized verbal stimuli, or stimuli that are easy to label, possibly biasing the type of cognitive process or strategy used by the subject. When

differences in visual processing between normal and RD readers are tested with various stimulus types, no sensory differences are found due to the type of material (Morrison, Giordani & Nagy, 1977). On the other hand, most cognitive studies have not varied the pertinent parameters of visual stimuli (e.g., spatial and temporal frequency) so as to uncover differences in visual processing. In fact, the few studies that have, report significant differences between normal and disabled readers (e.g., DiLollo et al., 1983). Put another way, it is important to keep in mind that failure to find differences between groups cannot be equated with demonstrating that the groups in question are comparable along the relevant dimension.

From a neural perspective, the sensory- versus verbal-deficit dichotomy might be moot. Distributed brain systems involved in cognitive skills such as reading almost certainly include processing streams that carry sensory information (for a cogent characterization of cognitive brain networks, see Mesulam, 1990). Moreover, organizational principles of the brain cross the boundaries of sensory systems, and one such principle might well be the existence of functional streams that are relatively segregated and act in parallel. In that sense, it is conceivable that a fast stream akin to the magnocellular division of the visual system would carry information about fast formant-transitions in the auditory modality. Thus, a deficit in the fast processing-stream could be a contributing factor to the phonological deficits frequently observed in reading disability (Felton & Brown, 1991; but see Hunt & Badawi, 1985). This is but an example of how sensory perturbations might be connected to strictly verbal phenomena: the operation performed by a given sensory mechanism might be a vital ingredient in a cognitive task, and contribute only marginally to another.

The present study was designed to test the relationship between brain-potential correlates of visual-spatial selective attention, and reading ability. Several considerations motivated the choice of the paradigm used in the experiment. First, new evidence had been accumulated suggesting that a sensory deficit might be a contributing factor to the verbal impairments present in reading disability. One subdivision of the visual pathway could be particularly involved, but there was some disagreement as to which stream it might be (cf. Harter, 1991; May et al., 1991). Previous findings also had indicated that reading-impaired subjects were normal or superior in processing peripheral stimuli (Anllo-Vento et al., 1990; Harter, 1991; Harter et al., 1989a; Harter et al., 1989b), thus partially supporting the absence of magnocellular damage. Yet, the extent to which sensory factors contribute to reading disability remains a highly controversial issue. Thus, a more precise characterization and replication of the results reported to date was needed to further evaluate the contribution of putative sensory factors to reading ability, and the manner in which these factors exert their influence.

Secondly, it was unclear what, if any, was the relationship between selective attention and reading ability. Most available studies of reading had not controlled for the incidence of ADD and, the few that did, provided conflicting results. For instance, while Holcomb et al. (1985) did not find differences in attentional modulation between normal and RD subjects, Harter and his group (Harter, 1991; Harter et al., 1988b; Harter et al., 1989) consistently have reported the opposite effects. In addition, the selection of a relevant spatial location seemed more likely to be associated with magnocellular than with parvocellular function. A primary purpose of this study, then, was to probe the hypothetical connection among reading, visual-spatial attention and the magnocellular system.

A hypothesis that was tested alleged that reading ability would be related to the brain activity evoked in a visual-spatial cueing task such as the one employed here. It was further hypothesized that differences between poor and good readers would be of two types: variations across groups over **time** would reflect the relationship of reading ability to the various operations included in the task, while differences in scalp **topography** would reveal distinctions in inter-hemispheric and intra-hemispheric activation between poor and good readers.

It was anticipated that the greatest differences in brain activity between poor and good readers would be found in response to the cue. This expectation was based on the assumption that cueing prompts a selective shift of attention towards the cued hemifield that should be associated with relatively greater magnocellular activation. Thus, poor readers should be characterized by a reduced amplitude in those potentials assumed to reflect the directing of attention in visual space. In accordance with previous results (Anllo-Vento et al., 1990), poor readers should show smaller CNV amplitudes than good readers, but the two groups also might differ in the magnitude of cue-related potentials appearing earlier in time (cf. Harter, 1991).

It was hypothesized that the effect of prior cueing on the response to the subsequent target also would be related to reading ability. Differences between poor and good readers, however, should be less pronounced in the processing of the target than in the previous processing of the cue. Target validity is assumed to reflect the enhancing effect of prior selective attentional-allocation on the processing of targets subsequently appearing at the cued location. Since the assumption maintained here claims that poor readers are less able to direct their attention selectively towards the cued location, they should not show, or show to a

lesser extent, the contingent enhancement in brain activity evoked by the relevant targets.

It has been shown previously, however, that the amplitude of the post-target N1 is comparable, or even larger, in RD or less-able readers than in normal controls (Anllo-Vento et al., 1990; Harter, 1991; Harter et al., 1989b). This evidence has been interpreted as reflecting a relatively heightened ability to process peripheral information on the part of poor or disabled readers (Harter, 1991; Harter et al., 1989b; Geiger & Lettvin, 1987). But the position taken here stipulates that target processing depends on prior attentional allocation, even when the target is peripherally presented. Consequently, the hypothesis, as it pertained to N1, was two-sided: this study tested whether reading ability was, in fact, related to enlarged or reduced N1 potentials.

Two other hypotheses that were evaluated here related to the topographical distribution of the differences in brain potentials found between reading levels. First, the largest disparities across reading levels were expected to be found over the parietal regions, since electrophysiological differences between normal and disabled readers, when found, have been largest there (Conners, 1970; Dainer et al., 1981; Holcomb et al., 1985; Preston et al., 1977; Symann-Louett et al., 1977). In particular, group differences in the amplitude of the LPD, or slow P3-like positivity, should be diminished in the poor reading group, particularly over the left hemisphere as in previous studies (Harter, 1991; Harter et al., 1988a, 1988b; Preston et al., 1977; Symann-Louett et al., 1977). Although this location is only partly consistent with other neuroanatomical and neurophysiological techniques, it is reliable in ERP recordings. Given the coarser spatial resolution of the ERP technique with respect to other brain-imaging methodologies, it would be unwise

to make deductions about the neural generator of these potentials based on their topographical distribution.

Secondly, poor readers should show a more widespread distribution of brain activity than good readers. This hypothesis is based on the results of several functional studies (Anllo-Vento et al., 1990; Rumsey, Berman, Denckla, Hamberger, Kruesi & Weinberger, 1987; Wood, Flowers, Buchsbaum & Tallal, in press), which have reported a broader distribution of cerebral activation, or a shallower anterior-posterior gradient of intra-hemispheric brain activity in disabled readers. These findings are interpreted as reflecting less localization of function, or a need for greater neural recruitment in disabled readers.

CHAPTER II

METHODS

Subjects

The study described below concentrates on the development of spatial selection in a subset of children from a group of 83 kindergartners believed to be at risk of developing a reading disability. In this section, the way in which the initial subject selection was conducted, and the make-up of the original group will be described. Then, the neuropsychological profiles of the group as a whole, and that of the subgroup participating in this study, will be compared.

At the time of their first assessment these children were in kindergarten and had not yet learned to read. They were screened from among all kindergartners in eight schools of the Winston-Salem city-school system. Selection procedures were based on teachers' evaluations of the subjects' ability to acquire reading skills, as well as on their performance in a series of neuropsychological tests that reportedly predict subsequent reading outcome (see Felton and Brown, 1989 for a complete explanation of the selection procedure). Those subjects whose IQs were below 80 (on the Otis-Lennon Mental Abilities Test) or who were rated as above-average to superior in their potential for reading success were not included.

The study reported here was conducted when these children were in the fourth grade. They already had been tested once each year, starting at the beginning of their first-grade year. Twenty-three subjects from this group agreed to participate in an additional experiment at the end of their standard yearly session. Five of these 23 subjects were not included in the final analysis, due to the presence of

systematic eye movements in their records: the oculomotor potentials that accompany a saccadic eye-movement distort significantly the recording of brain potentials. Thus, records showing eye-movement contamination had to be excluded.

The eighteen subjects whose data were evaluated in this experiment were 9 years and 9 months of age on average (range=9-2 to 10-7); their average IQ, as measured by the Weschler Intelligence Scale for Children-Revised (WISC-R), was 93.5 (range=77-109), while their average standard score by age on the Woodcock-Johnson Reading Cluster was 93.3 (range=64-114). Fourteen subjects were males and four were females. Table 1 compares the composition and test scores of the original group of at-risk kindergartners and the subgroup included in this study.

All subjects participating in this study had normal or corrected-to-normal visual acuity. At the beginning of the session, a rough estimate of each subject's acuity was obtained by means of a Snellen chart. In order to determine hand dominance, each child was asked to tell the experimenter the hand he/she used to draw or write, but handedness was not assessed systematically. Only one male subject reported being left-handed.

Stimuli and Task

As indicated above, the task reported here was administered at the end of the standard yearly session, on a volunteer basis. The larger longitudinal study explored the neurophysiological bases of reading disability and included five different tasks. All tasks were presented in a "video game" format so as to maintain the motivation of the subjects throughout the testing session. One of the five regular "games" included in the longitudinal project was very similar to the one presented here. All subjects, therefore, had prior experience with this task.

Table 1

Individual and Neuropsychological Characteristics of At-Risk Subjects in the Initial Group and Subjects in Study

	Original Group	Group in Study
N	83	18
Age (First grade)		
$\underline{M}$	6.55	6.59
$\underline{SD}$	(.48)	(.39)
Otis-Lennon IQ (Kindergarten)		
$\underline{M}$	99.58	96.67
$\underline{SD}$	(10.2)	(9.94)
PPVT-R (First grade)		
$\underline{M}$	90.62	87.41
$\underline{SD}$	(12.9)	(12.1)
Woodcock-Johnson Reading Cluster[a] (First grade)		
$\underline{M}$	93.1	93.7
$\underline{SD}$	(15.2)	(13.8)
WISC-R, Full Scale (Third grade)		
$\underline{M}$	96.15	93.5
$\underline{SD}$	(11.7)	(9.33)
Woodcock-Johnson Reading Cluster[a] (Third grade)		
$\underline{M}$	92.46	93.28
$\underline{SD}$	(12.9)	(14.3)

Note: PPVT-R = Peabody Picture Vocabulary Test-Revised
WISC-R = Weschler Intelligence Scale for Children Revised
[a]Standard Score by Age

For purposes of clarity, a diagrammatic representation of the task is included in Figure 1. A trial consisted of a sequence of two stimuli: a cue and a subsequent square flash defined as the target. The cue either conveyed directional information (a horizontal arrow pointing to right or left) or was neutral (a small circle). Both cues and targets were white, subtended approximately 42' of visual angle, and were flashed onto the black background of a video screen at a viewing distance of 56 cm. Stimuli were presented on a Color Graphics monitor and controlled by a 386 Compaq computer. The directional arrow-cue was presented at the fixation point in the center of the screen and randomly pointed to right or left (p=.5). Its direction was intended to make the subject orient or shift attention toward the cued hemifield, while maintaining fixation. One third of the time the cue was non-directional or neutral, indicating that the subject did not have to orient to either visual hemifield. The cue, whether directional or neutral, remained on throughout the 1500 ms of the recording epoch.

The target was presented 600 ms after cue onset and approximately 8 degrees lateral to the fixation point. Targets appeared randomly in the right or left visual field (p=.5), independently of the preceding cue. Subjects had to respond by lifting the index finger from a reaction-time key every time a target appeared in the visual field cued by the antecedent arrow. All children responded with the right hand.

A minimum of 900 ms elapsed following the presentation of the target stimulus and before the initiation of the next trial. The actual intertrial interval was longer than 900 ms whenever the subject took some time to depress the response key after lifting his/her finger in response to the previous target. Trials representing the six possible experimental conditions--determined by the type of
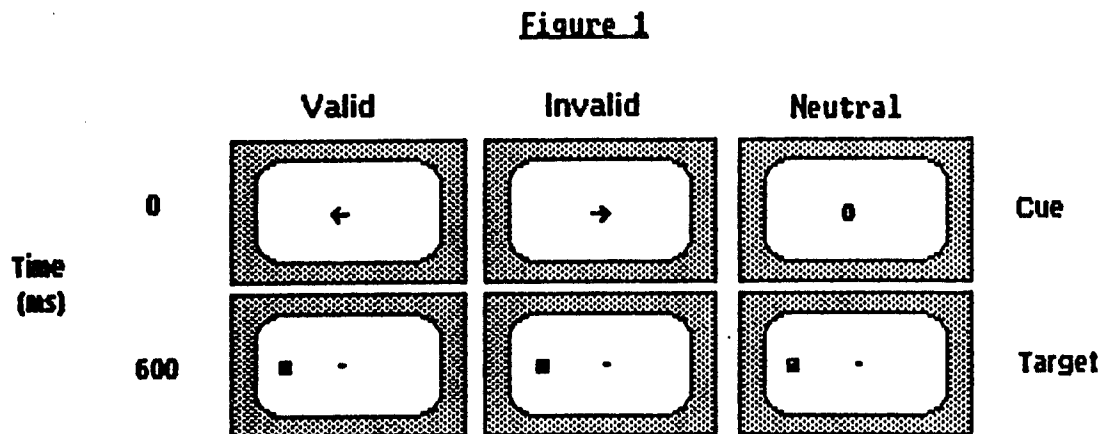
Figure 1

Figure 1. This figure exemplifies the nature of the task employed in the study. Subjects fixated the center of the screen, where the cue was presented. Six-hundred milliseconds later, a target appeared to the right or left of the fixation point. Figure 1 depicts the three types of conditions: **valid**, when the target appeared in the cued hemifield; **invalid**, when it appeared in the visual field opposite to that signalled by the cue; and **neutral**, when the cue was non-directional.

cue and the visual field of the target--continued to be presented randomly until approximately 24 artifact-free and behaviorally correct trials had been collected in each condition.

Speed and accuracy of the responses were recorded automatically by the computer. Finger-lift reaction times (RT) were considered a *hit* when they were emitted within 900 ms after target presentation. Responses to targets flashed in the invalid visual field were considered *false alarms* (FAs), and response times longer than 900 ms when targets appeared in the valid visual field were classified as *misses*. Children were given points for hits, and extra points for fast correct responses. At the end of the session, they could exchange points for toys or money.

Event-related Potentials and Electro-oculograms

The EEG was recorded for 1500 ms following the onset of the cue. Grass AC amplifiers were used to amplify the EEG, with high and low frequency cut-offs at 100 and 0.1 Hz, respectively. A 386 Compaq computer recorded and digitized the data at 50 Hz. International Electro-caps were used to position electrodes over the scalp. Brain potentials were recorded from seven locations within each hemisphere: occipital (O1-O2), parietal (P3-P4), central (C3'-C4'), temporal (T5-T6), anterior temporal (T3-T4), frontal (F3-F4), and lateral frontal (F7-F8). All electrodes were referenced to yoked ears, and electrode resistance was kept below 10,000 ohms.

Electrodes placed 1 cm to the left and 1 cm below the corner of the right and left eyes, referenced to the opposite ear, were used to monitor eye movements and blinks. Rejection of trials contaminated by movement artifacts was conducted on-line. The cut-off rejection value was set separately for each individual at the beginning of the session by calculating the range of voltage at each electrode

location while the subject remained immobile. The average voltage range was calculated from ten consecutive readings, first with the subject's eyes open, then closed. The rejection criterion was set as the maximum average voltage at any of the electrode locations while the subject's eyes were closed. A trial was rejected whenever the voltage exceeded this criterion or when the electro-oculogram (EOG) channels reached 50% of the criterion value for all other electrode locations. Trials that resulted in misses were also discarded.

Single-trial data were digitized on-line, and later stored on optical disks. The averaging was carried out by a computer program that included only trials where the subject had responded correctly.

Data Analysis

An averaged ERP waveform was obtained for each experimental condition and electrode location, totalling 96 waveforms per subject. From these waveforms, several ERP measures were extracted which were subsequently subjected to statistical analysis. The design used was a repeated-measures analysis of covariance with the between-group factor representing reading level (poor vs. good readers). When analyzing the pre-target measures, the within-subject experimental factors consisted of cue direction (right or left), electrode location within each hemisphere (occipital, parietal, central, temporal, anterior temporal, frontal and lateral frontal) and hemisphere (right or left). When analyzing the post-target effects, within-subject factors consisted of cue validity (validly or invalidly cued targets), visual field of the target (right or left), electrode location within each hemisphere (occipital, parietal, central, temporal, anterior temporal, frontal and lateral frontal) and hemisphere (right or left). In order to correct for any possible eye-movement distortions that might have remained undetected, the

EOG obtained at the eye contralateral to the hemisphere of recording was used as a covariate. The subjects' sex was not included as a between-subject factor in the model since the number of males was two times the number of females, and there were equal numbers of females in each of the two reading groups. For all ANOVAs, degrees of freedom were adjusted to correct for the sphericity problem associated with repeated-measures designs (Vasey & Thayer, 1987).

Several behavioral measures also were analyzed: average reaction times for right and left visual field responses, and percentages of hits, FAs and misses. These measures were subjected to univariate ANOVA tests. Additionally, an unbiased measure of sensitivity, d', and a measure of response criterion, , were calculated for each subject (Swets, 1964) and analyzed for differences between cueing conditions. Finally, relationships between behavioral and ERP measures were determined by computing Pearson and partial correlation coefficients, as appropriate.

Throughout the text, subjects falling above the median on the Woodcock-Johnson Reading Achievement Test are designated "good readers", while those below the median are called "poor readers". It is important to note that this is a mere label used for purposes of simplicity, and applied exclusively to the subjects in this study. As reflected in the previous neuropsychological description of the group, most subjects showed reading ability that was well within the normal range.

CHAPTER III

RESULTS

Figure 2 exemplifies the characteristic configuration of the group-averaged ERP waves obtained in this experiment. An individual averaged wave for each experimental condition was collected at each electrode location. Figure 2 displays in each cell the superimposed averaged waveforms for valid, invalid and neutral trials recorded at the occipital electrodes. The left and right columns correspond to the left and right hemispheres, respectively, while the rows represent the visual field where the target appeared. Thus, the top box of the left column contains the averaged response evoked over the occipital region of the left hemisphere by validly, invalidly and neutrally cued targets appearing in the right visual field. Note that six separate waveforms were obtained over each hemisphere for each electrode location. Since ERPs were recorded from a total of seven sites in each hemisphere, the number of ERP waves for each individual subject totalled 84 (7 electrode locations, 2 hemispheres, 3 types of cue, and 2 hemifields where the target could appear). The EOG recorded beside each eye brought the number of averaged ERPs to a total of 96 per subject. A display of all waveforms averaged across all individuals is presented in Figures A.1 and A.2 of Appendix A. Figure A.1 corresponds to trials in which the target appeared in the right visual field, and Figure A.2 to left field target presentations.

The results from this experiment are organized in two major sections. The initial portion reports the patterns of brain activity obtained on this spatial task and their association with reading ability. The second section describes the nature

Figure 2. Grand-average ERPs for all subjects recorded at the occipital sites. Superimposed waves correspond to the valid (——), invalid (——), and neutral (...) conditions. The top row depicts potentials evoked by stimuli appearing in the right visual field (RVF), while the bottom row corresponds to left visual-field stimuli (LVF). ERPs in the left and right columns represent brain activity recorded over the left (LH) and right (RH) hemispheres, respectively. Ordinate values are amplitudes of the ERP in microvolts (µV), while the abscissa depicts the latency of the potential in milliseconds. Negative polarity is up.
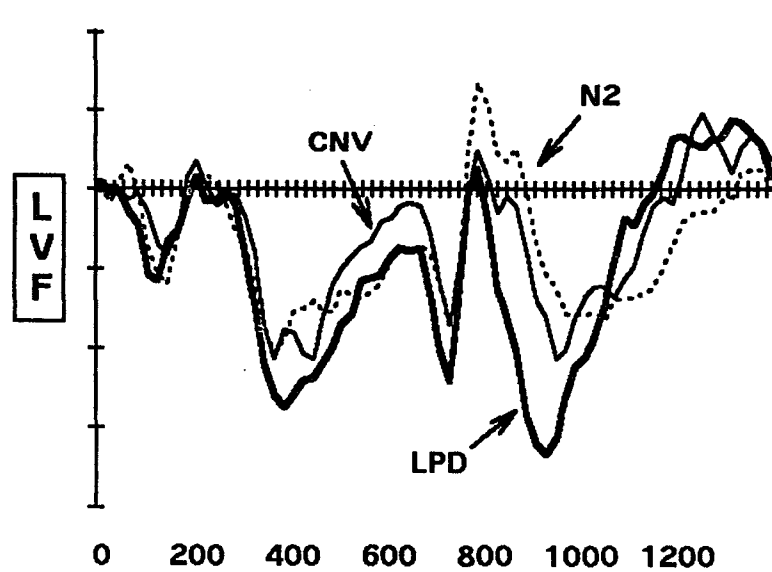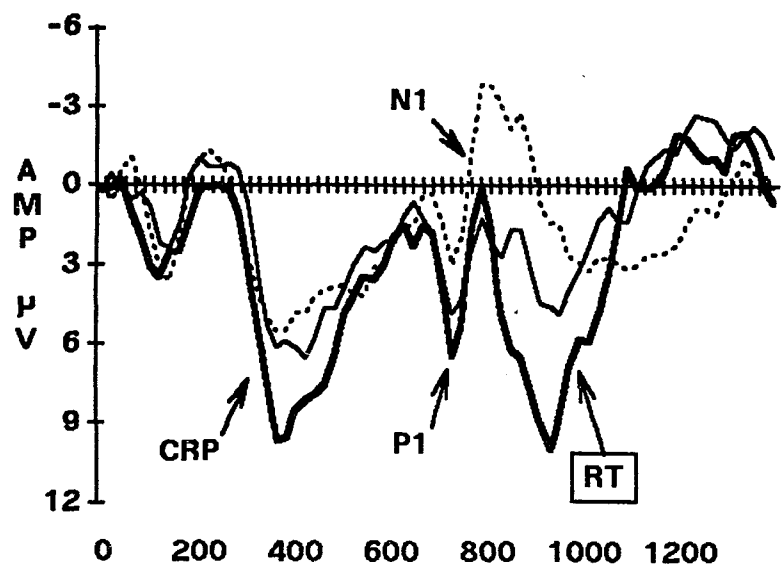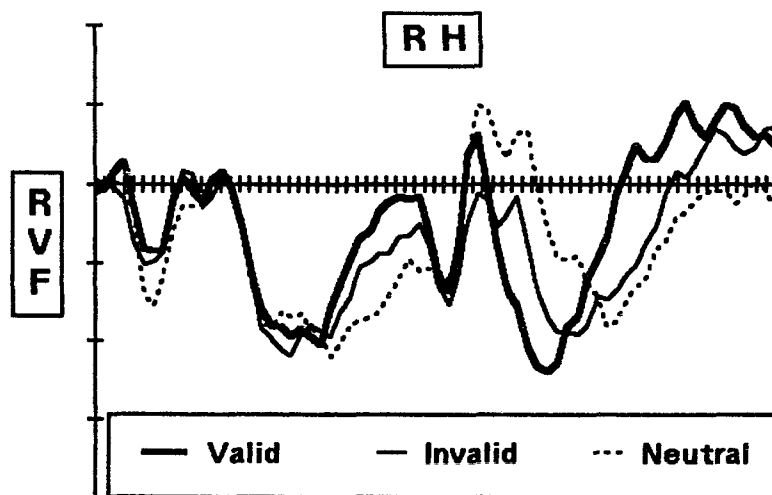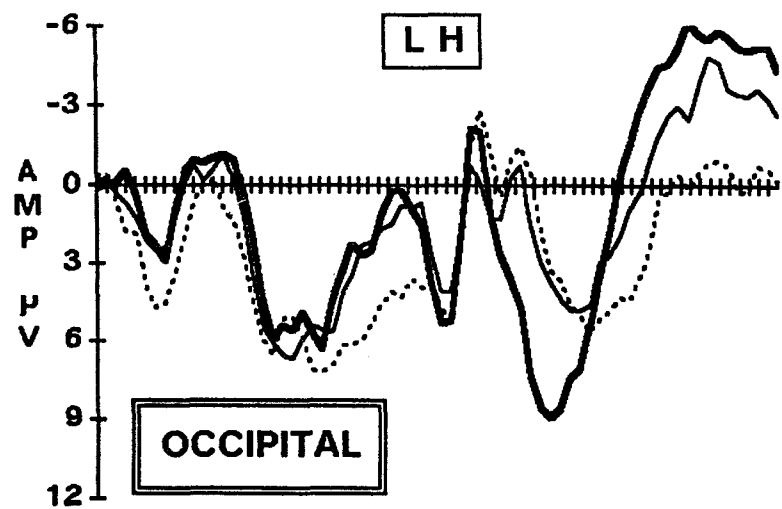
of the relationship between ERP and behavioral measures, and explores their connection to reading skills.

<u>Event-related Potentials</u>

This section concentrates, first, on the brain potentials evoked by the cue stimulus and preceding the onset of the target. It is during this interval that the subject should discriminate the cue, register its meaning, and direct attention in visual space according to whether the cue is directional or not. Consequently, cortical potentials evoked by the cue should reflect the point at which the brain first differentiates between directional and non-directional cues, and between the cued and uncued hemifields. Cue-related potentials also should reveal whether the subject selectively prepares to process visual information in the cued location. Later on, differences in brain activity following the presentation of the target stimulus should indicate how the presence of the earlier valid or invalid cue affects the processing of that stimulus.

While analyzing these findings, it is important to acknowledge the relationship between the cortical potentials recorded on this spatial task and the subjects' reading ability. The children in this study were selected originally on the basis of their potential risk for developing a reading disability, although they had not begun to read. By the time they were tested on this task, they were attending 4th grade and their reading skills could be determined. These 18 subjects were divided in two groups according to their reading performance in third grade. The median standard score on the Woodcock-Johnson Test of Reading Achievement was taken as the dividing line between poor and normal readers.

Potentials Evoked by the Cue

Within the first 150 ms, no significant differences between the potentials elicited by each of the three cues were expected to be found. The visual evoked potential during this latency range is primarily affected by the physical properties of the stimulus, which were kept nearly identical for all cues. (Recall that all cues appeared at the same central location, and were of equal size and luminance.) Following the processing of basic shape differences between cues during the first 150-200 ms, brain potentials falling between 200-700 ms may reveal selective processing of directional cues, and directing of attention towards the hemifield signaled by such cues. In order to test this hypothesis, two separate analyses were carried out on the amplitude of each one of the potentials evoked by the cue. The purpose of the first analysis was to establish whether the brain's response to directional and non-directional cues was the same or different. Consequently, all cues were included in the model. The second question of interest was whether different arrow directions resulted in distinct patterns of brain activity. To that end, the neutral stimulus, which should elicit no attentional shifts, was subtracted from the response to either a right or a left arrow and, thus, served both as an individual baseline and a control for the general increase in arousal that follows any warning signal.

In both cases, a repeated-measures ANOVA model was used to test for significant differences in brain responses related to the processing of the different cues. When testing for the difference between directional and non-directional cues, the three repeated measures were Electrode Site (occipital, parietal, central, temporal, anterior temporal, frontal and frontal lateral), Hemisphere (left and right), and Cue Type (arrow right, arrow left, and neutral). This analysis was

followed by a contrast between the directional arrows and the neutral cue. Reading level was not included in the model, since the focus of this test was the attentional task in itself, and inclusion of all subjects maximized statistical power. In the subsequent analysis, however, the model included one between-subject (Reading Level) and three within-subject factors (Electrode, Hemisphere and Arrow Direction). As stated above, responses to the neutral cue were subtracted from responses to either a right or a left arrow. These difference potentials (DERPs) were measured at several points during the interval between cue and target presentation, as specified below.

In order to control for the possible presence of undetected eye movements, which would hinder the distinction between attentional and oculomotor shifts, the EOG was used as a covariate. The EOG recorded at the contralateral eye was selected over ipsilateral recordings as the appropriate tracing for the measurement of eye movement contamination for a given hemisphere. The contralateral EOG shares less brain activity with the hemisphere being recorded, while yielding the same information as the ipsilateral EOG in terms of oculomotor potentials.

The average amplitude of the waveform over the initial 60 ms was used as a common baseline to calculate the amplitude of all deflections present in the interval between cue and target. This procedure helps compensate for any amplitude oscillations that may be present at the beginning of the waveform, and which may vary inconsistently across experimental conditions or electrode locations.

For the cue-related components, waveforms in response to the same arrow cue were collapsed across the two conditions defined by the hemifield where the subsequent target was presented. Since the target's location was varied randomly,

and it could not be anticipated by the subject, these trials were in effect replications of the cue condition. In averaging across the two target locations, each condition was weighed by the number of trials included in the average so as to minimize violations of homoscedasticity.

Four potentials were analyzed within the cue-target interval. The names assigned to the first two were preceded by a "C" (for "cue") in order to distinguish them from similar potentials evoked by the target later on: (1) *C-P120*, an early positivity peaking between 80-160 ms after cue onset; (2) *C-N240*, a negative deflection present at about 240 ms; (3) a *Cue-Related Potential*, a slow positive wave defined as the average amplitude of the potential ranging between 320-480 ms after the cue, and (4) *Contingent Negative Variation*, a negative slow wave immediately preceding the target, and spanning from 480 ms after cue onset until 60 ms following target presentation.

C-P120. This early potential was quantified as the highest peak in the deflection occurring between 80-160 ms after the onset of the cue. C-P120 was of positive polarity at the posterior scalp locations (occipital, temporal and parietal), and negative over more anterior regions (anterior temporal, central, and frontal; see Figure A.3 in Appendix A).

At this early latency, there were no indications of a difference in amplitude across the three cue types (Cue Type, $F(2,33)=.47, p=.627$). The contrast between directional and non-directional cues also failed to reach significance (Arrows vs. Neutral, $F(1,16)=.68, p=.423$).

Significant differences between right and left arrow cues also were lacking (Arrow Direction, $F(1,15)=.01, p=.91$) over both hemispheres (Arrow Direction x Hemisphere, $F(1,15)=.10, p=.75$). The response of the two hemispheres to the

right and left arrows appeared not to be uniformly distributed over the scalp, but variations in distribution did not reach statistical significance (Electrode x Hemisphere x Arrow Direction, $F(6,96) = 1.98$, $p = .076$). This three-way interaction probably reflected changes in the polarity of C-P120 in the posterior to anterior axis.

C-N240. Immediately after C-P120, a negativity was apparent at the posterior electrode locations simultaneously with a prominent anterior positivity. The peak amplitude of this deflection was calculated within the 180-280 ms latency range. The type of cue appeared not to influence the amplitude of C-N240 (Cue Type, $F(2,33) = 1.91$, $p = .164$), and no differences were found between directional and non-directional cues (Arrows vs. Neutral, $F(1,16) = 1.23$, $p = .284$).

The overall amplitude of C-N240 was greater for poor than normal readers (Reading Level, $F(1,15) = 5.24$, $p = .037$). This effect, however, did not interact with any other factor, and was the only significant difference between the two groups. Later on, it will become clear that this main group effect constitutes the first evidence of a negative shift in the over-all ERP waveform of the poor readers with respect to the normal readers.

The nature of the relationship between the direction of the arrow and the recorded hemisphere varied across the scalp (Electrode x Hemisphere x Arrow Direction, $F(6,96) = 3.02$, $p = .027$). Figure 3 contains the posterior to anterior scalp distribution of each of the four combinations of arrow direction and hemisphere of recording. Since these two latter factors did not interact significantly, the mean amplitude of C-N240 for all bars in each graph is comparable to that of any other graph. Careful examination of the differences across electrode locations,

Figure 3. Bar graph depicting the difference between the arrow and neutral responses for the C-N240 potential, averaged across all subjects. The top row corresponds to the right-arrow (RA) response, while the bottom row includes the left-arrow (LA) potential. The left and right columns, as in the previous figure, represent the ERPs recorded over the left (LH) and right (RH) hemispheres, respectively. The abscissa depicts the various electrode locations: occipital (O), parietal (P), central (C), posterior temporal (TP), anterior temporal (TA), frontal (F), and frontal lateral (FL). Amplitudes in microvolts (μV). Negativity up.

Fig. 3
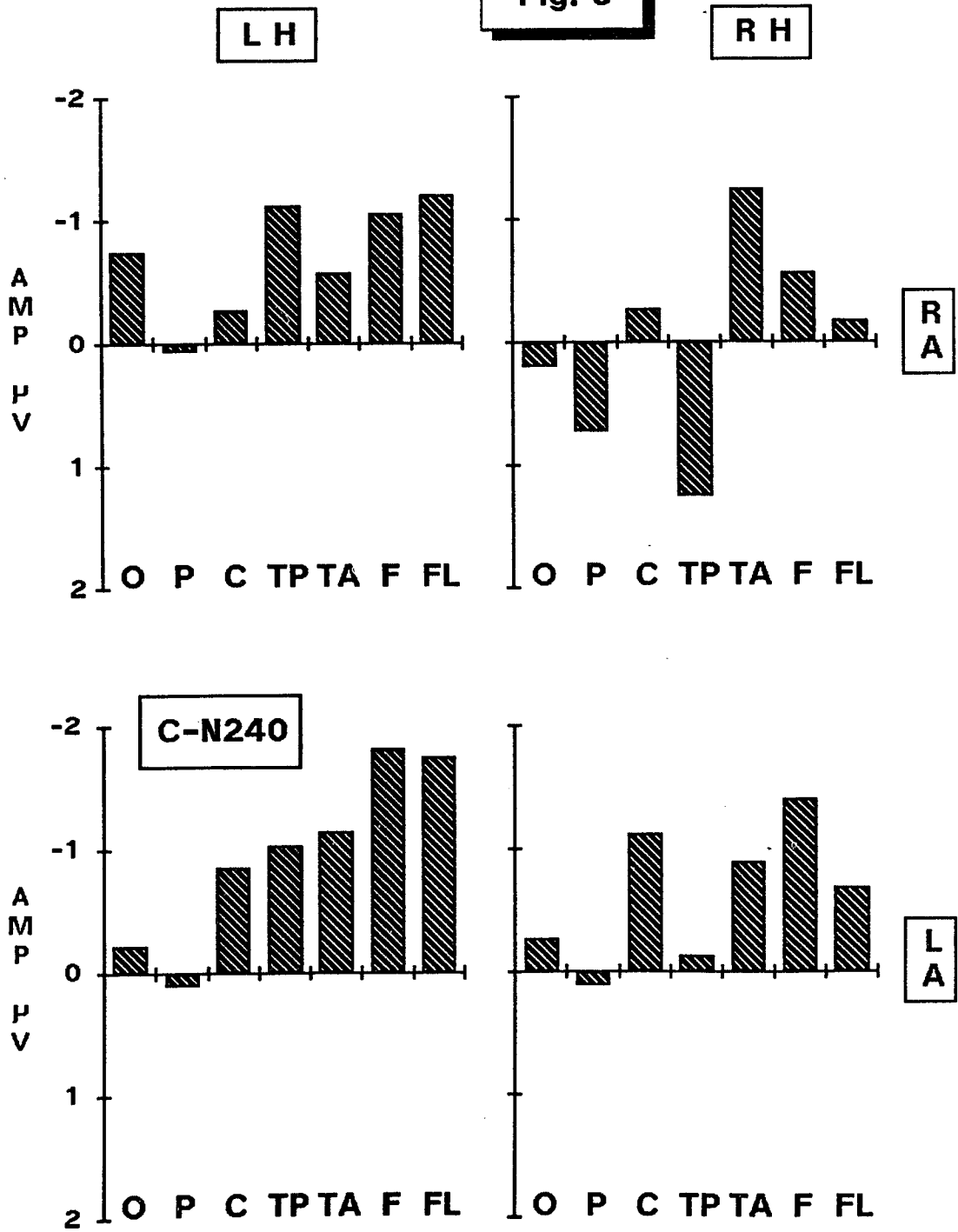
however, revealed that the posterior electrode sites behaved quite differently from the more anterior locations.

Over the occipital, parietal and, especially, the posterior temporal area, the difference in C-N240 amplitude between the right and left arrows was present over the right, but not the left, hemisphere. There was a suppression of the C-N240 evoked by the right arrow over the occipital, parietal and temporal-posterior regions of the right hemisphere. The statistical significance of this response pattern was tested with a contrast between the posterior (occipital, parietal and temporal posterior) and anterior (central, frontal, temporal anterior and frontal lateral) electrodes. The posterior-anterior contrast revealed the expected significant interaction between the direction of the arrow and the hemisphere from which the potential was being recorded (Posterior-Anterior x Hemisphere x Arrow Direction, $F(1,16)=5.02, p=.0396$).

Cue-Related Potential. The polarity of the potential obtained between 300-500 ms after cue onset was positive over the posterior regions of the scalp, and became gradually more negative towards the front of the head. This Cue-Related Potential (CRP) was quantified as the average amplitude of the waveform between 320-480 ms following the cue. A look at Figure A.3 in Appendix A shows how, over the posterior regions, the CRP evoked by the neutral condition remained closer to baseline than either the left or the right arrow responses. Since the magnitude of the CRP was greatest at the back of the head, this trend suggested that the CRP had an overall positive polarity, and justified the use of the neutral cue as a baseline when comparing right and left directional cues.

As in the case of the previous potentials, C-P120 and C-N240, the amplitude of the CRP did not vary as a function of the evoking cue (Cue Type,

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$F(2,33)=.30, p=.741$), or the directionality of the cue stimulus (Arrows vs. Neutral, $F(1,16)=.32, p=.581$) when tested across all subjects. Instead, the variation present in the data seemed to be significantly explained by changes in the EOG (EOG covariate for the Arrow Direction factor, $F(1,16)=8.7, p=.009$). The statistical significance of the EOG covariate indicates that there is no variation in the data that can be explained by any of the factors in the model, once eye movements are controlled. It further suggests that the precautions taken to control for eye movements might have not been sensitive enough to detect whether subjects had moved their eyes only slightly. It is not a coincidence, therefore, that the most significant contribution of the EOG covariate was found at this point in time, which corresponds roughly to the latency range for voluntary saccades in children (Miller, 1969).

The outcome markedly changed when the analysis was adjusted for reading level by dividing the whole group into poor and normal reading subgroups. In Figure 4, one can see the pattern of results observed in the normal reading group. These results may be contrasted with those recorded from poor readers, and depicted in Figure 5. The first noticeable difference between the two groups consists of a general reduction in amplitude in the poor readers, when compared to the normal subjects. The difference in activation between these two groups, however, was not consistent enough to attain statistical significance as a main effect (Reading Level, $F(1,15)=1.36, p=.26$). Despite the lack of differences across groups in the over-all magnitude of the potential, the pattern of interactions across electrode sites did discriminate between the two reading levels.

The most noticeable differences across groups were found over the posterior regions of the scalp, at the occipital, parietal and temporal electrode

Figure 4. Magnitude of the cue-related potential (CRP) response in good readers. Bars correspond to the difference in amplitude between each arrow direction and the neutral condition. Clear bars correspond to the right arrow (RA), while hatched bars depict the left arrow (LA) responses. Each graph represents a different electrode location, where the left-hemisphere (LH) responses are displayed on the left-hand side, and the right-hemisphere (RH) responses on the right. Note the difference in amplitude between the RA and LA responses over the posterior sites: occipital, parietal, and temporal posterior.
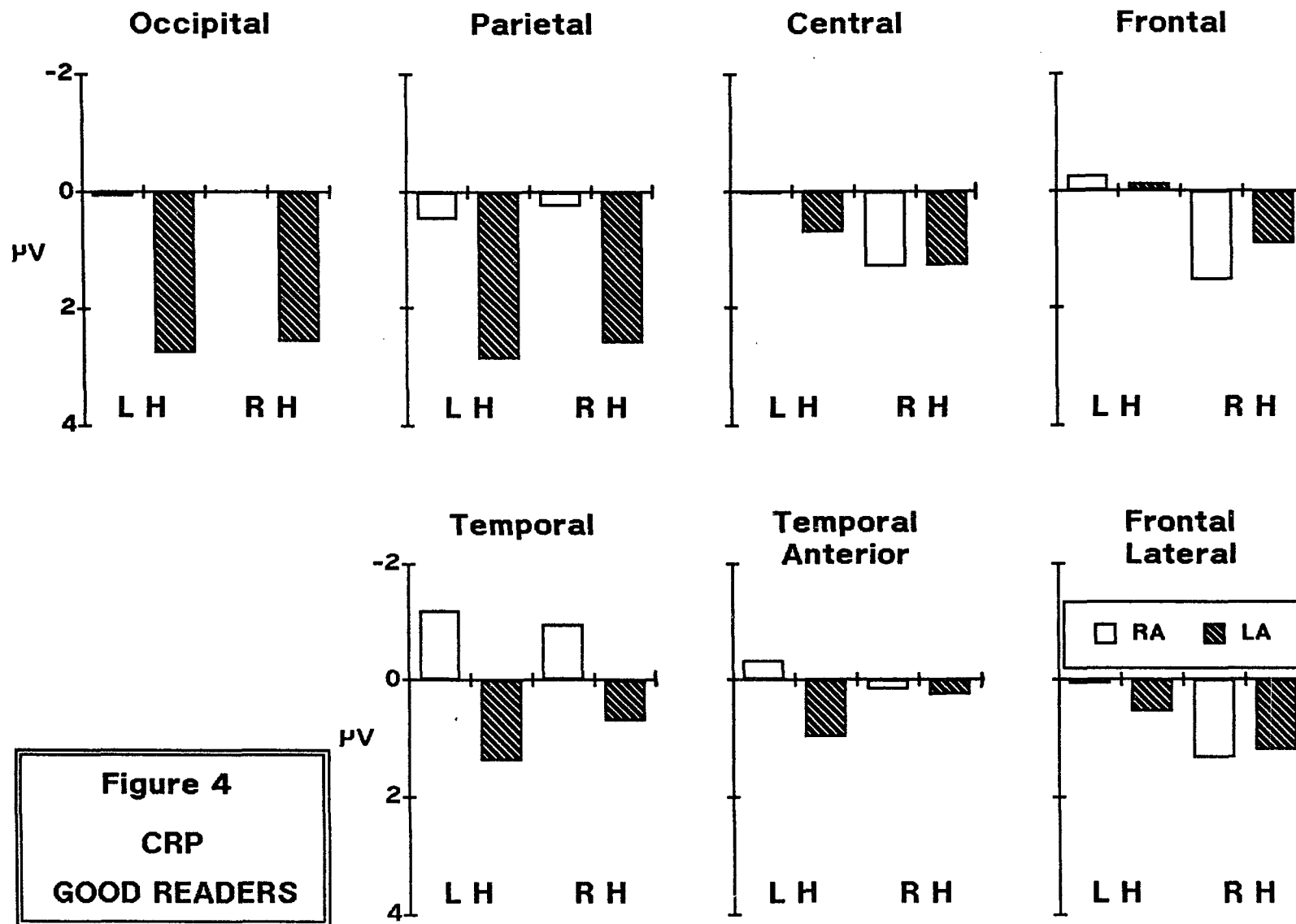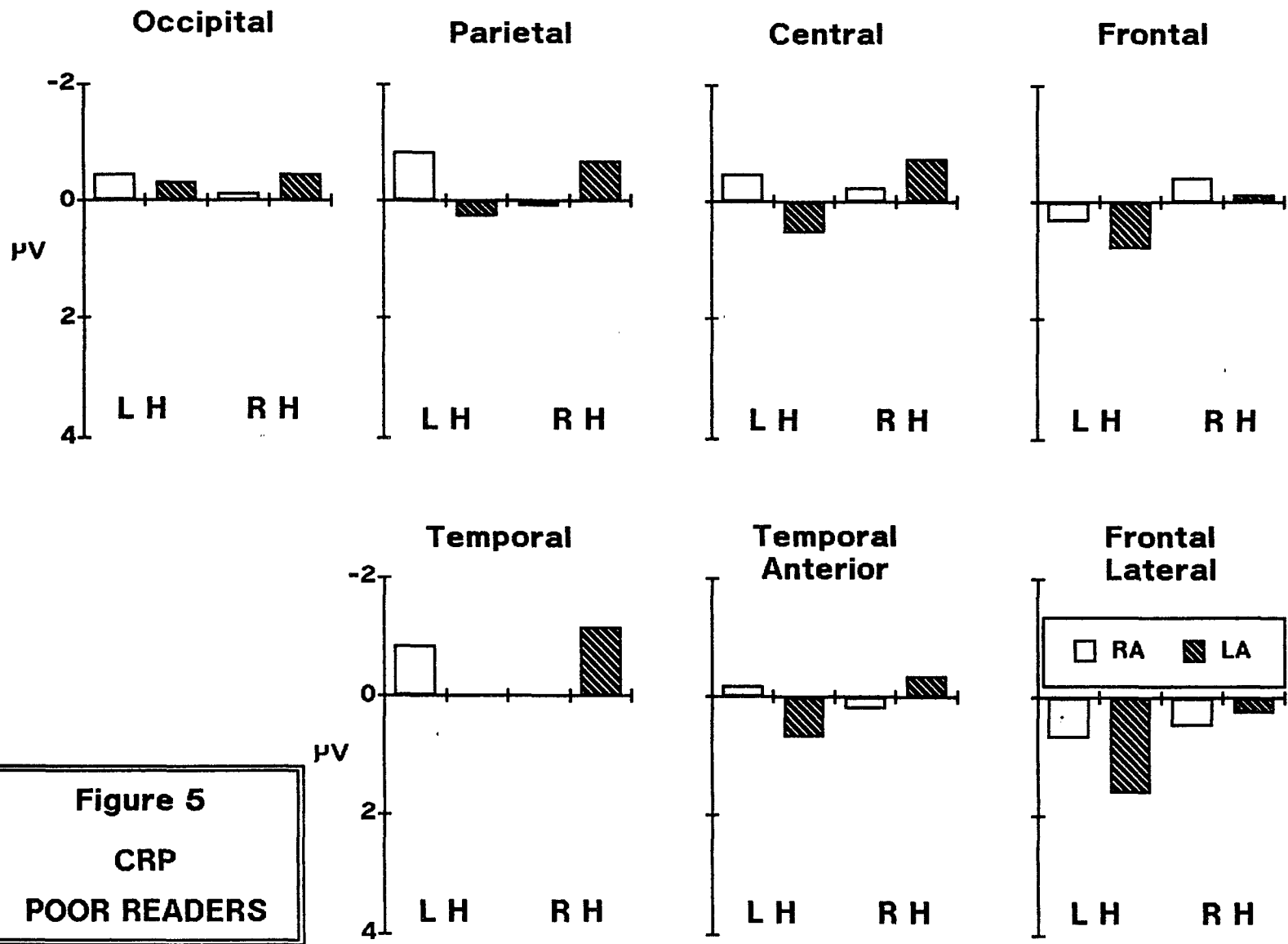
Figure 4
CRP
GOOD READERS

Figure 5. As in the previous figure, bars depict the magnitude of the cue-related potential (CRP), this time for poor readers. Notice the marked absence of differences between the CRP-responses to right (RA) and left (LA) arrows over the posterior electrode locations.

Figure 5

CRP

POOR READERS

locations. Normal readers showed greater activation in response to the left than to the right arrow over both hemispheres. The CRPs of the poor readers did not respond differently to the two arrow directions over either occipital or temporal areas. At the parietal electrodes, however, their responses were greater over the hemisphere ipsilateral to the cued hemifield. These effects can be seen by comparing Figures 4 and 5. Note that positive values indicate greater activation in response to directional than non-directional cues. Figures A.4 and A.5 of Appendix A depict the same interaction, this time in the evoked potentials themselves. When tested across all electrodes, the interaction only approached significance (Electrode x Arrow Direction x Reading Level, $F(6,96)=2.64$, $p=.072$). But when posterior and anterior sites were compared by means of a contrast, the F-statistic increased and the p-value gained significance (Posterior-Anterior x Arrow Direction x Group, $F(1,16)=4.45, p=.05$).

Contingent Negative Variation. Following the CRP, a slow negative potential emerged that extended beyond the presentation of the target. This negativity could be characterized as a contingent negative variation (CNV) (Cohen, 1969) related to the predictable presentation of the target. It was quantified as the average amplitude of the ERP between 480-660 ms following cue onset. Figure A.3 in Appendix A shows how the waveform evoked by the neutral cue was less negative than the responses to either arrow within this latency range.

The CNV was significantly more negative in response to the directional arrows than to the neutral cue (Cue Type, $F(2,33)=6.11$, $p=.0055$; Arrows vs. Neutral, $F(1,16)=9.57, p=.0070$). This difference in amplitude, associated with the directionality of the cue, was widely distributed over the surface of the scalp

(Electrode x Cue Type, $F(12,204)=1.64$, $p=.187$), but seemed most salient over the temporal sites, both posterior and anterior (see Fig. A.3 in Appendix A).

When the response to the neutral cue was subtracted separately from the right- and left-arrow responses, a significant difference was observed as well between the two arrow directions. Differences between right and left arrow, however, had a more circumscribed distribution than differences due to cue directionality (Electrode x Hemisphere x Arrow Direction, $F(6,96)=3.88$, $p=.004$). Figure 6 illustrates the character and distribution of this three-way interaction. As in previous figures, the bars represent the difference between the neutral cue and either the left (LA) or the right (RA) arrow. Note that the right arrow evoked larger responses than the left over both hemispheres, though this difference was most noticeable for the occipital, parietal and temporal posterior areas of the right hemisphere.

As in the case of the CRP, the pattern of CNV results was not the same for normal and poor readers. While the CNV in response to the right arrow was comparable across the two reading levels, normal readers showed significantly attenuated CNV amplitudes in response to the left arrow across both hemispheres (see Figure 7), but only over the posterior regions of the scalp (Electrode x Arrow Direction x Reading Level, $F(6,96)=5.31$, $p=.003$) (see Figure 8). In contrast, poor readers did not display differential activation across cues or hemispheres, in spite of their greater overall CNV amplitudes. In fact, as mentioned above in the case of C-N240, the waveforms of poor readers tended to be more negative than those of normal readers, but without a systematic or selective pattern for each distinct deflection. In general, differences between normal (Figure 7) and poor

Figure 6. The average contingent-negative variation (CNV) for all subjects at all electrode locations. As in previous figures, clear bars correspond to the right-arrow responses (RA), while the hatched bars stand for the left-arrow (LA) CNV potentials. Once again, the most noticeable differences between the responses to the RA and LA were recorded over the posterior electrodes.

Occipital   **Parietal**   **Central**   **Frontal**

Temporal   **Temporal Anterior**   **Frontal Lateral**

Figure 6
CNV
ALL SUBJECTS
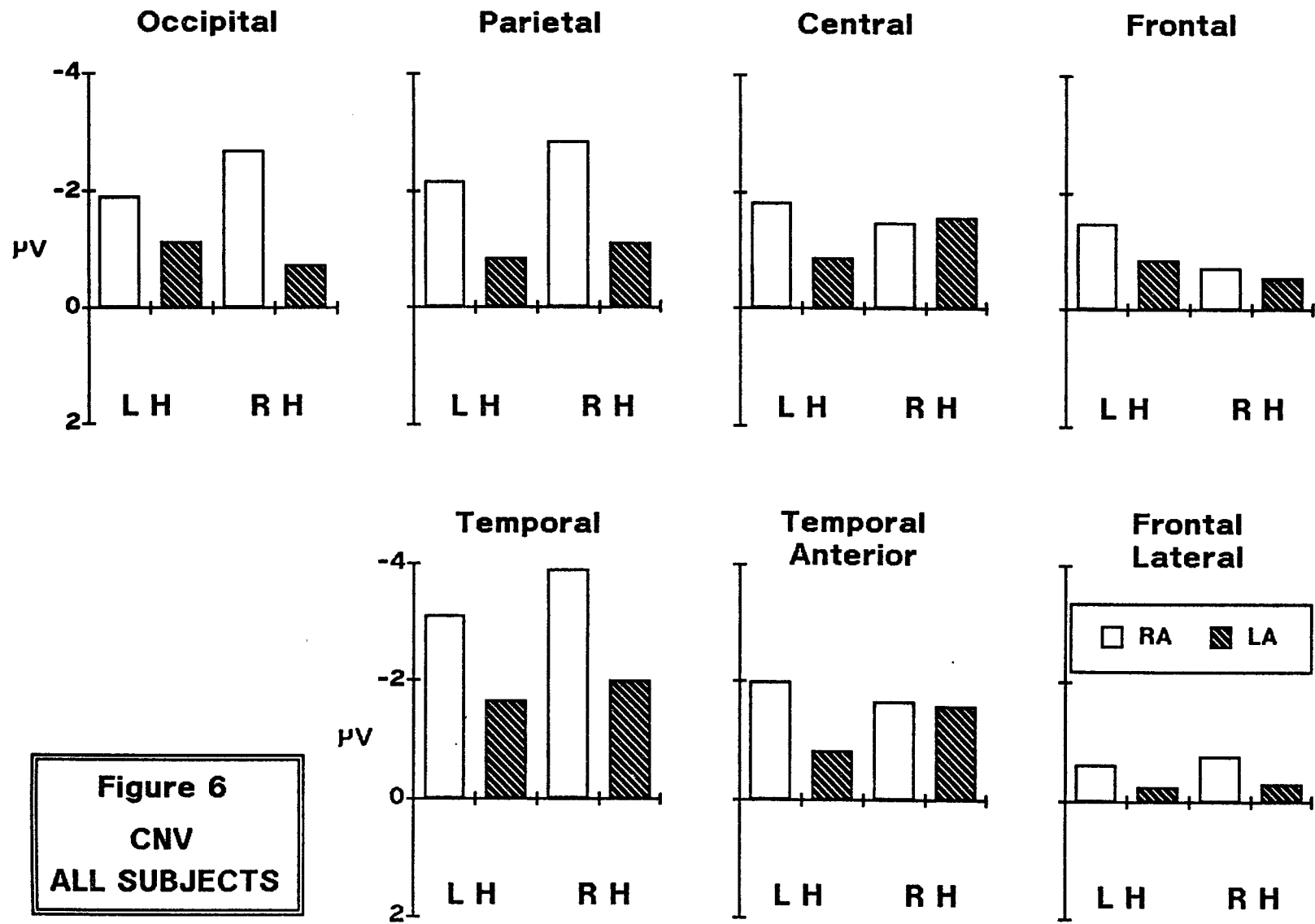
Figure 7. Magnitude of the contingent negative variation (CNV) in good readers. Bars correspond to the difference in amplitude between each arrow direction and the neutral condition. Clear bars depict right-arrow (RA) responses, while hatched bars represent the left-arrow (LA) CNVs. Each graph displays the potentials recorded over the left (LH) and right (RH) hemispheres at each electrode location.

Figure 7
CNV
GOOD READERS

Occipital Parietal Central Frontal

Temporal Temporal Anterior Frontal Lateral

RA   LA

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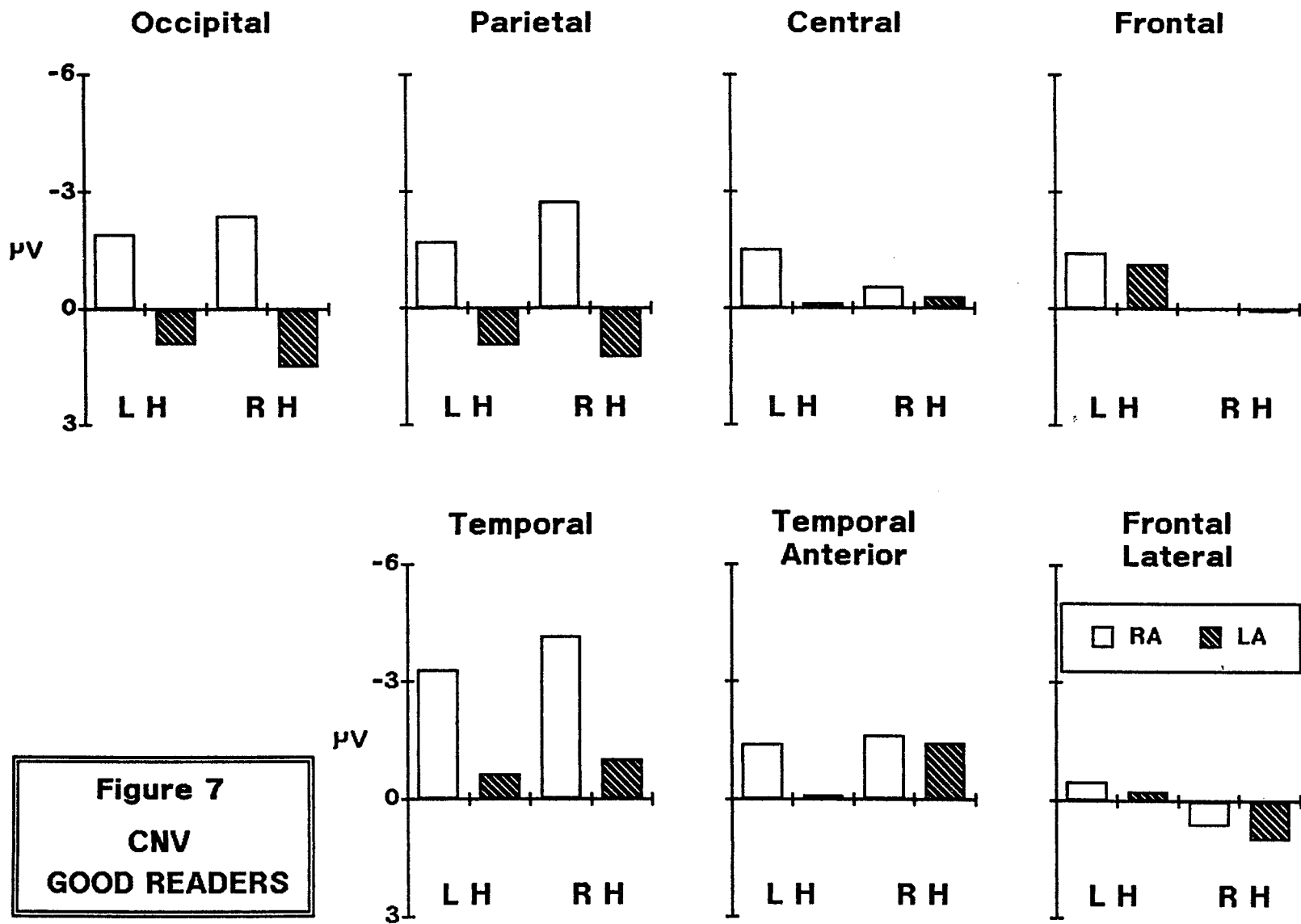
Figure 8. Magnitude of the contingent negative variation (CNV) in poor readers. Contrast these responses with those of good readers in the previous figure. Although the over-all amplitude of the CNV is larger for this group, no significant differences are observed between the responses to the right (RA) and left (LA) directional cues.

Occipital

Parietal

Central

Frontal

Temporal

Temporal Anterior

Frontal Lateral

□ RA ▨ LA

Figure 8

CNV

POOR READERS

60
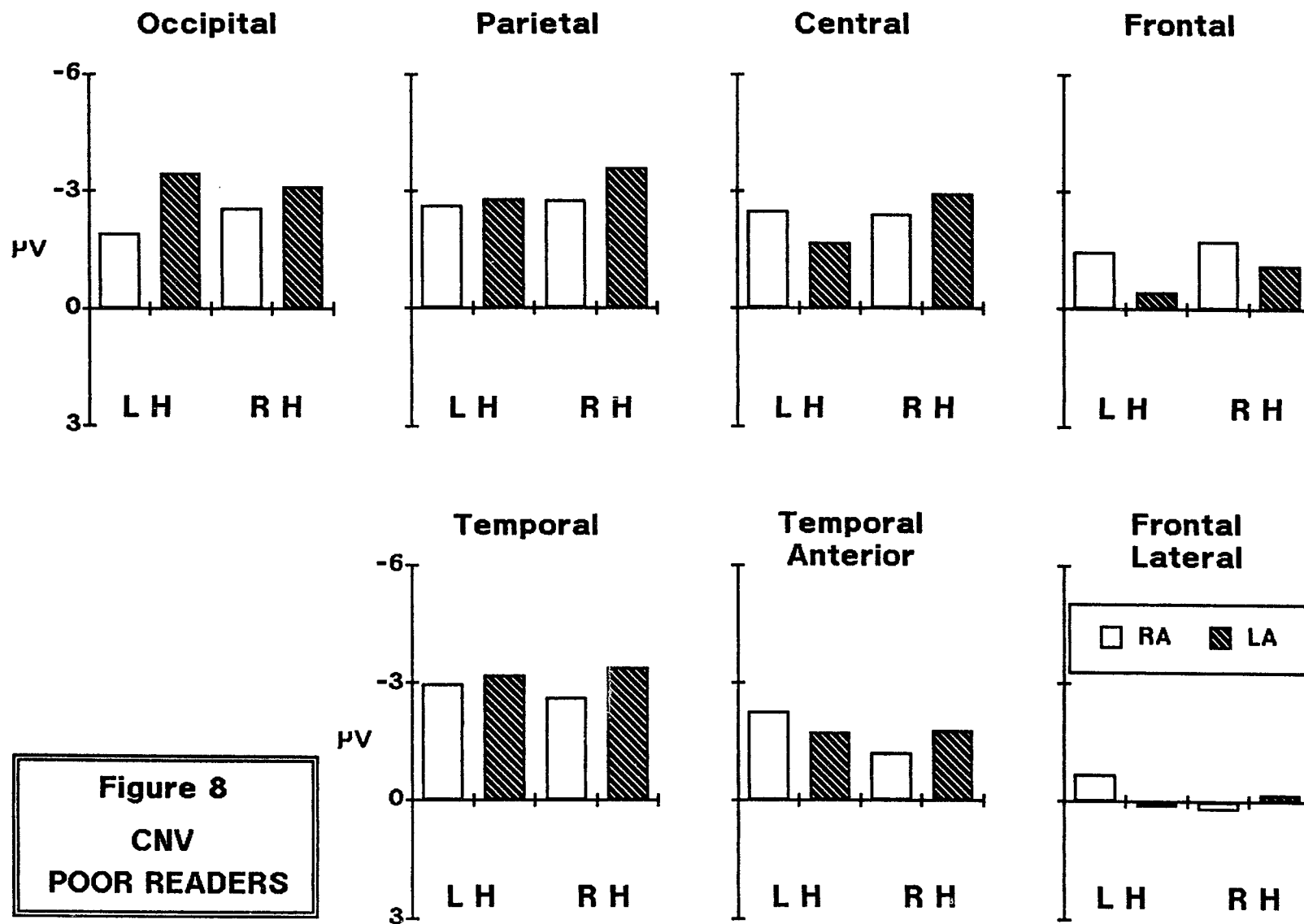
readers (Figure 8) were more accentuated in the CNV than in previous potentials evoked by the cue.

The results of an ulterior contrast between posterior and anterior electrodes revealed a remarkable difference between the two groups in their response to the right and left arrows (Posterior-Anterior x Arrow Direction x Reading Level, $F(1,16)=8.12$, $p=.0065$). The contrast confirmed the trend observed in the full model: the CNV pattern was, in fact, different for the two reading groups, but only over the posterior areas of the scalp. Differential processing of arrow direction was only observed over the occipital, parietal and temporal posterior regions, and only in normal readers. No differences were found between poor and normal readers over anterior areas of the scalp (see Figures 7 and 8).

ERPs to Targets Following Valid, Invalid and Neutral Cues

This section concentrates on the potentials that appeared between 600-1500, and were evoked by the target stimulus. The objective here is to verify that the attentional shift presumedly prompted by the cue affected the processing of the subsequent stimulus.

In a more traditional vein, a way to achieve this goal is to compare the response to a target following an arrow pointing in its direction (valid cueing) with the response evoked by the same target when the preceding cue pointed in the opposite direction (invalid cueing). In this case, the physical parameters of the target stimulus (shape, eccentricity, and probability of appearing in either the cued or uncued visual field) remain constant across conditions. Differences between validly and invalidly cued targets are attributed, therefore, to the spatial selection

set up by the preceding cue. It is that difference that is designated hereafter as the *validity effect*.

But the spatial distribution of the validity effect is not the only important aspect to consider. A second question of interest in looking at post-target potentials relates to the temporal processing of information that leads to the decision to respond made by the subject. The neutral cue plays a central role here in that its presence informs the subject from the outset that a response is not required. Conversely, when a directional cue is presented, the subject has to wait until the target stimulus appears, discriminate its location and, only then, decide whether or not to emit the contingent motor response.

Changes in the cortical potential evoked by the invalid condition should reflect this decision-making process. Until the discrimination of the target's location takes place, the invalid condition should resemble the valid response, since in both cases the subject was cued in one direction, where he/she expected the appearance of the target. But, following the discrimination of the target's location, the invalid condition should become similar to the response evoked by a neutral cue, since in either case the subject must refrain from responding in order to avoid a false alarm.

As in the case of the cue-related potentials previously covered, it is important to determine whether normal and poor readers differed in the selective processing of the target and the subsequent preparation for a motor response. So, once again, the repeated-measures ANOVA employed in the analysis of each post-target potential included reading level as a between-subject factor.

Several post-target deflections were measured in the interval between 600-1500 ms. For all of them, the baseline used was the average amplitude of the

waveform between 560-640 ms, that is just before and during the time the target stimulus was presented. The potentials obtained following the target resembled those reported for adult subjects in other visual selective attention studies (Hillyard and Picton, 1987): P1, N1, N2, and a late positive potential, followed by a slow negative wave that extended until the end of the recording epoch (see Figure 2).

P1 and N1. The earliest positive deflection, P1, peaked at approximately 120 ms following target onset, while the first negativity, N1, had its peak amplitude at about 180 ms. P1 could be identified only at the posterior electrode sites, so it was quantified at the occipital, parietal and temporal locations alone. The same was true for N1 (see Figure A.8 in Appendix A).

The initial statistical model compared the brain's response to validly and invalidly cued targets, without considering the neutral cue conditions. No significant validity effects were observed for either P1 (Cue Validity, $F(1,15) = 1.24$, $p = .28$) or N1 (Cue Validity, $F = .38(1,15)$, $p = .55$). That is, the amplitude of these two deflections was not modulated significantly by whether the target appeared in the validly or invalidly cued hemifield.

The validity effect was evident, however, in a peak-to-trough P1-N1 measure, where the peak amplitude of P1 was subtracted from the peak amplitude of N1. The use of this measure reduces the contribution of the ongoing slow wave related to prior preparation; P1-N1 reflects transient changes from 120 to 180 ms after target onset. When quantified in such fashion, the validity of the cue resulted in an increase of P1-N1 amplitude that was marginally significant (Cue Validity, $F(1,15) = 4.47$, p

the temporal sites, present only over occipital locations of the left hemisphere, and absent over the parietal area.

The amplitude enhancement reflecting cue validity differed between poor and normal readers. Poor readers showed greater validity effects than normal readers over the parietal and temporal areas of the left hemisphere (Electrode x Hemisphere x Cue Validity x Reading Level, $F(2,32)=5.06$, $p=.0170$). Figure 9 illustrates the nature and distribution of the amplitude modulation attributable to validity, as it relates to reading level. Each bar represents the difference in P1-N1 amplitude between the valid and invalid conditions. Greater negative values are evidence of greater P1-N1 validity effects. Notice how the greatest contrast between poor and normal readers is localized over the left parietal and temporal areas. Normal readers had a much more circumscribed validity effect, especially over the left hemisphere. In fact, group differences were not significant over the right hemisphere.

As already stated, the presence of validity effects and the group differences in those effects do not constitute the only consideration in the analysis of these data. It is also important to establish the point in the processing of the target at which differences across valid, invalid, and neutral conditions suggest that the subject has discriminated the target location and is deciding the appropriateness of a response. A contrast of P1-N1 amplitude between the invalid and neutral conditions was conducted to shed light on this matter. The analysis revealed significant differences between the two conditions at all posterior sites (Invalid-Neutral Contrast; Occipital: $F(1,15)=18.86$, $p=.0006$; Parietal: $F(1,15)=6.85$, $p=.0194$; Temporal Posterior: $F(1,15)=19.93$, $p=.0005$). P1-N1 amplitude was of comparable magnitude for the two groups at all sites (Invalid-Neutral Contrast x

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Figure 9. Bars depict differences in amplitude between the P1-N1 response

to the valid and the invalid targets, recorded over the left and right hemispheres.

Hatched bars correspond to poor readers, and clear bars to good readers. Note

that the P1-N1 responses were larger for the lower-reading group, especially over

the left hemisphere. Good readers, on the other hand, showed greater differences

in amplitude across electrode locations.

Fig. 9

Left Hemisphere

Right Hemisphere

P1-N1

Poor Readers

Good Readers

Occipital Parietal Temporal
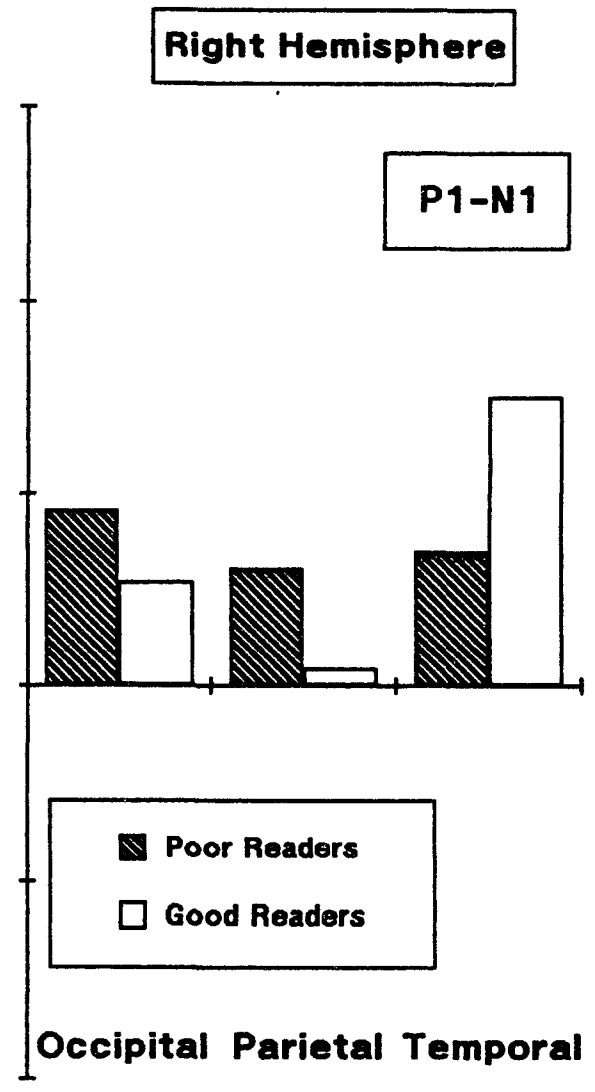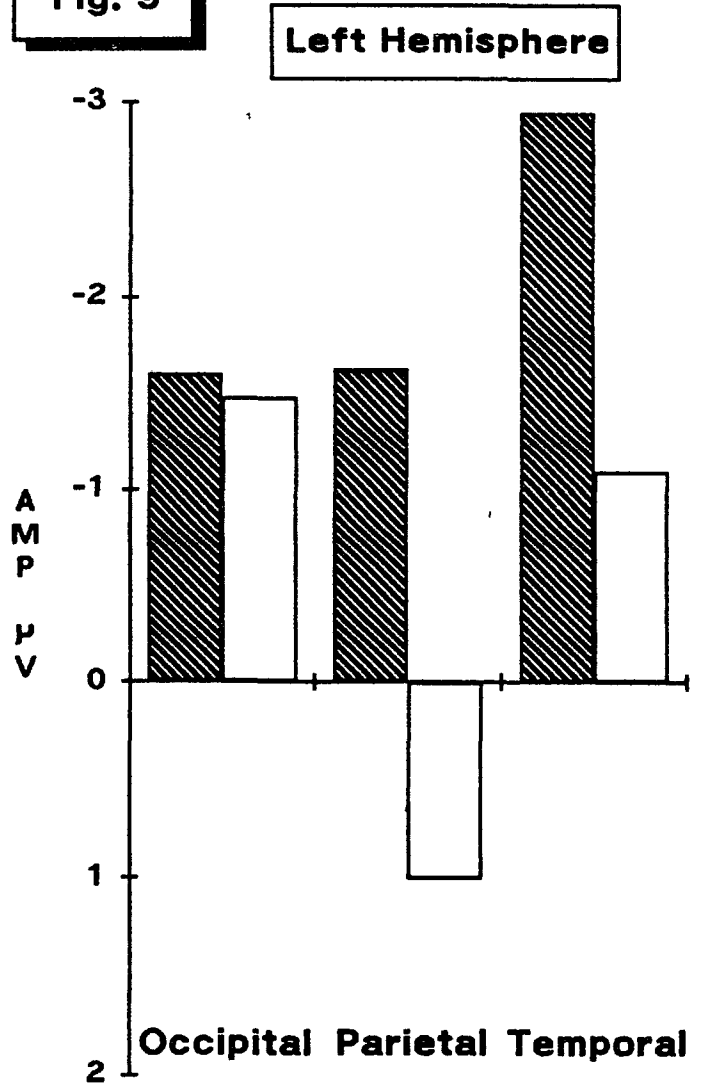
Occipital Parietal Temporal

Reading Level; Occipital: $F(1,15)=.10$, $p=.76$; Parietal: $F(1,15)=1.43$, $p=.25$; Temporal Posterior: $F(1,15)=.00, p=.95$). Thus, differences between the valid and invalid conditions indicated that discrimination of the target's location was taking place during this time interval (100-200 ms post-target). In contrast, the fact that invalid and neutral conditions did not resemble each other suggested that the decision to respond had not yet started for either reading group.

N2. This negative deflection was quantified as the peak amplitude of the waveform between 140-220 ms following the presentation of the target. The initial statistical analysis included the N2 response under the valid and invalid cueing conditions. Targets presented in the invalid hemifield evoked significantly larger negative deflections than validly cued targets (Cue Validity, $F(1,15)=31.69$, $p=.0000$). The most prominent activation of N2 amplitude due to validity was over the parietal and central areas of the hemisphere contralateral to the field where the target had appeared (Electrode x Hemisphere x Cue Validity x Visual Field, $F(6,96)=4.32, p=.0007$).

In this latency range, the morphology of the waveform showed a sharp turn of the valid condition from negative to positive polarity (see Figures A.9 and A.10 in Appendix A). In contrast, the invalid response became increasingly negative, deviating from the valid, and lagging the neutral condition. The difference in polarity between valid and invalid conditions is also present in Figure 10, where the bars representing the valid condition tend to have positive values, while most negative values correspond to the invalid condition.

Poor and normal readers differed in the N2 response to the target as a function of the preceding cue (Cue Validity x Reading Level, $F(1,15)=8.5$, $p=.0049$). In Figure 10, it is evident that poor readers showed smaller validity

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Figure 10. Valid (hatched) and invalid (clear) N200-responses evoked by right (RVF, top row) and left (LVF, bottom row) visual-field targets. Poor readers are depicted on the left column, while good readers are found in the right column. Notice how the valid and invalid responses tend to be of opposing polarity, particularly in the good readers. Electrode locations vary along the abscissa: occipital (O), parietal (P), central (C), frontal (F), temporal posterior (T), temporal anterior (TA), and frontal lateral (FL).
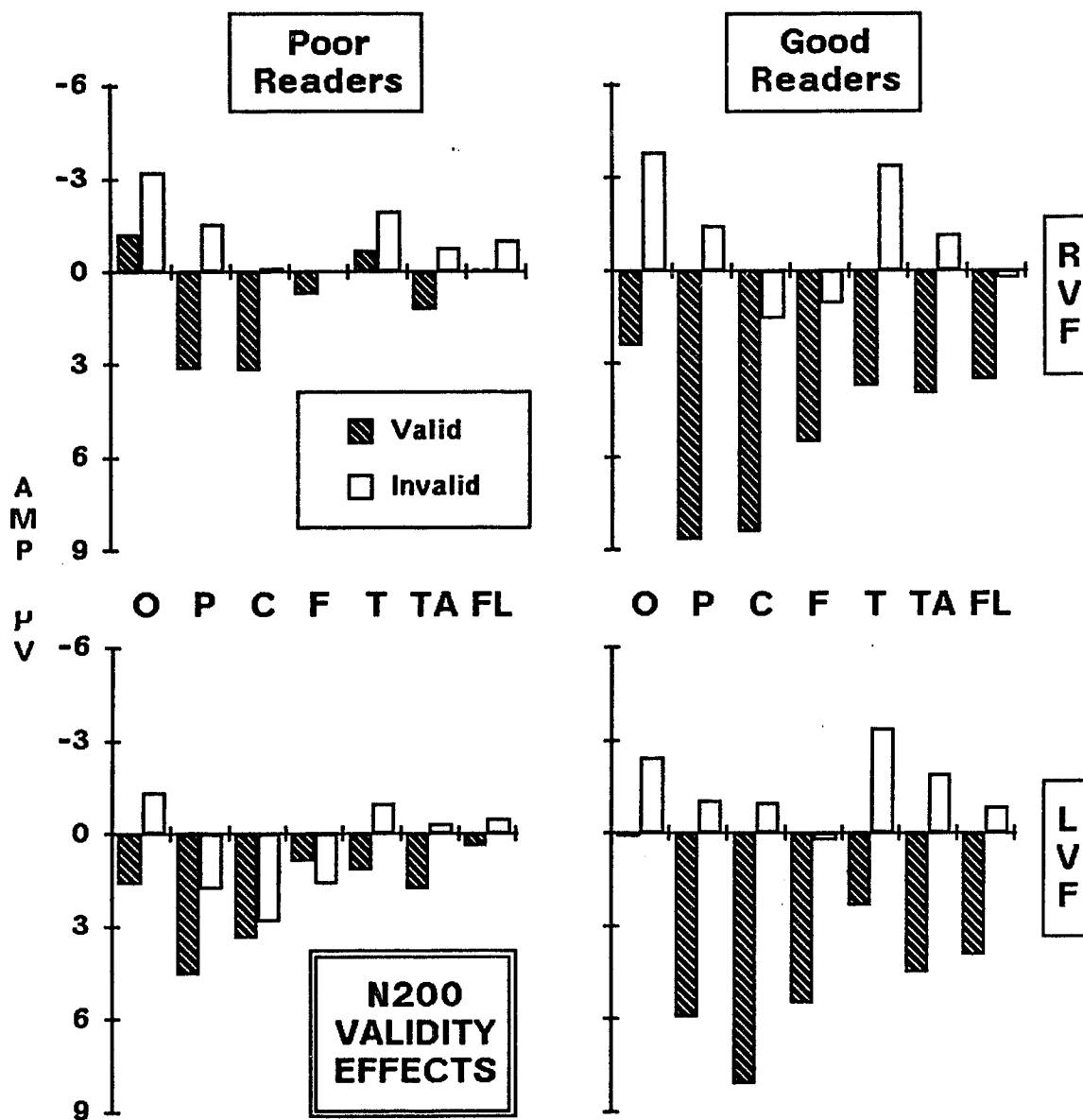
FIGURE 10

effects than normal readers. This trend was accentuated when responding to the left visual field targets. The N2 responses of normal readers had opposite polarities in response to valid and invalid target presentations, while poor readers only showed this reversal pattern in response to targets appearing in the right, but not the left, hemifield (Electrode x Cue Validity x Visual Field x Reading Level, $F(6,96) = 3.16, p = .0349$).

As in the case of P1 and N1 above, peak-to-baseline N2 values did not reveal the more interesting patterns of change of the invalid condition relative to the valid and neutral conditions. A P2-N2 peak-to-trough measure, including the neutral condition, was needed to detect the pertinent transient changes. P2-N2 was analyzed separately for the posterior and anterior electrodes, in keeping with prior reports on the two foci of this potential (Simson et al., 1977), and the latency differences observed between the front and the back of the scalp (see below). In accord with the N2 peak measure, P2-N2 validity effects were highly significant both at posterior $(F(2,32) = 15.69, p = .0000)$ and anterior electrodes $(F(2,32) = 20.17, p = .0000)$. Over the posterior half of the scalp, the modulation of P2-N2 amplitude due to cue validity did not differ between poor and normal readers (Cue Validity x Reading Level, $F(2,32) = 1.35, p = .27$). For both reading levels, P2-N2 was significantly smaller and more positive in response to the valid condition, and the invalid and neutral conditions were of negative polarity and comparable in amplitude. The pattern changed over the anterior electrode sites, where validity effects were not the same for the two reading groups (Cue Validity x Reading Level, $F(2,32) = 4.42, p = .0202$). These results are depicted in Figure 11, where the hatched bars represent average P2-N2 amplitudes for each of the three cueing conditions over the posterior electrodes. At these locations the valid

Fig. 11

Poor Readers

Good Readers

P2-N2

Posterior
Anterior

AMP

μV
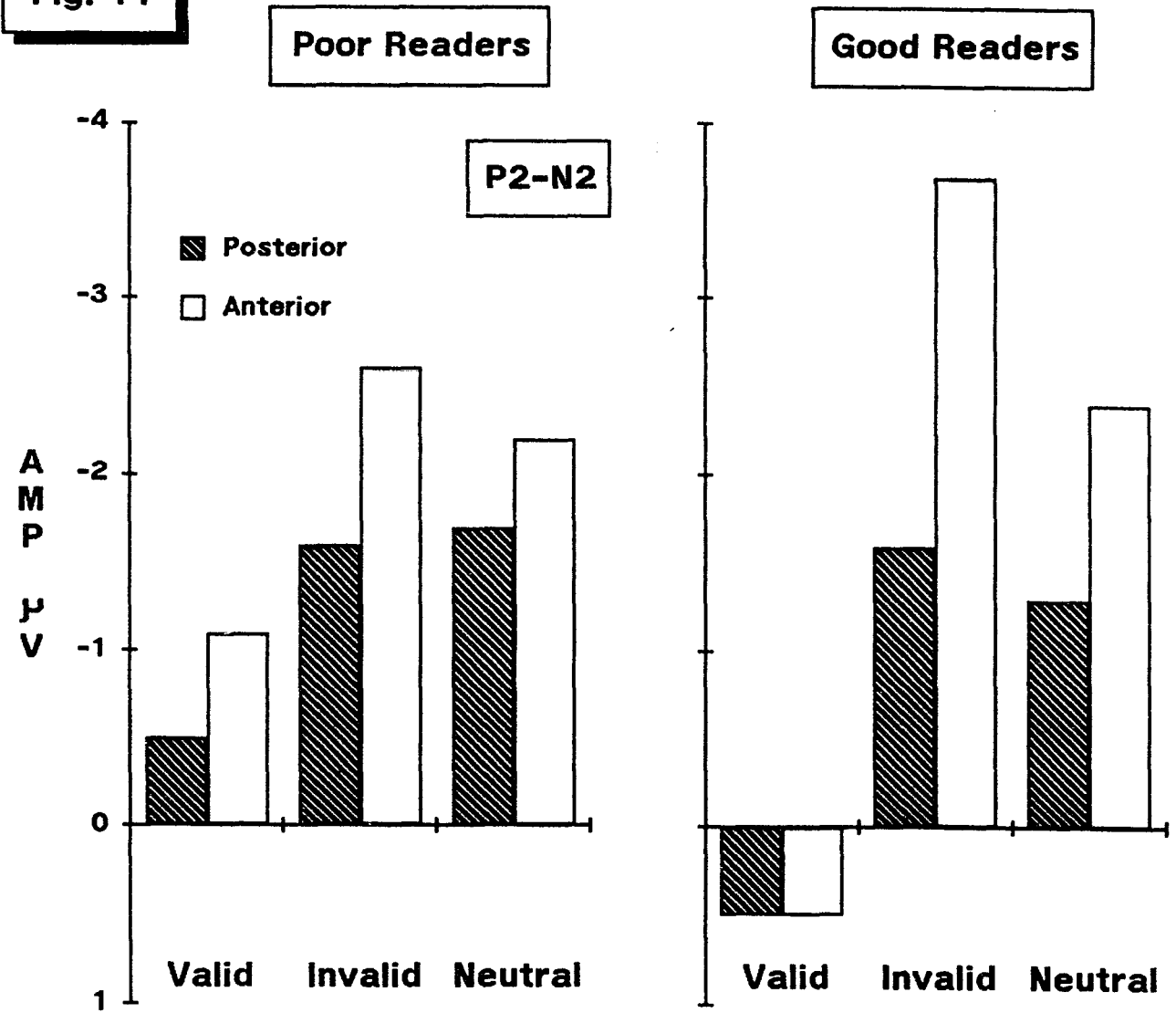
condition deviated in polarity from both invalid and neutral conditions, which did not differ significantly from each other. Over the anterior half of the scalp, however, the invalid condition was enhanced with respect to the neutral condition. But only normal readers showed such enhancement, as well as the difference in polarity between the valid and the other two conditions (Figure 11).

Late Positive Deflection. N2 was followed by the initiation of a marked and long-lasting positivity. As stated above, the valid condition had already turned positive in polarity, but it was not until approximately 260-280 ms beyond the target's onset that potentials evoked by the other two cueing conditions started becoming positive as well (see Figure A.8 in Appendix A). This large positive deflection, present through the 240-440 ms range, is probably a P3-like potential, given its latency and scalp distribution (Squires, Squires & Hillyard, 1975). Since it likely encompasses more than one source of brain activity, it was labelled *late positive deflection* (LPD).

LPD was quantified as the average amplitude of the waveform in two latency ranges: 280-360 ms (LPD1), and 360-440 ms (LPD2). In keeping with the analyses reported above, the valid and invalid conditions were tested first, then the three cueing conditions were included. The contribution of reading level to the results was assessed in both cases.

Figure 12 illustrates how the amplitude enhancement of LPD1 due to validity was highly significant, regardless of reading level (Cue Validity, $F(1,15) = 14.86$, $p = .0016$). Differences between validly and invalidly cued targets were largest over the parietal and central areas (Electrode x Cue Validity, $F(6,96) = 11.4$, $p = .0000$) of the left hemisphere (Electrode x Hemisphere x Cue Validity, $F(6,96) = 7.20$, $p = .0002$).

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Figure 12. Average group LPD1-potentials obtained in response to valid (clear) and invalid (hatched) target presentations in the right (RVF, top row) and left (LVF, bottom row) visual fields. The greatest validity effects are observed over the parietal and central areas. Electrode labels, on the abscissa, are the same as in previous figures.

Figure 12
LPD1
ALL SUBJECTS
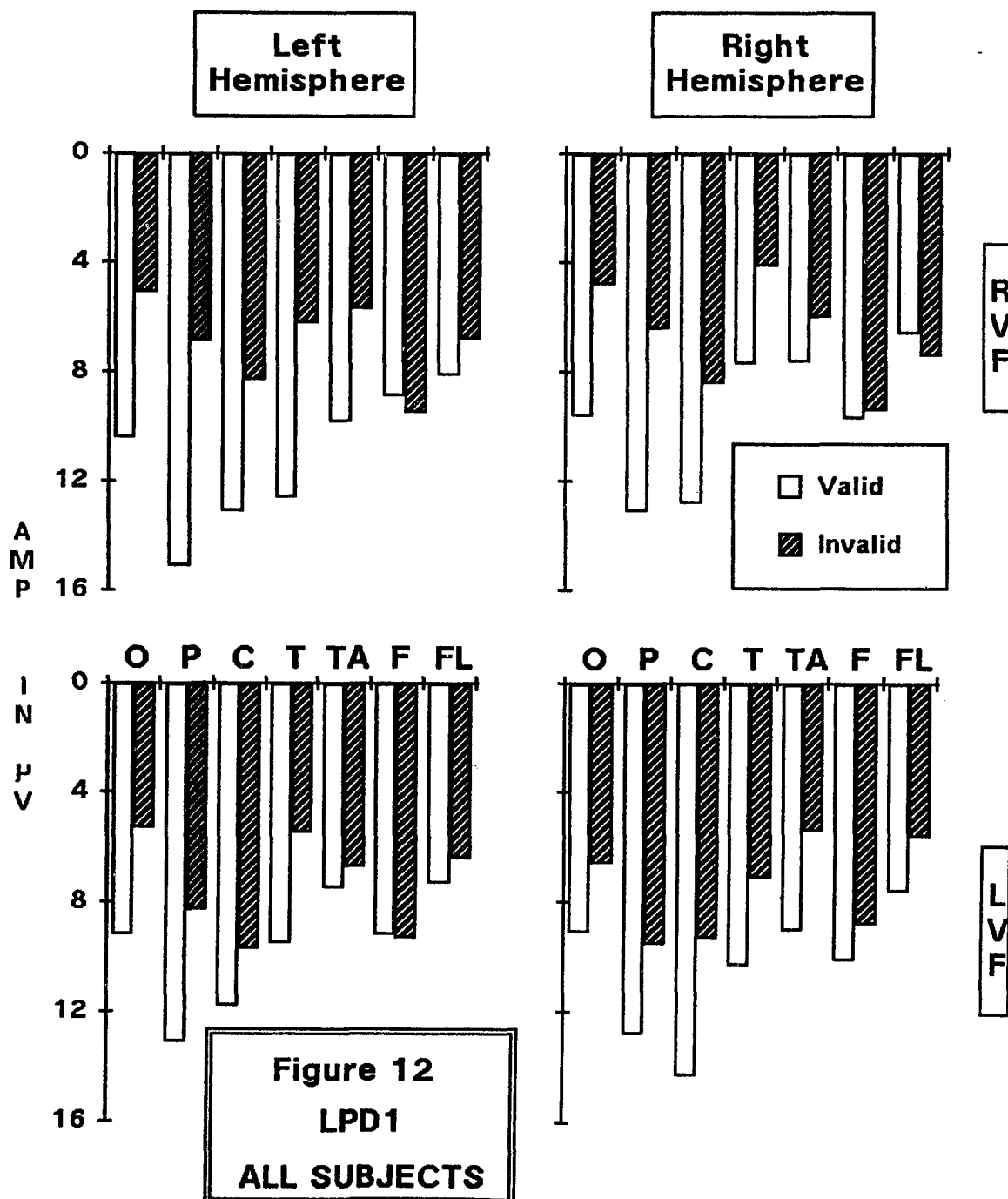
The increase in LPD1 as a function of validity interacted in a rather complex fashion with other factors. Several trends are particularly worth mentioning. Once again, poor readers exhibited validity effects that were smaller than those of normal readers. This trend, though present over most of the scalp, only reached significance at the central (Cue Validity x Reading Level, $F(1,15)=5.39$, $p=.0348$), frontal ($F(1,15)=4.68$, $p=.0471$), and anterior temporal ($F(1,15)=5.4$, $p=.0346$) electrodes, and was due to differences in the valid, and not the invalid, LPD1 response. Interestingly, group differences in LPD1 validity were similar to those of N2, in that they were present or heightened for the left visual field rather than the right visual field targets (Electrode x Cue Validity x Visual Field x Reading Level, $F(6,96)=3.58$, $p=.017$). The bars in Figure 13 represent differences between the valid and invalid conditions for poor and normal readers. The graphs on the left column illustrate the validity effect obtained over the left hemisphere for right (upper panel) and left (lower panel) visual field targets; the right-hand column reflects analogous validity effects for the right hemisphere. At the central and temporal anterior electrodes, the LPD1 validity enhancements of normal readers were greater over the hemisphere contralateral to the visual field of the target. Poor readers, while disclosing comparable validity effects for right visual field targets, differed from the normal subjects in showing diminished or absent LPD1 validity modulations for left visual field targets, especially over the left hemisphere (Hemisphere x Cue Validity x Visual Field x Reading Level, Central: $F(1,15)=8.31$, $p=.0114$; Temporal Anterior: $F(1,15)=8.41$, $p=.0110$). The same trend was present at other electrode sites, although without reaching significance.

Figure 13. Bars represent the magnitude of the validity effect (valid - invalid LPD1) for poor (clear) and good (hatched) readers over the parietal (Par), central (Cen), temporal anterior (TA), and frontal (Fro) locations.

Figure 13

LPD1
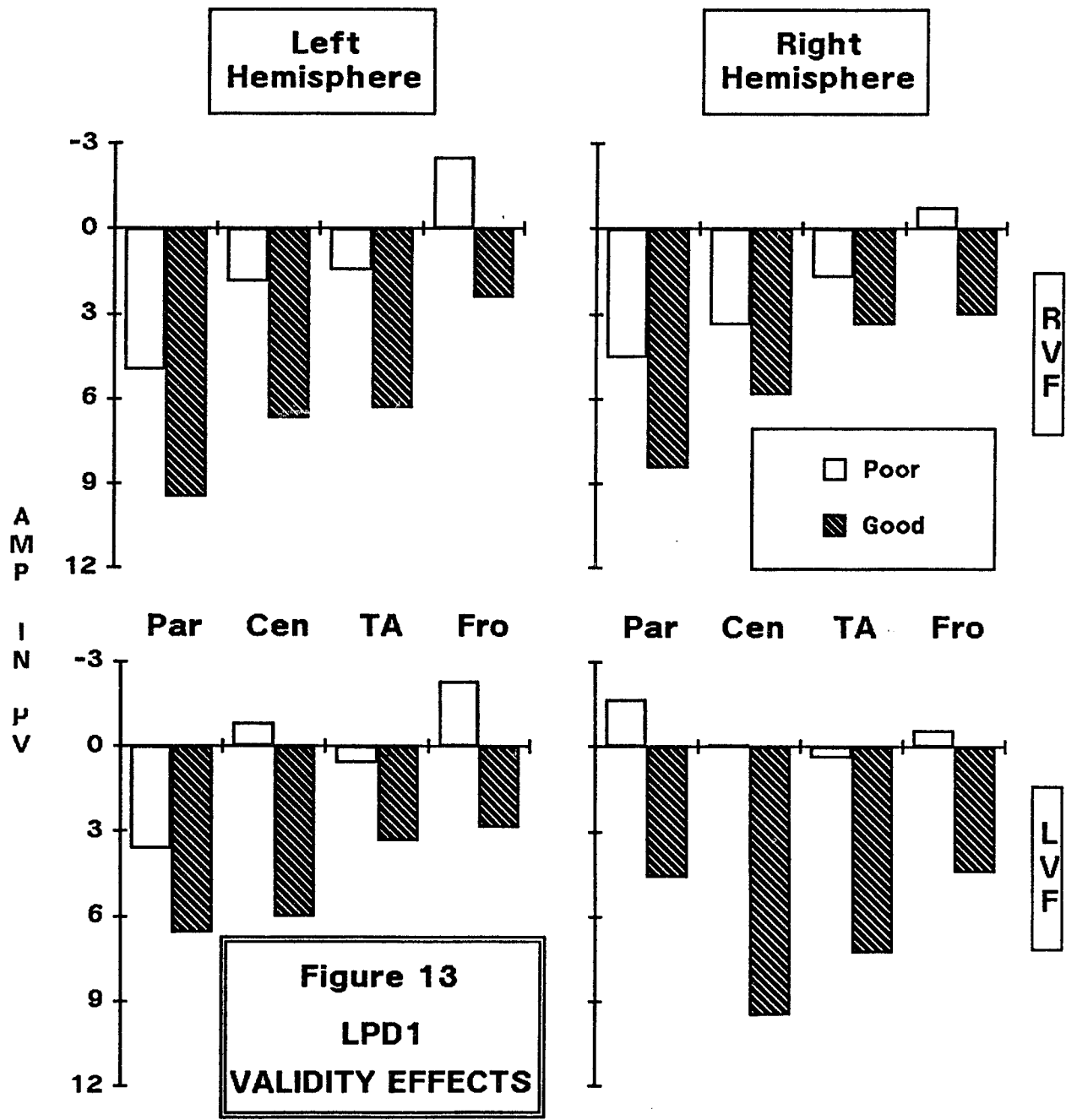
VALIDITY EFFECTS

As mentioned above, the second measure of this late positivity, LPD2, represents the average amplitude of the waveform between 360-440 ms after target onset. Unlike LPD1, LPD2 validity failed to reach significance as a main effect (Cue Validity, $F(1,15)=3.68$, $p=.0742$), probably due its circumscribed scalp distribution. LPD2 amplitude was enhanced as a function of cue validity exclusively at parietal and central electrodes (Electrode x Cue Validity, $F(6,96)=8.09$, $p=.0001$). Figure 14 illustrates how differences in amplitude between validly and invalidly cued targets were predominant over the left hemisphere (Hemisphere x Cue Validity, $F(1,15)=17.55$, $p=.0008$), where the distribution of the validity effect was broader, and extended to the occipital and temporal posterior areas (Electrode x Hemisphere x Cue Validity, $F(6,96)=3.89$, $p=.0016$).

Figure 15 shows how the over-all enhancement of LPD2 amplitude due to validity varied significantly between normal and poor readers, especially over the central electrodes (Electrode x Cue Validity x Reading Level, $F(6,96)=4.35$, $p=.0062$). Normal readers exhibited more pronounced validity effects than poor readers. Particularly interesting was the fact that, once again, the validity enhancement had a different visual field distribution for each reading group. Normal readers showed validity effects that were greater, or only significant, in response to right visual field targets. In contrast, poor readers displayed a marked left visual field predominance of the LPD2 validity enhancement. This group difference was only present or insinuated over the posterior electrode locations (Cue Validity x Visual Field x Reading Level; Occipital: $F(1,15)=6.66$, $p=.0209$; Parietal: $F(1,15)=3.97$, $p=.0649$; Temporal Posterior, $F(1,15)=8.2$, $p=.0118$).

Figure 14. Average LPD2-response evoked by valid (clear) and invalid (hatched) targets in response to right (RVF) and left (LVF) visual-field stimuli. Note the greater validity effect obtained over the left hemisphere (left-hand column).

Figure 14
LPD2
ALL SUBJECTS

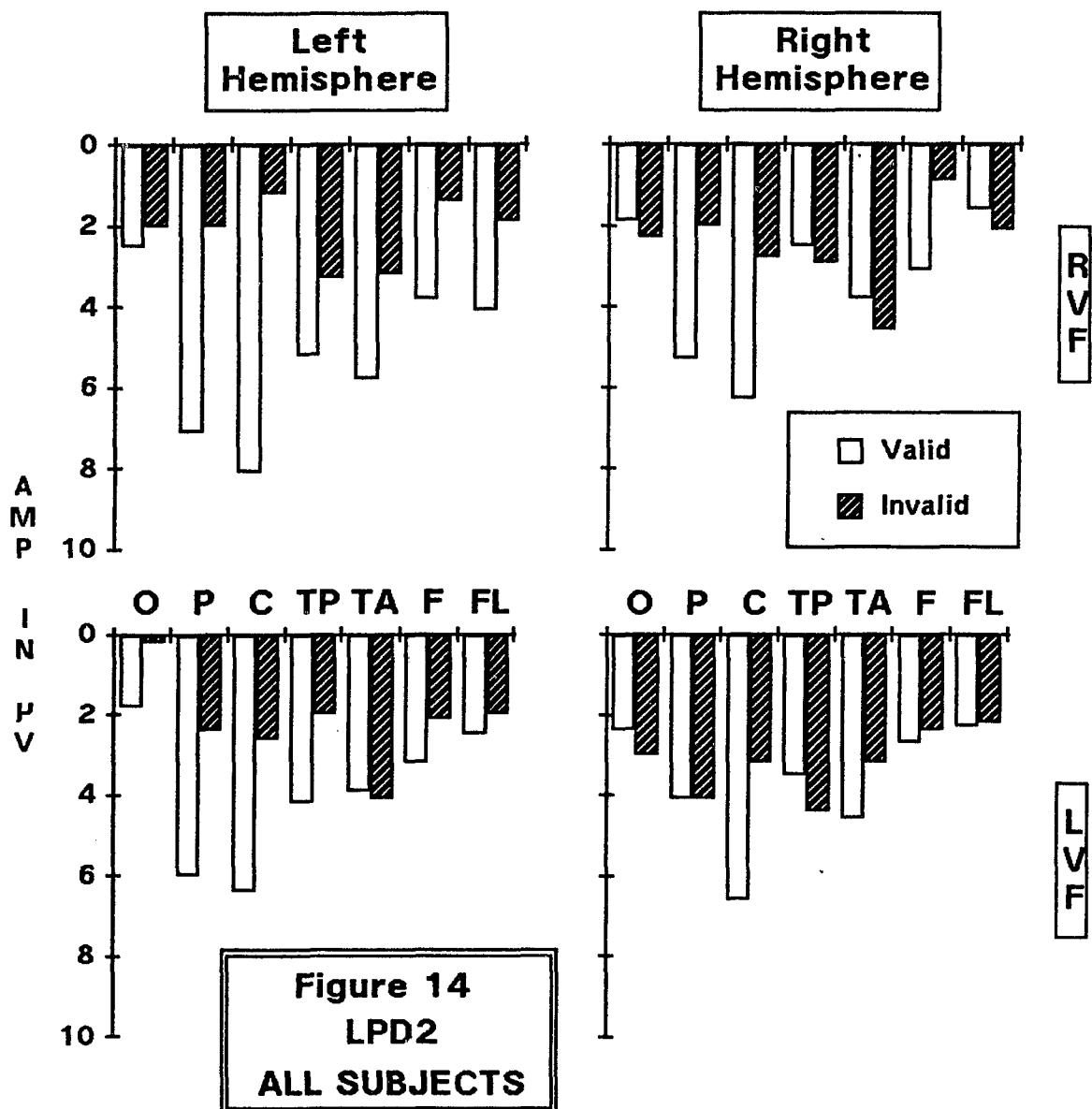*Figure 15.* Differences in the magnitude of the LPD2 validity-effect between poor (clear) and good (hatched) readers, obtained over the occipital (Occ), parietal (Par), central (Cen), and posterior temporal (Tem) electrode sites. Note that the good readers showed greater LPD2 validity-effects in response to right visual field (RVF) targets, while the opposite trend was observed for the poor readers.

Figure 15

LPD2
VALIDITY EFFECTS

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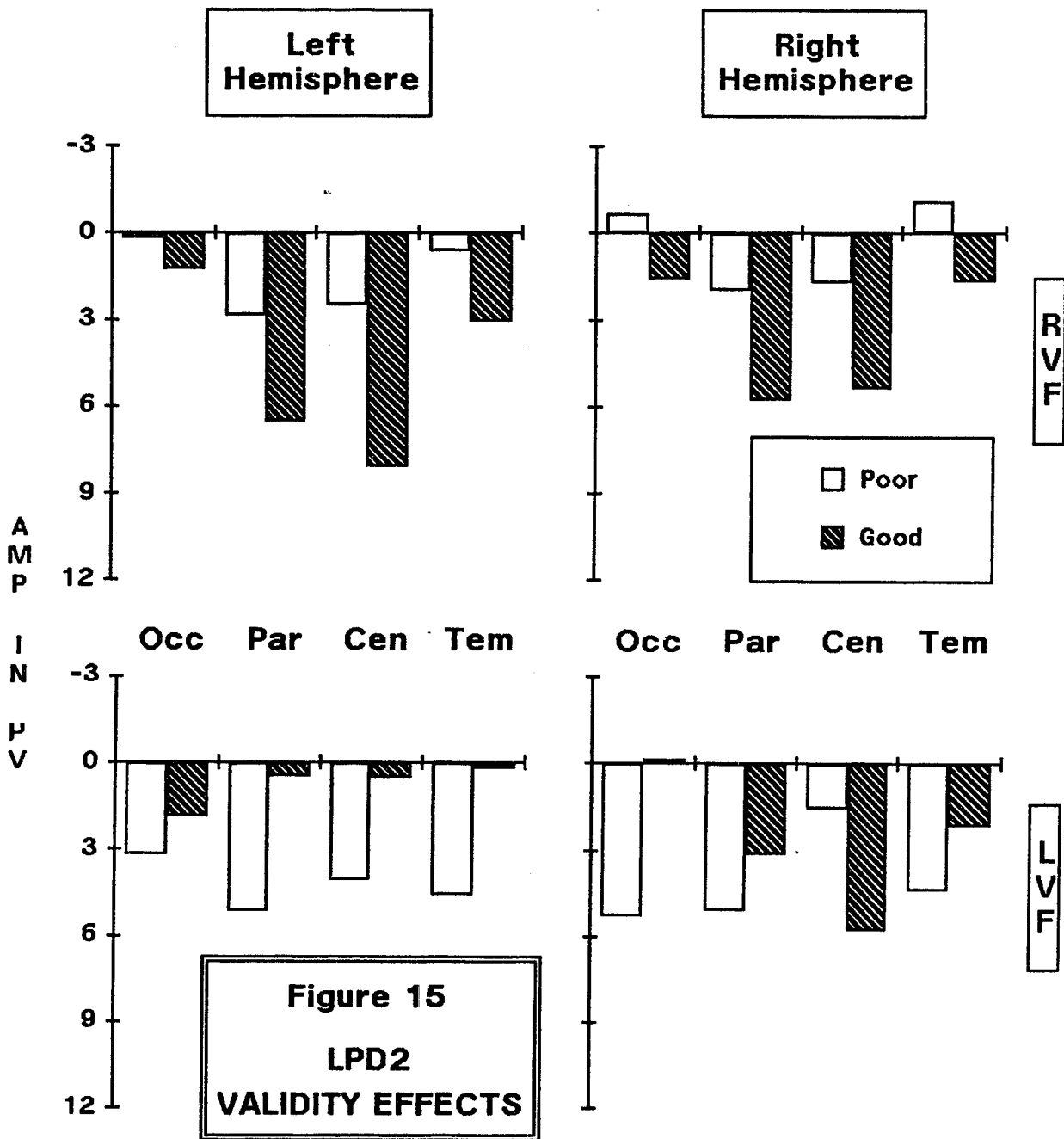
Latency Measures

So far, data analyses have concentrated on the effect of cue directionality or validity on the *amplitude* of the ERP. But it is also important to determine whether such factors affected the *speed* of information processing by measuring the latencies of the deflections presented above.

For reasons stated in the Introduction, this task only required a timed response to validly cued stimuli, so it was not possible to compute the reaction time benefit due to valid cueing which characterizes behavioral studies . A comparable analysis, however, was conducted on the latency of the ERP deflections that were sensitive to cue directionality or validity. Such analyses provide an estimate of the brain's ongoing temporal processing of directional and non-directional cues, on one hand, and validly, invalidly and neutrally cued targets, on the other.

The statistical model used to this end was similar to those already described in that it incorporated reading ability as a between-subject factor, though without the EOG measure as a covariate. ANOVAs were followed by contrasts between means in order to determine which levels of cue directionality (in the case of cue-related potentials) or validity (in the case of target-related potentials) were the source of significant latency differences for each deflection.

In examining latency results, it should be kept in mind that the sampling rate used here was rather slow (1 data point/20 ms), and probably affected selectively the signal-to-noise ratio of the early, transient deflections. In this light, it was not surprising to find no significant differences in latency between directional and non-directional cues for the **cue-related potentials C-P120 and C-N240.**

The peak latency of the CRP was computed within the 320-480 ms range previously used to calculate its average amplitude. Since only the posterior electrodes showed significant cue effects (see above), they alone were included in the analysis. In poor readers, the CRP peak latency was shorter over the right (405 ms) than over the left hemisphere (417 ms), while the opposite was true for normal readers (right hemisphere: 423 ms, left hemisphere: 413 ms) (Hemisphere x Reading Level, $F(1,16)=4.67$, $p=.0462$). This pattern of hemispheric responses interacted with the directionality of the cue: normal readers displayed faster latencies for directional than non-directional cues, but only over the left parietal area; poor readers, on the other hand, showed a directional advantage only over the right parietal region (Electrode x Hemisphere x Cue Type x Reading Level, $F(4,64)=3.18$, $p=.0192$).

The peak latency of the CNV, measured in the 480-660 ms interval, did not reveal any significant latency effects. Like the previous CRP, the latency of the CNV was only measured over the posterior half of the scalp. The lack of significant results may be related to the slow frequency of this cortical potential, which makes peak latency difficult to measure.

The validity of the cue affected the latency of all potentials evoked by the target. Latency decreased as a function of valid cueing starting about 120 ms after the presentation of the target; the peak amplitude of P1 was delayed when the target was invalidly cued in comparison to when it was either validly or neutrally cued (Cue Validity, $F(2,32)=5.43$, $p=.0093$). No differences were observed between P1 latencies to validly and neutrally cued targets. Changes on P1 latency due to validity were present only over the parietal and temporal posterior sites (Electrode x Cue Validity, $F(4,64)=3.31$, $p=.0159$). They also showed a reversed

hemispheric pattern across reading levels; validity effects were present over the right hemisphere in normal readers, and over the left hemisphere in poor readers (Hemisphere x Cue Validity x Reading Level, $F(2,32) = 4.04, p = .0296$).

N1 latency was modulated by the validity of the cue as well, though not in the same fashion as P1. N1 peak amplitudes also were fastest in response to validly cued targets, but this time neutrally cued targets were processed as slowly as the invalidly, rather than the validly, cued targets (Cue Validity, $F(2,32) = 7.10$, $p = .0028$). In contrast with P1 latency, validity effects on the latency of N1 did not differ across hemispheres, electrode locations or reading levels.

N2 latencies were significantly slower over the posterior regions of the scalp (293 ms) than over the anterior areas (264 ms) (Electrode, $F(6,96) = 6.58$, $p = .0006$). By the time N2 peaked, its latency already had become shorter for invalidly than neutrally cued targets, but exclusively over frontal and lateral frontal sites (Electrode x Cue Validity, $F(12,192) = 5.36$, $p = .0000$). Latency differences between validly and invalidly cued targets were not taken into consideration, since measurement of the N2 peak was hindered by the beginning of the LPD deflection (see Figures A.1 and A.2 in Appendix A).

The peak latency of LPD was measured between 240 and 420 ms following target presentation, thus encompassing the LPD1 and LPD2 ranges. Within this interval, LPD latency was always significantly shorter for validly cued targets (343 ms) than either invalidly (372 ms) or neutrally (373 ms) cued stimuli (Cue Validity, $F(2,32) = 12.58, p = .0001$) for both poor and normal readers. The effect of validity on latency was widely distributed over the scalp, and larger over the right than over the left hemisphere (Hemisphere x Cue Validity, $F(2,32) = 6.99$, $p = .0046$). Although there were no over-all latency differences between invalid and neutral

latencies, invalidly cued targets were associated with shorter latencies than neutrally cued stimuli over the occipital, parietal, and temporal areas of the head (Electrode x Cue Validity, $F(12,192) = 5.58, p = .0004$).

From the results reported in this section, it is clear that validly cued stimuli were processed faster than either invalidly or neutrally cued targets, regardless of reading level. In general, differences in latency between the two reading groups seemed to be characterized by reversals in the hemispheric lateralization of the effect.

Behavioral Responses, Reading, and ERPs

As stated in the Method section, subjects were required to respond to all validly cued targets with a timed finger-lift response. Reaction times (RTs), and the percentages of hits and false alarms were recorded separately for each experimental condition (see Table 2).

Signal detection measures of sensitivity (d') and response criterion ( ) were calculated as well (Swets, 1964). Faster responses were associated with higher sensitivity values ($r = -.78, p = .0001$), and unrelated to the strictness of the subject's criterion ($r = .12, p = .62$).

Reaction times for the two visual fields were very highly correlated ($r = .94$, $p = .0001$). Indeed, a glance at Table 2 reveals that the average RTs to right and left visual field targets were identical for both poor (389 ms) and good (399 ms) readers. Since a similar visual-field concordance was observed for the other behavioral indicators as well, the response averaged across visual fields was used to analyze the relationship among task performance, reading, and ERP measures.

Table 2

Means and standard deviations (in parenthesis)
for behavioral measures by reading level

		Good Readers	Poor Readers
Behavioral Measure			
Reaction times (ms)			
	RVF	399.2 (60)	389.4 (53)
	LVF	399.5 (58)	389.1 (58)
Hits (%)			
	RVF	97.2 (4)	95.8 (5)
	LVF	97.1 (3.5)	94.9 (5)
False Alarms (%)			
Invalid Targets	RVF	0.9 (1.8)	0.7 (0.9)
	LVF	1.3 (2.1)	1.0 (1.7)
Neutral Targets	RVF	1.6 (1.2)	0.6 (1.0)
	LVF	1.7 (2.6)	1.7 (1.4)
Sensitivity (d')			
Invalid Targets	RVF	4.8 (1.0)	4.4 (0.7)
	LVF	4.5 (0.9)	4.3 (0.8)
Neutral Targets	RVF	4.5 (0.8)	4.5 (0.8)
	LVF	4.5 (0.9)	4.1 (0.7)
Response Criterion ( )			
Invalid Targets	RVF	2.9 (2.5)	7.6 (10)
	LVF	3.4 (3.8)	6.1 (7.9)
Neutral Targets	RVF	1.8 (1.9)	5.5 (7.5)
	LVF	3.4 (3.9)	4.6 (7.3)

Note: RVF = right visual field; LVF = left visual field

Table 2 includes means and standard deviations for each measure according to reading level and visual field. Note that performance was almost flawless, probably heightened by experience with a similar task earlier in the session. The resulting ceiling effect, as well as the limited number of subjects inherent in the use of two reading groups, could account for the small behavioral differences observed between poor and good readers. Still, the relationship between task performance, brain potentials evoked by the task, and reading ability remains the question of interest in this study.

The effect of visual field and reading level on behavioral measures of response speed and accuracy were assessed by means of univariate ANOVAs. Not surprisingly, given the means reported in Table 2, no visual-field effect was found for any of the behavioral measures (all ANOVAs, $F(1,16) < 1$, $p > .05$).

Table 2 also reveals a counter-intuitive result: on average, poor readers were actually faster in the performance of the task than good readers. But they were also less accurate, as measured by their percentage of hits. The only other noticeable behavioral trend was the poor readers' higher average response-criterion, which suggests that they were more cautious in their responses. The ANOVAs showed that none of these between-group differences were significant.

The fact that behavioral responses in this task did not vary significantly across reading groups is disturbing. Several studies have found good readers to be faster and more accurate than poor readers in visual tasks not involving a spatial component (Solman & May, 1990; Williams et al., 1990). On the other hand, Harter (1991) has reported significantly faster RTs for poor readers in a visual-spatial paradigm similar to the one used here. It was anticipated, therefore, that the behavioral responses in this task would provide an observable basis upon which

to connect both reading and visual-spatial attention to a common physiological system.

Behavior did not seem to vary as a function of reading level, at least according to the ANOVAs reported above. However, since behavioral measures are continuous and any "group" cut-off applied to them would be arbitrary, the connection between behavior, ERPs and reading was investigated next with Pearson correlations. Indeed, there was some evidence of a connection between brain activity and behavior. Right hemisphere differences in P1-N1 amplitude due to target validity were inversely related to values for the invalid ($r = -.51$, $p = .031$) and, less so, for the neutral ($r = -.48$, $p = .04$) condition. Since P1-N1 is a negative potential, and its magnitude increases are reflected in greater negative values, the negative sign of the correlation indicates that larger validity effects were associated with less strict criteria. In terms of latency, neither P1 nor N1 peak latency effects were correlated with behavioral measures. Over the frontal region, however, the difference in peak latency between valid and invalid N2 potentials was related to the subject's sensitivity as measured by d' ($r = .64$, $p = .004$). Larger latency differences between valid and invalid conditions were associated with greater sensitivity.

Still, it is not surprising that post-target potentials are correlated with post-target behavior. The effect of pre-target shifts in attention on subsequent performance needs to be assessed as well. Thus, correlation coefficients were also calculated between cue-related potentials and behavioral measures. Unfortunately, none were significant. It would appear, therefore, that the connection between reading and brain activity reported above cannot be extended to observable behavior, at least not to the kinds measured in this study.

Recall that reading was a significant explanatory variable in the analysis of cue-related, pre-target potentials. The correlation between reading and pre-target ERP measures, therefore, may be confounding the statistical relationship between each and the behavioral measures. To examine this possibility, partial correlation coefficients were computed for two groups of variables. The first group included RT, the amplitude difference between the two arrow directions in the CRP range (CRP_d), Woodcock-Johnson reading scores, and EOG; the second, RT, CNV amplitude differences due to arrow direction (CNV_d), Woodcock-Johnson reading scores, and EOG. Both calculations revealed significant relationships (all p-values .01 or smaller) between reading and RT, as well as between each measure of brain activity and RT. The interaction between reading and brain activity, therefore, appears to have obscured the impact of each variable on behavior when examined in isolation.

The signs of the correlation coefficients revealed a complex, but interesting, pattern. Holding CNV_d or CRP_d constant, higher reading ability was associated with faster RTs. But it is important to remember, as emphasized earlier, that pre-target potentials do not stay constant as reading ability changes. In fact, the Pearson correlation coefficients between reading scores and CNV_d at the parietal electrodes were $r = .58$ ($p = .01$) for the left hemisphere, and $r = .60$ ($p = .008$) for the right hemisphere. The pattern was similar, although weaker, for the parietal CRP_d ($r = .40, p = .10$ for the left, and $r = .53, p = .03$ for the right hemisphere).

Together, partial and Pearson coefficients indicate that common variation in reading ability and amplitude differences of CRP and CNV due to arrow direction are associated with more rapid responses. This suggests that attentional shifts, as measured here, are associated with benefits in both reading and the task

examined. The relationship is more complicated, however, since there is evidence that subjects can "over-prepare" in response to directional cues. The analyses revealed negative partial correlations between RT, and both CRP_d and CNV_d ($p=.03$ and $p=.01$, respectively), indicating that increases in either of the pre-target arrow differences were actually associated with higher RTs once reading level was held constant. That is, subjects who displayed greater CRP_d or CNV_d than normally associated with their level of reading ability, performed the task more slowly than did those subjects with equal reading ability and less differential response to the arrow.

One final result needs to be reported. As expected, reading and IQ scores were highly correlated ($r=.64$, $p=.004$), and both independent of age (W-J and Age: $r=-.34$, $p=.16$; WISC-R and Age: $r=-.15$, $p=.56$). Consequently, all analyses reported above were conducted with IQ substituted for reading scores, since all the pertinent relationships could be due to general intelligence rather than reading ability. In all cases, similar trends were present, but statistically insignificant. Reading scores are not just replacing measures of general intelligence; reading ability seems to be associated with shifts in visual-spatial attention on its own.

CHAPTER IV

DISCUSSION

The results of this study will be interpreted within a simple model of attentional shifts, which provides a useful conceptual framework, and one that can be adapted and enriched as new findings become available. As we shall see, however, the evidence suggests a more complex explanation that will take further research to develop.

In applying the model to the paradigm used here, the first evidence of differential cue processing, as revealed by variations in brain activity, is taken to reflect the discrimination of all three cues. As the discrimination is completed, a shift of attention is initiated which is manifested in the form of an enhanced excitability of those areas of the brain encoding the cued location. Due to this increased excitability, a greater brain response is elicited by validly cued target stimuli than by stimuli appearing in the uncued hemifield.

The argument proceeds in four parts. The first two sections examine findings related to the visual-spatial task per se. The purpose is to attain a more precise characterization of: (1) the timing and location of the brain activity associated with a shift in attention, and (2) the impact that this selective preparation has on the brain's response to the subsequent target. In the third section the argument is extended to connect the ability to shift attention in visual space to the functional specialization of the where system in general, and the magnocellular stream in particular. Specifically, the magnocellular pathway is conceived as a critical determinant of our ability to shift attention in the absence of concomitant eye movements. The section ends by considering the differences in both brain and behavioral response patterns as a function of reading ability. The

fourth and last section discusses questions left unanswered, problems that should be addressed in the future, and the developmental implications of the findings.

The Effects of Spatial Cueing

One of the principal motivations of this investigation was to extend our understanding of the brain activity evoked by symbolic directional cues. Earlier studies have shown that the presentation of right and left arrow cues prior to the onset of a target results in differing hemispheric or topographical patterns of brain activity (Harter, 1991; Harter & Anllo-Vento, in press; Harter et al., 1989b). These patterns have been interpreted previously as reflecting shifts in attention, and they will be here as well.

The goal is to define more clearly the timing and pattern of ERPs that are associated with attentional shifts. To this end, two modifications of earlier paradigms were incorporated in the study. First, a non-directional circle-shaped stimulus was added to the right and left directional arrows to act as a "neutral" cue. The purpose was to establish a baseline for the brain activity evoked by the directional cues and subsequent target. Importantly, in the neutral condition the subject was instructed neither to shift attention, nor to respond to the target. The point was to isolate the portion of the ERP related to attentional shifts from that immediately preceding the presentation of a target which signals the contingency of a motor response. The second modification to the paradigm pertained to the control for the effects of eye movements, a vexing problem for any study of visual attentional shifts. Stricter eye-movement control was achieved in three ways: (1) the EOG was recorded separately from electrodes placed directly beside the right and left eyes; (2) a more conservative criterion was employed to exclude subjects

whose records displayed evidence of eye movements; and (3) an EOG measure was included as a covariate in all statistical analyses to control for any remaining electrophysiological activity that could be attributable to eye movements.

Most of the discussion of the experiment as a visual-spatial task concentrates on the group of subjects as a whole, without taking reading ability into consideration. It should be reemphasized that these subjects were tested four years after having been classified as at risk of developing a reading disability. The conclusions drawn here, therefore, must not be generalized casually to the general population. But this caution should not be overdrawn. Felton and Brown (1989, 1991) have reported that the measures used to classify these children as at-risk had little value in predicting subsequent reading outcome for the entire sample from which these subjects were drawn, once general intelligence was controlled. Moreover, the group examined here displays small differences in reading level and incidence of disability relative to the general population. Only two of the subjects (11%) met the criteria for the North Carolina's RD classification, a proportion within the normal range of incidence (Kolb & Whishaw, 1990). In addition, the group's Woodcock-Johnson reading scores, used here to measure reading level, did not differ significantly from the test's population distribution. Thus, while the special character of this sample must be kept in mind, it would be misleading to characterize the group as unusual in reading ability or incidence of disability.

To characterize brain activity associated with shifts in attention, brain potentials will be discussed in sequential order starting with the potentials evoked by the cue. As predicted, no significant amplitude or latency differences were found across cue types for C-P120. The result confirms one assumption of the design employed here, namely that minor physical differences among the three

cues would not elicit measurable variation in brain activity. More importantly, the result is consistent with the prediction that attentional shifts, as measured here, do not begin until at least 120 ms after presentation of the cue.

This conclusion needs to be connected to behavioral studies which report that performance-enhancing attentional shifts can be elicited by cues that are presented as little as 100 ms before the subsequent target (Posner, 1978, 1980). The two estimates, of course, are not contradictory. First of all, there is no disagreement that subjects begin sensory processing of the cue within the first 120 ms. In fact, the preceding conclusion regarding differences in the physical parameters of the cues rests implicitly on this assumption. At issue is when attention begins to shift in response to a cue.

Posner was interested in establishing the shortest interval between the cue and the target that allowed for a shift in attention to take place. He determined whether shifts in attention had occurred by comparing reaction times to validly and invalidly cued targets. But timed responses were recorded hundreds of milliseconds after the cue had been presented. Since attention can be directed by either sensory or stored visual cues, the shifts in attention detected by Posner could have occurred at any time between the presentation of the cue and the recorded response. The approach used here assumes that a shift in attention is associated with brain activity that can be measured instantaneously over the surface of the scalp. Obviously, this method permits more precise timing than a delayed motor response.

It could be argued, of course, that the timing of attentional shifts depends on the nature of the eliciting cue. It is certainly possible that the peripheral cues employed in most of Posner's studies (Posner, 1980; Posner et al., 1984, 1987)

prompted immediate, reflexive orienting responses that the symbolic central cues used here did not. In fact, peripheral and central cues result in temporal and structural differences in attentional allocation (Umiltà, Riggio, Dascola & Rizzolatti, in press). Also related to this question are recent experiments in split-brain patients (Holtzman, Volpe and Gazzaniga, 1984; Luck, Hillyard, Mangun & Gazzaniga, 1989) which indicate that two disconnected hemispheres can perform parallel visual search, but are unable to shift attentional focus independently in response to symbolic cues. Thus, peripheral and central cues could be processed differently, and involve distinct brain systems.

A second type of behavioral study, this time without cues, has also been interpreted as evidence that shifts in attention occur within 100 ms. Saarinen and Julesz (1991) asked subjects to identify a sequence of digits presented at different locations. They reported that subjects could recognize numeral order above chance level at interstimulus intervals as short as 33 ms. Because the digits were presented at different locations in the visual field, they concluded that attentional shifts could occur within this time window. Their results hinge critically on the assumption that performance so measured would be diminished if the subject did not shift attention. But in their design all digits appeared 1.5 from fixation, that is within the foveal region. It is certainly debatable whether degradations in the accuracy of stimulus recognition occur in the absence of shifts in attention within this area of the visual field. As a result, their conclusion about the timing of attentional shifts should be considered with some reservation.

The first evidence of brain differentiation across cues was observed at 240 ms. Significant differences in the amplitude of C-N240 were apparent over the posterior regions of the right hemisphere depending on whether the right or left

arrow had been presented. At the same time the average C-N240 amplitude evoked by the two directional cues was not significantly different from that elicited by the neutral cue. It appears, therefore, that subjects do not engage in a two-step discrimination process, where the directional cues are differentiated from the non-directional cue first, and then the particular arrow direction is distinguished. Instead, discrimination among all three cues seems to occur simultaneously.

The onset latency of C-N240 is roughly comparable to that of a previously reported *selection negativity*, presumed to index the selection of the relevant location of a target stimulus in adults (Harter & Aine, 1984; Harter et al., 1982). In this study, however, the initial evidence for the selection of the appropriate location **preceded** target presentation by about 350 ms. Moreover, this measure of selection was present even though all cues were presented at the same foveal position. In this sense, C-N240 could be interpreted as selection prompted by the symbolic character of the directional cue, rather than by its physical location.

Later in the pre-target interval, the evidence for attentional shifts was not consistent. Across all subjects there were no differences in brain activity between 320-480 ms that reflected processing of either directional versus non-directional cues, or right versus left arrows. Starting at 480 ms and until 100 ms after target onset, however, the CNV revealed significant differential processing of all cue types over the posterior regions of both hemispheres.

It would be senseless to argue on the basis of this evidence that attentional effects appear at 240 ms and disappear at 320 ms, only to reappear some 150 ms later. There is a simple explanation, but it relies critically on differences in brain activity between poor and good readers. These differences will be discussed in detail later, when reading ability is brought into the picture. For purposes of

exposition, however, I will simply remind the reader now that there were marked differences in the potentials of good and poor readers which started at the CRP (320-480 ms) and extended through the CNV range (480-660 ms). For the remainder of this section only the potentials evoked by the cue in good readers will be considered.

The CRP and CNV potentials of good readers suggest that a consistent and significant pattern of brain activity is associated with a directional cue prompting a shift in attention. Beginning at 320 ms and continuing through the CNV, the amplitude of the ERP evoked by the left arrow is relatively more positive than that elicited by the right arrow over the posterior regions of both hemispheres. Such a differential pattern of brain activity can only be associated with attentional shifts, because the physical characteristics of the cues and any preparation to respond are common to both arrows prior to the presentation of the target. Relative polarity differences found across cue potentials, however, cannot be interpreted too broadly. Less than a handful of studies have investigated the response of the brain to a visual-spatial cue, and it would be premature to reach a definitive conclusion.

Harter et al. (1989b) first characterized the brain potentials evoked by a directional cue in a study of eighty-six children between 6 and 9 years of age. There, the ERP evoked by a right arrow was subtracted from that evoked by a left arrow in order to isolate that part of the potential that could be related to an attentional shift in visual space. When this procedure was implemented, several slow waves were identified: a series of slow negativities which were larger over the contralateral hemisphere were followed by large positivities that also showed contralateral amplitude maxima. Subsequently, Harter and Anllo-Vento (in press) reported similar trends in young males. The present study, however, was the first

to include a non-directional cue which did not require a response. This new "baseline" provided a clearer delineation of the absolute polarity and latency range of the slow waves in question. The waveform obtained in response to the neutral cue revealed that a broad positivity with an onset latency of about 320 ms was followed by a broad negativity, beginning approximately 480 ms after the cue. This distinct pattern of brain responses led to a choice of measures slightly different from those employed before by Harter and his group (Harter, 1991; Harter & Anllo-Vento, in press; Harter et al., 1989b). Variations across studies in the resulting patterns of cue-related brain activity may be attributable to uncontrolled differences in the samples examined in these studies such as age, sex, hand of response, or handedness of the subjects. It should be emphasized, however, that all these studies found significant differences in brain activity as a function of arrow direction, and that the differences were consistently localized over the posterior half of the head.

Similar slow-potential distributions have been observed in a visual tracking paradigm by Deecke and his colleagues (Lang et al., 1984; Deecke et al., 1985). This group reported the presence of a slow negativity, named *directing attention potential* (DAP), which preceded the onset of tracking and continued for about 200 ms beyond the motor potential. The amplitude of the DAP was greatest over the parietal and occipital areas of the hemisphere contralateral to the visual field of stimulus presentation, rather than over the hemisphere contralateral to the hand of tracking. Consequently, the DAP was construed as a correlate of spatial attention, and not only of preparation to respond.

Unlike the difference between right and left arrow cues, amplitude modulations due to cue directionality were only present within the CNV range.

Starting approximately 500 ms after cue onset, the amplitude of the CNV evoked by either arrow was significantly larger than that elicited by the neutral cue. This difference was hemispherically symmetrical and widely distributed over the surface of the scalp. The non-directional cue informed the subject that no shift of attention or motor response were required in that trial. The presence of significant differences between directional and non-directional cues, in addition to the difference between right and left arrows, confirms that the neutral cue served as a signal to withhold a motor response, and not only as a signal not to shift attention.

To summarize, the present study provides the following characterization of brain events elicited during the cue-target interval: (1) for the group as a whole, differences among the three cues appear to be absent at C-P120, but by C-N240 the two arrow directions evoke significantly different amplitudes over the right posterior region, and (2) good readers show a significant difference across arrow directions that is established widely over the posterior region starting at 320 ms, and sustained through the CNV range. The findings in this paradigm suggest that attentional shifting most likely does not start until after 120 ms following the onset of the cue. By 240 ms the selection process has begun, and is first reflected as a differential response to arrow direction that is localized over the right hemisphere. The localization and transient nature of C-N240 suggest that subjects are selecting the cued location at this point in time, but have not shifted their attention yet. The actual shift of attention begins by 320 ms, and is sustained throughout the cue-target interval. The brain activity associated with the shift is observed over a major portion of the posterior region. The discussion of this interpretation and its

implications will be postponed until the findings associated with target validity are covered.

Target Validity

The evidence presented thus far supports the central assumption of the model on which this paradigm was based, namely that a directional cue would activate a shift of attention towards the relevant hemifield that would be reflected by differences in the brain's response to directional and non-directional cues, as well as between right and left arrows. The model further assumes that the processing of the subsequent target should vary depending on whether it appeared in the validly or invalidly cued location.

Targets that appeared at the valid location did, in fact, evoke larger potentials than targets that were invalidly cued. The differences in amplitude associated with target validity started 120-180 ms following target presentation, and continued to increase for the next 250 ms. Evidence of modulation of brain activity dependent on the validity of the target supports the interpretation that a spatially selective allocation of attention affects the neural processing of subsequent stimuli.

Earlier cueing studies in adult subjects have shown a consistent enhancement of the P1 and N1 deflections whenever the target appears in the cued visual field (Anllo-Vento & Harter, 1988; Harter & Anllo-Vento, in press; Harter et al., 1989a, 1989b; Mangun & Hillyard, 1988, 1990; Mangun, Hillyard & Luck, in press). The same effect has been reported in first-graders (Harter et al., 1989b) and young boys (Anllo-Vento et al., 1990; Harter et al., 1989a). In the present study the expected enhancement was only marginally significant. This

suggests either that other studies have overestimated P1-N1 target-validity effects, or that P1-N1 validity was reduced by the particular features of the current study.

Validity effects might be overestimated if eye movement is poorly controlled. If subjects move their eyes, even slightly, in the direction of the arrow, the subsequent target will fall closer to their fovea in the valid than in the invalid condition. Since stimulus eccentricity is a significant determinant of VEP amplitude, larger amplitudes being recorded in response to foveal than peripheral stimuli, eye movements would result in spurious validity effects. This possibility appears unlikely in view of the empirical data recently provided by Mangun and Hillyard (in press). They calibrated the magnitude of the average EOG evoked by saccades elicited by stimuli at different eccentricities, and concluded that a stimulus presented $0.36°$ to either side of the fixation point evoked an average EOG deflection in the 3.5-4.5 μV range. According to their results, and to the amplitude of the EOG records disclosed in previous studies (Harter & Anllo-Vento, in press; Harter et al., 1989b), undetected saccades would correspond to an average excursion of $0.18°$ under these recording conditions. Thus, eye movement seems an unlikely cause of decreased P1-N1 validity.

The alternative explanation to the marginal P1-N1 effects obtained here is related to the characteristics of the present study. As mentioned in the results section, the sampling rate was rather low (every 20 ms) and could have produced aliasing in the measurement of transient potentials such as P1 and N1. Although significant P1-N1 validity effects have been reported in studies that employed the same sampling rate (e.g., Harter et al., 1989a, 1989b), the number of subjects in those cases was much larger than in this one. In addition, attenuated P1-N1 validity could be due to the fact that here valid and invalid targets were

equiprobable, unlike in most studies of visual-spatial cueing (e.g., Anllo-Vento and Harter, 1988; Mangun & Hillyard, in press). Although it is possible that greater predictability of the target's location would yield greater validity effects, such effects have been obtained even when valid and invalid targets were equiprobable (Harter, 1991; Harter & Anllo-Vento, in press; Harter et al., 1989b). But, again, those studies included many more subjects than this one.

It would be wrong to infer that P1-N1 validity effects are missing on the basis of the relatively weak results in this study. Even here, robust target validity effects appear right after N1. Furthermore, P1 and N1 validity effects have been reliably obtained in previous research (Anllo-Vento & Harter, 1988; Harter & Anllo-Vento, in press; Harter et al., 1989a, 1989b; Mangun & Hillyard, 1988, 1990; Mangun, Hillyard & Luck, in press). The optional interpretation, therefore, would claim that cue validity enhanced the amplitude of P1-N1, though not powerfully. As we shall see later, P1-N1 validity interacted with reading in a predictable, yet enigmatic, fashion.

Beyond P1-N1, target validity significantly influenced the amplitude of all ERP deflections. Most interestingly, brain responses evoked by validly and invalidly cued targets, similar in topography for the first 200 ms, began to deviate from each other at the onset of N2, at which time the invalid response started to resemble the neutral condition, particularly over the posterior electrodes. Recall that a neutral cue indicated to the subject from the outset that a response would not be required on that trial. On the other hand, after a directional arrow the subject had to wait for the target to be presented, and determine its location before deciding whether or not to respond. Thus, timing of the underlying processes of discrimination and decision to respond can be approximated by

examining when brain activity in the invalid condition begins to look more like the neutral than the valid condition.

The point at which the valid and invalid conditions begin to diverge in polarity (about 200 ms after the target) could be taken to represent the end of location discrimination and initiation of the response. In this study, the brain's response to valid targets was always associated with the fastest latency, regardless of the potential being measured. This consistent trend suggests that the benefits of cueing are present from the beginning of target processing. It also indicates an automatic tendency to respond. Given the constant ISI, the subject learns to anticipate making a motor response as the time to target presentation approaches; but when the target appears in the invalid location, the motor response must be withheld. Interestingly, latency differences between the valid and invalid N2 were significantly related to the unbiased measure of sensitivity, d'. Relative ease in the discrimination of the target's location, as measured by d', was associated with a faster decision to respond to valid targets, a slower suppression of the response to invalid targets, or both. These results are in consonance with neurophysiological studies in behaving monkeys (Hyland, Chen, Maier, Palmeri & Wiesendanger, 1989; Dao-fen, Hyland, Maier, Palmeri & Wiesendanger, 1991), which show that neural firing in the supplementary motor area (SMA) and primary motor cortex (MI) precedes the onset of a manual movement by approximately 200-300 ms, and is related to reaction time.

Following N2, all conditions evoked a slow positivity continuing until about 440 ms after the target's onset. It is in this interval that the greatest validity effects were observed. The valid condition was associated with much larger amplitudes than either the invalid or the neutral conditions, particularly over the parietal and

central areas of the left hemisphere. The latency and distribution of LPD suggest that it constitutes a P3-like potential, and thus can be interpreted as a measure of closure (Verleger, 1988) or response evaluation (Donchin & Coles, 1988), which in this study immediately preceded the average reaction time (394.3 ms).

At the time the motor response is emitted, LPD displays a left-hemisphere predominance, probably due to the fact that all subjects responded with their right index finger. In that light, the difference in the distribution of the potential across the hemispheres is interesting. Over the right hemisphere, validity effects were constrained to the central and parietal areas; over the left hemisphere, which was contralateral to the hand of response, the LPD2 validity effect spread to the occipital and posterior temporal areas. Thus, premotor activity results in greater and wider neural activation immediately before and during the time of the response.

In the context of the behavioral literature (e.g., Downing & Pinker, 1985; Posner, 1978, 1980), validity effects represent the benefits in response time or accuracy accrued as a function of the imperative stimulus following a spatially informative cue. This study was designed to minimize the processing of irrelevant targets, and thus required no response when the target appeared in the invalid hemifield. Consequently, it was not possible to compare reaction times to validly and invalidly cued stimuli, and to calculate behavioral benefits.[1] But the advantages of cueing can still be assessed by means of a related measure: the latency of the LPD potential, which occurs in close temporal proximity to the timed response. In fact, validity effects were present in the latencies of all potentials, starting with P1, and got progressively larger as the time to respond

[1] The reaction times gathered in this study will be discussed below, since they were explained, in part, by reading ability.

neared. By LPD, the average difference in latency between valid and invalid targets, in favor of the former, was 29 ms. This estimate of validity is unexpectedly similar to the average RT-benefits showed by others (e.g., 20.5 ms in Mangun & Hillyard, in press; about 20 ms in Posner et al., 1984). But this reliable advantage of ERP latency to the valid versus the invalid condition was not paralleled by differences between invalid and neutral response-latencies, which would have supported a similar pattern of latency costs. Behavioral experiments have estimated the costs and benefits of spatial cueing with respect to a "neutral" condition (Berlucchi, Aglioti, Biscaldi, Chelazzi, Corbetta & Tassinari, 1989; Posner, 1978, 1980; Rizzolatti, Riggio, Dascola & Umiltà, 1987; Umiltà et al., in press), although the significance of such a "neutral" attentional state is not clear (Gawryszewski, Riggio, Rizzolatti & Umiltà, 1987). Here, most potentials evoked by the target did not show a significant latency difference between the neutral and the invalid conditions. Several features of this study could be responsible for this difference. For instance, no other behavioral paradigm has used central cues that are non-predictive, followed by only two peripheral target locations. More importantly, the subjects in previous studies were normal adults, rather than children. It is intriguing, in this light, that the only study found in the literature in which a similar paradigm was used with poor and good readers (Brannan & Williams, 1987) found behavioral costs only in normal young adults, while benefits were present in both the adult and young groups, and diminished in poor readers.

The beneficial effects of valid cueing found both with reaction times and LPD-latency measures seem more noteworthy than the differences in costs, especially when one considers that they are measures of brain activity and behavior, respectively, and that previous studies varied in other respects. This

finding lends credibility to the connection between brain and behavioral phenomena maintained here, and supports the idea that subjects had selectively directed their attention towards the cued location prior to the target's presentation.

In sum, the post-target validity effects obtained in this study confirm and extend previous reports: stimuli appearing at a cued spatial location evoke brain responses that are enhanced with respect to those elicited by uncued stimuli. The selective processing of validly cued targets starts between 120-180 ms after their presentation, and continues to increase past the average response time. In addition, the peak latency of all post-target potentials indicates that valid targets are processed faster than invalid targets starting soon after their onset. The relative courses of the ERP waveforms evoked under the valid, invalid and neutral conditions further suggested that the decision to respond occurred between 180-260 ms after the target's onset. Taken together, these results provide a brain correlate of the well-established behavioral benefits derived from directing attention toward a cued spatial location.

The Cueing Task: A final appraisal. The following is assessment of the results obtained in the present study that relate to visual-spatial attention. Both the innovations in design and the methodological improvements included in the study are evaluated. The contribution of the neutral cue is examined first, followed by an appraisal of the information provided by strict eye-movement control.

The neutral cue became useful in a rather unexpected way. It did not change significantly the conclusions drawn from similar studies--which did not include a neutral cue, though it did help to establish those findings more firmly. In

particular, the ERP response to the neutral condition confirmed the presence of differential brain activity in response to directional cueing. But, more importantly, the neutral cue served to better define and time two particular events: the onset of the differences in the processing of arrow direction, and the point at which the subject decided to respond or withhold a response.

Differences between left and right arrows started at about 240 ms and continued beyond target presentation, a latency range which does not differ substantially from previous estimates (Harter, 1991; Harter & Anllo-Vento, in press; Harter et al., 1989b). Without the neutral cue, however, it would not have been possible to separate the perceptual and motor aspects of the cueing response. It was the neutral cue that helped define part of the CNV as related to response preparation, starting approximately 500 ms after the cue. It also provided a frame of reference to time the decision to withhold the response following an invalid target.

The final contribution of the neutral condition is of a more technical nature. Previous studies (Harter, 1991; Harter & Anllo-Vento, in press; Harter et al., 1989b) had difficulties defining the polarity and timing of the potentials evoked by directional cues. It was clear that differences existed in the brain's response to left and right arrows, but there was no way to anticipate how the ERP would behave in response to a non-directional cue. The neutral cue included in this study served that purpose. It clearly showed the presence of two slow responses of opposite polarity: a slow positivity--the CRP, and a slow negativity that conformed to earlier characterizations of the CNV. This relatively more technical contribution will prove useful in analyzing the available developmental data from similar paradigms.

A second innovation in this study was a much more precise control of eye movement. Oculomotor potentials have always been a challenge for ERP research. Muscle potentials are orders of magnitude larger than brain potentials and, thus, distort the accurate recording of brain activity. Research on visual-spatial attention is particularly susceptible to this type of problem for two different reasons. First, most visual-spatial paradigms require processing of peripheral stimuli while the subject maintains fixation, and the tendency to make an eye movement in order to foveate the target is not always successfully suppressed. In addition, small, undetected eye movements result in different points of the retina being stimulated; the consequence is an increase or decrease in the amplitude of the ERP that could be mistakenly attributed to a covert shift in attention.

This study unquestionably established that eye movements are not responsible for the effects of either cueing or target validity. In the first place, several precautions were taken to insure a reliable detection of eye movements. Secondly, the EOG measure was included in the statistical analysis as a covariate and, although significant, it did not change the direction of the effects obtained with respect to those previously reported.

<center>Reading, Visual-Spatial Attention,

and the Magnocellular Pathway</center>

This section will begin with a succinct recapitulation of the logic behind this project. It will serve to place in perspective the subsequent, more detailed theoretical explanation, and to define the empirical and conceptual boundaries of the study. Later, the section will include an evaluation of the experimental evidence as it relates to reading ability, and it will end with a concise overview.

Recently, the idea that poor and disabled readers show deviant patterns of visual processing has gained renewed support (Brannan & Williams, 1987; Harter, 1991; Livingstone et al., in press; May et al., 1991). These empirical studies have been guided by new advances in our knowledge of the neurobiological bases of perception and their development. A particularly important concept has been the functional and structural differentiation of two parallel visual streams: parvocellular and magnocellular. Although some have claimed that parvocellular dysfunction underlies the visual deficits observed in poor readers (Harter, 1991; Geiger & Lettvin, 1987), the bulk of the evidence argues in favor of a magnocellular explanation (Brannan & Williams, 1987; Livingstone et al., in press; Lovegrove et al., 1980, 1986; May et al., 1991). The selection of a relevant location in visual space is likely to be mediated by the magnocellular system (see below). Therefore, variations in reading ability should be related to variations in visual-spatial attention to the extent that they both selectively engage the magnocellular pathway. The present study was motivated by the hypothetical connection between visual-spatial attention, reading, and the magnocellular stream. To provide an empirical test of this connection, the study probed the relationship between normal variations in reading ability and the ability to voluntarily shift attention in visual space.

This study adheres to the premotor interpretation of spatial attention (Rizzolatti et al., 1987; Tassinari, Aglioti, Chelazzi, Marzi & Berlucchi, 1987), which postulates a close connection between movements of attention and the programming of eye movements. In it, a covert shift of attention is envisioned as an efferent excitation of the neural mechanisms that mediate eye movements, and a concomitant suppression of the programmed motor response. This hypothesis

has derived, in part, from clinical neuropsychological data: damage to those brain structures related to eye movements often entails both oculomotor and attentional deficits (Heilman & Valenstein, 1972; Lynch, 1980; Robinson & Petersen, 1986). It is also supported by neurophysiological brain studies in alert, behaving monkeys, which show that some brain structures process visual and motor information concurrently. A clear case is the posterior parietal cortex, where neurons encode both a particular visual location and a movement towards the same location in extrapersonal space (Mountcastle, Lynch, Georgopoulos, Sakata & Acuna, 1975; Wurtz et al., 1980). Similar responses are obtained in other neural structures, both cortical (e.g., the frontal eye fields) and subcortical (e.g., the superior colliculus) (Oakley & Eason, 1990).

A different source of support for an oculomotor interpretation of attentional shifts comes from behavioral studies on the benefits and costs of cueing (for a review, see Umiltà et al., in press). The oculomotor hypothesis successfully explains, for instance, the *inhibition-of-return* phenomenon (Maylor, 1985; Posner & Cohen, 1984), whereby a stimulus presented after more than 300 ms at a peripherally cued location is processed more slowly than a stimulus appearing at some distance from the cue (Maylor, 1985; Posner & Cohen, 1984; Tassinari et al., 1987; Tassinari, Biscaldi, Marzi & Berlucchi, 1989). The model stipulates that, since the eye movement towards the cued location is suppressed, it will take more time to reprogram a motor or attentional movement towards that same location than towards a new, uninhibited one. Interestingly, the inhibition of return has a latency of about 300 ms, about the same as the onset of a saccade. The oculomotor interpretation also accounts for the presence of attentional *gradients*, that is, decreases in behavioral gain observed as the target appears increasingly

farther from the cued location (Mangun & Hillyard, 1988; Downing & Pinker, 1985). In this case, once attention has been shifted, the amplitude of the shift rather than its vector, has to be calculated, and the suppression extended farther with increasing distance from the cued location. Thus, the oculomotor model provides an appealing and parsimonious conceptualization of selective spatial attention.

Neural oculomotor activity is closely linked to the magnocellular pathway. It is obvious that sudden changes in luminance, particularly in the periphery, will elicit an orienting eye movement to center the attention-gathering event onto the better resolving power of the fovea. Stimulus changes of the kind that evoke an oculomotor response are most often fast and peripheral; magnocellular cells are designed to optimize the processing of this type of information. An oculomotor interpretation of attentional shifts entails the participation of the magnocellular pathway in visual-spatial attention. So far, then, the connection between selective attention and the magnocellular pathway has been made. The next step is to find the link between reading and the magnocellular stream.

The current evidence that reading disability is associated with a less effective magnocellular pathway has been summarized in the Introduction. This study extends the relationship beyond pathological cases to normal variations in reading ability: good readers tend to have well-developed magnocellular function, while poor readers have a less efficient magnocellular system. This is, of course, a simplified --though testable-- hypothesis. As stated before, the development of these ideas requires a better understanding of the relationship between the magnocellular and parvocellular pathways, and the role they play in reading and selective attention. In the interim, the present study set out to test an inference

derived from the discussion presented above. If selective spatial attention relies on magnocellular function, and poor readers have limited magnocellular resources, reading ability should be positively associated with measures of visual-spatial selection. The empirical evidence garnered in support of this interpretation is presented next.

The reader will recall that reading ability was significantly related to the pattern of brain activity evoked during the performance of the visual-spatial task. Here, the main trends observed during task performance for good and poor readers will be discussed within the conceptual framework already introduced. The supportive evidence will be presented first, and followed by a speculative explanation of divergent findings.

Sensory processing of the foveal cue did not differentiate good and poor readers: the visual potential evoked by the three cue types was comparable across reading levels. Group similarities in the brain's response to the cue continued beyond C-N240, and until the onset of the CRP. As you might recall, C-N240 was the first potential to reveal differential processing of arrow direction for the group as a whole. Therefore, the results suggest that poor and good readers alike discriminate the cue's shape and encode its meaning during the initial 300 ms.

Starting at the CRP, that is approximately 320 ms after cue onset, the responses of the two groups began to diverge. From this point through the presentation of the target, poor readers gave no indication that they were selectively processing arrow direction. That is to say, in poor readers brain activity evoked by the left arrow was no different from that evoked by the right arrow. Good readers, on the other hand, showed significant differences in the processing of the two directional cues. This finding is fundamental; it suggests a relative

inability to direct attention selectively on the part of the poor readers. Even more revealing is the latency of the result, since the time of CRP onset coincides approximately with the average latency of a saccade. According to the oculomotor model of attentional shifts, the suppression of the programmed eye movement mediating covert orienting should take place at this time. Suppressive brain activity is suggested by the fact that the CRP is a slow positive potential of the kind that has been shown to reflect cortical inhibitory activity (Birbaumer, Elbert, Canavan & Rockstroh, 1990). In addition, the greatest CRP differences between reading levels were found over the parietal cortex, an area known to encode both information about the spatial location of visual stimuli and imminent saccadic eye movements towards the same location (Lynch, 1980; Mountcastle et al., 1975; Wurtz et al., 1980).

Not only did poor and good readers differ in their processing of arrow direction, they also varied in the degree of preparation reflected by the amplitude of the CNV. In agreement with previous studies (Anllo-Vento et al., 1990; Jones & Mitchie, 1986), this preparatory potential was drastically reduced in the lower reading-level group. Interestingly, poor readers had relatively more negative and widely distributed CNVs than good readers. But they did not show the selective motor readiness indexed by CNV differences between directional and non-directional cues. This finding of relatively more widespread brain activation in poor readers has been previously reported in electrophysiological (Anllo-Vento et al., 1990; Harter, 1991), PET (Rumsey et al., 1987), and regional cerebral blood flow (rCBF) studies (Flowers, Wood & Naylor, 1991; Wood et al., in press). The reliable finding is one of shallower anterior-posterior gradients of intrahemispheric activation, reflecting less localized neural activity. Wood, Flowers, Buchsbaum

and Tallal (in press) have interpreted this trend as a sign of greater effort or less automaticity, since it was negatively correlated with accuracy in task performance.

By the time the target stimulus is presented, during the terminal portion of the CNV potential, poor and good readers are in very different states of preparation. The brain activity of good readers is localized, reflects a high degree of readiness, and a differential processing of arrow direction; poor readers, in contrast, appear not to have processed location selectively in spite of their widespread brain activation, and to be substantially less ready. The logical next question is whether such differences translate into improved target processing by good readers. The answer is yes.

The first sign of an advantage related to the prior degree of activation is the validity effect observed in the ERP response to the target. Good readers showed greater target validity effects than poor readers. Group differences in P2-N2 validity were present over the anterior electrodes, and indicated that, unlike good readers, poor readers did not process the invalidly and neutrally cued targets differently. Recall that P2-N2 was presumed to reflect the subject's decision to withhold a response. This interpretation is supported by the anterior distribution of the effect, since frontal brain structures are associated with decision-making and executive function (Kolb & Whishaw, 1990; Lezak, 1983). The scalp distribution of the differences across valid, invalid and neutral conditions suggested once again a greater degree of localization in good than poor readers: the P2-N2 validity effect was much larger over the anterior electrodes for the better readers, while for poor readers it was of similar magnitude over the front and back of the head.

Differences in validity effects across groups became even larger at the LPD range (280-360 ms post-target). A diminished effect of relevance in this latency

range has been previously reported in poor readers, not only in visual-spatial tasks (Harter, 1991; Harter & Anllo-Vento, in press; Harter et al., 1989a) but most often in letter- or word-identification paradigms (Harter, 1991; Harter et al., 1988b; Holcomb et al., 1985). Here, differences in target validity across reading levels were associated with greater LPD responses being evoked by valid targets in good readers; no group differences in LPD amplitude were found for the invalidly cued targets. The same result has been previously reported (Harter, 1991; Harter et al., 1989a), but was not found in an attentional study of RD and ADD children (Holcomb et al., 1985). A possible explanation for the discrepancy between these studies comes from differences in task demands. Holcomb et al. (1985) used a simple recognition task that could have measured novelty rather than selective attention.

There was an unexpected lack of demonstrable differences in LPD lateralization across reading groups. A diminished or absent P3-relevance effect had been found before over the left hemisphere of poor readers (Harter et al., 1988b; Preston et al., 1977), and was also expected here. Instead, both groups showed the same left-hemisphere lateralization of LPD validity, probably due to the right-hand motor response. The most likely explanation for this unanticipated negative result derives from the nature of the task. Harter et al. (1988b) used three discrimination tasks of increasing difficulty, all of which included letter and non-letter patterns. Preston et al. (1977) presented their subjects with three-letter words and unpatterned flashes, and instructed them to count the number of occurrences of a target word. In contrast, in a study which employed a visual-spatial task similar to the one used here (Harter, 1991), differences in P3 lateralization across reading groups were not demonstrated. But the reduction of

P3 validity effects was present there, just as in the present study. Although the conclusion that "verbal" tasks may be more likely to reveal cerebral asymmetries is tempting, it should be noted that the left-hemisphere LPD1 validity effect found here was positively correlated with the Woodcock-Johnson reading scores, indicating that larger LPD1 validity effects were associated with greater reading ability, despite the visual-spatial nature of the task.

So far only electrophysiological measures have been considered in evaluating the advantage of selective spatial preparation prompted by the cue. What about behavioral benefits? Those were also observed, though in a less straightforward fashion. Recall that the relationship between reading scores and differences in CRP and CNV amplitude due to arrow direction was complex. The variables were positively correlated, though not perfectly, and their common variations were associated with faster reaction times. But the part of brain activity that was uncorrelated with reading ability was associated with slower responding. This second result has not been previously reported in the literature, and suggests that there is an effective level of preparation, which is associated with enhanced performance in both reading and visual-spatial attention tasks. Beyond this level, however, performance in a spatial-attention task declines with greater preparation. Inverted U relationships of this kind are not unusual in psychology. In fact, the phenomenon obtained here could be an exponent of the Yerkes-Dodson Law, whereby increased arousal improves performance up to a middle range, beyond which it exerts the opposite effect. The finding of both effective and excessive components in preparation for an event underscores the complexity of the connection between brain function and observable behavior.

The final finding to discuss is certainly the most puzzling. Although poor readers showed very little evidence of preparation, their P1-N1 validity effects were greater than those of good readers, even if only over the left hemisphere. The result would be much less compelling if it had not been found in two other studies (Harter, 1991; Harter et al., 1989a). Harter et al. (1989a) tentatively interpreted it as reflecting superior spatial processing in poor readers, possibly due to brain reorganization following left-hemisphere insult. Harter (1991) extended the functional significance of this conjecture by noting that poor readers had faster reaction times than good readers. It should be remembered that the average reaction time was faster for poor readers than for good readers in this study as well, though not significantly. But the complexity of the relationship between brain activity and behavior noted above should warn us against endorsing simple explanations.

In fact, no evidence found in this study suggested that enhanced P1-N1 validity effects were associated with behavioral benefits. The results did not support the intuitive idea that greater preparation ought to result in greater P1-N1 validity effects being evoked by the target. The most reasonable speculation which can be drawn at this point is that the mechanism mediating the sensory response to the peripheral target is under a state of refractoriness following spatially selective preparation, so that less preparation results in a transiently heightened response. It would be misleading to suggest that this conjecture is more than minimally suggested by the evidence found in this study. Yet the P1-N1 validity enhancement, or suppression, appears to be a significant phenomenon in visual-spatial attention, and one that deserves further study.

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General Conclusions and Implications for Further Study

The study presented here set out to examine the hypothetical relationship between visual-spatial attention and reading ability by means of brain cortical potentials. The idea which motivated the investigation was the presumed connection of these two cognitive processes with a common, underlying brain mechanism: one of the specialized processing-streams of the visual system. Previous research had suggested the existence of a connection between a functional and structural dysfunction in the magnocellular subdivision of the visual pathway, and the presence of reading disability. But, at the time of testing, the children who participated in this study, though originally selected as at-risk of developing a reading disability, could not be differentiated in reading achievement from the normal population. Consequently, this is the first neurophysiological study that probed the presence of a connection between two cognitive skills --reading and visual-spatial attention-- in a group of children within the normal range of reading ability. The results obtained suggest that reading ability and disability might constitute a continuum of skill. Within this framework, reading

disability would be conceived as representing the bottom-end of the normal distribution, rather than a separate pathological population.

The single and most original contribution of this study derives from the relationship it revealed among brain activity, reading ability, and behavior. Greater differences between the brain potentials evoked by right and left directional cues were associated with both faster responding to a relevant target, and higher reading ability. One possible interpretation for this finding derives from the presumed role of the magnocellular pathway in mediating the transmission of a type of information that is fundamental in both reading and visual-spatial attention. As discussed above, a visual-spatial shift of attention might be conceived as an oculomotor program whose execution has been inhibited. Its likely premotor character ties it closely to the *where* system and the magnocellular stream. On the other hand, reading involves an automatic ability to direct and control eye movements in order to optimize lexical access (Pollatsek & Rayner, 1990). In addition, and as already mentioned, other cognitive operations or subcomponents included in the reading process, such as phonological decoding, might be mediated by a perceptually specialized stream with characteristics similar to those of the magnocellular pathway.

How close has this study brought us to positing a neurobiological theory of visual-spatial attention, reading ability, and their relationship? Not very close. It would be premature to articulate a broad conceptual model at this point. But this investigation, nevertheless, points to a profitable avenue: the joint consideration of brain and behavioral measures to evaluate cognitive processes that may be interrelated. In this case, the findings of this study suggest that reading partly depends on a proficient control of attentional shifts.

Of course, the inquiry should be furthered, and there are various logical next steps. The simplest would be to replicate these findings, or even extend them, with a group of truly disabled readers. Such a study would evaluate further the existence of a continuum of reading ability-disability, where the lower-end of the distribution constitutes the extreme exponent. It could also appraise more definitely the existence of a connection between reading disability and visual-spatial attention and, through it, suggest more strongly the involvement of the magnocellular pathway.

A more challenging project would be to characterize more directly and in greater detail the contribution of the magnocellular pathway to reading ability and disability. The visual-spatial task used in this study was rather complex; there are more basic and better-known features of the magnocellular stream that could be examined instead. In fact, there is an abundant number of behavioral studies that have tested this hypothesis and could be modified to permit the simultaneous recording of ERPs.

Last and most complex is the developmental avenue. Because visual-spatial attention skills may contribute to reading acquisition during a particular point in development, it would be important to study the relationship between these two cognitive skills over time. But there is another, more fundamental reason to address this question developmentally. If, in fact, the magnocellular pathway is associated with visual-spatial attention and reading, as proposed here, current knowledge as to its neurobiological development should also be taken into account. It is known that the magnocellular pathway is particularly sensitive to environmental influences, and its developmental course is delayed with respect to that of its parvocellular counterpart (Blakemore & Vital-Durand, 1984).

Magnocellular plasticity, therefore, is in agreement with models of developmental dyslexia, such as that of Gerschwind and Galaburda (1985), which claim that the etiology of the disorder is of a prenatal nature.

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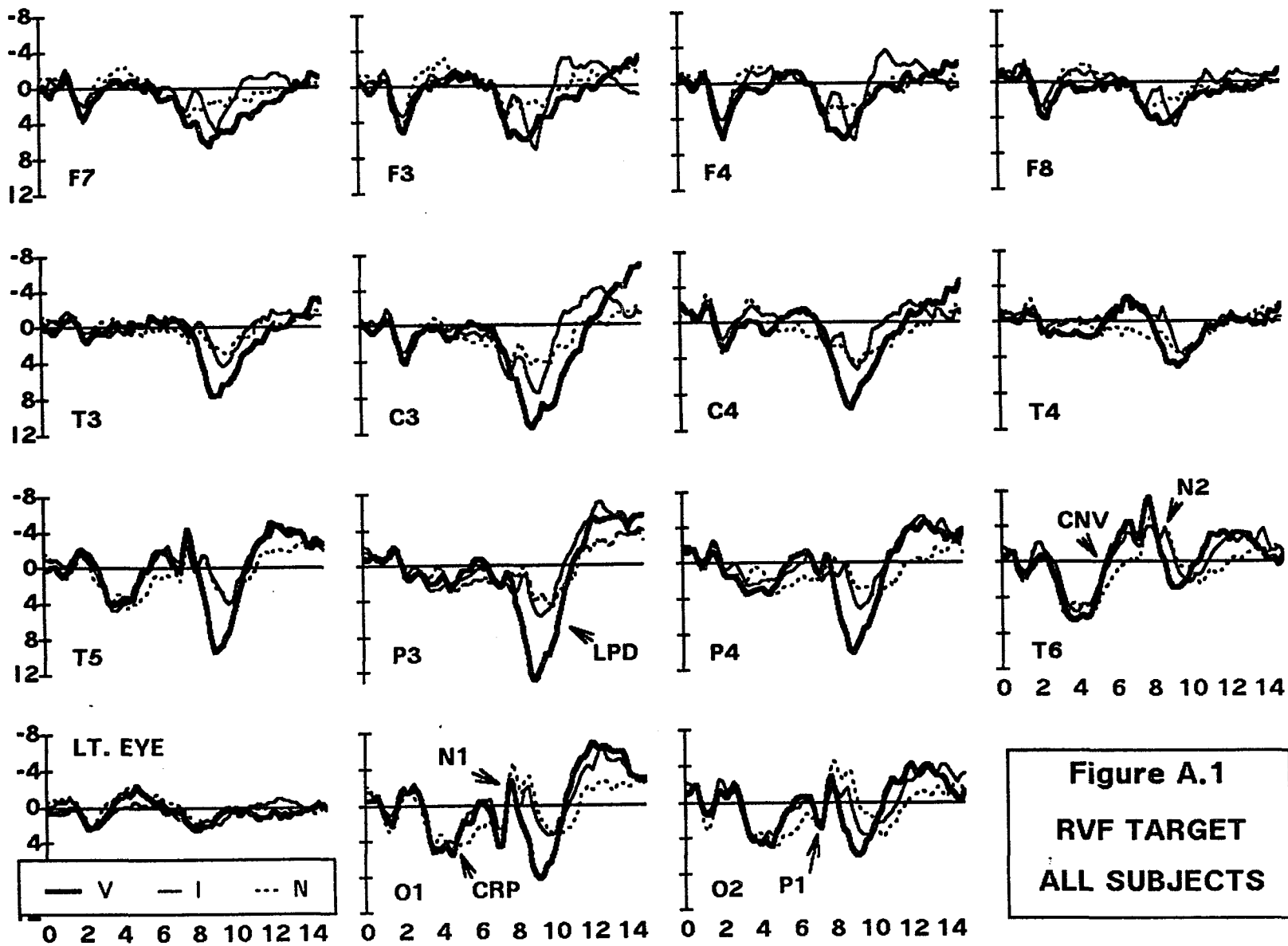Pavlidis, G. Th. (1981). Do eye movements hold the key to dyslexia? *Neuropsychologia*, **19**: 57-64.

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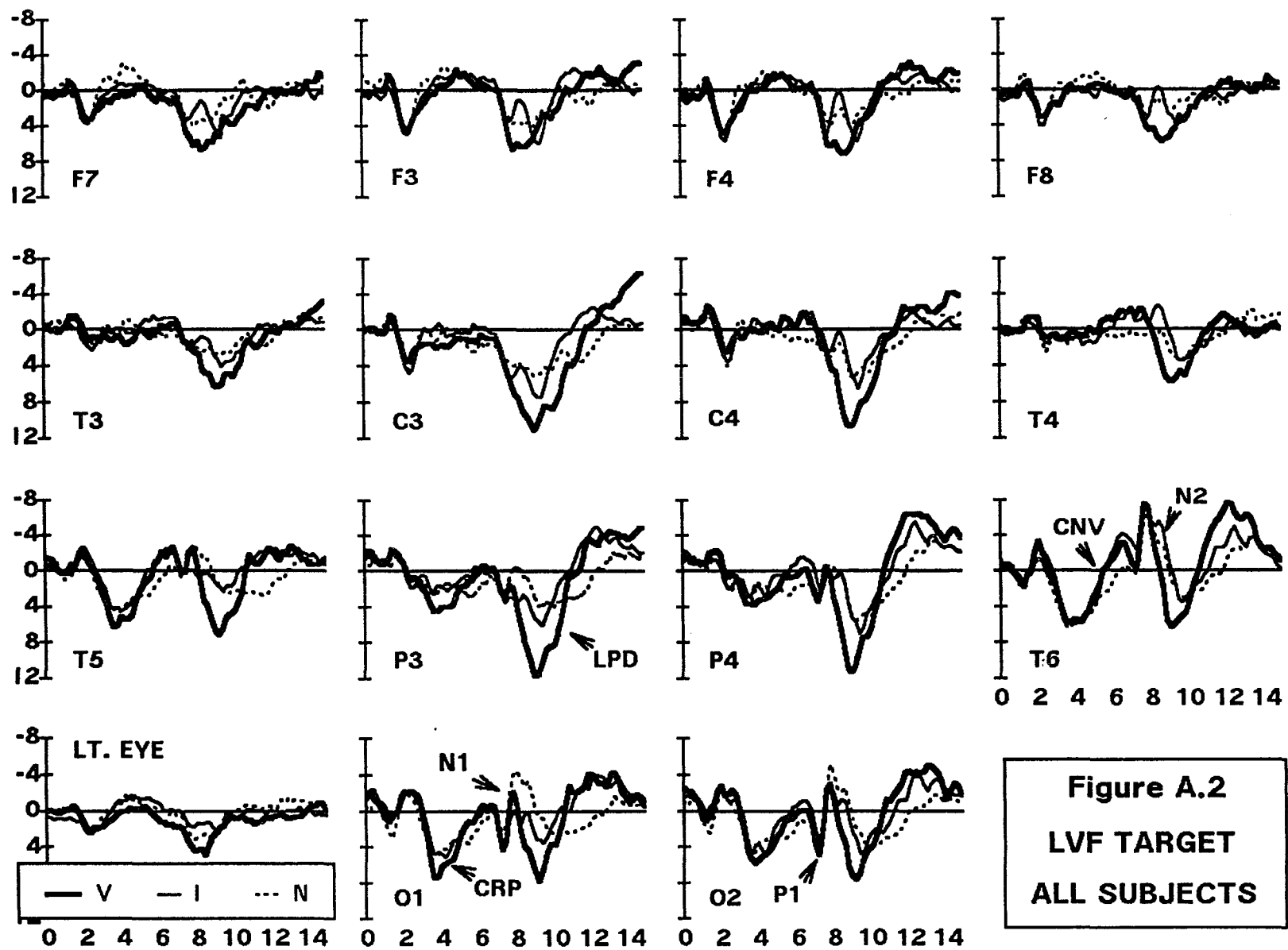
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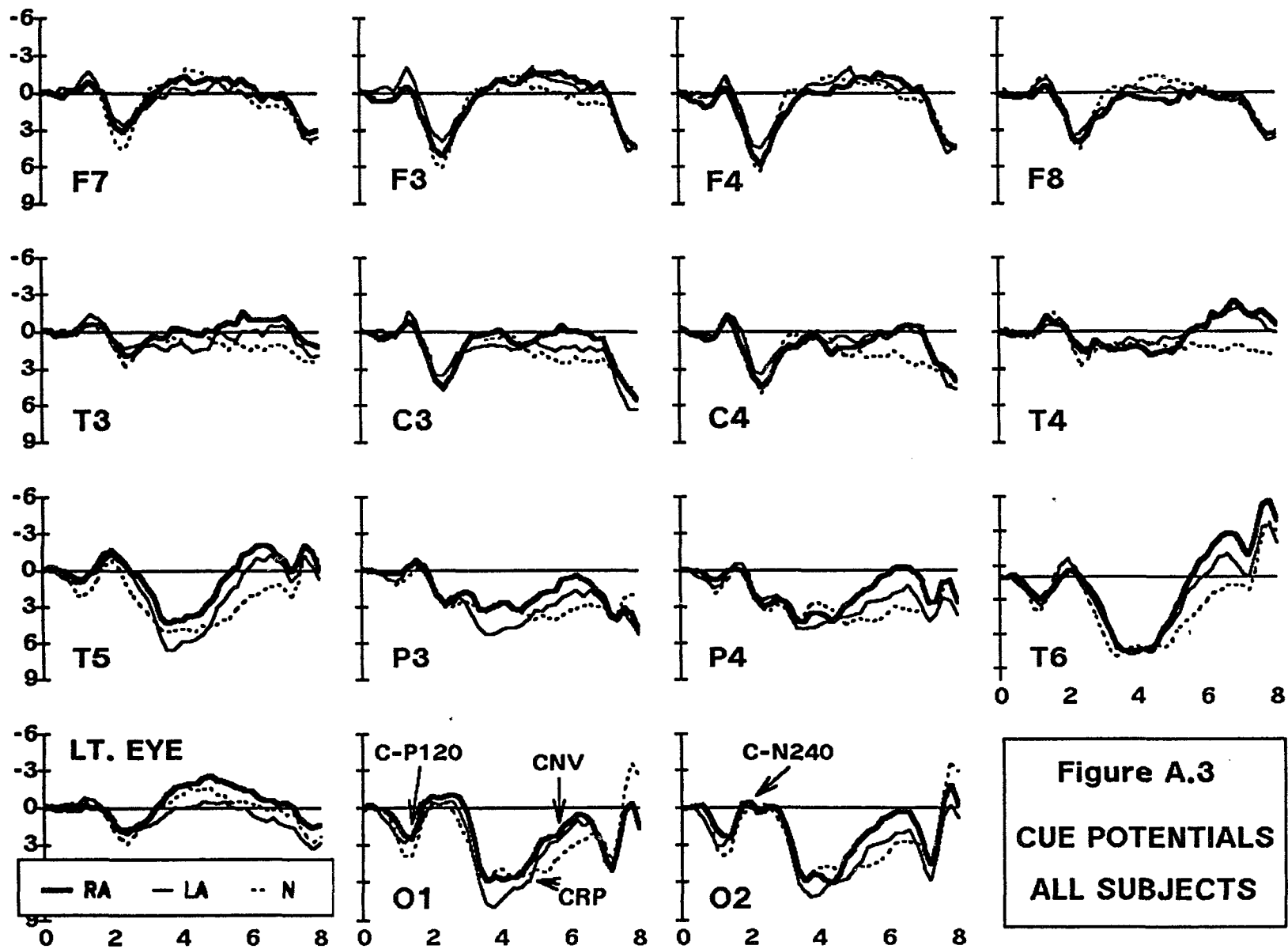
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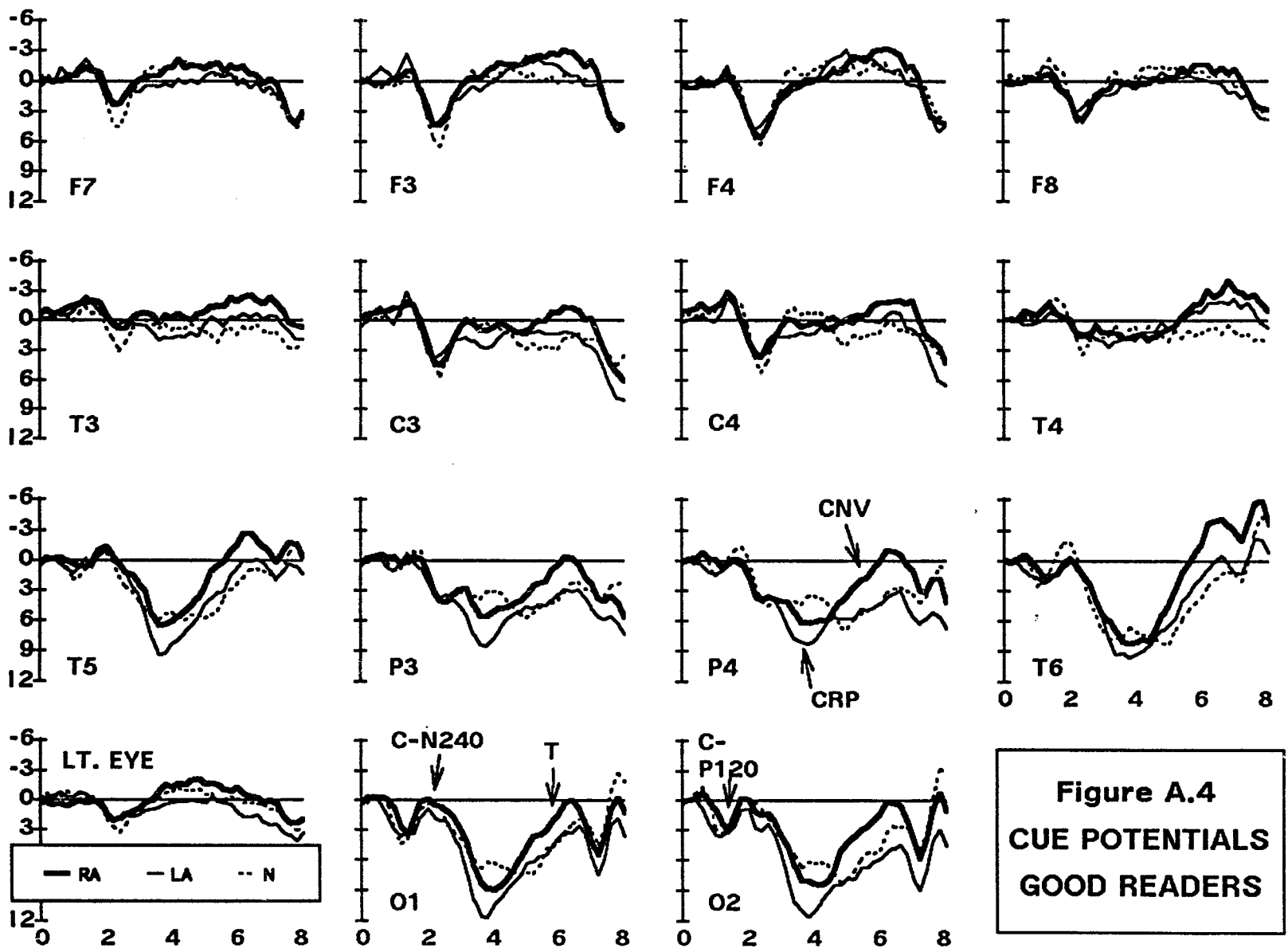
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APPENDIX A. FIGURES OF THE RAW WAVEFORMS

Figure A.1

RVF TARGET

ALL SUBJECTS

Figure A.2
LVF TARGET
ALL SUBJECTS

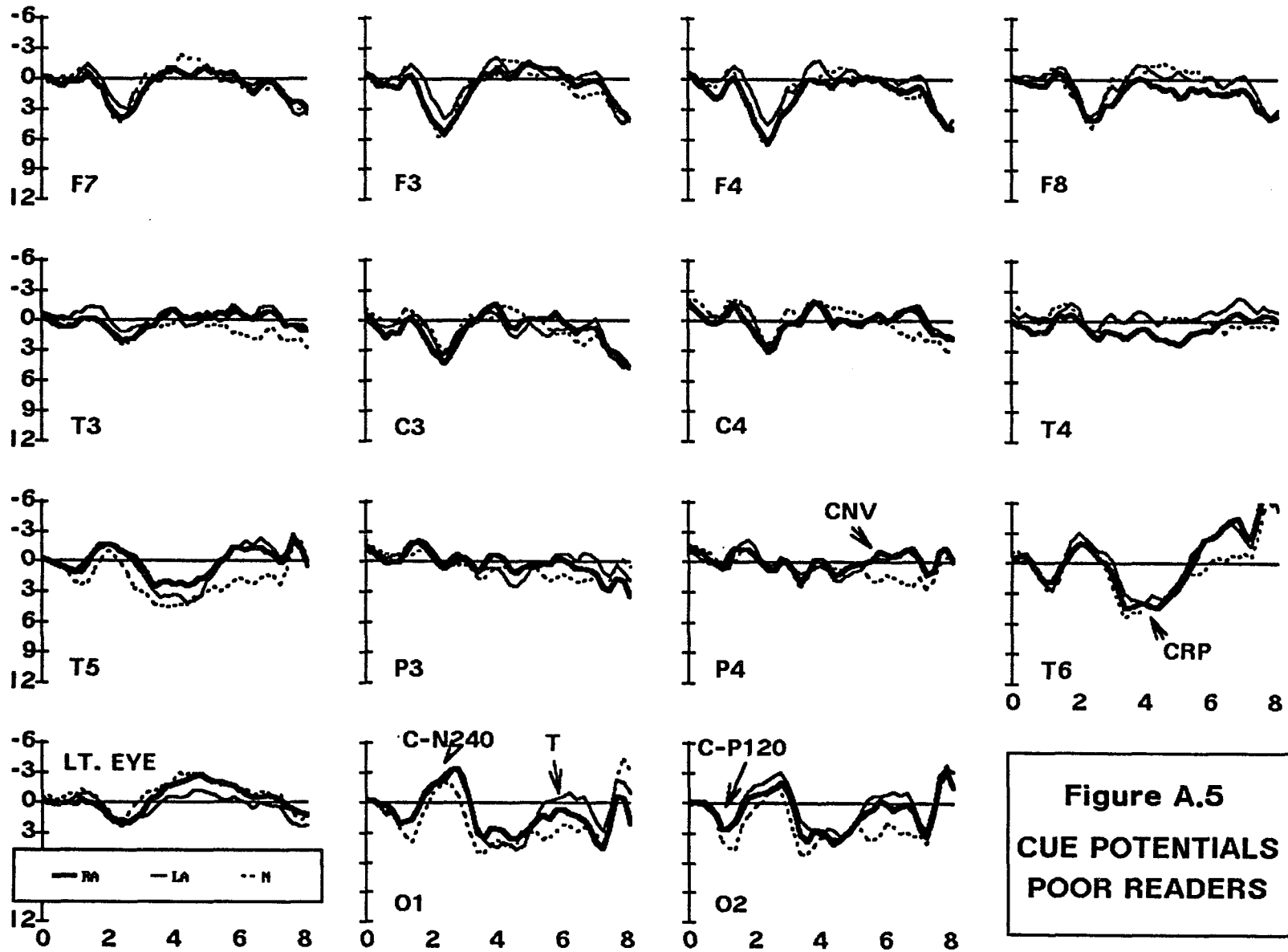Figure A.3

CUE POTENTIALS

ALL SUBJECTS

Figure A.4
CUE POTENTIALS
GOOD READERS

Figure A.5
CUE POTENTIALS
POOR READERS

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Figure A.6

CUE DERPs

ALL SUBJECTS

Figure A.7
TARGET
POTENTIALS

ALL SUBJECTS

Figure A.8

TARGET
POTENTIALS

POOR READERS

Figure A.9
TARGET
POTENTIALS

GOOD READERS