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Visual evoked potentials in dyslexics and normals: Failure to find a difference in transient or steady-state responses

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Abstract

We measured transient and steady-state checkerboard contrast-reversal visual evoked potentials (VEPs) in ten dyslexics, five patient controls, and 11 normals over a range of contrasts and luminances. Latency, amplitude, and phase measurements failed to distinguish the responses of dyslexics from those of normals or patient controls. Decreases in luminance or contrast resulted in an increased latency of the transient VEP in all groups, but these changes also did not distinguish the responses of dyslexics from those of the controls. Response variability was similar in dyslexics and normals, but was increased in subjects with attention deficit-hyperactivity disorder (ADHD). Performance on standardized psychometric testing did differentiate the dyslexics from controls, but did not correlate with VEP responses.

Keywords: Dyslexia, Magnocellular pathway, Visual evoked potential

Introduction

Whether dyslexia is a single disorder with a specific neurobiological etiology, a multiplicity of identifiable disorders, or one end of a continuum is a matter of great interest and heated debate (Galaburda, 1992; Shaywitz et al., 1992; Wood et al., 1991). Livingstone et al. (1991) recently reported a striking abnormality in the VEPs of dyslexics: a loss of responses under conditions of high temporal frequency, low luminance, and low contrast. Such an abnormality might escape detection by routine clinical VEP testing (Chiappa, 1990), which is typically done under conditions of lower temporal frequency, and higher luminance and contrast. Livingstone et al. (1991) interpreted this abnormality as a physiological correlate of a loss of magnocellular neurons, and suggested that this magnocellular loss was important in the pathophysiology of dyslexia. In view of the importance of this finding and the small sample size of their study, we set out to reproduce these observations, with an analytical method (Victor & Mast, 1991) which provided rigorous statistical criteria for detection of significant responses and significant differences between responses. We used steady-state and transient conditions essentially identical to those used by Livingstone et al. (1991): 16-Hz reversal rate, 2% contrast, 4 cd/m^2, 3-deg checks), as well as conditions of higher luminance and/or contrast. We found no evidence for a low-contrast, low-luminance VEP abnormality associated with dyslexia. Systematic dependence of transient VEP latency on luminance and contrast was observed in all subjects, but these changes also failed to distinguish dyslexics from controls.

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Methods

Subjects

Eleven of the patient subjects were respondents to a mailing to 60 learning-disabled patients seen by a pediatric neurologist (author RN). This group included nine dyslexic subjects and two attention deficit-hyperactivity disorder (ADHD) subjects. A tenth dyslexic patient responded to a notice concerning this study posted at a school which specializes in learning-disabled students. Three additional patient controls (two in the ADHD group and one with post-traumatic aphasia) were referred by a second pediatric neurologist.

For the purposes of this study, developmental dyslexics were defined as subjects of normal intelligence who presented with a history of unexpected reading disability. This assessment was made by a learning disability assessment team run by one of the authors (RN) or by the evaluation of the specialized school. Eight of the ten dyslexics fulfilled this criterion. Two of these eight subjects met "educational" criteria for dyslexia, defined

as a reading level of 1.5 to 2 years behind grade level (Duffy & Geschwind, 1987; Nass, 1991). The remaining two dyslexic patients had a history of head injury directly preceding their reading difficulties and constituted the subgroup of acquired dyslexics. The diagnosis of ADHD was based on teacher response to a standard teacher questionnaire (two patients) or neuropsychological evaluation (two patients).

Normal subjects were recruited from the local population. All subjects had visual acuity correctable to 20/30 or better. For the ten dyslexics, the age range was 8–46 years (median: 13.5 years). For the 11 normal subjects, the age range was 6–34 years (median: 12 years). The five non-dyslexic controls included four ADHD patients [age range 7–12 years (median: 9 years)] and one with post-traumatic aphasia, age 8 years. Informed consent was obtained in accordance with International Review Board (IRB) standards.

Visual evoked potentials

The stimulus consisted of an 8 × 8 array of checks, each subtending 3.3 × 3.3 deg, presented on a Conrac 7351 monitor modified to run at a frame rate of 135.16 Hz. The mean luminance of the display was 59 cd/m^2, and was reduced to 4 cd/m^2 for half of the runs by placing a gray Plexiglas® sheet in front of the display. Stimulus contrast $[(L_{max} - L_{min})/(L_{max} + L_{min})]$ was 20% or 2%. Luminances and contrasts were calibrated with a Pritchard 703 spectrophotometer. For transient VEP recording, the checkerboards underwent square-wave reversal at a rate of 0.528 Hz (reversal rate 1.056 Hz). For steady-state VEP recording, the checkerboards underwent sinusoidal contrast reversal at rates of 2.112, 4.224, and 8.448 Hz (reversal rates of 4.224, 8.448, and 16.89 Hz, here called "4 Hz," "8 Hz," and "16 Hz"). The control signals required to produce these stimuli (synchronization, blanking, and R, G, B drives compensated for nonlinear voltage/intensity characteristics of the display) were generated by specialized digital hardware (Milkman et al., 1980) interfaced to a DEC 11/73.

Gold-cup electrodes filled with electrode paste were placed on scalp sites for differential recording between C_Z and O_Z, with a ground at the mastoid. Viewing was binocular at 60 cm, with natural pupils and corrective lenses if necessary to maintain acuity at 20/30 or better. Signals were filtered (1–100 Hz), amplified (10,000×), and digitized by the DEC 11/73, which was synchronized to the visual display.

The recording sequence was composed of blocks of ten runs at a single luminance level. The first block was at a luminance of 59 cd/m^2, the second and third blocks were at a luminance of 4 cd/m^2, and the fourth block was a return to a luminance of 59 cd/m^2. The subject was allowed several minutes to adapt to each luminance change. Pupil size, measured after adaptation to luminance change, ranged from 3 to 4 mm for the high luminance blocks and from 5 to 6 mm for the low luminance blocks. Each ten-run block consisted of two subblocks of five runs, one at a contrast of 20% and one at a contrast of 2%. Each subblock consisted of a transient run, one of each of the three steady-state runs, and a repeat of the transient run. Each run was 35 s long, with the final 30 s saved for analysis. Thus, data for each of the four transient conditions (two contrasts × two luminances) were acquired in four trials of 30 s each, and data for each of the 12 steady-state conditions (three frequencies × two contrasts × two luminances) were acquired in two trials of 30 s each. Off-line analysis consisted of averaging the transient and steady-state responses across replicate trials. For the transient responses, latencies were determined by the interval between stimulus onset and the first major occiput-positive deflection (the P100), and amplitudes were determined by the size of this deflection as measured from the baseline. For the steady-state conditions, responses were quantified by Fourier analysis, and response variability was assessed by the T_{circ} statistic (Victor & Mast, 1991).

Neuropsychological testing

The Slosson Intelligence Test (Slosson, 1991), the Gates-MacGinitie Reading Test (comprehension only) (MacGinite, 1978), the Beery Visual-Motor Integration Test (Beery, 1982), and the Benton Verbal Fluency Test (Benton & Hamsler, 1976) were administered in the standard fashion on the day of the VEP studies.

Results

Transient responses

Examples of transient VEPs obtained under the four conditions of luminance and contrast are shown in Fig. 1. Under low-luminance, low-contrast conditions, there were no significant differences in amplitude (dyslexics: 15.5 ± 9.2 μV, controls: 16.3 ± 11.9 μV, $P > 0.05$ by t-test) or latency (dyslexics: 154 ± 9 ms, controls: 155 ± 10 ms, $P > 0.05$ by t-test). Exclusion of the five patients from the control population or the two acquired dyslexics from the dyslexic population did not cause any of these

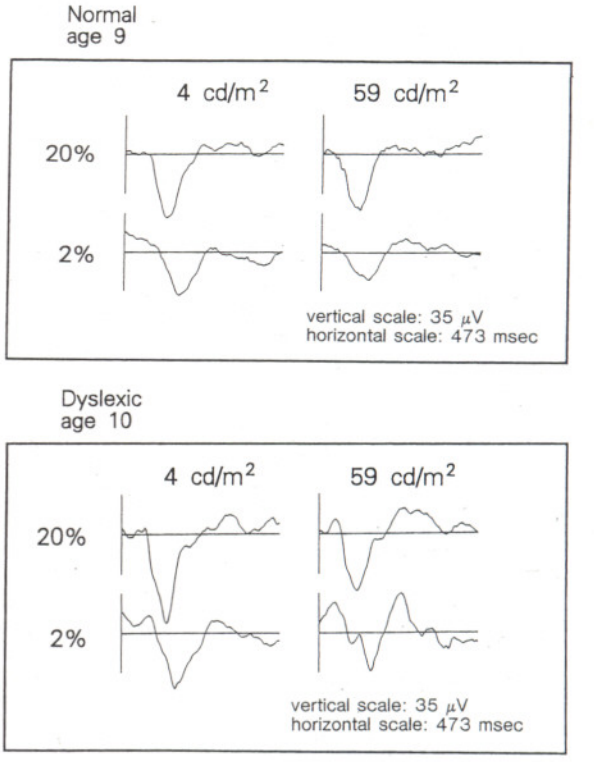

Fig. 1. Transient VEPs obtained from a typical normal subject and a typical dyslexic.

differences to become significant. Response components at a latency of 40 ms (Livingstone et al., 1991) were not seen in any subject.

Across groups (Fig. 2), subjects showed a general decrease in VEP amplitude with increasing age. There was a marked reduction in latency with increasing contrast or luminance. For 4 cd/m^2 and a contrast of 2%, the average latency was 154 ms. This latency decreased to 132 ms when contrast was increased to 20% and luminance was held constant at 4 cd/m^2. At 59 cd/m^2 and a contrast of 2%, the average latency was 138 ms. This latency decreased to 117 ms when contrast was increased to 20% and luminance was held constant at 59 cd/m^2. All of these differences were statistically significant ($P < 0.001$ by paired t-test for each comparison). However, there were no significant between-group differences of VEP latency at higher luminance and/or contrast. The size of the within-individual latency shifts with luminance or contrast increases also did not differ significantly between groups. Latencies and amplitudes of the two dyslexic patients who met "educational" criteria for dyslexia were typical of those of their age-matched controls.

Steady-state responses

Examples of steady-state VEPs obtained under the 12 conditions (three temporal frequencies × two luminances × two contrasts) are shown in Fig. 3. We initially consider steady-state responses elicited at low luminance, low contrast, and high temporal frequency (16 Hz). We used the T_{circ} statistic with a false-

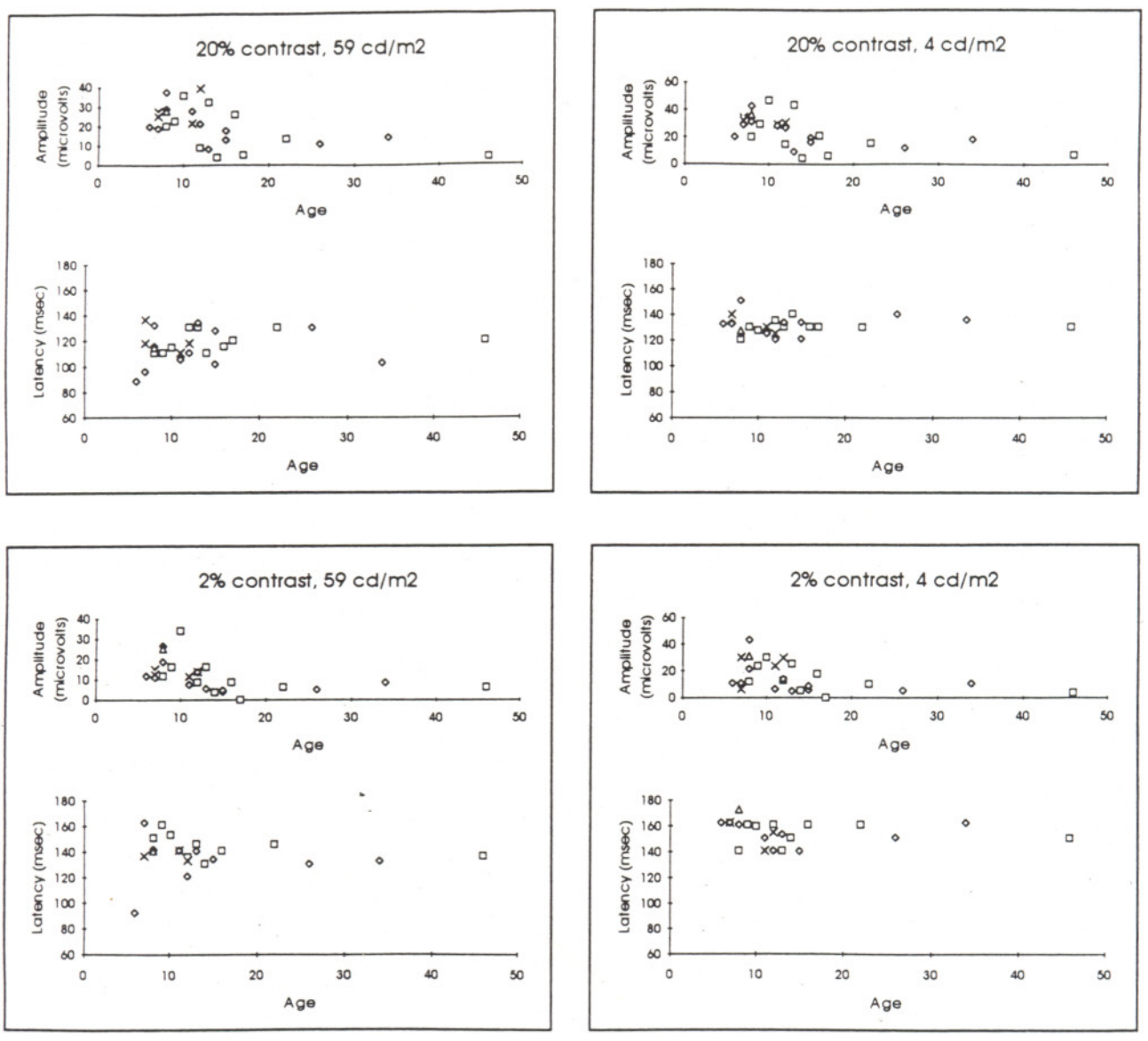

Fig. 2. Amplitude and latency of transient VEPs as a function of age.

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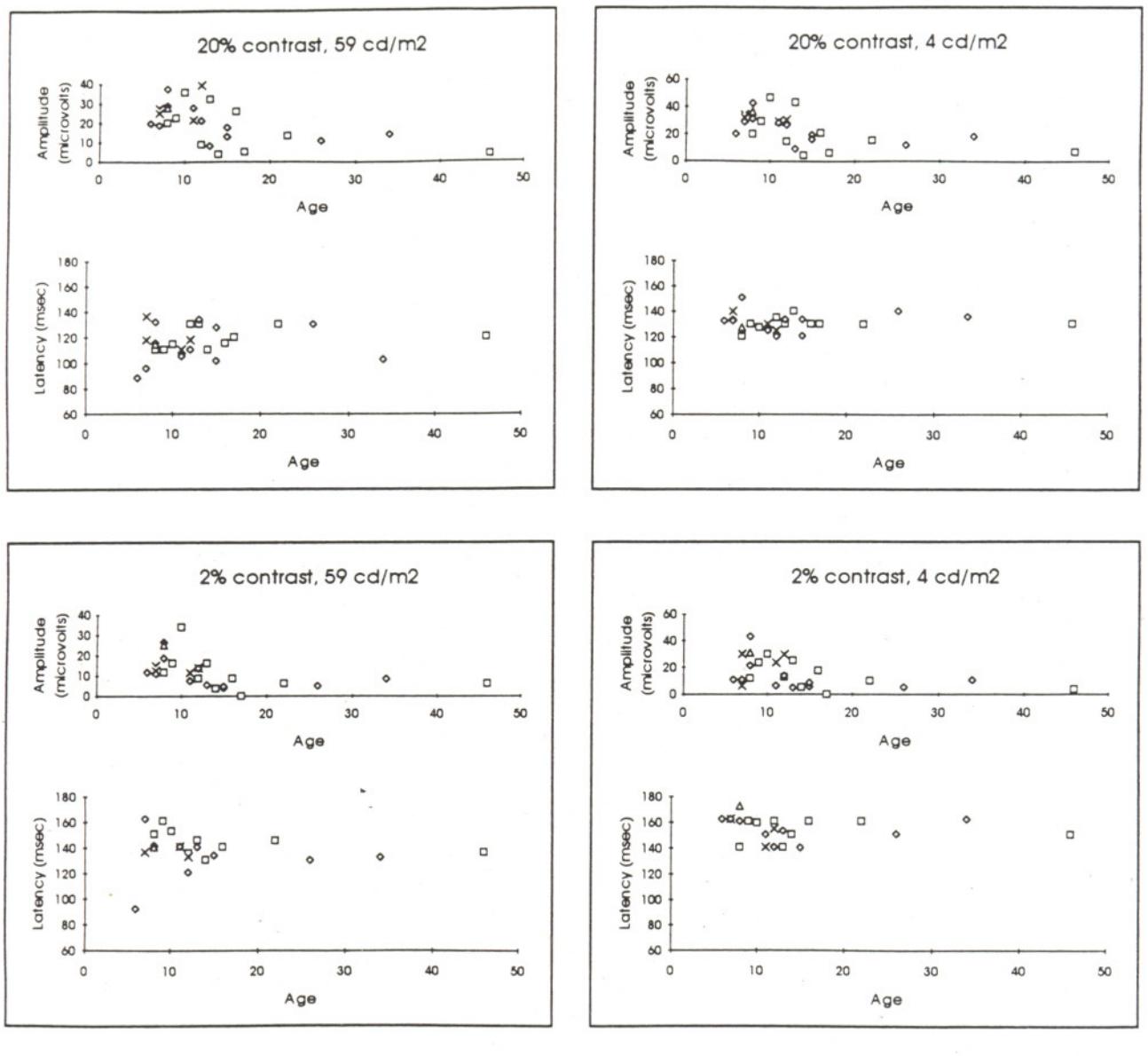

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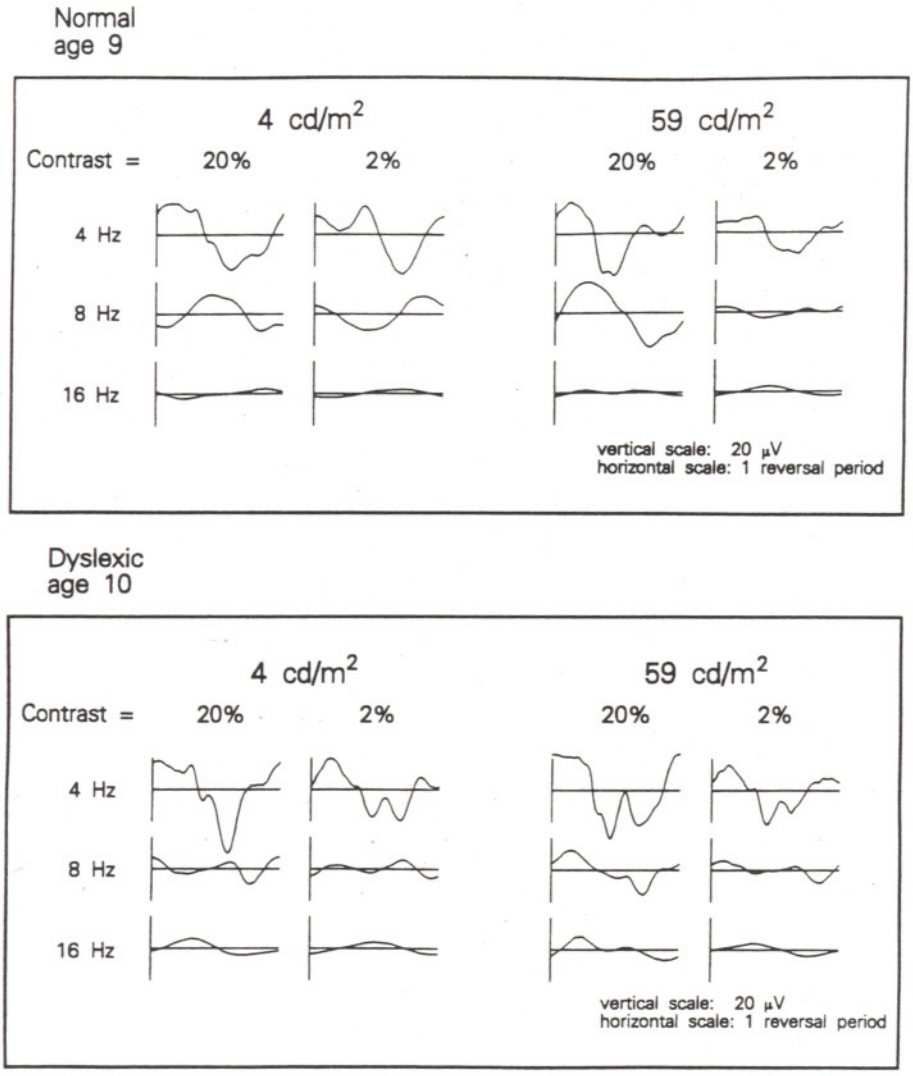

Fig. 3. Steady-state VEPs obtained from a typical normal subject and a typical dyslexic.

positive rate of 0.05 and 8-s data segments (Victor & Mast, 1991) to determine whether a steady-state response was statistically different from zero. Across groups, most subjects' responses were not significantly different from zero for this condition (16 of 26). The fraction of individuals in the dyslexic group who did have detectable responses to this stimulus (2 of 10) did not differ significantly from those in the other groups (8 of 16) (one-tailed $P = 0.13$ by Fisher's exact test). Neither of the two dyslexics who did have detectable responses to this condition were in the subgroup of acquired dyslexics. Of the two dyslexics who met "educational" criteria, one had a significant response to this condition, and one did not.

We considered the possibility that the lower frequency of detectable responses among dyslexics might represent a suggestion of a distinguishing characteristic, even though it did not reach statistical significance. One approach to this question is to ask whether a similar finding would persist, had we analyzed only one-half of the data from each subject. In this case, two of ten dyslexics would have had detectable responses, and three of the 16 non-dyslexics would have had detectable responses—essentially identical fractions. A more systematic approach to comparison of the steady-state responses is to quantify them by their Fourier coefficients (last panel of Fig. 4), and to compare the Fourier coefficients across groups *via* the Hotelling T^2 statistic (Anderson, 1958). This statistic assumes that the Fourier coefficients, considered as a pair of numbers (real and imaginary parts), have a bivariate Gaussian distribution within each group, but that the covariance matrix of this distribution is unknown. With this statistic, we found no significant difference between groups of the steady-state responses elicited at 16 Hz, low luminance, and low contrast between groups (dyslexics *vs.* non-dyslexics: $P = 0.381$; dyslexics *vs.* normals, $P = 0.549$). Thus, there is no suggestion of a difference of responses of dyslexics and non-dyslexics under the steady-state conditions employed by Livingstone et al. (1991).

We also compared steady-state responses obtained at the other 11 conditions studied (see Fig. 4). The responses in the dyslexic group were compared with either all non-dyslexics, or the normal group alone. The values of the T^2 statistic (Table 1) revealed no significant differences under *a posteriori* statistics: of the 22 P values, one was less than 0.05. These findings were not substantially changed by exclusion of the two acquired dyslexics, or of all but the two dyslexics who met "educational" criteria.

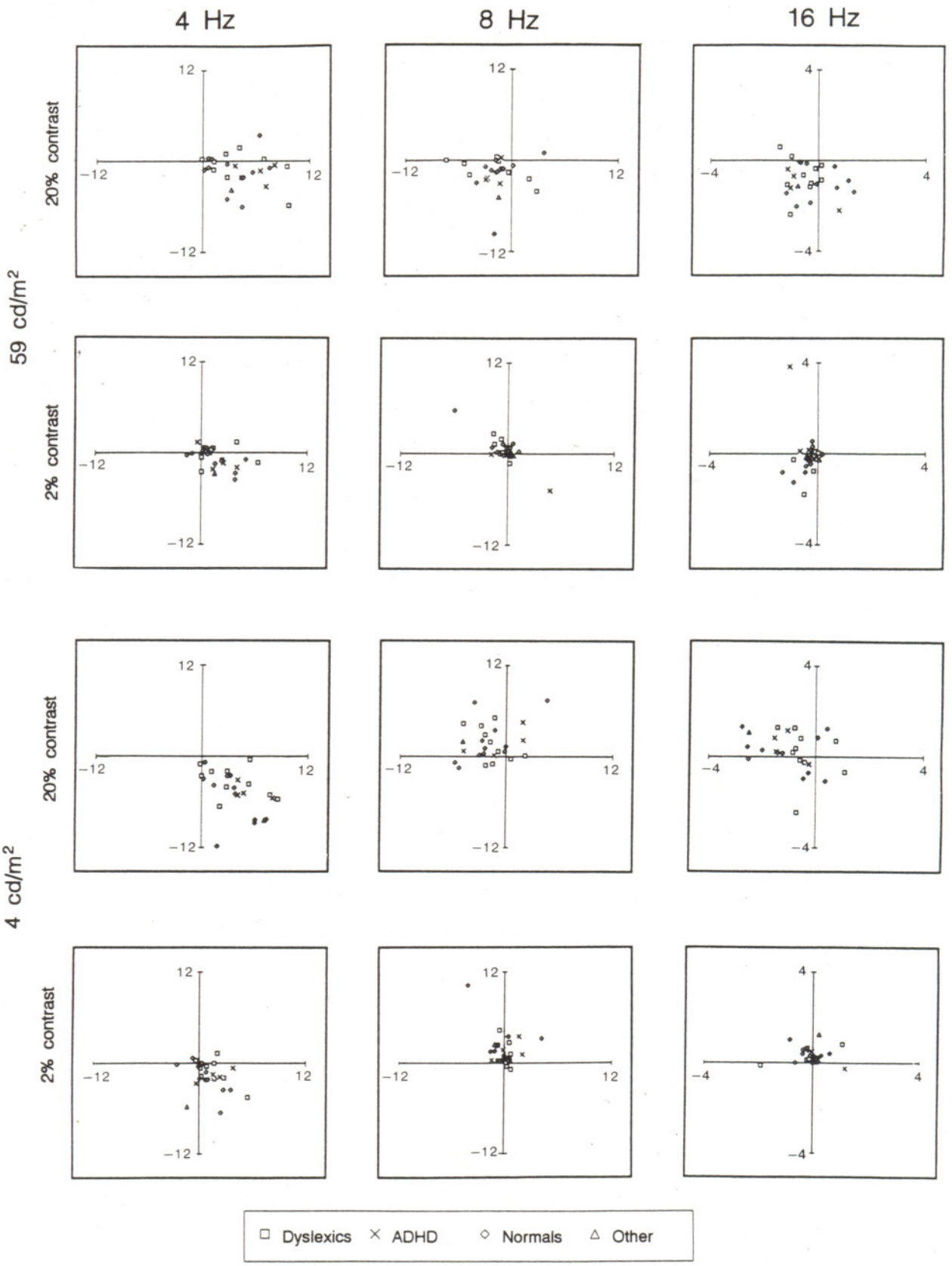

Fig. 4. Steady-state VEPs obtained from the study population. The VEP from each subject is quantified by its Fourier component at the reversal frequency. The real and imaginary part of each Fourier component is plotted as a vector in the complex plane.

Table 1. *Significance levels of differences between steady-state responses and response variabilities of dyslexics and controls*

	Dyslexic *vs.* Non-Dyslexic		Dyslexic *vs.* Normal		ADHD *vs.* Normal	
	P^a	P^b	P^a	P^b	P^a	P^b
59 cd/m^2						
20% contrast						
4 Hz	0.813	0.272	0.798	0.566	0.219	0.314
8 Hz	0.696	0.071	0.831	0.172	0.792	0.633
16 Hz	0.428	0.685	0.317	0.304	0.581	0.035*
2% contrast						
4 Hz	0.331	0.102	0.536	0.357	0.985	0.167
8 Hz	0.871	0.049*	0.791	0.123	0.098	0.126
16 Hz	0.258	0.525	0.326	0.829	0.194	0.041*
4 cd/m^2						
20% contrast						
4 Hz	0.153	0.288	0.189	0.828	0.264	0.061
8 Hz	0.861	0.168	0.822	0.329	0.619	0.362
16 Hz	0.137	0.379	0.271	0.974	0.836	0.102
2% contrast						
4 Hz	0.149	0.121	0.047*	0.576	0.446	0.023*
8 Hz	0.431	0.075	0.451	0.311	0.780	0.050
16 Hz	0.381	0.734	0.549	0.551	0.501	0.010*

*$P < 0.05$.
[a]Difference between means.
[b]Difference between response variabilities.

Response variability

A possible confounding factor in comparing the presence of detectable VEP signals across groups might be a difference in the variability of the VEP response in each individual. Contributors to variability in the measured VEP response include ongoing EEG independent of the stimulus, stimulus-elicited changes in the EEG which are not phase-locked to the stimulus (Mast & Victor, 1991), and contamination by nonelectroencephalographic sources of noise, such as scalp electromyographic signals and movement artifact. For each individual, variability of the steady-state EEG was assessed by the T_{circ} statistic, and comparisons of the individual levels of variability across the study populations were performed by the t-statistic (applied to the logarithms of the variability estimates of the responses from each individual). Steady-state response variability (Table 1) was similar in dyslexics and normal controls ($P > 0.05$ in all of 12 conditions), but was significantly greater in ADHD subjects than in normal controls ($P < 0.05$ in four of 12 conditions).

Correlation with psychometric testing

We considered the possibility that VEP measures might correlate with formal measures of reading ability or other psychometric tests. We constructed a VEP index that expressed the relative attenuation of the response under conditions of high temporal frequency, low luminance, and low contrast:

$$\log\left\{\frac{[\text{response amplitude (16 Hz, 2\% contrast, 4 cd/m}^2)]}{[\text{response amplitude (2 Hz, 20\% contrast, 59 cd/m}^2)]}\right\}$$

Since the index is based on within-individual response ratios, it will minimize the effect of inter-individual differences in overall amplitude. Fig. 5 shows scattergrams of the relationship of this VEP index to the psychometric scores. As seen in Table 2, the psychometric scores indeed separate the dyslexic subjects from the controls despite possible influences of remediation on the dyslexic individuals' scores; however, this separation is not correlated with the VEP index.

Discussion

We found that checkerboard reversal VEP responses elicited under standard testing conditions or conditions designed (Livingstone et al., 1991) to favor magnocellular contributions were similar in dyslexics and nondyslexics. VEP responses reported (Livingstone et al., 1991) to be selectively absent in dyslexics were also absent in normal subjects. We now consider possible explanations for the discrepancy between our results and those of Livingstone et al. (1991). It is unlikely that differences in stimulus characteristics underlie this discrepancy. The spatial characteristics of the visual stimuli were essentially identical to those used by Livingstone et al. (1991). The slight differences in temporal frequency cannot account for the discrepancy in our results, since Livingstone et al. reported reduction of responses over a frequency range from 5 to 15 Hz, with the most dramatic effect at the highest temporal frequency.

The subjects in the Livingstone et al. (1991) study were adults; our subjects were predominantly children. This raises the possibility that the previously reported abnormalities are a consequence of altered visual experience. Furthermore, dyslexics likely represent a heterogeneous population, and differences in diagnostic criteria therefore need to be considered as well. However, it is highly unlikely that the discrepancy between the present study and that of Livingstone et al. (1991) was due to the definition of dyslexia or to the selection of subjects, because (1) we found that the transient response claimed by Livingstone et al. (1991) to be selectively absent in dyslexics was also absent in normal subjects, and (2) we found no difference between the dyslexics who met "educational" criteria and those who did not. The dyslexics in the present study were diagnosed by historical criteria of unexpected reading difficulties, and did not necessarily meet the educational system criteria of a reading ability of 1.5 to 2 years behind grade level. The biological validity of such criteria remains to be established (Duffy & Geschwind, 1987; Nass, 1991).

A third possible explanation for the discrepancy in results is a difference in technical aspects of VEP recording. However, the recording sites (C_Z and O_Z) were the same in both studies, and amplification and filtering were similar.

A final difference between our study and that of Livingstone et al. (1991) is the method of data analysis. In our study, data were saved without averaging, and Fourier components were calculated not only from the entire data segment but also from subsegments. This procedure enabled us to measure both the size of the average steady-state response and its variability. Comparison of average response size and variability in turn provided a rigorous criterion for when signal was present (Victor & Mast, 1991). In the absence of this procedure or its equivalent, it is not possible to distinguish between a Fourier component in the averaged record which is driven by the stimulus, and one which simply reflects the ongoing electroencephalogram and nonelectroencephalographic noise sources.

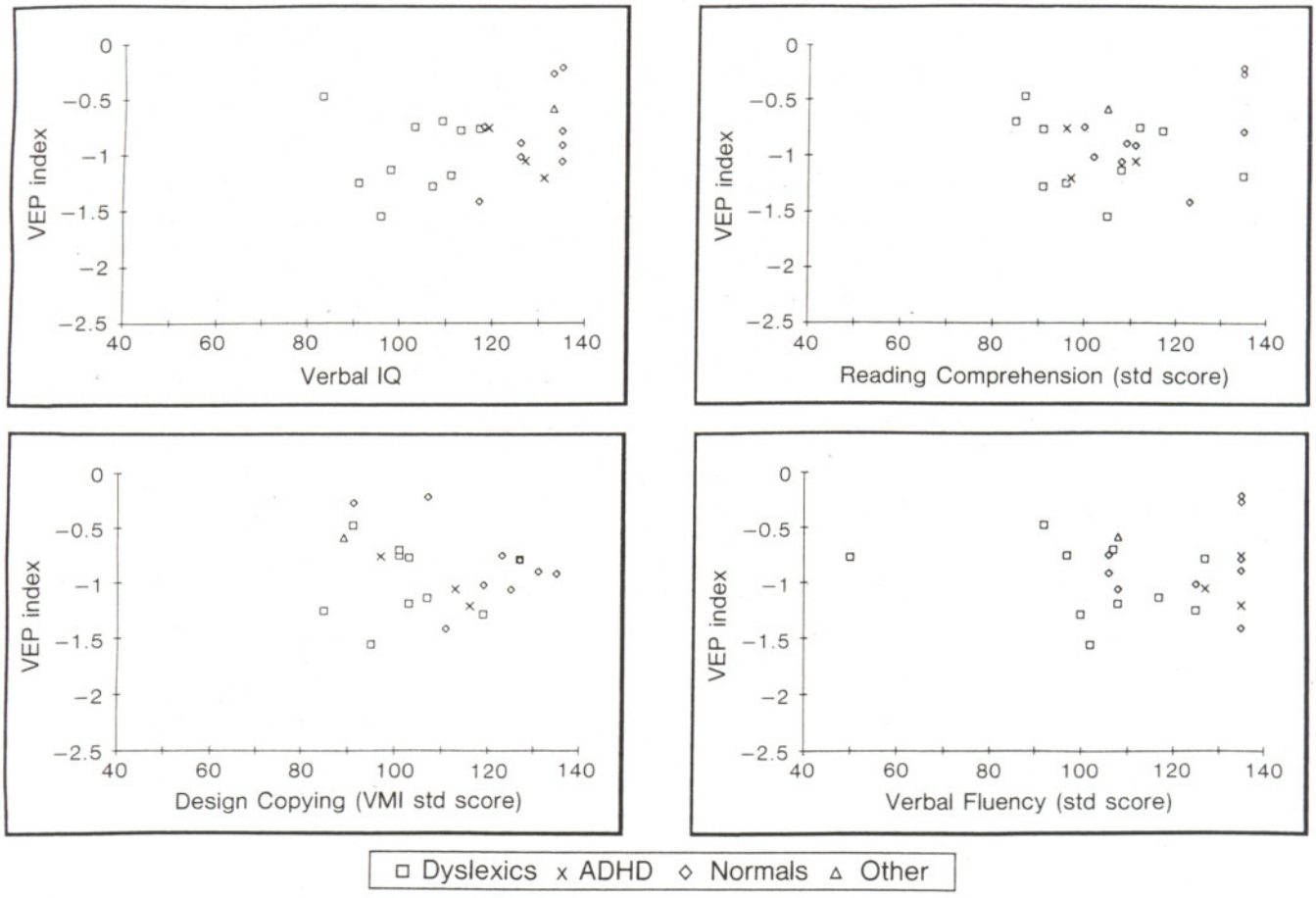

Fig. 5. Relationship of VEP index to psychometric test scores.

Table 2. *Psychometric scores and their correlations with the VEP index*

	Neuropsychological testing			
	Verbal IQ	Reading comprehension	Design copying	Verbal fluency
Dyslexics				
Mean	102.8	102.7	103.2	102.5
S.D.	10.7	15.8	12.4	21.8
ADHD				
Mean	115.0	101.3	102.7	122.7
S.D.	21.9	8.4	14.5	19.5
Other				
Mean	133.0	105.0	89.0	108.0
S.D.	—	—	—	—
Normals				
Mean	128.9	117.6	118.8	124.4
S.D.	7.4	14.6	13.7	13.7
t-test for significance between groups				
Dyslexic *vs.* non-Dyslexic	<0.001[a]	0.096	0.131	0.008[a]
Dyslexic *vs.* Normal	<0.001[a]	0.024[a]	0.063	0.020[a]
Dyslexic *vs.* ADHD	0.115	0.855	0.951	0.029[a]
ADHD *vs.* Normal	0.006[a]	0.021[a]	0.038[a]	0.758
Correlation of VEP measure with psychometric scores				
VEP index	0.222	0.184	−0.184	−0.016
P value	0.137	0.187	0.816	0.532

[a] $P < 0.05$

There are two caveats that should be emphasized. The first is that the sensitivity or specificity of this (or any other) VEP paradigm for the detection of magnocellular deficits is unknown. Secondly, despite the negative results reported here, we cannot conclude that we have ruled out the role of visual sensory abnormalities in dyslexia, or of M-cell deficits in a subpopulation of dyslexics. There are several reports of visual sensory abnormalities in dyslexia (Lehmkuhle et al., 1992; Stuart & Lovegrove, 1992; May et al., 1991; Lovegrove et al., 1990), which this study does not purport to negate. However, in view of our clearly negative findings, it is unlikely that the basis of these abnormalities is a loss of magnocellular function readily manifest in the VEP that is causally and specifically related to dyslexia in general.

Acknowledgments

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