

INFORMATION TO USERS

This was produced from a copy of a document sent to us for microfilming. While the most advanced technological means to photograph and reproduce this document have been used, the quality is heavily dependent upon the quality of the material submitted.

The following explanation of techniques is provided to help you understand markings or notations which may appear on this reproduction.

1. The sign or "target" for pages apparently lacking from the document photographed is "Missing Page(s)". If it was possible to obtain the missing page(s) or section, they are spliced into the film along with adjacent pages. This may have necessitated cutting through an image and duplicating adjacent pages to assure you of complete continuity.
2. When an image on the film is obliterated with a round black mark it is an indication that the film inspector noticed either blurred copy because of movement during exposure, or duplicate copy. Unless we meant to delete copyrighted materials that should not have been filmed, you will find a good image of the page in the adjacent frame. If copyrighted materials were deleted you will find a target note listing the pages in the adjacent frame.
3. When a map, drawing or chart, etc., is part of the material being photographed the photographer has followed a definite method in "sectioning" the material. It is customary to begin filming at the upper left hand corner of a large sheet and to continue from left to right in equal sections with small overlaps. If necessary, sectioning is continued again—beginning below the first row and continuing on until complete.
4. For any illustrations that cannot be reproduced satisfactorily by xerography, photographic prints can be purchased at additional cost and tipped into your xerographic copy. Requests can be made to our Dissertations Customer Services Department.
5. Some pages in any document may have indistinct print. In all cases we have filmed the best available copy.

University
Microfilms
International

300 N. ZEEB RD., ANN ARBOR, MI 48106

8123023

KENNIFF, SUSAN MELISSA

VISUAL TEMPORAL PERCEPTION PROCESSING DEFICIT AND
NEUROPSYCHOLOGICAL TEST PROFILES IN ADULT DYSLEXICS

City University of New York

PH.D. 1981

University
Microfilms
International 300 N. Zeeb Road, Ann Arbor, MI 48106

Copyright 1981

by

Kenniff, Susan Melissa

All Rights Reserved

VISUAL TEMPORAL PERCEPTION PROCESSING DEFICIT AND
NEUROPSYCHOLOGICAL TEST PROFILES IN ADULT DYSLEXICS

by

Susan Kenniff

A dissertation submitted to the Graduate Faculty
in Psychology in partial fulfillment of the
requirements for the degree of Doctor of
Philosophy, The City University of New York

© COPYRIGHT BY

SUSAN KENNIFF

1981

This manuscript has been read and accepted for the Graduate Faculty in Psychology in satisfaction of the dissertation requirements for the degree of Doctor of Philosophy

April 30, 1981 Louie J. Gerstman
date Chairman of the Examining Committee

May 1, 1981 Martin L. Hoffman
date Executive Officer

Dr. Louis Gerstman

Dr. Elkhonon Goldberg

Dr. Jeffrey J. Rosen
Supervisory Committee

The City University of New York

Abstract

VISUAL TEMPORAL PERCEPTION PROCESSING DEFICIT AND NEUROPSYCHOLOGICAL TEST PROFILES IN ADULT DYSLEXICS

by

Susan Kenniff

Advisor: Professor Louis J. Gerstman

This tripartite study of adult developmental dyslexics and normal reader control subjects was designed to evaluate neuropsychological test battery (NTB) profiles; to investigate visual field (VF) and center field (CF) visual temporal perceptual processing performances; and finally, to determine correspondences between the clinical and experimental research areas examined in these two adult reader groups.

Employing stringent diagnostic criteria, 10 adult developmental dyslexics were selected and matched with 10 normal readers for age (18-26), sex (male), handedness (right), race (Caucasian) and education (at least high school graduate). A comprehensive NTB compiled to delineate dyslexic and normal reader profiles across a broad range of functional processes assessed reading, verbal (excluding reading), visual, sensory/motor and arithmetic performances. The verbal identification experiment

(tachistoscopic analogue) included four task presentations separated by rest periods. The first and fourth presentation consisted of a bilateral mode condition (stimuli counterbalanced in the two VF) with the second and third intervening presentations represented by unilateral mode conditions (VF stimuli occurring exclusively in either the LVF or RVF for each condition). In ten minutes a total of 580 single-digit stimuli were presented CF, flashed for 15 msec in rapid succession; of these 580 digits, 540 were nonzeros and 40 were zeros. Each CF zero, presented at irregular intervals, was accompanied by a nonzero digit simultaneously flashed for 15 msec in either the LVF or RVF. Interstimulus timing of 1 sec was maintained when only CF stimuli were presented and this timing was extended to 1.5 sec when simultaneous CF/VF stimuli occurred.

Analysis of adult dyslexic NTB profiles relative to those of normal readers in this investigation and comparison of adult dyslexic psychometric and achievement test performances to those found in clinical research of child developmental dyslexics both provide strong evidence for persistence of functional deficits (in extent and kind) into adulthood. This finding of continued functional impairment, in the context of dyslexia etiology, fails to support the "maturational lag" theory and neither justifies nor refutes a "cerebral deficit" hypothesis; in addition, slight evidence is offered for the possibility of significant melioration of the dysfunction through remediation.

Essentially comparable dyslexic and normal reader overall VF performances occurred in this experiment with no consistently defined VF preferences identifiable, across unilateral and/or bilateral mode conditions, in either reader group. In contrast significant group

differences, despite the dyslexic large within-group variability, were manifested in CF performance across both unilateral and bilateral mode conditions. Almost complete absence of overlap in score distribution between the two reader groups further demonstrated the singularity and severity of dyslexic CF impairment. The rate of stimulus presentation variable discriminating VF and CF experimental task demands and the disparity of VF and CF performance results in dyslexics relative to those of normal readers taken together support the conclusion that dyslexics experience specific difficulty in a verbal identification task (involving no memory component) which necessitates the processing of visual-verbal stimuli (of short duration) presented CF regularly, successively, and rapidly. This marked dyslexic CF performance decrement may be attributed to a temporal perceptual processing deficit, possibly symptomatic of a subtle dysfunction in rudimentary foveal perception.

Correlation profiles, particularly within the dyslexic group, showed a positive relationship between CF performance ability and reading skills; this relationship, consistent and circumscribed in dyslexics, suggests a latent perceptual and/or temporal mechanism integral to both CF performance and reading process demands.

With appreciation for the financial support rendered by
the Joseph P. Rosen Foundation on behalf of this research project.

TABLE OF CONTENTS

	PAGE
ABSTRACT.....	iv
ACKNOWLEDGMENT.....	vii
LIST OF TABLES.....	xi
LIST OF FIGURES.....	xiv
Chapter	
I. INTRODUCTION	
Developmental Dyslexia--A Pragmatic	
Conceptualization.....	1
Neurological Theories of Developmental Dyslexia	
Orton--Defect in Organization of Cerebral	
Function.....	6
Geschwind--Bilateral Parietotemporo-	
Occipital Maldevelopment.....	10
Interhemispheric Articulation Dysfunction.....	16
II. RELATED RESEARCH	
Hemispheric Visual Processing Capacity.....	42
Laterality and Developmental Dyslexia.....	47
Hemispheric Vigilance.....	59
Temporal Order Perception.....	66
Research Hypotheses Under Investigation.....	70
III. METHODS	
Subjects.....	72
Apparatus.....	78

	PAGE
General Experimental Design.....	85
Procedure.....	89
Scoring.....	91
IV. RESULTS	
Neuropsychological Test Battery.....	93
Reading Tests.....	94
Verbal Tests (Excluding Reading).....	94
Visual Tests.....	96
Sensory/Motor Tests.....	98
Arithmetic Test.....	98
Experimental Task.....	98
Analysis I.....	99
Analysis II.....	103
Analysis III.....	106
Analysis IV.....	111
Analysis V.....	111
Analysis VI.....	111
Correlation Analysis of NTB and Relevant	
Experimental Task Measures.....	113
Reading Tests.....	115
Verbal Tests (Excluding Reading).....	115
Visual Tests.....	118
Sensory/Motor Tests.....	120
CF Experimental Task Measures.....	122
Addendum.....	125
V. DISCUSSION.....	126

	PAGE
APPENDICES	
A. Individual Dyslexic and Normal Reader NTB Profiles.....	146
B. Computer Program Printout of Experimental Task.....	187
C. Equipment Design Description.....	199
D. Luminosity Measurement Data.....	211
NEUROPSYCHOLOGICAL TEST BATTERY REFERENCES.....	222
REFERENCE LIST.....	224

LIST OF TABLES

TABLE	PAGE
1. Mean Age, Education & Intelligence Test Scores by Reader Group.....	75
2. Mean Reading, Spelling & Arithmetic Test Scores by Reader Group.....	76
3. Additional Mean NTB Test Scores by Reader Group.....	77
4. Statistical Summary of Univariate t Tests of Reading Scores for the Two Reader Groups.....	95
5. Statistical Summary of Univariate t Tests of Verbal (Excluding Reading) Scores for the Two Reader Groups...	97
6. Split-Plot ANOVA of Incorrect VF Identifications on the Experimental Task as a Function of Group (Dyslexic, Normal Readers), Time (Bilateral 1, Bilateral 2), Rate (3, 10, 18, 27), and Field (Left, Right).....	100
7. Results of the Simple Time X Field Interaction in the Dyslexic Group.....	102
8. Split-Plot ANOVA of Aggregate VF Deficit Scores on the Experimental Task as a Function of Group (Dyslexic, Normal Reader), Time (Bilateral 1, Bilateral 2), and Field (Left, Right).....	105
9. Split-Plot ANOVA of Correct-But-Late VF Responses on the Experimental Task as a Function of Group (Dyslexic, Normal Reader), Time (Bilateral 1, Bilateral 2), and Field (Left, Right).....	107
10. Split-Plot ANOVA of Incorrect VF Identifications on the	

TABLE	PAGE
Experimental Task as a Function of Group (Dyslexic, Normal Reader), Field (Left, Right), and Rate (3, 10, 18, 27).....	108
11. Results of the Simple Rate X Field Interaction in the Normal Reader Group.....	110
12. Split-Plot ANOVAs of Incorrect CF Identifications, Correct-But-Late CF Responses and Aggregate CF Deficit Scores on the Experimental Task as a Function of Group (Dyslexic, Normal Reader), and Time (Bilateral 1, Bilateral 2).....	112
13. Split-Plot ANOVAs of Incorrect CF Identifications, Correct-But-Late CF Responses and Aggregate CF Deficit Scores on the Experimental Task as a Function of Group (Dyslexic, Normal Reader), and Field (Left, Right).....	114
14. Correlations Among Reading Tests and Significant Correlations Between Reading Scores and Other NTB Scores for Dyslexic and Normal Reader Groups.....	116
15. Correlations Among Verbal Tests (Excluding Reading) and Significant Correlations Between Verbal Scores and Other NTB Scores for Dyslexic and Normal Reader Groups.....	117
16. Correlations Among Visual Tests and Significant Correlations Between Visual Scores and Other NTB Scores for Dyslexic and Normal Reader Groups.....	119
17. Correlations Among Sensory/Motor Tests and Significant	

TABLE

PAGE

Correlations Between Sensory/Motor Scores and Other NTB Scores for Dyslexic and Normal Reader Groups.....	121
18. Correlations Among CF Scores of the Experimental Task and Significant Correlations Between CF Scores and NTB Scores for Dyslexic and Normal Reader Groups...	123

LIST OF FIGURES

FIGURE		PAGE
1.	Group X Time X Field Interaction on Bilateral Mode Condition with Mean Incorrect VF Scores.....	101
2.	Group X Time X Field Interaction on Bilateral Mode Condition with Mean Aggregate VF Deficit Scores.....	104
3.	Group X Field X Rate Interaction on Unilateral Mode Condition with Mean Incorrect VF scores.....	109

I. Introduction

Developmental Dyslexia--A Pragmatic Conceptualization

The conceptual overview of dyslexia which has guided this research postulates a language disorder that renders the acquisition of reading skills--the ability to derive meaning from the written word--extremely difficult (Critchley & Critchley, 1978). The condition, essentially cognitive in nature, constitutes a deficit in the capacity to elicit information from the symbolic representation implicit in the written word. This view of language as "verbal-symbolic-representational behavior" considers vital the "symbol-processing" element in human learning; thus information must, "as it progresses cognitively from reception to expression, be transduced into representational units and coded on the basis of meaning" (Myklebust, 1978, p. 3). Dysfunction in this area--dyslexia--is manifested initially in the form of a learning disability which deters the acquisition of reading skills and later as a severe impairment to reading performance. Among the performance problems may be included word recognition--inability to pronounce an unfamiliar word or to distinguish between similar words; the reversal of individual letters and word order; difficulty in tracking--maintaining focus on the correct place or repositioning focus from line to line; and repetition, substitution, addition, and omission of letters and words in reading text (Critchley, 1970).

An effort to move from defining dyslexia in general terms to describing the actual group designated poses inherent problems. Proliferation of terms and controversy in agreeing on a distinct

categorization has been noted by both theoreticians and clinicians in this field (Spreeen, 1976). From an operational perspective the dyslexic, experiencing his particular reading dysfunction, is characterized as undergoing extreme difficulty in the identification of single written words, as well as difficulty in analyzing their component sounds. Other considerations that separate dyslexics from backward readers require a differentiation of relative syndromes; thus dyslexics with marked decoding problems must be distinguished from backward readers with adequate decoding skills but poor comprehension (Vellutino, 1978, 1979, chap. 2; also see Fischer, Liberman & Shankweiler, 1978). Rutter and Yule (1975), in perhaps the most thorough series of epidemiological research projects yet undertaken with handicapped children, identified and studied two distinct groups reading well below grade level (any correspondence with Vellutino's two groups has yet to be determined). The general reading backwardness group included those children attaining test scores on reading accuracy and comprehension significantly below their chronological age level, regardless of IQ; the specific reading retardation group (dyslexics)¹ included those children scoring significantly below that reading level predicted on the basis of both age and IQ. Their findings indicated meaningful differences between these two syndromes with the former group, in contrast to the dyslexics, exhibiting

¹The terminology preoccupation exhibited by various researchers in this field--particularly their attempt to differentiate between dyslexia and specific reading disability--is counter-productive "because the renaming of a problem implies greater precision than understanding warrants; it is a disservice to the diagnostician as well as the patient, in that we ourselves come to believe we have said something more than ordinary language serves to convey" (Eisenberg, 1979, p. 48).

a more equal sex distribution, the presence (in some individuals) of overt neurological disorder, a greater incidence of neuro-developmental deficits, and better educational prognosis in terms of reading and spelling.

Diagnosis and classification of a dyslexic assumes adequate native-language reading instruction "in kind and amount sufficient for his peers" (Eisenberg, 1978, p. 41). The single defining inclusionary guide to characterize functional dyslexics is the manifestation of a severe reading performance deficit relative to individual age and intelligence (Benton, 1975). Developmental dyslexia study, based on extensive clinical and experimental research, has categorized the dyslexic--utilizing absolute differences (Rabinovitch, 1954), an achievement quotient (or ratio) (Myklebust, 1967), or a multiple regression formula (Rutter & Yule, 1975)--as an individual with average or above-average intelligence who is functioning well below reading level expectancy.

Exclusionary factors that disqualify potential dyslexic subjects include low IQ as well as sensory, neurological and emotional disabilities and environmental disadvantages (Vellutino, 1979). These considerations, while anything but arbitrary, are intended to avoid the problem of introducing extrinsic variables that influence results and blur the investigation focus. Measurement of intelligence is negatively influenced by reading difficulties. The realization "that given tests of intelligence may be differentially related to reading skills and subskills" (Vellutino, 1979, p. 15) further compounds the problem. Visual acuity and other ocular deviations, as well as auditory dysfunctions, can create reading difficulties of a secondary nature that are subject to physical correction and are not endemic to dyslexia.

Gross neurological deficits affecting the ability to abstract and generalize--skills important to reading--are also considered secondary causes of reading disability and thus these nonreaders are not included in dyslexia studies. Finally, the study of individuals with a history of severe emotional and social problems, as well as distinct socioeconomic disadvantages, is subject to cause-effect conflicts influencing reading-test performances. Admittedly, cultural factors may coexist with particular reading disabilities--including dyslexia--but the obstacles to a clear analysis of the causal relationship involved tend to eliminate such disadvantaged individuals as valid dyslexic subjects.

Critchley and Critchley (1978), in their analytical attempt to define dyslexia, proposed that "it probably represents a specific maturational defect which tends to lessen as the child grows older, and is capable of considerable improvement, especially when appropriate remedial help is afforded at the earliest opportunity" (p. 149). Implicit in this conception is the view that performance patterns of older dyslexic readers should qualitatively resemble those of younger normal readers (Duane, 1974). This model, advocating the possibility of the dyslexic eventually "outgrowing" his disability, places Critchley and Critchley with that group (de Hirsch, Jansky & Langford, 1966; Satz & Van Nostrand, 1973) who conceive of developmental dyslexia as the result of a delay in the maturation process of the cerebral cortex. Commensurate delay in "those crucial early sensori-motor and later conceptual-linguistic skills which are intrinsic to the acquisition of reading" (Satz, Friel & Rudegeair, 1974, p. 176) serves to retard the "sequential and the hierarchical stages" (Satz, Friel & Goebel, 1975, p. 108) involved in the reading process.

In contrast, the model put forth by Reitan (1964) and Doebling (1968) posited a cerebral "deficit" as an etiological explanation for difficulties in the acquisition of reading skills. Benton (1975) categorized the "deficit" explanation of this dysfunction into two broad classes: the first postulates a "focal maldevelopment" (see Bastian, 1898; Geschwind, 1965; Hinshelwood, 1900; Morgan, 1896) of the brain while the second concentrates on a defect "in the overall organization of cerebral function" (p. 31) (see Orton, 1925, 1937). The former deficit view offers no expectation that young dyslexics will ever "catch up" (Rourke, 1976).

On occasion studies have attempted to follow up previously diagnosed dyslexics, but the collection of significant data has been virtually nonexistent (Trites & Fiedorowicz, 1976). Spreen (1970) had early deplored this dearth of clinical and statistical information; during the 1960s, those few researchers who involved themselves with the academic fate of childhood dyslexics concentrated on their relative success or failure in high school, college, and life in general (Balow & Blomquist, 1965; Preston & Yarrington, 1967; Rawson, 1968; Robinson & Smith, 1962). Unfortunately, such research failed to examine the reading, spelling, and writing levels to collect verifiable test data on any changes---improvement or otherwise---that might have occurred. More recent follow-up studies that have reported reading-performance progress data (Boder, 1973; Frauenheim, 1978; Rourke, 1975; Rutter & Yule, 1975; Rutter, Tizard, Yule, Graham & Whitmore, 1976; Trites & Fiedorowicz, 1976) appear to indicate only slight to moderate improvement in reading, spelling, and writing abilities as dyslexics mature---although their academic status appears to have progressed unimpeded despite their

handicap. Regrettably, Trites and Fiedorowicz (1976) note, completion of high school cannot be used as "the criterion of reading proficiency" (p. 50). Thus, although early remediation may improve some reading and related skills, and many dyslexics appear to develop compensatory techniques to circumvent this handicap, investigation reveals the persistence of residual deficits as well as a less than uniform improvement curve (Frauenheim, 1978). Older dyslexics continue to exhibit reading problems, despite earlier remediation attempts, as well as problems with writing, punctuation, copying, spelling, and word sequence.

Neurological Theories of Developmental Dyslexia

Orton--Defect in Organization of Cerebral Function

Samuel Orton first advocated a relationship between cerebral dominance and what is now termed developmental dyslexia in the second decade of the twentieth century. His original research was based on some 20 years of "interest and study from the literature, in the clinic, at the autopsy table and in the laboratory, of cerebral localization and of aphasias" (Orton, 1937, p. 11). His primary interest concerned "disorders and delays in the acquisition by children of the various functions of the language faculty" (Orton, 1932, p. 614) and, from this investigation, Orton hoped to ascertain those methods of treatment most effective in overcoming such dysfunctions. From 1925 to 1945, conducting extensive clinical testing with children, Orton's research produced an awareness of the contemporary view of hemispheric dominance and its

relationship to various language functions including speech, reading, and writing. The reading process itself was viewed as a complex cognitive procedure--one of many subserved by a single dominant hemisphere.

By 1925, Orton, while discussing "congenital word blindness," rejected the implication of this terminology as descriptively inappropriate for children who undergo unusual difficulty in learning to read. Proposed in its place was the term "strephosymbolia"--turning or reversal of words or signs. In this seminal analysis, Orton described the disability as one of "confusion, because of reversals, in the memory images of symbols resulting in a failure of association between the visually presented stimulus and its concept" (p. 610). His analysis of this condition contrasts with the pathological-factor diagnosis--cortical agenesis in the angular gyrus region of the left hemisphere--previously expounded by Hinshelwood (1917). Orton (1945/1966) rather viewed the dyslexic reading syndrome as a "physiological failure to establish the normal pattern of unilateral cerebral dominance" (p. 273), this failure deterring the acquisition of skills and negatively affecting performance in reading as well as spelling and writing.

Orton's theory is based on a three-level model of the visual cerebral cortex. These three levels correspond to different histologic types of cortex that are distributed on the mesial and lateral aspects of each of the two cerebral hemispheres. The first level, the "visual perceptive," consists of the striate areas of the two hemispheres. These two initial visual-stimuli-receptive areas operate independently and, unless bilateral lesions occur resulting in blindness, continue to function unilaterally should one be destroyed. The second level, which

Orton referred to as the "visual recognitive," consists of the parastriate areas. The function of this level involves reception of the visual stimuli transmitted from the first cortical zones. Here recognition of meaning of the visual-object-image occurs. As with the first cortical level, these areas in the two hemispheres operate independently and only through their bilateral destruction can visual object agnosia and similar defects in stimuli recognition result. The final level--the "visual associative"--is not specifically visual in function, not a direct receptor of the original stimulus, but rather this area, mediated by the parieto-occipital portion of the brain, subserves the visual identification and decoding of symbolic stimuli. Unilateral damage, specifically in the dominant hemisphere (usually the left) of this area, produces a condition in which "awareness of objectivity and recognition of the concrete meaning of objects [the printed word] are both retained" (Orton, 1925, p. 605), but their abstract or associative meanings are lost.

Optimum efficiency on this third level, in contrast to the two preceding levels, demands dominance of function in a single hemisphere and corresponding functional suppression in the remaining hemisphere. Single hemisphere control of the language faculty is evidenced by loss of understanding, speech, and writing should severe damage in language areas of this dominant hemisphere take place. Similar damage to the same areas of the nondominant hemisphere, which appear morphologically equivalent, produces no comparable dysfunction, thus providing evidence that the "more complex functions underlying language" involve "the problem of cerebral dominance" (Orton, 1928a, p. 288). Because of this functional specialization Orton (1945/1966) theorized that reading skills

and performance require a "complete elision of memory records" (p. 273) existing in the nondominant hemisphere so that antitropic or mirror-image information does not compete with reading--the "symbol-processing" interpretive operation occurring in the dominant hemisphere. The three visual cortical levels are descriptively rendered using a schematic analogy in which "the arrival platform of visual perceptive would give awareness, the overflow into the recognitive would add concrete meaning of the object or symbol and into the associative would add abstract or symbolic meaning through interlinking with sensory data from other fields" (Orton, 1925, p. 605).

From this trilevel model Orton (1928b) postulated his theory of "the obstacle to the acquisition of reading in children of normal intelligence" (p. 1051)--dyslexia. This dysfunction is the resultant of the failure of a single hemisphere to assume dominance in the perception of written word-symbols. The incomplete elimination of one set of memory traces influences response by causing confusion in the decoding process and serving "as an impediment to facile associative linkage" (p. 1051), which on the third level is necessary for the interpretive function. Orton was less than dogmatic in relating his theory of cerebral dominance and reading to other aspects of motor laterality. He cautioned against attempting to draw too rigid a parallel in associating reading disability with a particular handedness pattern. Acknowledging that a large proportion of dyslexics encountered are right-handed, right-eyed, and right-footed as well, such "distinctly unilateral motor patterns . . . does not preclude the possibility of a confusion of dominance in the parts of the cortex which have to do with

the reading process, and one sees the same symptoms as in those who do have confusion in the motor patterns" (see discussion section of Lynn's article, 1942, p. 1064).

A sense of optimism is found in Orton's prognosis of dyslexia and the possibilities of remediation. The conception of dyslexia as "a physiologic variant rather than a general mental defect or a specific brain defect" (Orton, 1928c, p. 1098) provides a solid theoretical basis for the assumption that proper remedial treatment can result in increased acquisition of reading skills and improved reading performance. Orton envisioned corrective measures aimed at counteracting antitropic or mirror-image interference from the nondominant hemisphere by training for orientation in "a consistent direction of reading," which implies "a consistent selection" (p. 1098) of competing visual engrams. Orton's final years were spent in continued research and in the training of teachers, although the remedial methodology devised--essentially supportive academically and emotionally--has never provided an actual technique that could truly correct this language impairment.

Geschwind--Bilateral Parietotemporo-Occipital Maldevelopment

Attribution of dyslexia to bilateral structural deficits in the parietotemporo-occipital or angular gyrus region has been advanced by several researchers in the field (Benton, 1975). Perhaps the most substantive overview and formulation is provided by Norman Geschwind (1962, 1965) who conceived of the dyslexia syndrome as resulting from a maldevelopment--"probably bilateral"--in the angular gyrus region.

Major aspects distinguishing the human inferior parietal lobule from that of the higher primates--phylogenetically nearest to man in the evolution of the nervous system--involves differences in development, structure, and function. In Critchley's (1953/1971) comparative analysis of studies with higher primates the human inferior parietal lobule which includes the angular and supramarginal gyri was shown to be more complexly structured than the rudimentary counterpart found in the primates. Late evolutionary development of this area in humans is characterized by a highly variable gyral structure (Critchley, 1953/1971) as well as late myelination of nerve fibers (Flechsig, 1901; Yakovlev, 1967). De Crinis (1934) had earlier observed that full development of this region's neuronal dendritic processes occurs relatively late in postnatal life and Yakovlev (cited by Geschwind, 1965) had since ascertained a similar cytoarchitectonic maturation. In addition, Yakovlev indicated that this area contains few thalamic afferents. Because individual structural differences in man are more often found in this posterior parietal area than most other cortical regions, Benton (1975) suggested a late and still incomplete evolutionary development.

From a functional perspective the major afferent and efferent nerve fibers of this region primarily interconnect with other cortical regions rather than subcortical areas (Critchley, 1953/1971). Such a plexus constitutes an "association area" and, since these connections are received from adjacent association areas, Geschwind (1965) referred to it as "an association of association areas" (p. 274). The resulting assemblage involves visual, auditory, and somesthetic impulses drawn from adjacent cortical secondary association areas and therefore functions as an anatomical-integrating-mechanism for higher-order

information processing. Theoretically this structure serves as a "way station between the primary sensory modalities and the speech area" (Geschwind, 1965, p. 276), serving to receive, integrate, and process crossmodal or intersensory stimuli. Conceivably this synthesis (in the dominant hemisphere) provides the basis for the acquisition and retention of reading skills as well as other language functions.

Geschwind (1962, 1965) conceived a neurological model of dyslexia which was derived from a reconstruction of the clinical and anatomical findings in Dejerine's (1891, 1892) seminal case histories of two patients with acquired alexia. The first patient illustrated the clinical syndrome of "pure alexia with agraphia"; the second patient's syndrome of "pure alexia without agraphia" provided a more circumscribed functional disability for comparative study. Geschwind hypothesized that analysis of these two case histories--the clinical and anatomical findings of which were subsequently confirmed and elaborated upon in more recent alexia studies (Ajax, Schenkenberg & Kosteljanetz, 1977; Benson & Geschwind, 1969; Damasio, 1977; Greenblatt, 1977; Vincent, Sadowsky, Saunders & Reeves, 1977)--offered a simple model for understanding those anatomical mechanisms contributing to acquired reading disabilities (that occur in the absence of other significant aphasic disturbances). This model in turn provided support for the identification of an anatomical substrate integral to the acquisition of reading skills in children. Thus, through methodical analytic extrapolation, Geschwind posited a possible neuroanatomical dysfunctioning mechanism responsible for developmental dyslexia.

Dejerine's (1891) first study involved a mature male who exhibited

a right hemianopia and loss of ability to read and write in the absence of other significant neurological disabilities. Autopsy eventually revealed an entirely normal brain with the exception of a lesion comprising "the inferior three-quarters of the [left] angular gyrus, terminating at a point on the external perpendicular fissure . . . penetrating to the occipital horn of the lateral ventricle . . . and destroying the major portion of Gratiolet's optic radiations" (p. 199). The following year, Dejerine (1892) proposed, based on this finding, that the left angular gyrus area functionally subserves what he termed "le centre de memoire visuelle des lettres" (p. 87) and that a lesion in this region in adults results in a loss of earlier acquired reading skills as well as writing skills.

Dejerine's (1892) second study involved a stroke patient who, in addition to sustaining a right hemianopia, suffered from a marked inability to read letters, words, and musical notation but still possessed the capacity to write and read using kinaesthetic cues. Autopsy here revealed that this syndrome could be attributed to a "lesion of the white mass of the left occipital lobe . . . and of the splenium of the corpus callosum . . . isolating the right occipital lobe from the left angular gyrus" (p. 89). From this finding Dejerine concluded that in alexia without agraphia, a resultant of a disconnection of the intact right visual cortex from the left angular gyrus (concurrent with left visual cortical damage), visual input is obstructed from reaching the "visual memory centre for words." Dejerine therefore proposed that since the left angular gyrus--"visual memory centre for words"--in this patient had remained intact, writing skills were retained; since fiber

connections from the somesthetic system to the left angular gyrus were also preserved, reading using kinesthetic cues was possible.

Geschwind (1965) utilized information derived from his own clinical studies to reinforce and extend Dejerine's anatomical findings and functional conclusions on the two alexias--that with agraphia attributable to a functional destruction of the "visual memory centre for words" and that without agraphia attributable to a disconnection of vital visual input to this "visual memory centre for words." Geschwind noted that an analysis of spelling performance in patients with acquired alexia reveals yet another functional aspect of the left angular gyrus region. An inability to spell is common to the alexia with agraphia syndrome, while in the pure alexia without agraphia syndrome, where the angular gyrus remains intact, the patient retains an ability to spell. Thus the "interchange" area of written and spoken language, Geschwind concluded, is located specifically in the left angular gyrus region which is designed for "transmitting visual-auditory cross-modal associations in both directions" as well as "the retention of 'rules of translation' from written to spoken language" (p. 279). Destruction of the left angular gyrus area and subsequent "loss of visual word memory returns the patient to the state of being illiterate; lack of reading, writing and spelling and incomprehension of spelled words are all components of this more primitive state" (p. 279).

Thus Geschwind's research into the neuroanatomy and function of the angular gyrus region, as well as the then current knowledge of the acquisition of reading skills and reading performance behavior,

led him to propose that developmental dyslexia may be attributed to structural impairment, "probably bilateral," in this area of the brain.² Geschwind's emphasis on bilateral dysfunction may be traced to the untenability of a unilateral deficit model (Hinshelwood, 1917) since hemispheric functional plasticity in relation to the development of language skills (evidenced in hemispherectomy studies) has been shown to possess both equipotential (Basser, 1962; Lenneberg, 1967; Zangwill, 1964) and transferrable capacities (Basser, 1962; Carlson, Netley, Hendrick & Prichard, 1968; Gardner, Karnosh, McClure & Gardner, 1955; McFie, 1961). Young children, especially infants, have demonstrated an easy adaptability in the transfer of language functions from an impaired cerebral hemisphere to the healthy one. While language lateralization occurs some time between childhood (Krashen, 1972, 1973; Krashen & Harshman, 1972) and puberty (Basser, 1962; Lenneberg, 1967; Obrador, 1964), the ontogenesis is a gradual process. Despite a general specialization of language functions in one dominant cerebral hemisphere, hemispherectomy research has disclosed the ability of the nondominant hemisphere to assume language control if pressed into service, the relative adjustment increasing in difficulty and complexity dependent on

²Geschwind's (Galaburda, LeMay, Kempler & Geschwind, 1978; Galaburda, Sanides & Geschwind, 1978; Geschwind, 1979) most recent work, begun in 1968 (Geschwind & Levitsky) utilizing precise anatomical measurement to examine right/left structural hemispheric asymmetries in different language areas of neurologically intact brains at autopsy, has increasingly focused on the absence of asymmetry (implying lack of a single dominant hemisphere) as a possible causative factor in some developmental disorders of language function, including dyslexia. Relative sizes of these areas in the two hemispheres, Geschwind tentatively has theorized, may also contribute to childhood learning disorders.

the age of the patient (Basser, 1962).

Interhemispheric Articulation Dysfunction

The perspective that views dyslexia as the resultant of interhemispheric articulation dysfunction assumes an overview in which all higher mental functions involve "highly organized circuitry in action" (Sperry, 1967, p. 714). Sensory stimuli, after reception and internalization, are transmitted to both hemispheres of the human brain. As described by Michael Gazzaniga (1970), these hemispheres are connected by "the largest and most mysterious information transmission system in the world--the corpus callosum" (p. 1). Conceivably during interhemispheric communication, four billion impulses (calculated at an average fiber transmission frequency of 20 impulses per second) could transverse the corpus callosum every second, a linkage system at least as effective as intracerebral tracts connecting the internal structures of each hemisphere (Eccles, 1973, chap. 6).

Papanicolaou (1980), in his average evoked potential (AEP) study of cerebral processes during auditory language tasks, examined the phenomenon of bilateral divergence--"opponent processes in dynamic interaction." Mutual excitatory and inhibitory influences, common to the "functioning of symmetric organismic structures" (p. 278)--in this instance the two cerebral hemispheres--interact to insure increased performance efficiency in terms of task specialization. Animal (Asanuma & Okuda, 1962; Berlucchi, 1965, 1966; Bremer, 1967; Eidelberg, 1969; Jung, 1961) and human (Shaltenbrand, Spuler & Wahren, 1970, 1972) electrophysiology studies have demonstrated that reciprocal excitatory

and inhibitory influences are mediated by the callosal commissure; animal (Doty, 1969; Trevarthen, 1974) and human (Bogen, 1979; Gazzaniga, 1970; Sperry, 1968a) commissurotomy studies have also indicated that a callosal disruption, resulting in a failure to monitor such opposing forces, can produce often conflicting behavioral responses. These polarized biological processes, theory holds (Sperry, 1974; also see Kinsbourne, 1974a, 1974b), are activated by either right or left hemisphere, the assignment determined by task specialization demands. The appropriate hemisphere for handling a particular procedure becomes the dominant one and exerts an inhibitory influence on its counterpart "to increase task performance efficiency" (Papanicolaou, 1980, p. 279). Evidence to support this thesis in the case of complex cognitive linguistic tasks is offered in commissurotomy study (Sperry, 1974), in aphasic study (Kinsbourne, 1974b), in hemidecorticate study (Dennis & Whitaker, 1976), and also in neurologically intact adults (Papanicolaou, 1980). Papanicolaou concluded, concerning cerebral specialization for language, that such assignment is functional and dynamic, involving the reciprocal excitation and inhibition of neuronal impulses, rather than structural, static, and strictly localized. Systemic impairment involving "complexities of cerebral interrelationship" (Money, 1966, p. 40) provides yet another theoretical basis for developmental dyslexia. Early adherents to this view (Crinella, Beck & Robinson, 1971; Money, 1966) maintained that such a breakdown of cerebral interhemispheric integration could conceivably result in learning behavior deficits, including inability in some children of otherwise normal or above normal intelligence to acquire commensurate reading skills.

Gazzaniga's (1974, 1978) dynamic-model conceptualization of

cerebral dominance involves two cognitive systems juxtaposed in the cerebral hemispheres, each assuming either a dominant or nondominant relative position depending on the perceptive task undertaken. In dyslexia, imperfect cerebral dominance, evidenced by an absence of visual field specialization (Gazzaniga's "interpretation" of Olson, 1973), is ascribed by Gazzaniga (1974) to a "poorly developed central control system [corpus callosum]" (p. 375) which fails to assign priority. Gazzaniga interpreted Orton's (1928b, 1937) dyslexia-syndrome "antitropic engrams" or "memory traces" in the nondominant hemisphere as a disruptive competing system which, "when working in parallel" (p. 376) with the dominant hemisphere, produces behavior responses that are both slowed and confused. These two "decision systems" are activated upon receipt of the same or parallel stimuli which then produce competing responses. Insufficient inhibition by one "system" and failure of a dominant focus to establish may result in severe behavioral disabilities. In the rare case where bilateral speech does not interfere with language, Gazzaniga (1978) attributed this positive behavioral response to the outcome of an efficiently functioning corpus callosum; thus this commissure serves to prevent problems with bilateral language "by providing interhemispheric speech synchronization or inhibition of the [interfering hemispheric] mechanism" (p. 81).

The developmental language function sequence evolves as the brain matures, subject to various internal and external influences. The early language encoding mechanisms emerge bilaterally and later consolidate in the dominant hemisphere (Basser, 1962; Lenneberg, 1967). (Definitive research to explain the maturational process by which the [usually] left hemisphere assumes the dominant role in normal language processing

remains to be undertaken by neuropsychologists as well as geneticists and researchers in affiliated fields.) During this early stage in brain maturation, since the interhemispheric neuronal communication system--principally the fibers of the corpus callosum which have not completely myelinated (Yakovlev, 1967)--is not fully operational, Gazzaniga (1974, 1978) viewed the young child as being in a state functionally similar to that of a split-brain condition. Because callosal nerve fibers innervating homologous language areas of the two hemispheres are not yet operationally effective, each hemisphere stores its engrams independently. Therefore, flexibility of the nondominant hemisphere to assume control of the language process in cases of severe injury to the dominant hemisphere most often occurs in infants and young children (Basser, 1962). Following the later period, in which language assignment is largely isolated in a single dominant hemisphere, subsequent injury to that hemisphere, with few exceptions and then to a limited degree, results in an inability of its nondamaged counterpart to develop or take over normal language skills. If, during this developmental process, internal and/or external influences impair the structure or capacity to function of the "cortical control system [corpus callosum]," Gazzaniga postulated, the resultant lack of a defined cerebral dominance would disrupt normal linguistic development. Such dynamic cortical disruption provides yet another cerebral dysfunctional mechanism to account for the developmental dyslexia syndrome.

A more detailed and expanded examination of this dyslexic model and the component elements which comprise it necessitates an examination of recent ontological studies involved with the language process. Language development has been described as originating as a bilateral function and later evolving toward dominance in the left hemisphere. This transition

occurs in successive stages from birth extended over a period judged to be anywhere from 5 to approximately 15 years (depending on the research study consulted). Those stages prior to complete hemispheric specialization of language, particularly reading, should not be conceived, cautioned Bakker, Smink and Reitsma (1973), "as a more primitive, diffuse mode of function." Rather, these neuropsychologists posited each stage in the ontological maturation "as a necessary episode in the development and acquisition of certain language skills" (p. 302). In their dichotic study testing the correlation of ear dominance and reading ability--a positive relationship between the two had been predicted (Satz & Sparrow, 1970) and found (Bryden, 1970; Satz, Rardin & Ross, 1971)--Bakker et al. had observed that the gradual acquisition of reading skills can be impaired should lateralization occur prematurely; conversely, their findings indicated that a lag in lateralization during the later stages of reading development can also adversely affect reading performance. Data drawn from this study suggested that progress in the acquisition of reading skills coincides with a gradually increasing lateralization. Since early reading requires the integration of both lingual and nonlingual information, right hemisphere participation serves a useful purpose and, Bakker et al. (1973) hypothesized, the effective development of this skill "may be facilitated by bilateral hemispheric processes" (p. 309). Reading deficits may stem from incidents of premature lateralization and test results of older children in this study point to improved reading performance coinciding with left hemisphere lateralization only after early reading strategies become more automatic and reading fluency is achieved.

The hemispheric strategies required in the acquisition of reading skills appear to be different from those strategies concerned with

later fluent reading performance (Sadick & Ginsburg, 1978). Initial introduction to reading skills involves visual and spatial components--(usually) right hemisphere attributes--as well as the lingual function (Smith, 1971); distinguishing letter forms is essentially a visual-spatial task and posits the need for bilateral hemispheric cooperation in this early stage. In fact, as Hellige & Webster (1979) observed in their experiment (adult subjects) on the relative hemispheric roles in the continuum of letter processing, during the initial stages the right cerebral hemisphere proves more efficient at "extracting relevant features of letters" (p. 653) when they are perceptually masked, despite the letter association in a language (reading) context. Sequencing of letters, words and finally sentences--requisite elements in reading fluency--are, however, essentially (usually) left hemisphere tasks which diminish the right hemisphere contribution in this later stage. Tomlinson-Keasey and Kelly (1979) in their tachistoscopic study of hemispheric specialization, utilizing a visual channel modality, confirmed the Bakker et al. (1973) analysis, but extended the reading lateralization period which culminates at that time the individual attains "the highest levels of language skills" (p. 105). In the acquisition of a second language presenting a new alphabet, Silverberg, Bentin, Gaziel, Obler and Albert (1979) suggested renewed right hemisphere involvement in the initial learning stages; again, as familiarization occurs and proficiency increases, the right hemisphere role diminishes. This second-language study concentrated on adolescent subjects; relatively little reading research has been conducted with normal adult populations. Of particular relevance, however, is the Ornstein, Herron, Johnstone and Swencionis (1979) EEG experiment with

normal adult readers concerning right hemisphere participation during narrative and expository reading tasks; their rather qualified results nevertheless ascertained that, while language is primarily controlled by the left hemisphere, "there may be certain aspects of the process of reading which differentially [involve] the right hemisphere" (p. 401).

In contrast to the limited research on lateralization priorities among normal adult readers, there has been considerable exploration into the right hemisphere role in the reading process observed in brain-damaged adults. Two sources of evidence investigating right hemisphere linguistic capacity are derived from examination of patients sustaining unilateral brain damage (including hemispherectomy cases) and commissurotomy or disconnection patients. The ability to recover language skills in aphasic patients who have sustained left hemisphere damage, Searleman (1977) in his overview of right hemisphere linguistic capacities asserted, can be attributed to the right hemisphere's assumption of these functions (Critchley, 1962; Gowers, 1887; Russell & Espir, 1961; Zangwill, 1960); this causality is valid provided that such a recovery is not traceable to the actual release of previously suppressed cortical tissue or to compensatory operations assumed by undamaged adjoining cortical tissue. Subsequent injury to the right hemisphere in these "recovered" aphasic patients results in permanent aphasia (Nielsen, 1946).

Heilman, Rothi, Campanella and Wolfson (1979), in their research on Wernike and global aphasia without alexia syndromes, reviewed three case studies of patients with left hemisphere lesions "who could not repeat, name, or comprehend speech, but who could comprehend written language." One of these three aphasic patients subsequently sustained a right hemisphere lesion and lost the ability to read--evidence negating the

possibility of left cortical tissue compensation. Heilman et al. conceptualized the mechanism by which these aphasics utilize the right hemisphere to "extract semantic information from iconic images" without undergoing the normal "phonological processing" (p. 129). Their explanation conceived of two hemispheric strategies concerned with reading: the left hemisphere decodes written language performing both integration and interpretation by transforming the graphic elements into meaningful phonemic units; the right hemisphere, basing its integration and interpretation of the graphic symbols in a more visuospatial mode, is able to extract semantic meaning from the graphic symbols bypassing the phonemic transformation. These researchers theorized that both reading strategies normally may be employed with the left hemisphere assuming dominance when the words are sequenced in complex syntactic units. The right hemisphere's visuospatial decoding strategy is more suited to understanding single words or word groups devoid of complex syntactical context. Faglioni, Scotti and Spinnler (1969), from their research on linguistic and perceptual letter recognition tasks (both requiring semantic decoding) in patients sustaining unilateral hemispheric damage, offered data to confirm this thesis: while letter symbols are processed specifically by the left hemisphere when presented in a conventional syntactical mode, if such presentation is made in a "perceptually complicated form" (p. 132), the participation of the right hemisphere in their identification is critical. Although Lenneberg (1974) maintained that in over 90 percent of patients with left hemisphere parietotemporal lesions language disorders result, Locke (cited by Lenneberg) had observed that patients with unilateral right hemisphere lesions in homologous areas experience great difficulty in

interpreting what they read. All this evidence on the potential and actual right hemisphere secondary involvement with "higher integrating functions" (p. 635), particularly in the language process (including reading), appears convincing.

Infants and young children subjected to left hemispherectomies usually have demonstrated an ability either to develop or transfer language skills to the intact hemisphere without significant performance loss (Basser, 1962; Carmichael, 1966; Falconer, 1960; Hodgson, 1967; Obrador, 1964; Smith & Sugar, 1975), affirming the functional plasticity of the right hemisphere. Relatively few adults, however, have undergone left hemispherectomies (Hecaen & Albert, 1978); in addition, the shortness of their postoperative life-spans has prevented extensive research in this area. After such an excision in adults, considerable language disturbance results, which characterizes the diminished flexibility with age of the right hemisphere to assume control of this function. In a single case, a 47-year-old male (Smith, 1966; Smith & Buckland, 1966), left hemisphere dominant preoperatively, maintained some residual speech ability and relatively more verbal comprehension as well as reading and writing proficiency, but deficits were still considerable. After two years, although Smith's patient was no longer classified aphasic, researchers were prevented from further testing because of his death. Dimond (1972), commenting on the Smith case study, distinguished between the speech and language activities; he pointed out that in left hemispherectomized adults there is a "potential for expression of linguistic capacities through means other than speech." His view conceives of adult language as exceedingly complex; for this reason an extended postoperative time period would be necessary before

the remaining nondominant hemisphere might assume control of this "advanced and complicated function" (p. 104). A child's brain under similar circumstances appears more flexible because the language process at this stage is infinitely less demanding; thus, Dimond contended, it would be incorrect to assume that with age the adult brain undergoes a complete loss of functional plasticity.

The second source of evidence concerning right hemisphere linguistic capacity involves commissurotomy or disconnection patients. . Such research is of considerable advantage in the study of hemispheric specialization, making possible measurement and comparison of relative performance in an individual with each hemisphere operating independently. A subject's performance can be tested on a particular task to ascertain response dominance and differential performance strategies for each hemisphere with minimal contamination from its counterpart. In addition, medical history, general background and past experience as well as current mental and physical status can be eliminated as inter-subject variables--inherent in similar laterality studies examining normals or unilateral brain-damaged subjects--when testing for right/left hemisphere differences. Gazzaniga's (1970) early findings in testing of commissurotomized subjects revealed fairly consistent cerebral language-behavior patterns. Gazzaniga found that tachistoscopic presentation of words directed exclusively to the nondominant (right) hemisphere could be received and internalized, but the subject was unable to communicate his response in either speech or writing; recognition or comprehension could only be expressed through nonverbal responses. Such verbal and linguistic impairment was not evident when information was received and processed in the dominant (left) hemisphere. The expressive language

component, Gazzaniga surmised, seems "to be organized almost exclusively in the left hemisphere" (p. 125). This highly lateralized organization, in terms of the ability to respond to verbal stimuli, contrasts with the bilateral capacity to receive and interpret language, both written and spoken, although the left hemisphere still appears to be better adapted for this process. This language-dominant hemisphere, noted Sperry (1974), "is also the more aggressive, executive, leading hemisphere in the control of the motor system" (p. 11); since both speech and writing utilize this system, it is a critical factor in language expression.

Comparative commissurotomy studies, including those concentrating on hemispheric linguistic capacities, are restricted by inter-subject differences, particularly preoperative, epilepsy-induced, extracallosal damage and its suspected differential effects on cerebral organization of language. In addition, the relatively recent and continuing development of assessment techniques as well as new and modified instrumentation in this field are still in their formative stages. Since language is essentially a left hemisphere process, the nature and quality of the role played by the right hemisphere in these cases is difficult to assess. Special testing procedures and measures involving nonverbal forms of expression are necessary to determine right hemisphere participation as "a conscious system in its own right, perceiving, thinking, remembering, reasoning, willing, and emoting, all at a characteristically human level" (Sperry, 1976, p. 18). In an effort to investigate the possibility that both hemispheres may be "conscious simultaneously in different, even in mutually conflicting, mental experiences that run along in parallel" (Sperry, 1974, p. 11), variables affecting test validity and data reliability must be taken into

consideration. Possible subject compensatory techniques influencing the reliability of experimental data in commissurotomy hemifield studies include: 1) interhemisphere behavioral cross-cuing strategies (Gazzaniga & Hillyard, 1971; Levy-Agresti, 1968); 2) encroaching bilateral representation of center-field (foveal) vision (Butter & Norrsell, 1968); 3) suppression of the usually dominant hemisphere response by interhemisphere or subsystem-mediated intrahemisphere attentional reversals (Trevvarthen & Sperry, 1973); 4) undivided subcortical integration or transfer influencing performance (Gazzaniga & Hillyard, 1971; Trevvarthen, 1970); and 5) interference, possibly created by conflicting demands attributable to a common motor and attentional apparatus, from the contralateral hemisphere (Levy, Nebes & Sperry, 1971; Nakamura & Gazzaniga, 1977).

One positive result derived from such experimentation, however, is the refutation of Geschwind's (1965) view that "word blindness" or "word deafness" would result in the right hemisphere if it were disconnected from its language processing counterpart. Although essentially silent and for the most part inferior in all tasks concerning language or linguistic performance, numerous commissurotomy studies have provided evidence that the right hemisphere can process some grammatic-syntactic structures at both the word and short phrase levels (Zaidel, 1976, 1978), can mediate the spelling and sometimes the writing of simple words (Gazzaniga, 1970; Gazzaniga, LeDoux & Wilson, 1977; Levy, Nebes & Sperry, 1972) can receive and respond to short spoken commands and descriptions (Nebes & Sperry, 1971, Zaidel, 1978), and can at times even express itself initiating speech (Levy, Trevvarthen & Sperry, 1972). Among the linguistic processes which commissurotomy studies indicate the

right hemisphere has proven less than adept must be included an ability to analyze phonetically spoken or written input (Levy, 1974; Levy & Trevarthen, 1977; Springer & Gazzaniga, 1975; Zaidel, 1978), an ability to retain and therefore comprehend nonredundant sentences in which word sequence is important and context is complex (Zaidel, 1976, 1978), and general response competence in the forms of verbal and written expression (Gazzaniga, 1970; Gazzaniga & Sperry, 1967; Sperry, 1968b).

The corpus callosum, largest and most important of the cerebral structures subserving interhemispheric integration, undergoes a morphological development yet to be definitively described. Most prominent of the fiber tracts in the human brain, the central core of the corpus callosum functions as a major commissure--"strands of nerve fibers crossing the median sagittal plane and joining the two cortices" (Selenes, 1974, p. 112). In 1965 Locke and Yakovlev obtained evidence, at autopsy, of two different forms of callosal fibers: the corticocortical or commissural fibers and the decussating transcallosal fibers projecting from the cingulum to the contralateral corpus striatum. The commissural fibers, located in the center of the corpus callosum, link, with either homotopical or heterotopical connections (Ironside & Guttmacher, 1929), homologous areas of the two cortices and are the principal focus in this analysis of intercerebral integration and its possible influence on the language process involved with reading. The other transcallosal decussating fibers, located at the dorsal and ventral aspects of the corpus callosum, project subcortically; their role, if any, in the interhemispheric communication system is still unknown.

The callosal tract is composed of very slender fibers, "the

myelinated ones having only thin myelin sheaths" (Tomasch, 1954, p. 125). Individual fiber sizes range from 1.5μ to 5μ , the more numerous in the lower range. The number of these fibers per square millimeter, in the adult human brain, achieves its highest density in the area of the genu (sharply bent transitional region between the main body and the rostrum) and the splenium (posterior region), ranging as high as 300,000 fibers per square millimeter. The genu region contains a larger proportion of nonmyelinated fibers relative to the splenium area which contains a more substantial number of small fibers with thin myelin-sheaths. Tomasch's estimate for humans, corresponding to the proportion of spinal nerve roots in dogs (Davenport & Ranson, 1931), ascertained an average ratio of 1.4:1, myelinated in contrast to unmyelinated callosal fibers. The actual number of callosal (commissural and decussating) fibers, derived by determining the total surface area of this structure, has been estimated from this study to be about 190,000,000; therefore, even if this figure represents all the cortical nerve-cells giving rise to callosal fibers, it involves only slightly more than two percent of the total nerve cells (Thompson [1899] calculated at 9,200,000,000) in the cerebral cortex.

The process of commissuralization in the corpus callosum, described by Rakic and Yakovlev (1968) in their thorough and methodical study of its development, evolves from the enfolding of the dorsal section of the lamina reuniens into a median groove which forms, through the fusion of its sides, into a massa commissuralis. This structure "becomes the bed for the ingrowth of the commissural fibers of the corpus callosum" during the 11th and 12th weeks of embryonic development. These early fibers penetrate the cellular bed of the massa commissuralis, eventually

forming the "definitive callosal commissural plate." Future caudal growth and alteration of configuration occurs as the fibers extend toward and across the midline, some time between the 18th and 19th fetal weeks. At this point the corpus callosum achieves both form and position corresponding to its mature state, differing only in rostrocaudal extent and thickness. Further differentiation coincides with and is influenced by changes in "the surface configuration of the cerebral hemispheres" (p. 57). Among the callosal fibers crossing the geometric midplane in the massa commissuralis are the commissural as well as the decussating transcallosal fibers projecting from the cingulum. Subsequent growth in the body of the callosum occurs at a relatively even rate prenatally and postnatally. The genu region forms more rapidly before birth and parallels the rapid initial spread of the isocortical plate over the frontal poles of the hemispheres. The splenium area in contrast develops more rapidly postnatally and such formation "correlates with the accelerated growth and differentiation of the temporo-parietal and occipital lobes" (p. 68).

Hewitt (1962), refuting the generally accepted model for maturation of the corpus callosum, produced anatomical evidence indicating that this incipient interhemispheric communication structure at birth "is incompletely established and only bears a superficial resemblance to the adult form" (p. 357); thus this tract, early in life, is not yet able to function fully as it will following completion of maturation. The callosal fibers, individually and as a plexus, are still undergoing development at five months following birth (Luttenberg, 1965), supplying additional evidence of its inability to serve as a completely operational interhemispheric transmission system for neuronal impulses

at this time. Recent behavioral study of young children (three to five years) has confirmed a "developmental increase in functional communication" (Galín, Johnstone, Nakell & Herron, 1979; also see Galín, Diamond & Herron, 1977; O'Leary, 1980) between the two hemispheres. Finally, histochemical studies (Luttenberg, 1966; Yakovlev & Lecours, 1967) demonstrate that through early childhood the slender thread-like callosal commissural fibers lack the myelin sheaths necessary for optimal function. Keynes (1979), in his recent study of nerve impulse transmission, observed that conduction is accelerated by the presence of such insulating sheaths composed of fatty myelin. The structural modification process--myelination of the fibers--allows "a 25-fold reduction in fiber diameter for the attainment of a given conduction velocity" (p. 126).

Callosal commissural fiber myelination may begin as early as the eighth fetal month (Luttenberg, 1966) or as late as the fourth postnatal month (Yakovlev & Lecours, 1967), and the rate of the process itself varies throughout the callosal regions. Yakovlev and Lecours described a "cycle of myelogenesis" which relates to the maturation progress in any particular area of the brain: "the cycle reflects and, in a sense, defines the position of a fibre system or region in the hierarchy of functional organization of the developing nervous system" (p. 6). Although the decussating transcallosal fibers begin myelination and are well on their way to maturation long before myelinated commissural fibers are found in the core of the corpus callosum, these latter fibers continue to show a gradual gain in intensity of staining and density. The relative progress of this regional myelination process in the callosal commissure first appears in the splenium, the forceps major

and tapetum, spreading gradually from this posterior area toward the genu, the rostrum and forceps minor. The final maturation point has yet to be definitively ascertained, but Yakovlev and Lecours maintain that myelination of commissural fibers is essentially completed by the end of the first decade although this process extends even into the second decade. Salamy (1978), in his study of maturational trends in commissural transmission timing, found that the ipsi-contralateral transmission latency difference correlated significantly with age (difference progressively declined with increasing age) through the first 13 years of life, which concurs with Yakovlev and Lecour's callosal "cycle of myelogenesis" timetable. Thus Salamy's findings confirm the histochemical contention that "commissural transmission attains symptote by the end of the first decade of life" (p. 1410).

Discussion of the relationship between structure and function of the callosal communication system will concentrate on those callosal connections of the visual cortex which are the basis for interhemispheric integration of visual perception and learning, both relevant to the reading process. Such an analysis may conveniently be divided into an examination "of the mechanisms of sensory integration across the vertical midline of vision . . .[and]the process of interhemispheric transfer" (Berlucchi, 1972, p. 371) of complex visual information.

In the first instance the corpus callosum plays a vital role integrating sensory visual input between the receptive fields of the two cortices, an important factor in the establishment of perceptual continuity across the vertical midline of vision. The neuropsychological issues pertinent to an analysis of this process have thus far been primarily investigated through anatomical and electrophysiological

experimentation with cats (Berlucchi, Gazzaniga & Rizzolatti, 1967; Hubel & Wiesel, 1967; Makarov, 1968-69); although anatomical studies with rats (Heimer, Ebner & Nauta, 1967; Nauta & Bucher, 1954), rabbits (Hughes & Wilson, 1969) and monkeys (Cragg & Ainsworth, 1969; Spatz, Tigges & Tigges, 1970; Zeki, 1969, 1970) have produced evidence of interspecies differences, the same conclusions are fundamentally valid with respect to callosal connections of the vertical meridian in the visual cortex. Only those visual cortex subdivisions of area 17, 18, and 19 involved with the visual fields adjoining the vertical midline appear to be reciprocally connected, either homotopically or heterotopically, through the splenium of the corpus callosum (Berlucchi, Gazzaniga & Rizzolatti, 1967; Hubel & Wiesel, 1967). In addition, from a synthesis of anatomical and electrophysiological evidence, Berlucchi (1972) maintained the unlikelihood of establishing a simple one-to-one exchange "between any of the cytoarchitectonic areas of the visual cortex and the interhemispheric callosal projections" (p. 376).

The response properties of splenial callosal units have been studied through microelectrode analysis--single cell recordings--and found to correspond closely to those neurons of areas 17, 18, and 19 in the visual cortex which Hubel and Wiesel (1962, 1963, 1965) have identified and subdivided into "simple, complex and hyper-complex" cell types (Berlucchi, Gazzaniga & Rizzolatti, 1967). "Simple" cells respond to an optimally oriented line in a narrowly defined location; "complex" cells respond to stimulation which is relatively independent of the precise position of the stimulus, although orientation remains critical; "hyper-complex" cells--a still higher order cell type--respond to stimulation where line length as well as other geometric

characteristics of the stimulus may be important. Anatomical studies have revealed that discrete lesions in either area 17 or 18 result in corresponding terminal fiber degeneration in contralateral areas 17, 18, and 19 (Garey, Jones & Powell, 1968; Hubel & Wiesel, 1965; Makarov, 1968-69; Wilson, 1968); callosal projections from area 19 appear to connect with contralateral area 19 as well as the contralateral suprasylvian area (Heath & Jones, 1970). Intracellular techniques have also demonstrated that electrical stimulation of the contralateral visual cortex and corpus callosum can antidromically activate certain neurons in areas 17 and 18 (Toyama, Matsunami & Ohno, 1969). Where the left optic tract had been severed, left hemisphere cortical responses--registered through electrical recordings near the 17-18 border of the visual cortex--could only be elicited by the presentation of light stimuli along the vertical midline of the visual field (Choudhury, Whitteridge & Wilson, 1965; Vebayeva, Whitteridge & Wilson, 1967). That these cortical responses may rely on callosal transmission from the right hemisphere is supported by the observation that following symmetrical contralateral cortical cooling or by cutting or cooling the splenium region these responses cease.

The discovery that area 17--which contains only "simple" cells involved with transmission of simple responses corresponding to the initial phase in the processing of visual input--contributes callosal fibers led Berlucchi (1972) to conclude that "callosal connexions of the visual cortex [do not only] selectively transmit higher order information" (p. 376); rather these "simple" units also make their contribution to the process of visual interhemispheric perceptual integration. Whitteridge (1965) suggested that these latter vertical

meridian callosal connections--as distinguished from those involved with transfer, integration, and coordination of complex visual experiences (Sperry, 1968a)--are required during the initial phase of the perceptual process. Sensory integration--the ability to perceive straight lines originating from the frontal plane intersecting the fixation point and crossing the vertical midline--necessitates the activation of appropriate neural receptors in the respective visual cortices of the two hemispheres; for the two transmitted segments of the line to be recognized as a single continuum some form of linkage crossing the vertical midline--callosal connections--must offer the neural analogue to perceptual cohesion. Transmission across this callosal linkage occurs so rapidly in man that, according to Whitteridge, historically its existence has gone relatively unnoticed; the normal transmission speed and efficiency characteristic of its function has seldom produced any problems which have heretofore warranted extensive investigation. Split-brain patients under examination, however, were consistently incapable of recognizing whether broad lines crossing from one hemifield into the other through the fixation point were either unbroken or separated in the middle (Gazzaniga, Bogen & Sperry, 1965). Such evidence creates a less than definitive (other forebrain commissures had also been sectioned) case for callosal connections of the visual cortex as the cortical tract establishing this form of linear continuity. Hubel and Wiesel (1965, 1967) did theorize, however, that elemental visual material transmitted by the corpus callosum both extends the visual fields across the midline and also serves in the creation of more complex visual fields involved in the establishment of neural integrity for rudimentary visual perception.

Normal bilateral function concerned with "visual experience" must involve interhemispheric transfer, integration, and coordination of higher order information (Berlucchi, 1974; Sperry, 1970; Wilson, Culver, Waddington & Gazzaniga, 1975); the forebrain commissures, particularly the splenium region of the corpus callosum (Bogen & Bogen, 1969) and in at least some individuals the anterior commissure (Gazzaniga, 1978, chap. 2), provide the major components subserving these functions. Because medical considerations eliminate the possibility of basic neurophysiological experimentation (usually involving invasive techniques) on normal human subjects, the study of "visual interhemispheric differentiation and interaction" (Berlucchi, 1974) has necessitated rather indirect methods. Experimentation utilizing subjects who have undergone commissurotomy has proven to this point an effective source of data on the corpus callosum component in the interhemispheric communication system. Sperry (1967), in describing the effects of such surgery, observed "the establishment of two entirely separate mental domains within the same cranium" (p. 716). Complete separation of the hemispheres permits material--experiences learned and remembered--to be isolated to a single hemisphere, inaccessible to its cerebral counterpart: "two separate realms of conscious awareness, two separate sensing, perceiving, thinking, and remembering systems" (p. 717). Visual perception in such test subjects is cerebrally isolated, stored in either one of the two separate visual worlds. Failure of the corpus callosum to transmit engrams from one hemisphere to the other produces a response in which objects viewed through the right half of the visual field "are registered in mental experience and remembered quite separately from things seen in the other half of the visual field"

(Sperry, 1968b, p. 725). Thus, each of the hemispheres--Sperry's "separate inner worlds"--becomes its own repository for visual images and memories, a phenomenon not possible in testing normal subjects.

Myers and Sperry's (1958) early research, utilizing feline subjects, on interhemispheric communication through the corpus callosum determined that the cortex in visual receptive regions not only received visual stimuli from the retina but also integrated and transmitted the significance of this experience to the contralateral hemisphere. Evidence indicated "a posterior localization of the visual gnostic transfer function" (p. 302) supporting the conception "of the primarily homologous nature of the callosum interconnections" (p. 303). The facility with which nerve impulses travel from one sensory or association area to its contralateral homologue, demonstrated electrophysiologically by Bremer (1958), is compatible with Myers and Sperry's findings as well as Meyers' (1956) earlier conclusion based on a two-phase experiment which demonstrated "the importance of the corpus callosum in the integration of the two hemispheres in visual learning and transfer" (p. 363). Following callosal section Myers (1956) had observed an inability to transmit simple perceptual impressions from one hemisphere to the other. His second application involved completely conflicting discriminations introduced separately to each hemisphere; no indication of interference occurred, from which Myers ascertained that the two hemispheres independently subserve visual learning and recall--such independence absent in cats with a functional corpus callosum.

Normal bihemispheric integration of perceived and learned visual information of necessity involves the reception of stimuli selectively channeled into each hemisphere of the intact brain; transmission of

neuronal impulses then make this experience accessible to hemispheric counterparts through the callosal and, on occasion, anterior commissures. The form in which this information is transferred under ordinary circumstances and the nature of callosal functioning in neurologically intact humans undergoing natural experiences is well beyond the scope of present experimental techniques (Bogen & Bogen, 1969). Nevertheless, Sperry's (1967, 1974) general assumption that the normal individual, having viewed an object in the left hemifield and later been presented with this same object in the opposite hemifield, undergoes a sense of recognition---even familiarity---appears irrefutable. The two half-fields of vision are somehow integrated within the brain into a unified whole through mediating cerebral mechanisms which may be delimited and identified in terms of location and function. Sperry's (1974) tentative overview, following his research on the split-brain subject's ability to mediate conscious experience independently and/or concurrently in each of the separated hemispheres, offers little more than a speculative theoretical basis for the method by which the intact corpus callosum in normals might serve "to harmonize and complete duplicate conscious processes" (p. 18). The hypothesis that each hemisphere processes stimuli reception and integration in essentially different ways, with the callosal transmission of engrams providing an experiential unification, reflects on even larger issues. Identification of the exact procedure by which subjective experience is perceived, internalized, integrated, coordinated, and synthesized into thought at this point is largely metaphysical; but, within the context of the mind-brain issue, Sperry envisioned, "more than any other cerebral system, the interhemispheric commissures and their cortical associations

continue to offer promise in the search for an eventual direct correlation between the phenomena of complex subjective experience and known variables in specified neural structures" (p. 18).

An additional function attributable to the corpus callosum--apart from those operations concerned with sensory perception and transfer of higher forms of information--involves the maintenance of bilateral synchrony of cortical electrical activity. Bremer (1966) theorized that the original purpose of the corpus callosum in its phylogenetic evolution may have been to sustain "a dynamic equilibrium between the two neo-cortices" (p. 208). Electroencephalogram (EEG) potentials, simultaneously recorded from symmetrical cortical points of the two hemispheres in a normal brain, essentially correspond in shape, amplitude and duration (Berlucchi, 1966). Experimental research (again with cats) examining the participation of the corpus callosum in preserving bilateral cortical synchrony of the EEG, revealed that EEG spindles recorded from homologous contralateral sites ceased to remain in phase following severance of the callosal commissure (Berlucchi, 1966; Bremer & Stoupel, 1957; Claes, 1939; Magni, Melzack & Smith, 1960). A general acceptance based on experimentation conducted during the 1950s and 1960s proposes that, in a normal EEG record, alpha waves recorded from homologous cerebral surfaces possess a consistent phase relationship. Evidence for this assumption has been derived from data accumulated by cross-correlation analysis of spontaneous or evoked alpha rhythms in animals (Berlucchi, 1966; Brazier & Barlow, 1956) and men (Barlow, 1961; Brazier & Casby, 1952; Cooper & Mundy-Castle, 1960; Liske, Hughes & Stowe, 1967; Walter, Rhodes & Brown, 1966). Their results point to "a constant phase relationship between alpha waves from the two

hemispheres with one or the other leading by not more than a few milliseconds" (Hoovey, Heinemann & Creutzfeldt, 1972, p. 337). Such a close phase relationship may be attributed to callosal interaction between the two hemispheres.

Hoovey et al. (1972) in 1970 developed a computer method to permit a more accurate measurement of interhemispheric phase coherence between bilateral individual simultaneous alpha waves. Test results, while not negating this previous research, nevertheless found considerable variation between individuals with regard to mean interhemispheric intervals. In subjects with congruent alpha synchrony, the amplitude correlation between simultaneously recorded right and left alpha waves was high, and (in terms of timing) leading right/left peak intervals of alpha pairs varied continually. Although these data suggest a less than symmetrical EEG correspondence, the concept of a general interhemispheric synchrony maintained by continuous callosal activity, "insuring the bioelectrical synergy of the two hemispheres" (Berlucchi, 1966, p. 355), remains a pragmatic construct.

Relative to this project is the research on alpha synchronization and dyslexia pursued by Martinus and Hoovey (1972) investigating children 8- to 11-years-old. The subject population was subdivided into three classes: a group experiencing special difficulty with attention or concentration, a second group of diagnosed dyslexics, and a third normal control group. Testing revealed that most of the control subjects underwent "a quantitative increase of alpha activity" as well as "an improvement in inter-occipital synchrony" (p. 353) when attempting to resolve an auditory discrimination problem. The clinical groups, however, exhibited a decrease in synchrony and dyslexic subjects tested inferior

with regard to alpha enhancement when compared to the normal control group as well as that group with specific attentional or concentration difficulties. Martinus and Hoovey concluded that the alpha-wave activity increase corresponded to a "more general activation process," whereas, the improvement of synchrony between inter-occipital alpha pairs bore a definite relationship "to the partial function of [acoustic discriminatory] attention tested" (p. 355). In commenting on the conclusion drawn by Martinus and Hoovey, Hughes (1978) posited the possibility that their results offer neurophysiological evidence concerning the relationship between alpha-wave synchrony and the attentional factor, both "crucial" components to an analysis of the dyslexia syndrome.

II. Related Research

Hemispheric Visual Processing Capacity

The mode by which the two cerebral hemispheres of the normal human brain function in perceptual reception and analysis of visual stimuli has been extensively investigated during the last fifteen years. Dimond (1972) and Dimond and Beaumont (1974) reviewed pertinent research, particularly the experimentation by Elithorn and Barnett (1967) on the cerebral lateralization of information and the independent organization of response by a single hemisphere, toward understanding the process by which the "two hemispheres work together and integrate their functions" (1974, p. 58). Dimond and Beaumont then proposed a model in which each hemisphere serves as an "interface" with the independent capacity to perceive and analyze multiple forms of information. The essential thesis formulates a view that, when the two hemisphere "interfaces" are functioning concurrently to integrate perceptual input, the processing capacity of the human brain is increased relative to each hemisphere operating in isolation unsupported by the other. Experimentation (Dimond, 1970, 1971, 1972; Dimond & Beaumont, 1971a, 1972a, 1972b) to support this hypothesis involved the functional isolation of each hemisphere to determine its capacity to code and classify visual information independently. The limits of the perceptual load input, as measured by percentage correct and/or reaction-time data, could then be compared to that with both cerebral hemispheres functioning concurrently as a perceptual analyzing system.

Dimond (1970) designed an experiment in which two signals were

simultaneously flashed either to the same hemisphere or one to each hemisphere. Reaction-time data revealed that projection of both signals to a single hemisphere resulted in a significant block to the timing of subject's second response. (This same effect resulted when the signals occurred consecutively with only a brief interval [Dimond, 1972].) When separate signals were directed to different hemispheres simultaneously, however, the degree of interference was significantly lessened. The blocking of response latencies at the hemisphere level "indicates something of the limitations of function of each cerebral hemisphere as an information system" (Dimond, 1972, p. 118). Theoretically this disruption is attributable to an "overload" in the analytical processing by a single hemisphere. Such interference occurs when either hemisphere of the brain is "overloaded." Each hemisphere "interface" receives and analyzes the information before a third phase links and "cross-compares" the perceptual load between the two cerebral hemispheres. Thus inter-hemispheric transference or sharing may not occur until the original receptor hemisphere has completed its analytical phase. The interference caused by both signals arriving simultaneously in the same hemisphere delaying subsequent analysis of the second signal, postulated Davis and Schmidt (1971, 1973), produces a "refractory effect" in the analytical process, this delay of sufficient duration "to exceed the extra callosal transmission time required for the comparison between information presented separately to the two hemispheres" (1971, p. 345).

Further experimentation with more complex tasks involving word recognition (Dimond, 1971) and digit identification (Dimond & Beaumont, 1971a, 1972a) as well as reaction-time responses to nonverbal material (Dimond & Beaumont, 1972b) produced comparable results: utilization of

the two hemispheric analyzing systems demonstrated a far greater capacity than that of a single hemisphere system suggesting "that the processes of perception are relatively time-consuming and an advantage is to be gained where use can be made of the double interface of the brain" (Dimond & Beaumont, 1974, p. 62). Thus, although each cerebral hemisphere "possesses its own limited 'central' decision mechanism," both decision mechanisms are "capable of working in parallel to enhance performance" (Dimond, 1972, p. 127)--dual information processing systems functioning in tandem to effectuate more sensitive and sustained operation.

In an attempt to explore the nature of this cerebral organization as well as theories of attention and capacity limitations in hemispheric information processing, Moscovitch and Klein (1980) designed a series of four experiments to validate the hypothesis that "perceptual interference effects would be greatest among items that are processed by the same hemisphere than among items processed by different hemispheres" (p. 590). Relative hemispheric independence and capacity limits were tested by deliberately overloading each hemisphere with material that "it is specialized to process, quite apart from the sensory field to which the stimulus is presented" (p. 591). Analysis of experimental data demonstrated that identification of peripheral stimuli is inferior and therefore interference greater when both CF and VF stimuli are specialized for processing by the same hemisphere--distinguished from CF and VF stimuli of contrasting modes, each of which is "typically processed by a different hemisphere" (p. 599). Thus "material-specific" stimuli compete for processing by the most qualified hemisphere creating interference through overload in that hemisphere's "central" processing

mechanism. That stimulus recognition interference was not dependent on or influenced by hemispheric response bias was also determined, thus indicating that this interference effect occurs early in the information processing continuum--the result of competition between "material-specific" stimuli "for the specialized, limited-capacity, hemispheric mechanisms that support . . . earlier [perceptual and/or analytical] functions" (p. 596).

Research specifically investigating relative CF and VF performance in the receptive-analytic-response process continuum has been extremely limited. Tangential studies focusing on other issues, however, have examined aspects of this relationship but have utilized widely varying approaches and methodologies which offer little opportunity to compare or apply their findings. Nevertheless, review of four such experiments provides some insight into relative CF and VF processing performance. Mancuso, Lawrence, Hintze and White (1979) in their recent study (utilizing adult, right-handed, male, normal subjects) examined the influence of central fixation stimuli on "peripheral recognition asymmetry within the 'controlled fixation' paradigm" (p. 118). Their findings indicate that altering the mode (verbal/nonverbal) of CF fixation stimuli can significantly influence VF recognition performance. Of particular interest is their serendipitous discovery of the "unexpectedly large number of errors in identification of the central stimuli" (p. 119). Berlucchi, Brizzolara, Marzi, Rizzolatti and Umiltà (1974) (utilizing adult, right-handed, male, normal subjects) investigated lateral asymmetries in attention affecting significant differences in VF perception. Their experimental design was intended to examine Kinsbourne's (1970, 1972) hypothesis that the hemispheric specialization

dichotomy found in verbal and nonverbal lateralized tasks can be attributed to corresponding lateral asymmetries in hemispheric distribution of attention. While the results of Berlucchi et al. failed to support this hemispheric attentional bias hypothesis, they did confirm (from analysis of both reaction-time data and accuracy scores) the superiority in performance of central to peripheral presentations. Although statistical analysis of VF errors to CF errors validated expected significant differences, using verbal stimuli the combined CF error score (70) relative to the combined VF error score (116) was considerable. Bouma and Legein (1977) examined CF and VF recognition of isolated letters, embedded letters and words employing a subject population of 20 right-handed dyslexic children (18 males and 2 females) and a control group of 20 average readers. At a visual angle of 1°--visual acuity approximately 50% of foveal value--both groups scored equally well CF and VF in the recognition of isolated letters. With embedded letters, however, both groups experienced slightly more difficulty in the CF, and VF recognition scores decreased dramatically for both groups with the dyslexics demonstrating a significantly greater VF decrement than the controls. Although VF word recognition scores for both groups were higher than those achieved in the embedded-letter task, the dyslexic group again experienced more difficulty in both CF and VF presentations; the contrast between the two groups was significant for VF recognition. In all these tasks, CF/VF recognition patterns differed only in degree and not in substance--CF performance was consistently superior to that of VF performance for all presentation modes with both dyslexics and normal readers.

In a comparative experiment in which 24 right-handed, male

adolescents were divided into three categories--normal readers, auditory-linguistic dyslexics, visual-spatial dyslexics³--Pirozzolo (1979) ascertained that, in the identification of three-letter words, the three groups performed equally well CF--mean percentage 97, 97, 96--but significant distinctive patterns occurred as the visual angle was widened from 0° to 1° to 2°. As the visual angle widened, correct word identification decreased in the case of all three groups; the deterioration in performance, however, relative to the normal readers, accelerated to a greater degree in the right VF for the auditory-linguistic dyslexics and deteriorated even further in both VF for the visual-spatial dyslexics.

Laterality and Developmental Dyslexia

Techniques for exploring the nature of functional lateralization and developmental dyslexia--from the early research of Hinshelwood and Orton to well past the midcentury--were limited primarily to examination and analysis of motoric laterality measurements (handedness, eyedness, footedness). Extensive clinical observation and evaluation of behavior in brain-damaged patients, correlated with neuroanatomical data provided

³Implicit in "classic" definitions of developmental dyslexia is the assumption of a single, homogeneous syndrome (Benton, 1978). During the last two decades, however, numerous researchers, through analysis of reading and spelling performances (Boder, 1970, 1971, 1973; Ingram, Mason & Blackburn, 1970; Johnson & Myklebust, 1967) and from examination of the occurrence of concomitant disabilities in oral language, motor skill, perceptual capacity and short-term memory (Denckla, 1977, 1979; Denckla & Rudel, 1976a, 1976b; Kinsbourne & Warrington, 1963; Mattis, 1978; Mattis, French & Rapin, 1975), have consistently proposed and classified at least two distinct subgroups of dyslexics--characterized by predominance of either auditory or visual symptomology.

at autopsy, supplied the rationale for utilizing these lateral motoric preferences to test neurological theories proposing either aberrant cerebral organization or cerebral dysfunction as possible neural mechanisms responsible for developmental dyslexia. Although some early research indicated reversed, inconsistent, or poorly established motoric lateralization preference patterns in dyslexics (Burt, 1950; Galifret-Granjon & Ajuriaguerra, 1951; Harris, 1957; Ingram, 1959, 1960; Macmeekken, 1939; Monroe, 1932; Orton, 1937; Vernon, 1957), more recent studies have failed to show a definitive relationship between left-handedness and/or deviant hand-eye preference and severe reading disability (Balow, 1963; Belmont & Birch, 1965; Coleman & Deutsch, 1964; de Hirsch, Jansky & Langford, 1966; Satz & Friel, 1974; Shankweiler, 1963; Zurif & Carson, 1970). In the early 1960s more sophisticated noninvasive techniques and methods--such as lateralized tachistoscopic presentation, dichotic listening, dichaptic presentation, and intra- and interhemispheric electrophysiological procedures--were developed for investigating cerebral lateralization and were subsequently applied in dyslexia research. With these innovative laterality assessment techniques, more direct approaches to the study of cerebral dominance patterns could be undertaken to examine current neurological theories--incomplete lateralization, hemispheric defect or maturational lag, interhemispheric articulation dysfunction--and the dyslexia syndrome.

Lateralized tachistoscopic presentation investigating cerebral dominance in terms of the reading component in language processing has produced supportive (although inconclusive and often confusing) evidence for these neurological theories. Particularly relevant to this project

is a review of laterality research specifically directed toward reading-- those tachistoscopic experiments investigating verbal recognition/identification and those electrophysiological studies recording brain activity during actual reading performance; for pragmatic reasons and limited applicability to this project's experimental design, those tachistoscopic experiments involving memory and/or learning components and those electrophysiological studies not directly examining brain activity during reading tasks, as well as dichotic listening studies which focus on the speech component in language processing and dichaptic presentation studies testing kinesthetic recognition/identification of verbal stimuli, have been excluded from analysis in this review.

Pirozzolo and Rayner (1979) in their investigation of cerebral asymmetries tested 18 right-handed males (mean age 12.5 years) grouped as nine normal readers (reading at grade-level or slightly above) and nine auditory-linguistic dyslexics (reading diagnostic-test scores at least two years delayed). Three tachistoscopic tasks were administered, each utilizing a different stimulus mode--faces, lower and uppercase words--and required nonverbal identification responses; elicited VF performances demonstrated a left visual-field (LVF) superiority for both groups in the facial recognition task but a significant lack of asymmetry for dyslexics in the word-recognition tasks. Accuracy scores for word-recognition tasks in both groups, however, were comparable. Such results, Pirozzolo and Rayner proposed, lend support to Orton's "incomplete lateralization" hypothesis; comparable word-recognition scores for both groups also argue against a "word-perception defect" in this dyslexic subtype. In another experiment Pirozzolo (1979), comparing CF and VF word-recognition performances among normal readers

and two dyslexic subgroups, maintained that normal readers and visual-spatial dyslexics were more accurate in identifying right visual-field (RVF) words while auditory-linguistic dyslexics showed an essentially symmetrical performance pattern for both VF; all groups performed equally well in CF word identification. Examination of Pirozzolo's mean percentage correct data, however, fails to corroborate his analysis of the visual-spatial dyslexic performance: their identification score (mean percentage correct) of words located at a visual angle of 2° or a combined score (average mean percentage correct) of words located at visual angles of 1° and 2° support a relatively symmetrical VF performance pattern. In addition, this dyslexic subtype consistently scored much lower in both VF than either the normal readers or the auditory-linguistic dyslexics.

Olson's (1973) tachistoscopic experiments, employing both unilateral and bilateral presentations, examined VF word-recognition patterns in good readers (50 children not differentiated by sex or handedness, age range 7-11, reading at or up to three years above their grade-level, at least normal intelligence and English as their first language) and poor readers (43 children not differentiated by sex or handedness, age range 8-13, reading six months or more below grade-level--including 12 bilinguals, seven with an IQ less than 90, and six with known emotional problems). A significant RVF preference for word identification occurred in both reader groups including an even more "enhanced right field preference for bilateral field presentations" (p. 343). The youngest unimpaired subgroup of poor readers (nine children, age range 8-9--not among those with IQ, emotional or bilingual problems), however, failed to demonstrate any VF superiority in this task, which Olson attributed

to a possible neural maturational delay in "hemispheric specialization for language processing" (p. 349). The heterogeneity of subject selection for both groups, the differential inclusion of "behaviorally" disadvantaged children in the poor reader group, and the failure to establish any valid criteria for subject matching between the two groups greatly reduces the possibility of extrapolating from these findings useful information in the study of dyslexia.

A general RVF superiority was also observed by Marcel, Katz and Smith (1974) in their tachistoscopic identification task--in which subjects were asked to identify words or as many letters as they could discern--testing poor readers and good readers. Subjects, 20 boys and 20 girls all between 7.6 and 8.7 years old, were subgrouped based on sex and relative scores as measured by the National Foundation for Educational Research Reading Test. Although poor readers performed as well as good readers on LVF word identification, overall results revealed for each group a significant RVF superiority in both word and letter identification--the degree of asymmetry more sharply defined in good readers. These researchers therefore concluded that, in subjects of the same age, good readers may be distinguished from poor readers by the relative magnitude of asymmetry in "cerebral hemispheric specialization of language functions" (p. 131).

The degree of differential asymmetry as an indicator of reading skill was further studied by Kershner (1977) in a tachistoscopic task with bilateral presentation for word identification. Kershner's subject population (33 right-handed children, 10 years of age) was divided into three groups--gifted children, good readers, dyslexics--categorized by a) high IQ and exceptional academic ability, b) average IQ with high-

average reading scores, and c) average IQ with at least two years reading retardation and no signs of emotional problems, gross brain injury or sensory handicaps. All three groups were left-hemisphere dominant for word identification, although the mean RVF/LVF ratio of lateralization was greatest for the gifted (14.91/2.41), substantial for the good readers (14.36/4.27) and significant but to a lesser degree (11.6/5.3) for the dyslexics; the combined mean VF accuracy scores did not vary appreciably among the groups. This study, statistically controlling for group differences in IQ and for reading levels as interacting variables, offered evidence to support the hypothesis that dyslexia is related to "nonintellectual, lateralized word-processing functions" (p. 65). Thus the adequate acquisition of reading skills necessitates "enhanced left-hemisphere decoding of written language" while reading disability may be associated with the "prevalence of a right-hemisphere-based perceptual coding strategy" (p. 61) which interferes with reading skill acquisition and performance. Although McKeever and Huling (1970) in an earlier study, based on a similar paradigm, with seventh-grade normal and exceptionally poor readers (three to four year reading delay) found a highly significant RVF effect and a marked left-hemisphere advantage in both groups, their subsequent data analysis fails to account for the superior overall and equally asymmetrical performance (evident in the raw-score results) shown by the poor reader group on this word-recognition task.

In his later and more extensive investigation with VanDeventer (1975), McKeever designed a series of lateralization experiments (conducted over a two-year period) to study the performance of chronic dyslexics for possible defects occurring at "the level of simple

language stimulus processing" (p. 363); a second issue under consideration was dyslexic cerebral dominance patterns for language. Nine male, right-handed, adolescent dyslexics (reading two or more years below grade-level, extensive reading remediation background, five with familial dyslexic histories--exclusionary factors included subnormal intelligence, visual or hearing deficits, cultural, economic or educational deprivation, bilingual background, neurological or medical problems, and primary emotional or behavioral problems) and nine normal readers were matched except for IQ (mean differential +12.2) and reading level (at grade-level or above). The unilateral and bilateral tachistoscopic word-recognition tasks and the vocal reaction-time task were administered and repeated one year later with modification: stimulus exposure durations were increased to enhance general recognition levels for both groups and to eliminate the LVF "floor" effect manifested by dyslexics in the bilateral word-recognition presentation. Both word-recognition test sessions elicited significantly greater RVF and LVF accuracy scores from the normal reader group on unilateral and bilateral presentations. In the first test session, each group performed significantly better in the RVF on both presentations but with some relative RVF decrement on the bilateral presentation; the dyslexic LVF performance in the bilateral presentation, however, exhibited a "floor" effect while the normal reader LVF mean score was maintained across presentation modes. One year later in the second test session word-recognition performances (after stimulus duration had been increased from an average 28.65 msec to 100 msec) yielded significantly better overall scores for both groups. Dyslexics showed no asymmetry on the unilateral presentation but significant asymmetry in favor of the RVF for the bilateral section;

this asymmetry was the result of a LVF recognition decrement as the RVF performance remained consistent. Normal readers showed significant asymmetry for both presentations and maintained LVF and RVF accuracy scores. In the vocal reaction-time task (letter identification), although responses for each group in both test sessions were significantly faster for the RVF, dyslexics demonstrated significantly longer latencies than those of normal readers for both VF. In contrast to the findings of Berlucchi et al. (1974) testing normal subjects, CF stimuli (given only in the first test session) elicited greater latencies than VF stimuli for both groups, although dyslexic performance was again markedly inferior to that of normal readers. A nonverbal tachistoscopic task (introduced in the second test session) examined manual reaction-time responses to unilateral dot presentation; no significant intergroup latency differences or intra/intergroup VF preferences resulted. A dichotic-listening task--recall of bilaterally presented digit series--was also introduced in the second test session. Although the results demonstrated significantly superior recall, normal readers over dyslexics, for both auditory channels, each group was left-hemisphere dominant on this task and the right-ear/left-ear mean difference (or degree of asymmetry) was essentially the same for each group.

McKeever and VanDeventer's general conclusions for the most part appear substantive: chronic dyslexics possess "clear left hemisphere language lateralization;" dyslexics are "impaired in their processing of brief, simple language stimulation" (p. 374); the forms of dyslexic impairment in visual and auditory language processing differ in character from those of nondyslexic equally disabled readers (see McKeever & Huling, 1970); and, dyslexics demonstrate "clear auditory short term

memory impairment for dichotic digits" (p. 375). The convoluted rationale for McKeever and VanDeventer's final conclusion--that dyslexics possess "an additional deficit of left hemisphere visual association area function"--lacks a valid basis in terms of their tachistoscopic experimental design and the results derived. The absence of any statistical correlation between dichotic listening deficits and impairment in word-recognition task performances, their blurring of distinction between auditory and visual language processing strategies, and finally their failure to take into account the additional memory component in the dichotic listening task, all serve to call into question the basis on which these researchers promulgate this final hypothesis. Worth noting is a study with a similar experimental tachistoscopic paradigm conducted by Teng and Sperry (1973) with split-brain subjects. Visual-field performance patterns for the split-brain subjects (particularly with manual responses) appear to conform to or parallel those VF patterns that occurred with McKeever and VanDeventer's dyslexic subjects: left hemisphere performance superior to right hemisphere performance, unilateral presentation superior to bilateral presentation, and right hemisphere deficit more pronounced than left during bilateral presentation. Teng and Sperry attributed the highly significant right-hemisphere response suppression when the left hemisphere is simultaneously stimulated to a "neglect" of LVF stimuli during bilateral presentation--a phenomenon not unlike that occurring with McKeever and VanDeventer's dyslexic subjects, particularly with shorter stimulus exposure durations.

Employing psychophysical techniques in their tachistoscopic study, Gross, Rothenberg, Schottenfield and Drake (1978) investigated duration

thresholds for unilateral VF letter identification with normal readers and dyslexic children. Fourteen dyslexics (at least two years behind grade-level on the Gray Oral Reading Test) and 15 normal readers (at or above grade-level)--age range 10-13.5, IQ 90 or above, normal or corrected vision, and no evidence of gross neurological deficits or severe emotional problems--were essentially matched for age, IQ and sex (two girls in each group). Although three dyslexic subjects were left-handed, Gross et al., in reporting individual data, notated these subjects' results and, in addition, when appropriate, withdrew this variable in their statistical analyses. Individual letter-identification thresholds (from durations which produced 100% correct to those producing chance 25% correct performance--with two intermediate identification "values") for each subject were determined by utilizing the "method of constant stimuli." The researchers then designated that duration threshold--calculated from regression lines--which yielded 62% correct identification (midpoint between 100% and chance 25%) for each individual as the basis for intra- and intergroup comparisons. Significantly higher threshold averages over both VF occurred for the dyslexics (mean value 47 msec) than for the normal readers (mean value 13 msec); absolute differences in thresholds between the VF were also significantly greater with dyslexics (averaging 11.4 msec) than with normal readers (averaging 1.3 msec). These intergroup differences were even more pronounced when the three left-handed dyslexics were excluded from their statistical analyses. Although direct comparisons between studies utilizing different methods must be approached cautiously, lack of correlation between intelligence measures and any threshold measure for either group in this study appears analogous to Kershner's (1977)

finding, in his word-recognition hemifield study, of no significant relationship between IQ and laterality preferences in gifted children, good readers or dyslexics. In Kershner's study, however, dyslexic reading performance seemed to be related to less lateralized word-processing strategies; Gross et al. found that less lateralized threshold durations for letter identification correlated with higher reading performance levels in normal readers--no relationship was found between reading level and laterality measures in their dyslexic group. Based on examination of their data, Gross et al. concluded that the significantly higher duration thresholds and/or perceptually asymmetrical performances occurring among the dyslexics indicated that "psychophysically determined visual functions in these children were not equivalent to those in normal readers" (p. 713). In addition, Gross et al. tentatively attributed their dyslexic subjects' marked VF differences in perceptual duration thresholds to a partial impairment in interhemispheric transfer of visual information--although, they conceded, alternative explanations could also be formulated.

The EEG study undertaken by Leisman and Ashkenazi (1980) involved 20 dyslexic children (7-10.9 years of age, 19 boys and one girl, an IQ ranging from 90-124, no evidence of ocular pathology or eye movement abnormality as well as no mental retardation or overt neurological deficiencies--each subject at least two years retarded in reading) originally subgrouped according to Boder's (1973) clinical classification; but, since the EEG spectral data demonstrated no significant differences between these subgroups, dyslexic data were pooled for the statistical analyses. The 20 normal readers in the control group were essentially matched for age and IQ although this group contained 16 boys and 4

girls reading at or above grade-level. The International 10-20 system (Jasper, 1954) for electrode placement was employed and EEG activity was recorded during three experimental tasks: rest-eyes closed and rest-eyes open while subjects underwent continuous performance tests (Leisman, 1973, 1974); during confrontation naming of projected items from the Stanford-Binet test; and, while subjects read paragraphs from the Spache Diagnostic Reading Tests. The resultant EEG data were then subjected to time series, cross series spectral estimation and coherence analyses "to determine the amount of sharing between any two wave trains" (p. 158). Results, particularly from the rest-eyes closed condition, demonstrated that dyslexics show a frequency spectrum--recorded from the parieto-occipital leads--consistently unlike that of the normal readers. This finding, Leisman and Ashkenazi suggested, may be attributed to a difference in functional organization between dyslexics and normal readers in this area of the brain. In addition, coherence values for normal readers provided evidence of greater shared activity between homologous regions in each hemisphere reaching significance at the (P_3O_1/P_4O_2) locations. Dyslexics in contrast demonstrated greater intrahemisphere coherence values during all tasks, which attained significance for the rest-eyes closed condition and at the (P_3O_1) location. From examination of the data these researchers proposed that a greater sharing--and perhaps superior communication--exists between the two cerebral hemispheres in normal readers and that comparative lack of sharing between hemispheres in dyslexics contributes to their reading impairment. The relative autonomy of the right hemisphere in dyslexics--"perhaps equivalent to the left" (p. 163)--could therefore conceivably be interfering with the left/right dominance relationship characteristic

of reading skill acquisition and performance achievement.

Utilizing essentially the same EEG techniques and data analysis procedures, Sklar, Hanley and Simmons (1972, also see 1973) had earlier tested 12 dyslexics (10 boys and 2 girls, age range 7-18) and 13 controls (11 boys and 2 girls, age range 7-16). The test situations involved the monitoring of EEG activity for 10-sec periods while the subjects were at rest, eyes closed; attentive, eyes open; performing mental arithmetic; reading word lists; and, reading text. The tasks which most discriminated between dyslexics and normal readers were the rest, eyes closed and the reading text conditions, which these researchers focus on in their analyses of the results. Sklar et al. found, directly related to Leisman and Ashkenazi's (1980) subsequent EEG experiment, that during these two conditions coherence between intrahemisphere leads (O_2T_4/T_4F_4) averaged higher in dyslexics than normal readers whereas coherence between symmetrical regions (P_3O_1/P_4O_2) across the intercerebral midline tended to be higher in normal readers. The higher coherence of intrahemisphere EEG activity in dyslexics and the contrasting higher coherence between homologous regions of the two hemispheres in normal readers appears once again to point to the interhemispheric communication system--callosal transmission and its possible dysfunction as a contributing source of reading impairment.

Hemispheric Vigilance

Impairment of attention has been proposed as an etiological factor for academic underachievement in children classified learning disabled, hyperactive, and/or minimally brain-damaged and has been extensively investigated (Ackerman, Dykman & Peters, 1977; Cohen & Douglas, 1972;

Douglas, 1976; Doyle, Anderson & Halcomb, 1976; Dykman, Ackerman, Clements & Peters, 1971; Dykman, Ackerman & Oglesby, 1979; Kinsbourne, 1973; Senf and Freundl, 1971; Sheer, 1976; Sroufe, Sonies, West & Wright, 1973; Sykes, Douglas & Morgenstern, 1973; Wender, 1971) with results that have been generally inconsistent. In at least one study (Margolis, 1972, cited by Jerison, 1977), a significant positive relationship had been demonstrated between reading ability (but not IQ) and vigilance performance in children; however, attention--particularly vigilance--remains essentially an unresearched area in those learning disabled children specifically diagnosed as developmental dyslexics. Research, conducted in the last decade (Dimond, 1976, 1977, 1979; Dimond & Beaumont, 1971b, 1973; Ellenberg & Sperry, 1979, 1980; Heilman & Van Den Abell, 1979, 1980; Warm, Schumsky & Hawley, 1976), focusing on intra- and interhemisphere vigilance performance patterns, offers a new perspective and approach to the study of cerebral lateralization of function in the dyslexia syndrome. These vigilance studies--testing normal subjects and/or split-brain patients--have consistently produced findings of differential hemispheric performance patterns which reflect on both this project's theoretical basis and experimental design. Two major determinations drawn from these investigations propose that "there are two different hemispheric vigilance systems of the brain" (Dimond, 1977, p. 341) and that "the cerebral commissures [most likely the splenium of the corpus callosum] force the two hemispheres to work together and maintain attentional unity in the intact brain" (Ellenberg & Sperry, 1980, p. 411). In addition, Dimond (1977) maintained that independent examination of right and left hemisphere vigilance patterns and examination of the system by which the two halves of the brain

function together to sustain vigilance performance are relevant to an exploration and understanding of the success or failure "of the mechanisms of consciousness to respond to the stimuli they receive" (p. 358).

In the first experiment designed to investigate differential intrahemisphere vigilance performance with normal subjects, Dimond and Beaumont (1971b) tested manual response to light signals flashed infrequently at irregular intervals and distributed evenly between the two hemisphere channels. No difference between the right and left hemisphere was revealed in terms of "detections," but overall performance declined significantly in both hemispheres during the extended 80-min test-period. More important, false positive responses (response by subject when no signal had been flashed) occurred significantly more often in the left-hemisphere channel than its counterpart. This difference in sensitivity between the right and left hemispheres led Dimond and Beaumont (1973) to perform the experiment again, but modified so as to present the signals to a single VF for the duration of the task. In this subsequent testing, the left hemisphere was significantly superior in the detection of stimuli, although performance by this hemisphere showed a steady decline over time. Performance in the right hemisphere, however, began at and culminated in a significantly lower detection rate relative to the left hemisphere, but consistent performance was maintained throughout the task. This resultant contrast, Dimond and Beaumont concluded, attests to the existence of two vigilance systems within the human brain--"a primary system operating initially at a high level, showing decrement with time, and associated with the left hemisphere, and a secondary system showing no decrement, but operating

at a lower level, associated with the right hemisphere" (p. 265). Vigilance research conducted by Heilman and Van Den Abell (1979, 1980) with normal subjects supports the assignment of right hemisphere dominance for the mediation of cerebral "activation" in the intact brain. Cognizant of Dimond and Beaumont's (1973) model of two "watchkeepers" in the human brain, Heilman and Van Den Abell (1979) performed a vigilance experiment designed to separate the "watchkeeping" function from activation--defined as "physiologic readiness to respond" (p. 315). Statistical analysis of experimental findings disclosed that LVF warning signals (red diode) significantly reduced the CF stimuli (green diode) reaction-time responses--a greater reduction than when the same warning signals were presented to the RVF. This right hemisphere dominance for activation of attention in normal subjects has been further examined by Heilman and Van Den Abell (1980)--as well as its possible application as the mechanism underlying hemispheric asymmetries of inattention ("neglect") in brain-damaged patients. Employing the same experimental paradigm, these researchers also recorded each subject's EEG while performing the task and found that the left parietal lobe desynchronized--"a psychophysiologic correlate of attentional arousal" (p. 329)--"mainly" after RVF warning signals; the right parietal lobe, however, desynchronized "equally" following RVF or LVF warning signals. This variance provides additional evidence of right hemisphere dominance for attention.

Dimond (1976, 1977, 1979) simplified the original version of the Dimond and Beaumont (1971b) visual vigilance task and created comparable tasks modified for auditory and tactile modalities; he then tested

split-brain patients to determine vigilance performance capacity for each hemisphere in relative isolation and to investigate experimentally attentional anomalies previously noted in split-brain patients (Levy, 1977; Sperry, 1974; Zaidel & Sperry, 1974; Zangwill, 1974). These lateralized vigilance tasks were admittedly easy: both normal subjects and partial-commissurotomy patients (splenium intact) experienced little difficulty in achieving perfect or near perfect scores. Total-commissurotomy patients, however, demonstrated a "rapid and apparently inexorable decline in performance" (1977, p. 353). A second result for this group was a clear right-hemisphere superiority in signal detection--reaching significance in the visual and tactile modalities. Marked deficits in conscious attention were also observed in the form of extensive lapses in awareness--no response whatsoever to a series of stimuli--of such dimension to be termed "black holes of consciousness" (Dimond, 1976, 1977); such attentional gaps extended over several seconds and occurred most often in the left hemisphere following the first 10 minutes of vigilance testing. Distinguishing between selective and sustained attention, Dimond (1979) proposed "different mental mechanisms" for the processing of these two attentional operations. The poor left hemisphere and relatively superior right hemisphere detection patterns exhibited by total-commissurotomy patients offer evidence for "the association of sustained attention with the functions of the right hemisphere" (p. 49). In addition, because impaired vigilance performance did not occur on these tasks with partial-commissurotomy patients, Dimond (1977) speculated that the splenium serves to "hold intact the fabric of the attentional process" (p. 357), its retention a requisite for the maintenance of consciousness.

In a recent study by Ellenberg and Sperry (1979) on sustained attention capacity following commissurotomy, testing procedures were divided into two series of tasks--each demanding sustained, focused attention; the series differed "in the amount of active involvement on the part of the subject." The first set consisted of tactual sorting tasks which necessitated active concentration with sustained, self-paced manual responses "to an unvarying, tedious, but attention-demanding task." The second set involved tactual signal-detection tasks corresponding to Dimond's (1977) paradigm, which required more passive vigilance participation--"manual responses to random, experimenter-paced tactual signals" (p. 422).

Performances of the total-commissurotomy patients on the passive signal-detection tasks showed marked deterioration after 10 to 30 minutes; normal subjects experienced no difficulty on these tasks and maintained sustained attention for 60 minutes. These findings correspond to performance patterns observed by Dimond (1976, 1977, 1979) with split-brain patients and normal subjects on similar tasks. The Ellenberg and Sperry commissurotomy patients, however, were capable of sustaining "prolonged concentration and alertness" over a test period extending to one hour when attending to "monotonous sorting tasks in which performance involved repeated sensory discriminations and sorting decisions with continual motor activity" (p. 434). Uninterrupted proprioceptive and external stimulation inherent in the performance of these tasks necessitated an active engagement which apparently served to maintain sustained attention in this group. Thus those "central mechanisms" facilitating concentration and sustained attention in the absence of external reinforcement, Ellenberg and Sperry suggested, appear to involve

commissural transmission in the intact brain and are greatly attenuated after total severance of the commissures. Their finding of an absence of consistent asymmetrical performance in any of the three groups tested on either the passive signal-detection tasks or the active sorting tasks contrasts with Dimond's (1979) conclusion that the right hemisphere is dominant for sustained mental concentration. Motor left/right dysynchrony was observed during bimanual sorting tasks with total-commissurotomy patients but not with partial-commissurotomy patients. This lateral independence of cerebral mechanisms which operated concurrently but separately with total-commissurotomy patients and the absence of such motor dysynchrony in partial-commissurotomy patients and normal subjects, Ellenberg and Sperry speculated, may be attributed to the lack of splenial connections which normally serve to unify "attentional components of cognitive processing" (p. 437).

To further verify this hypothesis, Ellenberg and Sperry (1980) designed an experiment to compare the relative capacities of commissurotomized and normal subjects for "division of voluntary attention between two simultaneous prolonged and attention-demanding tasks involving lateralized input and performance with left and right hands" (p. 412). In attempting to perform two different or conflicting tasks presented simultaneously and independently--stimuli introduced to separate hemispheres--normal subjects experienced considerable difficulty in overcoming the tendency toward bilateral integration of attention seemingly intrinsic to cerebral organization and function in the intact brain. Severing of the cerebral commissures, however, allowed patients to operate and maintain two separate attentional systems, each capable of stimulus processing and individual response.

This ability to sustain independent attentional systems for extended periods "despite continual conflicting left and right motor activity" was found only in total-commissurotomy patients. Thus experimental data derived provide strong evidence that normally attention is restricted to a single focus; the cerebral commissures operate to enforce cerebral integration in which the two hemispheres function together as "a single unified attentional system." That this condition is characteristic of partial-commissurotomy patients and that they, like normal subjects, demonstrated marked performance deficits on "dissimilar bimanual sorting tasks" (p. 416) led Ellenberg and Sperry to conclude that the splenium alone retains the capacity to secure and maintain that cerebral integration necessary for attentional unity.

Temporal Order Perception

Theoreticians and clinical researchers (Bannatyne, 1966; Critchley, 1970; Johnson & Myklebust, 1967; Orton, 1937; Vernon, 1957) in the field of developmental dyslexia have long considered the possibility of impairment in perception and retention of temporally organized stimulus patterns as a contributing factor. Experimentation (Bakker, 1970, 1972; Corkin, 1974; Rudel & Denkla, 1976; Sterritt & Rudnick, 1966; Zurif & Carson, 1970) designed to assess temporal order perception in dyslexics, however, is of relatively recent origin. A major problem which has recurred in this research area has been the inclusion of extraneous and often confounding cognitive variables--particularly encoding and memory processes--which make impossible a direct examination of temporal order perception. These experiments focusing on

deficiencies in sequential perception as a causative factor in developmental dyslexia have consistently--in their task demands--necessitated serial order recall. Such tasks, Vellutino (1977) cautioned, require longer stimulus durations and interstimulus intervals "than those used in studies of the threshold values necessary for perception of serial order" and thus serve to "assess short-term memory and not perception" (p. 341).

In the second section of an early and seldom cited two-part study testing normal subjects, Taubman (1950b) examined sensory discrimination performance--"essentially the judgment of the number of successively presented short . . . light flashes" (1950a, p. 167)⁴--by varying both the number of stimuli presented (1 to 10 light flashes) and the "intrastimulus" time interval (.500, .333, .250, .200, or .143 sec). Data collected from this study testing the limits of visual temporal order perception were used to assess thresholds and the effects of "intrastimulus" time intervals relative to actual and judged number of stimuli presented. At the .500 sec interval there existed a "perfect correspondence between the judgment scales and the stimulus continuum" (p. 197). Decrements in ascertaining stimuli number, however, occurred as the "intrastimulus" time interval was decreased and as a greater number of stimuli were flashed. In addition, percent correct--"mean stimulus which could be perceived without error at the various time intervals" (p. 207)--steadily declined with shorter time intervals after an initial dramatic drop-off (10 to 2.4 light flashes) which

⁴In the first section of this two-part study Taubman (1950a), testing the same subjects using a similar experimental design, investigated their ability to discriminate rapid, successively presented series of short tones.

occurred when the "intrastimulus" time interval was decreased from .500 to .333 sec. This seminal research together with studies by Hirsh (1959) and Hirsh and Sherrick (1961)--investigating absolute interstimulus thresholds employing psychophysical techniques--provides methodologies for examining fundamental elements of temporal order perception.

Tallal (1980), concentrating on primary perceptual processing deficits as a possible contributory factor in reading dysfunction, designed an experiment from which the verbal processing factor was eliminated. Drawing from earlier studies of dysphasic children, conducted with Piercy (1973, 1974), which employed auditory perceptual tasks--"association, discrimination, sequencing and rate perception" (p. 181)--Tallal tested elemental aspects of temporal perceptual processing in reading-impaired children. These experimental tasks provided innovative investigative techniques to study "in more detail" earlier research findings (Bakker, 1970, 1972; Corkin, 1974; Rudel & Denkla, 1976; Sterritt & Rudnick, 1966; Zurif & Carson, 1970) of temporal order processing deficits among dyslexic children. Statistical analyses of test results demonstrated that a "subgroup" (9 subjects) of the reading-impaired children (4 girls and 16 boys, age range 8-12 years, 1 year or more delayed in reading, Verbal, Performance and Full Scale IQ 85 or above, normal hearing and visual acuity, no gross emotional disturbance) experienced significant difficulty with temporal pattern perception, attributable to a "primary" perceptual deficit which influenced "the rate at which they [could] process perceptual information" (p. 193). This difficulty transcended limited ability to sequence since, in a task requiring discrimination between two nonverbal stimuli,

significant group differences were also revealed when the interstimulus time interval was shortened. With relatively prolonged interstimulus intervals, however, performance accuracy on the sequencing task (perception of temporal order requisite) and the discrimination task (no perception of temporal order requisite) was comparable for both normal readers and reading-impaired children. Therefore, an increase in rate of perceptual processing on these tasks--rather than perception of temporal order--appeared to elicit significant performance deficits in the "subgroup" of reading disabled children. This last observation led Tallal to conclude that stimuli presentation rate may significantly influence higher level perceptual functions "by interfering with the discrimination of the stimulus items to be processed" (p. 194). This primary perceptual dysfunction affecting "rapid acoustic analysis" could also underlie the temporal processing disorders--including sequencing and serial memory--which in previous studies have characterized dyslexic children. Tallal further stressed the absence of similar experimentation with other sensory modalities, particularly visual, thus disallowing the specific assignment of "rate of presentation in temporal order perception" (p. 194) as a major factor in the genesis of the dyslexia syndrome.⁵ Significant correlations between reading

⁵Denckla and Rudel (1976b), using rapid automatized naming (R.A.N.) exercises, differentiated dyslexic children from nondyslexic, learning-disabled children as well as normal readers by their deficits in "automatization" of verbal responses to visually presented stimuli (colors, numbers, letters, and objects). These researchers conceded, however, that rate of processing may have only partially influenced their results and that "attentional drift, poor speech-encoding, slow verbal retrieval, and subtle visual processing delays" (p. 477) must all be considered as well.

subskills, particularly phonetics, and tasks requiring rapid auditory perception (and nonsignificant correlations between these rapid auditory tasks and age, Verbal IQ or Performance IQ) indicate a direct, positive relationship between the acquisition of reading skills and adequate rapid auditory perceptual processing. Tallal's application of her findings to an analysis of the dyslexia syndrome is of questionable value since her subject population contained some severely reading-impaired children--possibly dyslexic--and some children only slightly reading delayed. Her failure to identify the reading-impaired "subgroup" (which performed significantly below normal limits on the rapid auditory perceptual tasks) and to analyze this "subgroup" data relative to the other reading-impaired subjects as well as the normal reader group also limits any conclusive implications drawn from these results.

Research Hypotheses Under Investigation

Three aspects of this research project distinguish it from previous experimentation in the field of dyslexia:

- 1) The experimental task--successive, rapid presentation of CF stimuli occurring concurrently with variable, infrequent presentation of VF stimuli--has no counterpart in previous dyslexia research;
- 2) The technology involved in implementing this experimental design represents an innovative approach to the investigation of visual temporal perception;
- 3) Extensive neuropsychological testing and experimentation

with a population of adult developmental dyslexics has been relatively unexplored.

For these reasons specific hypotheses are deemed inappropriate. More general assumptions which appear both reasonable and defensible may be subgrouped into five areas:

- 1) Neuropsychological test performance profiles of adult developmental dyslexics would indicate comparable deficit patterns to those of child developmental dyslexics;
- 2) Dyslexics would demonstrate significantly different VF identification and/or correct-but-late response patterns from normal readers, which could conceivably provide laterality evidence in support/refutation of neurological theories of dyslexia;
- 3) Dyslexics would encounter considerable difficulty in identifying and/or quickly responding to visual stimuli presented continuously and rapidly CF;
- 4) A comparison of performances with adult dyslexics and normal readers on this rapid-paced CF perceptual/VF vigilance task should yield data and information on relative CF/VF processing patterns;
- 5) Correlations between the NTB and experimental task performances in each reader group as well as comparisons/contrasts between the two groups should distinguish profile patterns and correspondances between the two investigative areas.

III. Methods

Subjects

Through extensive screening a subject population of 10 adult developmental dyslexics and 10 adult normal readers was selected for this study. These subjects were drawn from the metropolitan New York area, possessed comparable education backgrounds, and were recruited from academic institutions or educational assistance programs. Each subject received substantial (\$70 for two sessions) financial remuneration for participation in the experiment and was provided with an evaluation of his neuropsychological test results. The demographic qualifications involved a matching for age, as well as uniform sex, handedness and race. An attempt to match test subjects for IQ was not undertaken since the experimental task was essentially noncognitive. Using the Mann-Whitney U test, Full Scale IQ, $U = 11.5$, $p < .025$, and Verbal IQ, $U = 7.0$, $p < .005$, scores were significantly greater for normal readers than dyslexics; Performance IQ, $U = 21.0$, $p = .167$, scores, however, were not significantly different. Subjects were limited to ages 18 through 26 so as to focus on adult dyslexics, a population which previously had received relatively slight attention (Benton, 1970; Trites & Fiedorowicz, 1976). Because the experimental task was designed to examine possible differential hemisphere functioning, recent evidence of significant differences in male/female hemisphere performance patterns (Johnson & Harley, 1980; Rebert & Mahoney, 1978; Witelson, 1976) provided the rationale for restricting the population to either males or females. The relative prevalence of dyslexia among

males (Benton, 1975; Critchley, 1970) was the reason for restricting this study to male subjects. To minimize the possibility of right hemisphere or bilateral language representation, as well as other confounding hemisphere processing differences, only right-handed subjects were chosen (Beaumont, 1974; Bryden, 1965; Goodglass & Barton, 1963; Subirana, 1969). By limiting the population exclusively to Caucasians a surface homogeneity was achieved and cultural variables were somewhat lessened (Vellutino, chap. 2, 1979).

Criteria for inclusion in the dyslexic group required a previous diagnosis of dyslexia or primary reading disability; a record of reading remediation; a reading score equal to or greater than one standard deviation below Full Scale IQ and six grade levels or more below educational level on one or more of three reading tests⁶ (Gray Oral Reading Test; Wide Range Achievement Test; Woodcock Mastery Reading Test); and, word identification skills more impaired than reading comprehension. Applicable to both the dyslexics and the normal readers were Verbal, Performance and Full Scale IQ scores equal to or greater than 90; no evidence of "gross" brain damage; no primary (i.e. psychotic) psychiatric profile; and, English as a first language.

From preliminary screening and review of available assessment files (for potential dyslexic subjects), 12 normal readers and 12 dyslexics were finally selected for participation in this study. Medical and developmental histories were collected to establish both individual and group profiles. For the purpose of comprehensive testing of reading skills and related cognitive functions, as well as identifying

⁶ All three reading-test scores for each dyslexic subject were three grade levels or more below educational level.

and defining individual (Appendix A) and group parameters (Tables 1, 2 & 3), the following neuropsychological test battery (NTB) was administered:

1. Two laterality preference questionnaires,
 - a. Oldfield's (1971) Edinburgh Handedness Inventory,
 - b. Coren, Porac & Duncan's (1979) Behaviorally Validated Self-Report Inventory to Assess Four Types of Lateral Preference,
2. Wechsler Adult Intelligence Scale (WAIS),
3. Wide Range Achievement Test (WRAT) (reading, spelling, and arithmetic sections),
4. Purdue Pegboard (3 trials of right, left, and both hands and 1 assembly trial),
5. Woodcock Mastery Reading Test (letter identification, word identification, word attack, word comprehension, and passage comprehension sections),
6. Knox Cube Test (2 trials),
7. Raven Standard Progressive Matrices,
8. Short paragraph written on any subject matter they chose,
9. Double Simultaneous Stimulation,
10. Gray Oral Reading Test,
11. Boston Naming Test,
12. Rey-Osterrieth Complex Figure Test,
13. Spreen-Benton Sentence Repetition Test.

Medical and developmental histories, subsequently confirmed by results on the NTB, offered strong evidence of possible brain damage in two dyslexic subjects, thus eliminating them from this study. One normal reader's developmental history revealed--a disclosure by the

Table 1
MEAN AGE, EDUCATION & INTELLIGENCE TEST SCORES BY READER GROUP

	Dyslexics (N = 10)	Normal Readers (N = 10)
AGE	19.9 ± 2.4	19.6 ± 2.5
EDUCATION	13.1 ± 1.1	14.0 ± 1.4
WAIS FS IQ	105.7 ± 8.3	122.2 ± 12.3
WAIS VIQ	101.0 ± 7.7	120.8 ± 11.7
WAIS PIQ	111.3 ± 10.9	120.9 ± 12.1
Information	9.0 ± 1.9	12.8 ± 1.8
Comprehension	12.2 ± 2.6	14.5 ± 2.5
Arithmetic	8.9 ± 2.0	13.3 ± 3.6
Similarities	11.8 ± 1.5	12.7 ± 1.8
Digit Span	7.9 ± 1.7	13.3 ± 3.4
Vocabulary	9.2 ± 2.1	12.5 ± 2.3
Digit Symbol	9.6 ± 1.0	14.0 ± 2.3
Picture Completion	13.7 ± 2.5	13.9 ± 2.6
Block Design	11.5 ± 3.5	13.2 ± 2.8
Picture Arrangement	11.6 ± 1.8	11.7 ± 2.1
Object Assembly	12.0 ± 2.2	12.8 ± 3.4

Table 2
MEAN READING, SPELLING & ARITHMETIC TEST SCORES
BY READER GROUP

	Dyslexics (N = 10)	Normal Readers (N = 10)
WRAT		
Reading	6.4 ± 2.0	14.8 ± 2.1
Spelling	5.4 ± 1.8	11.7 ± 2.2
Arithmetic	6.4 ± 1.2	9.5 ± 3.4
WOODCOCK ^a Total Score	6.3 ± 2.9	12.8 ± 0.2
Letter Identification	9.7 ± 4.2	10.9 ± 3.2
Word Identification	5.8 ± 2.6	12.9 ± 0.0
Word Attack	3.5 ± 1.0	12.4 ± 0.9
Word Comprehension	8.1 ± 3.7	12.8 ± 0.3
Passage Comprehension	7.4 ± 3.5	12.8 ± 0.2
GRAY ORAL ^b	4.5 ± 2.3	12.0 ± 0.0

Note. Grade level mean scores are reported for these tests.

^aMaximum grade level score for this test = 12.9.

^bMaximum grade level score for this test = 12.0.

Table 3

ADDITIONAL MEAN NTB TEST SCORES BY READER GROUP

	Dyslexics (N = 10)	Normal Readers (N = 10)
<u>Verbal</u>		
BOSTON NAMING	71.0 $\pm$ 6.0	80.0 $\pm$ 4.1
SENTENCE REPETITION	13.4 $\pm$ 2.0	16.0 $\pm$ 1.8
<u>Visual</u>		
RAVEN	43.4 $\pm$ 6.3	50.9 $\pm$ 6.2
KNOX	12.5 $\pm$ 1.2	14.0 $\pm$ 2.7
REY-OSTERRIETH		
Copying	33.3 $\pm$ 1.4	34.5 $\pm$ 1.1
Memory	18.6 $\pm$ 6.4	22.0 $\pm$ 5.8
<u>Motor</u>		
PURDUE PEGBOARD ^a		
Right Hand	29.7 $\pm$ 22.6	39.9 $\pm$ 14.7
Left Hand	31.9 $\pm$ 29.3	49.3 $\pm$ 21.6
Both Hands	30.0 $\pm$ 25.0	42.4 $\pm$ 17.1
Assembly	67.1 $\pm$ 26.2	83.3 $\pm$ 12.7
<u>Sensory</u>		
DOUBLE SIMULTANEOUS STIMULATION		
Total Errors	2.6 $\pm$ 2.8	1.2 $\pm$ 2.1
Right Errors	1.7 $\pm$ 2.0	0.9 $\pm$ 1.2
Left Errors	0.9 $\pm$ 1.1	0.3 $\pm$ 0.9

^aPercentile scores reported here (using 3 trials each for right, left and both hands and 1 trial for assembly) are based on Male Purdue Pegboard Norms (industrial applicants).

mother unknown to her son--that he had been switched from left to right handedness as a young child and therefore he was excluded as a subject. Another normal reader was also dropped as a subject since he produced test results in the "brain-damaged" range on the Purdue Pegboard and demonstrated severe memory deficits on both visual and verbal NTB tests.

Apparatus

The experimental task required a visual display system with the capacity to present stimuli--a series of 580 single-digit numbers center field (CF), 40 of which were accompanied by a simultaneous single-digit number in right or left visual field (VF)--in rapid succession with accurate timing and consistent luminosity. The stimuli were to be flashed for 15 msec at 1-sec intervals; the intervals were to be extended to 1.5 sec when the CF stimulus was accompanied by a VF stimulus.⁷ The rapid-presentation rate with accurate interstimulus timing in this experiment necessitated design and construction of a specialized computer system interfaced to a light-emitting-diode (LED) display.

The computerized visual display system design objectives may conveniently be subdivided into seven separate and distinct functions:

- 1) Digit sequence stored for checking and/or modification if necessary, as well as made easily accessible for repeated presentations; digit sequence organized in three sets, one set representing the CF stimulus and the other two sets representing right and left VF stimuli;

⁷These intervals were of sufficient time to prevent visual masking (Stanley, 1976; Stanley & Hall, 1973).

- 2) Program to provide three presentation modes: unilateral presentation equally to both right and left VF, unilateral presentation only to the right VF, unilateral presentation only to the left VF;
- 3) Digital display components in three fields on neutral background, exact digit sources not visible when unlit; distance between CF and two VF components adjustable to achieve desired visual angle;
- 4) Means provided to control display intensity, intensity constant with time and temperature; no perceptible differences in the relative intensities of the three digital displays;
- 5) Control supplied for both stimulus duration timing and interstimulus timing, CF interstimulus timing (1-sec) controlled separately from CF/VF interstimulus timing (1.5-sec), CF and VF digits displayed together to appear and disappear simultaneously; all timings accurate and repeatable;
- 6) Responses to simultaneous presentation of CF and VF stimuli recorded and measured by elapsed time from stimuli onset until subject's vocal response to CF stimulus and interim timing between CF and VF vocal responses, taped record of audio signals of stimuli onset times together with subject's vocal responses throughout experimental task;
- 7) Upon completion of each digit sequence presentation, subject's timed responses to simultaneous CF/VF stimuli made available for transcription.

The Radio Shack TRS-80 level II, a microcomputer system with 16K of memory, level II BASIC language and tape cassette recorder (Realistic CTR-80) for storage and retrieval of program, was chosen to retain the digit sequence and control the timing of the experiment. The original design concept involved display of the digit sets using the computer's video terminal with the timing of the experiment controlled by a timing loop in the program. Unfortunately, upon examination, the TRS-80 microcomputer disclosed major systemic deficiencies in experimental design performance requirements. The intensity and size of the video display were rather unstable, the largest variation attributable to powerline voltage changes. Measurement of the speed of BASIC programs indicated that even the tightest possible loop required approximately 2 msec for a single execution; longer program sections required disproportionately more time. This factor severely limited timing precision, particularly of the stimulus duration. In addition, the raster scan of this standard video display refreshed a single point on the display only every 16.7 msec. Thus, even if the stimulus duration could be precisely timed by the program, the nature of the video display would introduce considerable variation.

To overcome these obstacles special hardware was designed and constructed providing a stable, responsive display and a standard time base for the experiment. An interface was built to transfer data between this hardware and the computer. Finally, portions of the program were written in machine language instead of BASIC to increase program execution speed.

Three LED digital display components (Hewlett-Packard type 5082-7730) were mounted in a 48 by 31 by 18 cm wooden box. The center

display component and the decoder-driver electronics were mounted on a circuit board in the center of the unit. A pin-point light source (IDI white mini-dot light [Model B473054] covered by black tape with minute cutout) was placed directly below the midpoint of the center display component. The other two display components were mounted on separate circuit boards connected to the center board with flexible cables. These boards were fastened to an aluminum rail with spring clips to adjust the distance between the outer displays and the center display. This entire unit was enclosed behind a sheet (6 mm thick) of transparent amber acrylic plastic so that the display components were visible only when lit. The LED components employed were 0.3 inch, red, seven-segment displays; their 7.62 by 5.18 mm italicized numeric characters (slanted 10°) generated a bright, continuously uniform light. These single-digit displays also provided a high contrast ratio⁸ and a wide viewing angle. Each display was composed of eight light-emitting diodes optically spread to form seven individual segments and a decimal point (the latter not utilized in this experiment). The LED display brightness was temperature sensitive. To overcome this variable factor a temperature compensation circuit was included on the board with the

⁸A spectral emission test performed using a Jarrel Ash Monochrometer (with the filter removed and operating at a display intensity control setting of 3/16) demonstrated that the spectral output peaked at the HP specified wavelength of 655 nm. In addition, utilizing the hardware filter from this experiment (6 mm amber acrylic plastic), a filter transmission test performed using a Jarrell Ash Monochrometer with Gamma Scientific Standard Light Source #626 established that the filter transmission (obtained by dividing the output of the second scan [filter interposed between the lamp and the detector] by the output of the first scan [only standard lamp]) wavelength output peaked very strongly in the red and accounted for the excellent contrast of the display.

decoder-driver electronics. Since this circuit was contained within the unit itself, good compensation could be achieved for ambient temperatures from 15° to 35°C.⁹ This circuit also provided power-supply regulation for the displays so that brightness was not dependent on power-line voltage. In order to minimize variations in brightness from one display component to another, these displays were manufacturer tested and grouped according to brightness; the three displays used were all from the same group.¹⁰ Both digital data and power for the electronics were obtained from the computer interface.

The electronics in the computer interface received data from the computer's data buss and converted it to the appropriate form to drive the displays. Activation of the display generated an audio tone. Each time a subject was tested a stereo tape was made using a Techtonics M33 deck; the subject's vocal responses, via an independent microphone

⁹ Display thermal sensitivity was measured using a Gamma Scientific Photometer (Model DR-14) with a photopically corrected PMT detector (Model D-46A) and a Photomicroscope (Model 700-10-37) equipped with a .006 inch diameter probe which focused on one segment of the center digit. This measurement was performed with the display operating at the intensity control setting of 3/16. Temperature within the display box (manipulated using either a fan or auto-transformer, controlled, register-heater and measured with a thermometer) was stabilized for 5 minutes at each of five temperatures ranging from 20° to 41°C. Output results, recorded at each of these five temperature settings, indicated that over a temperature range of 5° the variation in output was $\pm 5\%$ (much less than can be perceived by the average person).

¹⁰ Brightness uniformity between displays was measured using a Gamma Scientific Photometer (Model 2020-1 Serial #1155; PM Tube type 100 Serial #73756). This measurement was performed with the filter removed and the display operating at an intensity control setting of 3/16. Uniformity between displays was very good with the average output per segment varying less than 6% from the overall average segmental output (far less than can be detected by the human eye). The small differences could be attributed to slight positional alterations in measurement spacing.

(Electro-Voice dynamic omnidirectional professional Model 635A), were recorded on the first channel, and the audio tone was recorded on the second channel. The brightness of the displays was controlled by turning them on and off at a 12.8 kHz rate and varying the ratio of on time to off time. Maximum brightness could be reduced in 1/16 increments by selective setting of four switches on the interface board. The selected level was easily repeatable and did not interact with the temperature compensation circuitry. Two potential disadvantages of this method involved the possibility that the image might separate into many discrete images during rapid eye movements, and some jitter might be introduced into the stimulus interval timing. Both disadvantages were overcome by the high 12.8 kHz chopping rate. From a viewing distance of 91 cm or less, a smooth image could be maintained for eye movement rates not exceeding 128 radians/sec; and the jitter in the stimulus timing was less than .078 msec. Data were also fed into the computer from two sources via the electronics in this interface: first, from the relay contacts on the voice operated relay switch (VOX [Grason-Stadler Model E7300A]) used to measure timing of subject's vocal response onsets; and second, from a crystal-controlled 5 msec time-base generator included on the same circuit board as the interface electronics.

Two computer programs were written to control the experiment. The stimulus timing and the measurement of the subject's responses were processed by a machine language program; all other functions where execution time was not critical were directed by a program written in BASIC. The data displayed and values for stimulus duration, CF interstimulus time, simultaneous CF/VF interstimulus time, and VOX masking time (length of time after the display was turned on during

which the VOX status was not checked) were part of the BASIC program and could easily be modified by the experimenter. During the experiment each of the three presentation modes was available for selection. The BASIC program then delivered the data to be presented; reordered it when required to change the presentation mode; checked the left and right digits and substituted 15 in the program--such substitution permitting digit transfer to the appropriate VF (either right or left); and stored the data in an array in the computer's memory, accessible to the machine language program. The timing values were converted from msec to the nearest 5-msec unit and were also stored in memory. Control then passed to the machine language program which retrieved from memory the first digit set to be displayed and sent it to the data latch in the computer interface. This program then synchronized itself to the time base generator and activated the display for the specified length of time. Following display presentation the program determined whether a CF stimulus or simultaneous CF/VF stimuli were to be displayed and waited the specified time interval before retrieving the next digit set and reactivating the display. When simultaneous CF/VF stimuli were displayed, the timings of the first two VOX actuations--which occurred after the VOX masking time but before the next CF stimulus was presented--were recorded in an array in memory. After all stimuli (620 digits) had been presented, control was returned to the BASIC program. The second part of the BASIC program retrieved VOX timing values recorded in memory and displayed them to the experimenter. The results could be reviewed as required and then either the same presentation could be rerun or the presentation mode could be changed before proceeding.

General Experimental Design

The experimental design included four presentations utilizing three presentation modes. The distinction among the three modes involved the manipulation of only one variable--sidedness of the VF stimuli. The digital stimuli sequence employed in each of the four presentations was the same; piloting with normal readers and one dyslexic subject had indicated no memory carryover. Each presentation section was separated by a rest period. In the experimental task subjects were shown digital displays (tachistoscopic analogue) and required to identify them. In the first presentation VF stimuli occurred unilaterally with equal frequency in both VF; in the second presentation VF stimuli occurred in only one VF--either right or left; in the third presentation VF stimuli occurred in only that VF not examined in the previous presentation (order of the second and third unilateral only presentation modes was counterbalanced across subjects in each group); and, the fourth presentation consisted of a repeat of the bilateral mode given in the first presentation for score comparison and additionally to serve as a check for eye strain and fatigue effects.

Each of the four presentations consisted of 620 single-digit numbers.¹¹ In ten minutes a total of 580 numbers were presented CF,

¹¹Previous research and experimentation (Shilder, 1944; Stanley, 1976; Vellutino, 1972; Yeni-Komshian, Isenberg & Goldberg, 1975) had demonstrated that dyslexics experience no significant disadvantage in the identification of numeric symbols in contrast to their consistent inferior performance in the identification of letter symbols (Kershner, 1975; Shilder, 1944; Stanley, 1975; Stanley & Hall, 1973)--when compared with normal readers.

flashed for 15 msec in rapid succession;¹² of these 580 numbers, 540 were nonzero numbers 1 through 9, and 40 were 0 accompanied by a single-digit number which was simultaneously flashed for 15 msec in either the right or left VF.¹³ The 40 VF stimuli consisted of five single-digit numerals--3, 5, 6, 8, and 9--selected as more perceptually difficult symbols in the identification process.¹⁴ Interstimulus timing of 1 sec took place when only CF stimuli were presented and this timing was extended to 1.5 sec when simultaneous CF/VF stimuli occurred.

The following counterbalancing statements were employed to support stimuli arrangement in this experimental design:

1. Four simultaneous CF/VF presentation intervals (SPI)--each 3rd, 10th, 18th and 27th trial--were utilized, and each of these intervals occurred once in every minute;

¹²The rapid presentation of an extended series of single digits (1,800 at 1-sec intervals and 3,600 at 1/2-sec intervals) was designed by Corcoran (1963) for a vigilance task in which sleep-deprived subjects were required to verbally identify "signal" sequences (20/40 random sets of 3 odd digits).

¹³Although tachistoscopic research investigating VF digit recall (Hines & Satz, 1971; Hines, Satz, Schell & Schmidlin, 1969), identification of number sequences (Bryden, Dick & Mewhoet, 1968; Hatta & Dimond, 1980), and digit-identification reaction-time (Geffen, Bradshaw & Wallace, 1971; Gordon & Carmon, 1976) have all found consistent RVF superiority, unilateral single-digit VF identification studies (Drozdenko, 1979; Kimura, 1966; White, 1969), which involve essentially no memory component or reaction-time constraints, have failed to demonstrate such preferential lateralization. Any significant asymmetrical response patterns that occur in this experiment could therefore be attributed to other factors--possibly to differential hemisphere attention or vigilance performance.

¹⁴Observation and analysis of pilot test results demonstrated that certain VF numerals--particularly 1, 2, 4, and 7--were more easily identified by subjects; this phenomenon is conceivably related to that occurring with certain alphabet letters, as documented by various investigators (Harcum, 1964; Miller & Butler, 1980; Moscovitch & Catlin, 1970).

2. No SPI followed itself;
3. Each SPI preceded and followed each of the other SPI with generally uniform frequency;
4. VF numerals used--3, 5, 6, 8, and 9--occurred equally often and, in the bilateral presentation mode, occurred equally often in the right and left VF;
5. In every block of nine CF stimuli (excluding 0), each of the numerals--1 through 9--was used;
6. The CF numeral never repeated itself at the seams of the final numeral of the preceding set and the initial numeral of the following set;
7. The CF numeral preceding or following the simultaneous CF/VF stimuli was never the same value as the VF stimulus;
8. In the two bilateral presentation modes, five left and five right VF stimuli occurred at each of the four SPI.

Visual angles delimiting the VF stimulus were calculated by determining the distance from subject's naision to the CF display component and the distance from the midpoint of the CF digit to the inner- and outer-most edges of the italicized VF digit: visual angles subtended, gradually widened during piloting to increase task difficulty, (see Heron, 1957; Ruediger, 1905; Woodrow, 1938), were established at $4^{\circ}58'$ for the inner edge and $5^{\circ}01'$ for the outer edge of the VF digit.

The LED display intensity control setting on the interface unit was maintained at 3/16. Utilizing this display control setting, digital

luminance (with filter intact) measured approximately 10.8 microcandles,¹⁵ consistent across all subjects, for the duration of the experimental task. This brightness level, derived through piloting, was found to produce the least degree of eyestrain while permitting optimum viewing perception. To prevent an autokinetic movement phenomenon, a constant fixation point (pin-point of light) was established directly below the midpoint of the CF numeric display component. In addition, to soften contrast between the darkened room and the stimuli flash, as well as assist subject's visual orientation within the room relative to the LED display unit, an ambient light source (60-watt red light-bulb shielded by a white glass dome and further screened by a black panel) was placed behind the LED display unit.

¹⁵ Display segment luminous intensity was measured using a Gamma Scientific Photometer (Model 2020-1 Serial #1155; PM Tube type 100 Serial #73756). This measurement was performed with the filter removed and the display operating at an intensity control setting of 3/16. Output data were recorded with a microcandela probe mounted vertically above the segment tested (three segments from each digit were examined). The photometer and probe had been calibrated against a Gamma Scientific Standard Lamp (Serial #626). Luminous intensity data indicated good uniformity (5%) between digital segments with an absolute value of 40 microcandelas (a function of the stability of the standard light source used, probably accurate within 20%). To ascertain the filter transmission of LED spectral emission loss a further test was performed using a Gamma Scientific Photometer (Model DR-14) with a photopically corrected PMT detector (Model D-46A) and a Photomicroscope (Model 700-10-37) equipped with a .006 inch diameter probe which was focused on one segment of the center digit. The display intensity control setting was again fixed at 3/16. With the filter removed the sensitivity of the detector amplifier was adjusted to read 100. The filter was then placed on the display unit reducing the output recorded to a reading of 27 for an average LED emission filter transmission of 27%. Thus luminous intensity with the filter intact could be computed at approximately 10.8 microcandelas. In addition, this gross transmission of the filter to the emission of the display closely matched the filter characteristic in the spectral region of interest.

A white noise generator (IVIE Electronic Model IE-20B)¹⁶ was used to transmit an undistorted continuous level of white noise through a stereo headset (Realistic Model LV/10) thus serving to mask extraneous room noises and maintain subject's attention during performance of the experimental task. Attenuator setting of this white noise generator was fixed at -20 dB emitting a level 62.5 mv RMS.

Procedure

Subjects, tested individually, were initially briefed with a uniform description of the task to be performed and were familiarized with the apparatus involved.

"We are going to perform the experiment four times. Each segment runs 10 minutes and there will be 20-minute breaks between the first and second, second and third, and third and fourth segments.

The experiment will be performed with a very low level of light. In order to accustom your eyes to the semi-darkness, prior to each segment of the experiment I will turn off the overhead lights and we will sit and talk for a while until your eyes have made an adequate adjustment to the semi-darkness.

For this experiment it is necessary that you place your head on the chin rest with your forehead resting against the bar; once the experiment begins, maintain this same position. The chin/head-rest stand should not move at any time during the experiment. If this should happen, please tell me immediately.

When you respond to what you see on the screen, your responses will be recorded through this microphone.

During the experiment you will also wear these earphones. Just before the experiment begins, white noise will be

¹⁶This white noise generator contained a calibrated output level with a cumulative attenuator error not more than ± 0.5 dB for loads equal to or greater than 600 OHMS.

turned on and you will hear this sound through the earphones for the duration of the experiment.

In the experiment a series of single-digit numbers--0 through 9--will appear at the center of the screen. These numbers will flash on the screen very rapidly, one after the other. You should focus your attention on these numbers and say them out loud as you see them. Should you miss one or fall behind, do not stop. Continue to report the numbers as you actually see them. Whenever the number 0 appears at the center of the screen it will be accompanied by another number. Please first say 0 and then the other number (for example 0-1, 0-2, 0-3, and so on). If you are ever unsure of the other number, try and guess what number it might be. Just below where the numbers at the center of the screen will appear there is a pin-point of light which will remain on for the duration of the experiment. This pin-point of light acts as a guide. Once the experiment begins you should focus your eyes at the center of the screen just above the pin-point of light. The center numbers will flash on just above it."

The subject was then seated and correctly positioned with his head placed in the chin/head rest (part of a standard ophthalmological slit-lamp apparatus mounted on a specially designed adjustable wood stand). This chin/head rest served to hold the subject's head centrally positioned at eyelevel with the stimulus source and also to maintain a constant distance (84 cm) from the subject's naison to the LED display unit. With measurement from the subject's naison to the visual display unit checked, he tried on the earphones, adjusted them to his head size, and was introduced to white noise sound. After acclimating subject to white noise, the earphones were removed, the overhead lights extinguished and, during the subsequent dark-adaption period (approximately 7 minutes), he was placed at ease. Following dark adaption the subject was instructed to put on the earphones (the white noise was activated), place his head in the chin/head rest (the computer loaded the program for the particular presentation), focus his eyes just above the fixation point of light at the center of the screen and report the numbers as he actually saw them

(both the tape recorder and VOX were operational). Subsequent to a brief sample trial of the experimental task intended to insure understanding, each subject underwent a sequence of four presentations, each presentation separated by a 20-minute rest period as well as a 7-minute dark-adaption period. Upon completion of each presentation the subject freed himself from the chin/head rest, removed the earphones and left the test room to relax during each of the interim periods. During these interim periods CF/VF response latencies (signaled by the VOX with timings subsequently registered and stored in the computer) were transcribed.¹⁷

Scoring

Tapes of the experiment--stereophonically recorded stimuli-onset audio signals and subject's verbal responses--were utilized to compute individual error and lateness scores. Following testing of all subjects in the experiment, these tapes were played to insure accurate scoring of correct and incorrect identifications. The same criteria served for the scoring of CF and VF stimuli. Incorrect identifications included four distinct types: 1) misnaming--substitution of an incorrect number for a correct number; 2) omissions--elision of a numeric stimulus from the proper sequence; 3) intrusions--insertion of extra number and/or

¹⁷ Although the VOX unit remained operational throughout testing, certain deficiencies were revealed in its performance which rendered the derived results invalid. Intended to signal initiation of subject's verbal responses to CF and VF numerals on simultaneous CF/VF stimuli presentations, the VOX proved erratic in performance and unreliable in its closure-onset signal to subject's verbal response. For these reasons a decision was made to eliminate from consideration and analysis data drawn from this source.

reversal of numeric sequence; and 4) false-starts--partially incorrect or incorrect identification corrected. To measure correct-but-late responses to both CF and VF stimuli, the tapes were replayed--at a slower than normal speed--to separate and accurately ascertain timing between the stimulus/or/stimuli-onset audio signal and initiation of the subject's verbal response. The criterion in effect for a correct-but-late response involved the failure of the subject to start a correct verbal response either prior to or simultaneously with the next audio signal of stimulus/or/stimuli-onset. When the subject's response was both incorrect and late, only a single scoring of this response was entered; it was categorized as incorrect.

IV. Results

A tripartite analysis of data gathered from the two research areas of this project was undertaken. In the initial section the Neuropsychological Test Battery (NTB) was investigated to determine the existence of contrasting test profiles between the dyslexic and normal reader groups. The second section involved analysis of data derived from the experimental task to ascertain those variables influencing performance as well as to distinguish those variables differentiating the two reader groups. In the final section relationships of NTB scores and relevant performances on the experimental task for each reader group were examined to delineate correspondences within and between the two investigative areas.

Neuropsychological Test Battery

Tests comprising the NTB were grouped and analyzed under the following subcategories--reading, verbal (excluding reading), visual, sensory/motor and arithmetic. Differences distinguishing the dyslexic group from the normal reader group were then examined for each of these subcategories; a multivariate test (Hotelling T^2) was used to determine whether the profile for any set of mean scores for the dyslexic group differed from that of the normal reader group. When profiles of mean scores of a set for the two reader groups were found to be significantly different (probability of type 1 error $< .05$), the multivariate critical t value (square root of critical T^2) was computed and used as the critical value for the univariate t tests to establish which neuropsychological test or tests in that subcategory had contributed to this

significant finding.

Reading Tests:

This subcategory contained scores from three reading tests: Woodcock Mastery Reading Test,¹⁸ Gray Oral Reading Test and the reading section of the WRAT. The multivariate F statistic for this set of scores, corresponding to T^2 of 138.1 {i.e., $F = T^2 [N-P/P(N-1)]$ where N = total number of subjects and P = number of dependent variables in set}, was $F(3, 16) = 40.9$ which is significant at $p < .001$. Comparison of the critical t value with the univariate t -test results for the three reading examinations revealed large significant group differences between dyslexics and normal readers on all tests (see Table 4). With the Woodcock and Gray Oral relatively large group differences also occurred between standard deviations which is attributable to the normal reader group's near maximum performance on these two tests. Dyslexic scores on all the reading tests were well below the maximum and no overlapping of score distribution between the two reader groups took place, further demonstrating the severity of this particular dyslexic group's reading impairment.

Verbal Tests (Excluding Reading):

This subcategory contained scores from four verbal tests:

¹⁸ A separate nonparametric statistical procedure (Mann-Whitney U test) was used to determine significance of mean score differences between reader groups on the Woodcock subtests. All subtests were significant at $p < .001$ except Word Comprehension which was significant at $p < .01$. These data support the thesis that reading comprehension was relatively less impaired in the dyslexic group than word decoding or identification reading skills (mean scores of these subtests for both reader groups are reported in Table 2 of the Methodology section).

Table 4
STATISTICAL SUMMARY OF UNIVARIATE t TESTS OF READING SCORES
FOR THE TWO READER GROUPS

Reading Test	Reader Group	Mean	s.d.	<u>t</u>	<u>p</u>
WOODCOCK ^a	Dyslexic	6.3	2.9	7.1	.001
	Normal	12.8	0.2		
GRAY ORAL ^b	Dyslexic	4.5	2.3	10.4	.001
	Normal	12.0	0.0		
WRAT READING	Dyslexic	6.4	2.0	9.2	.001
	Normal	14.8	2.1		

^aMaximum grade level score for this test = 12.9.

^bMaximum grade level score for this test = 12.0.

WAIS VIQ,¹⁹ spelling section of the WRAT, Boston Naming Test and Spreen-Benton Sentence Repetition Test. The multivariate F statistic of group differences for this set of scores, corresponding to T^2 of 51.2, was $F(4, 15) = 10.7$ which is significant at $p < .001$. Comparison of the critical t value with the univariate t -test results for the four verbal examinations revealed significant group differences between dyslexics and normal readers on three of the four tests (see Table 5). Univariate test results therefore demonstrated that dyslexic subjects were also deficient in language skills other than reading: spelling, naming of objects (drawings) and some additional verbal skills--Digit Span, Arithmetic, Information and Vocabulary--examined in the Verbal section of the WAIS.

Visual Tests:

This subcategory contained scores from five visual tests: WAIS PIQ,²⁰ Raven Standard Progressive Matrices, Knox Cube Test and Rey-Osterrieth Complex Figure Test (copying and memory section). The multivariate F statistic of group differences for this set of scores was $F(5, 14) = 2.2$ ($T^2 = 13.9$) which is not significant ($p = .12$).

¹⁹ A separate nonparametric statistical procedure (Mann-Whitney U test) was used to determine which mean scores on the WAIS Verbal subtests differed significantly between the two reader groups. Significant group differences at $p < .01$ were revealed on four WAIS Verbal subtests but no significant group differences occurred on the Comprehension and Similarities subtests (mean scores of these subtests for both reader groups are reported in Table 1 of the Methodology section).

²⁰ A separate nonparametric statistical procedure (Mann-Whitney U test) was used to determine whether mean scores on the WAIS Performance subtests differed significantly between the two reader groups. A highly significant group difference at $p < .001$ for Digit Symbol occurred in the absence of significant group differences on any of the other WAIS Performance subtests (mean scores of these subtests for both reader groups are reported in Table 1 of the Methodology section).

Table 5

STATISTICAL SUMMARY OF UNIVARIATE t TESTS OF
VERBAL (EXCLUDING READING) SCORES FOR THE TWO READER GROUPS

Verbal Test	Reader Group	Mean	s.d.	t	p
WAIS VIQ	Dyslexic	101.0	7.7	4.47	.05
	Normal	120.8	11.7		
WRAT SPELLING ^a	Dyslexic	5.4	1.8	7.04	.001
	Normal	11.7	2.2		
BOSTON NAMING	Dyslexic	71.0	6.0	3.93	.05
	Normal	80.0	4.1		
SENTENCE REPETITION	Dyslexic	13.4	2.0	3.07	N.S.
	Normal	16.0	1.8		

^aA grade level mean score is reported for this test.

Sensory/Motor Tests:

This subcategory contained scores from one sensory and four motor tests: Double Simultaneous Stimulation (DSS) and the four conditions of the Purdue Pegboard. The multivariate F statistic of group differences for this set of scores was $F(5, 14) = 1.3$ ($T^2 = 8.3$) which is not significant ($p = .32$). Exceptionally large standard deviations on all these tests for both reader groups undoubtedly contributed to this lack of a significant difference. In addition, although all subjects in this study had been verified right-handed using standard handedness laterality measures, mean percentage correct scores for both groups (see Table 3 of Methodology section) were nonsignificantly higher for the left hand than the right hand on the Purdue Pegboard Test.

Arithmetic Test:

One test of arithmetic ability was subjected to separate analysis: the arithmetic section of the WRAT.²¹ With only one variable in this subcategory the multivariate F test is the same as T^2 and the standard univariate F test (or squared t test). The univariate F statistic of group differences for this score was $F(1, 18) = 7.5$ which is significant at $p < .02$.

Experimental Task

VF and CF performances were each scored based on number of incorrect identifications and correct-but-late responses. A series of

²¹The WAIS arithmetic subtest score was included as part of the WAIS VIQ score to facilitate reference to this aggregate result as an indicator of verbal ability.

split-plot ANOVAs²² were conducted on this data and, whenever additional analyses were indicated, post hoc tests were performed.

Analysis I:

A split-plot ANOVA was conducted for bilateral conditions in which incorrect VF (IVF) scores were examined as a function of Group (dyslexic, normal reader), Time (bilateral 1, bilateral 2), Rate (3, 10, 18, 27) and Field (left, right). Only two of the fifteen effects were found to be significant (see Table 6): one main effect--Rate, $F(3, 54) = 3.51$, $p < .03$, and one three-factor interaction--Group X Time X Field, $F(1, 18) = 8.33$, $p < .01$. The Rate effect could not be explained with pair wise comparisons using the Tukey HSD test. The three-way interaction illustrated in Figure 1 demonstrates, across the two bilateral conditions of the experimental task, an absence of consistent LVF or RVF performance in dyslexic and normal reader groups; slopes of the lines plotted in Figure 1 also indicate that, in each reader group, the differences between LVF and RVF incorrect identifications reversed from the first to the second bilateral presentation of this task. The simple Time X Field interaction was significant for dyslexics, $F(1, 9) = 6.44$, $p < .04$, but nonsignificant for normal readers (see Table 7). Thus over time (with two intervening unilateral presentations) the difference between the number of LVF and RVF incorrect identifications was significant for dyslexics but not for normal readers. Each of these simple interactions was examined further in terms of the simple, simple effects of Field (within Group and Time).

²²"In the AV-experimental design literature, these designs [subjects within groups by conditions] are variously called 'split-plot,' 'randomized blocks factorial,' and . . . 'subjects nested within groups repeatedly measured on a factor'" (Cohen & Cohen, 1975, pp. 412-413).

Table 6

SPLIT-PLOT ANOVA OF INCORRECT VF IDENTIFICATIONS ON THE EXPERIMENTAL TASK AS A FUNCTION
OF GROUP (DYSLEXIC, NORMAL READER), TIME (BILATERAL 1, BILATERAL 2),
RATE (3, 10, 18, 27), AND FIELD (LEFT, RIGHT)

Source	Sum of Squares	Degrees of Freedom	Mean Square	<u>F</u>	Tail Prob.
MEAN	432.45	1	432.45	49.98	0.000
GROUP	4.05	1	4.05	0.47	0.503
ERROR	155.75	18	8.65		
TIME	1.25	1	1.25	3.13	0.094
TG	0.05	1	0.05	0.12	0.728
ERROR	7.20	18	0.40		
RATE	9.18	3	3.06	3.51	0.021*
RG	2.58	3	0.86	0.99	0.406
ERROR	47.00	54	0.87		
TR	0.63	3	0.21	0.37	0.775
TRG	1.43	3	0.48	0.84	0.477
ERROR	30.45	54	0.56		
FIELD	0.80	1	0.80	0.68	0.421
FG	1.25	1	1.25	1.06	0.317
ERROR	21.20	18	1.18		
TF	0.20	1	0.20	0.41	0.529
TFG	4.05	1	4.05	8.33	0.010**
ERROR	8.75	18	0.49		
RF	4.68	3	1.56	2.44	0.074
RFG	0.63	3	0.21	0.33	0.806
ERROR	34.45	54	0.64		
TRF	3.33	3	1.11	1.68	0.182
TRFG	1.08	3	0.36	0.54	0.655
ERROR	35.60	54	0.66		

* $p < .05$

** $p < .01$

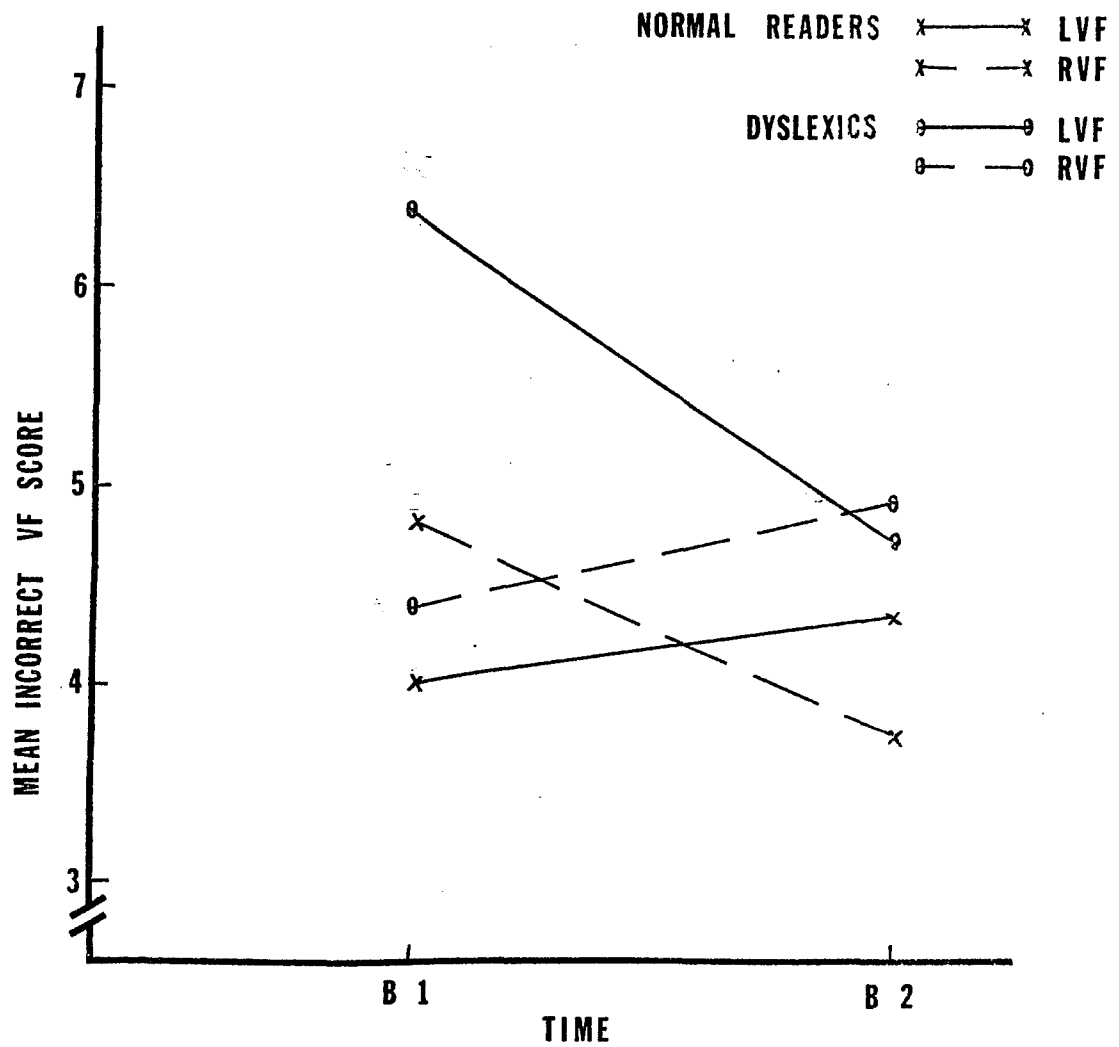


Figure 1. Group X Time X Field interaction on bilateral mode condition with mean IVF scores.

Table 7
RESULTS OF THE SIMPLE TIME X FIELD INTERACTION
IN THE DYSLIXIC GROUP

Source	Sum of Squares	Degrees of Freedom	Mean Square	<u>F</u>	Tail Prob.
MEAN	260.10	1	260.10	31.57	0.000
ERROR	74.15	9	8.24		
TIME	0.90	1	0.90	3.12	0.111
ERROR	2.60	9	0.29		
RATE	7.25	3	2.42	2.90	0.053
ERROR	22.50	27	0.83		
TR	1.75	3	0.58	0.94	0.435
ERROR	16.75	27	0.62		
FIELD	2.03	1	2.03	1.22	0.299
ERROR	14.98	9	1.66		
TF	3.03	1	3.03	6.44	0.032*
ERROR	4.23	9	0.47		
RF	2.23	3	0.74	1.01	0.402
ERROR	19.78	27	0.73		
TRF	3.93	3	1.31	1.48	0.242
ERROR	23.83	27	0.88		

* $p < .05$

Only the Field effect within the first bilateral presentation in the dyslexic group was significant, $F(1, 69) = 4.68$, $p < .05$; dyslexics made significantly more LVF than RVF incorrect identifications on this bilateral presentation.²³ Although Figure 1 indicates that dyslexic LVF performance on the first bilateral presentation was markedly inferior to that of normal readers, all simple, simple Group effects (within Field and Time) were nonsignificant.²⁴

Analysis II:

An analogous split-plot ANOVA was conducted for bilateral conditions in which aggregate VF deficit (AVF) scores (IVF + correct-but-late VF [CBLVF]) were examined as a function of Group, Time and Field.²⁵ The three-way Group X Time X Field interactions (note Figure 2) remained significant in this analysis, $F(1, 18) = 6.23$, $p < .03$ (see Table 8); however, all post hoc test results on the simple Time X Field interactions and the simple, simple Field effects within each reader group as well as the simple, simple Group effects were nonsignificant. Addition of the CBLVF scores to the IVF scores resulted in the elimination of significance for the two post hoc tests found in the dyslexic group in the previous analysis using only IVF scores. An analogous split-plot ANOVA of Group X Time X Field for bilateral conditions, computed with only CBLVF scores revealed that significantly

²³ Among the 10 dyslexic subjects 7 performed better in the RVF, 2 performed equally well in each VF and only 1 performed better in the LVF.

²⁴ The within group variability apparently was too great to overcome the small number of observations that went into estimating the Group effect and its error term.

²⁵ The Rate factor was excluded from this analysis since, on bilateral conditions, the number of CBLVF scores for either group was too small to be considered reliable within interval rates.

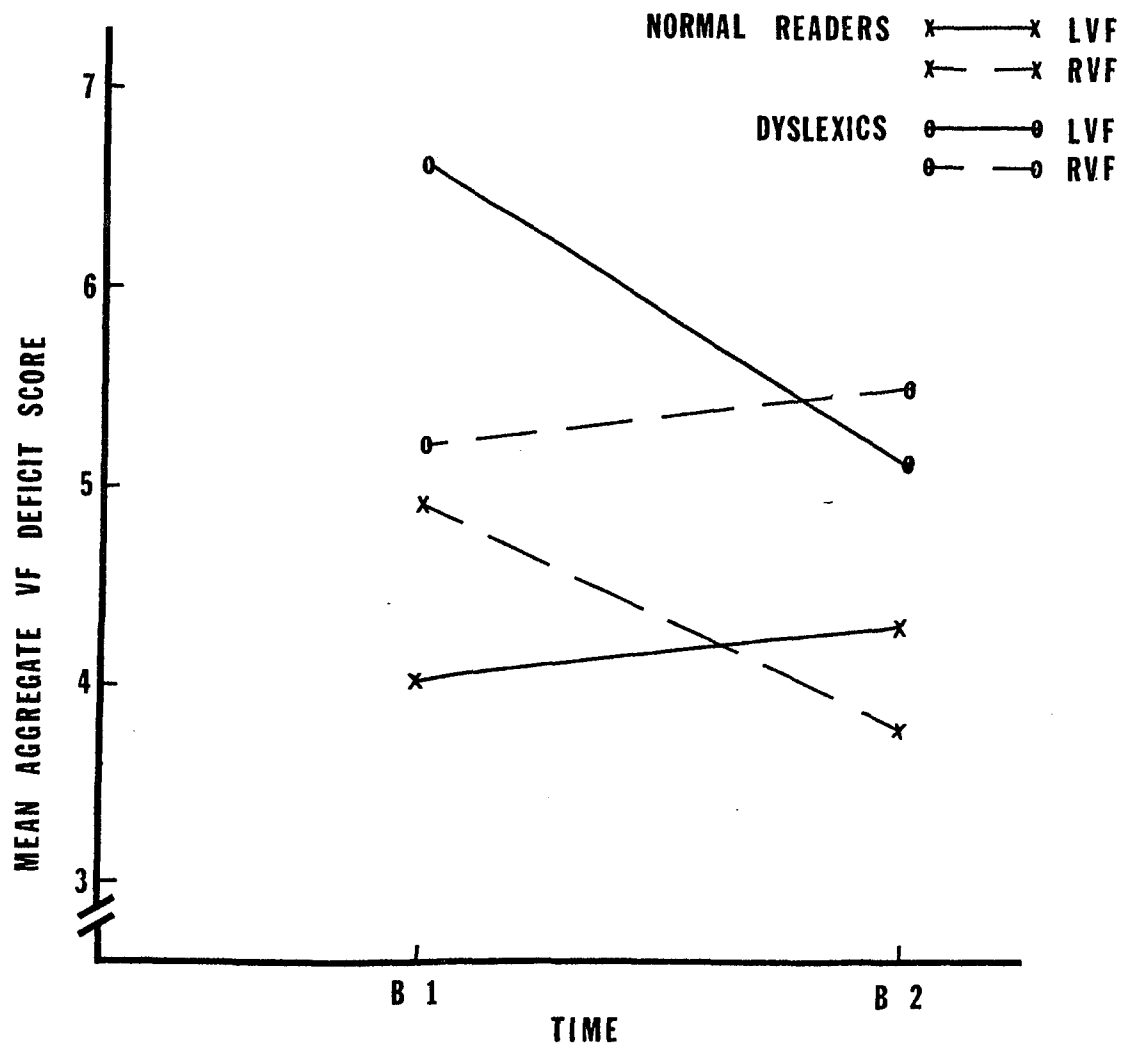


Figure 2. Group X Time X Field interaction on bilateral mode condition with mean AVF scores.

Table 8

SPLIT-PLOT ANOVA OF AGGREGATE VF DEFICIT SCORES ON THE EXPERIMENTAL TASK
 AS A FUNCTION OF GROUP (DYSLEXIC, NORMAL READER), TIME
 (BILATERAL 1, BILATERAL 2), AND FIELD (LEFT, RIGHT)

Source	Sum of Squares	Degrees of Freedom	Mean Square	<u>F</u>	Tail Prob.
MEAN	1940.45	1	1940.45	53.98	0.000
GROUP	36.45	1	36.45	1.01	0.327
ERROR	647.10	18	35.95		
TIME	5.00	1	5.00	2.74	0.115
TG	0.20	1	0.20	0.11	0.744
ERROR	32.80	18	1.82		
FIELD	0.45	1	0.45	0.10	0.759
FG	2.45	1	2.45	0.53	0.476
ERROR	83.10	18	4.62		
TF	0.20	1	0.20	0.10	0.759
TFG	12.80	1	12.80	6.23	0.023*
ERROR	37.00	18	2.06		

*p < .05

more CBLVF responses were made to RVF stimuli, $F(1, 18) = 6.82$, $p < .02$, and that dyslexics made significantly more of these late responses than normal readers, $F(1, 18) = 4.89$, $p < .05$ (see Table 9). The relative differences between LVF and RVF scores for dyslexics on the two independently scored VF deficit measures (IVF and CBLVF) counterbalanced and thus resulted in a lack of significance on the post hoc tests in this AVF analysis.

Analysis III:

A split-plot ANOVA was conducted for unilateral conditions in which IVF scores were examined as a function of Group (dyslexic, normal reader), Field (left, right) and Rate (3, 10, 18, 27). Two of the seven effects were found to be significant (see Table 10): one main effect--Rate, $F(3, 54) = 2.87$, $p < .05$, and one three-factor interaction--Group X Field X Rate, $F(3, 54) = 3.80$, $p < .02$. The Rate effect could not be explained with pair wise comparisons using the Tukey HSD test. The three-way interaction illustrated in Figure 3 indicates high performance variability among the four rate intervals in normal readers, as well as marked differential VF performance among the rate intervals in this group. The simple Field X Rate interaction did reveal a significant Rate effect, $F(3, 27) = 3.06$, $p < .05$, and a significant Field X Rate effect, $F(3, 27) = 3.60$, $p < .03$, for the normal reader group, but not for dyslexics (see Table 11). Examination of the simple, simple Field effects within Group and Rate revealed only one significant Field effect, at the level of Rate 3 in the normal reader group, $F(1, 9) = 6.33$, $p < .04$; normal readers made significantly more LVF than RVF incorrect identifications with Rate 3--the shortest rate interval. Examination of the simple, simple Group

Table 9

SPLIT-PLOT ANOVA OF CORRECT-BUT-LATE VF RESPONSES ON THE EXPERIMENTAL TASK
 AS A FUNCTION OF GROUP (DYSLEXIC, NORMAL READER), TIME
 (BILATERAL 1, BILATERAL 2), AND FIELD (LEFT, RIGHT)

Source	Sum of Squares	Degrees of Freedom	Mean Square	<u>F</u>	Tail Prob.
MEAN	6.05	1	6.05	7.31	0.015
GROUP	4.05	1	4.05	4.89	0.040*
ERROR	14.90	18	0.83		
TIME	0.0	1	0.0	0.0	1.000
TG	0.0	1	0.0	0.0	1.000
ERROR	6.00	18	0.33		
FIELD	1.25	1	1.25	6.82	0.018*
FG	0.45	1	0.45	2.45	0.135
ERROR	3.30	18	0.18		
TF	0.20	1	0.20	0.64	0.433
TFG	0.20	1	0.20	0.64	0.433
ERROR	5.60	18	0.31		

* $p < .05$

Table 10

SPLIT-PLOT ANOVA OF INCORRECT VF IDENTIFICATIONS ON THE EXPERIMENTAL TASK
AS A FUNCTION OF GROUP (DYSLEXIC, NORMAL READER), FIELD (LEFT,
RIGHT), AND RATE (3, 10, 18, 27)

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Prob.
MEAN	926.41	1	926.41	56.42	0.000
GROUP	12.66	1	12.66	0.77	0.392
ERROR	295.56	18	16.42		
RATE	14.07	3	4.69	2.87	0.045*
RG	1.52	3	0.51	0.31	0.818
ERROR	88.29	54	1.64		
FIELD	0.31	1	0.31	0.09	0.767
FG	1.41	1	1.41	0.42	0.527
ERROR	60.91	18	3.38		
RF	3.97	3	1.32	1.11	0.353
RFG	13.57	3	4.52	3.80	0.015*
ERROR	64.34	54	1.19		

* $p < .05$

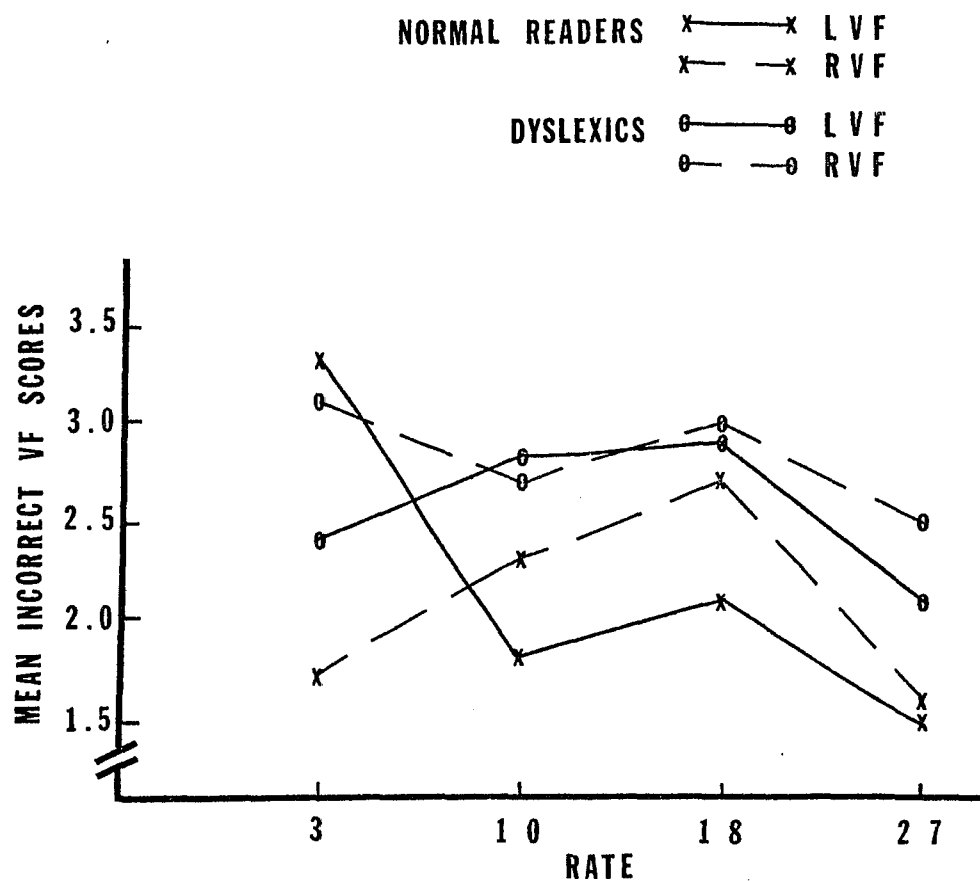


Figure 3. Group X Field X Rate interaction on unilateral mode condition with mean IVF scores.

Table 11

RESULTS OF THE SIMPLE RATE X FIELD INTERACTION
IN THE NORMAL READER GROUP

Source	Sum of Squares	Degrees of Freedom	Mean Square	<u>F</u>	Tail Prob.
MEAN	361.25	1	361.25	22.08	0.001
ERROR	147.25	9	16.36		
RATE	11.05	3	3.68	3.06	0.045*
ERROR	32.45	27	1.20		
FIELD	0.20	1	0.20	0.05	0.820
ERROR	32.80	9	3.64		
RF	15.70	3	5.23	3.60	0.026*
ERROR	39.30	27	1.46		

* $p < .05$

effects within Field and Rate, however, revealed no significant differences between dyslexics and normal readers.

Analysis IV:

An analogous split-plot ANOVA was conducted for unilateral conditions in which AVF scores were examined as a function of Group and Field.²⁶ All effects in this analysis were nonsignificant. An analogous split-plot ANOVA of Group X Field for unilateral conditions, conducted with only CBLVF scores, also revealed nonsignificant results. In these analyses VF performances on the unilateral conditions were comparable in the two reader groups.

Analysis V:

A series of three split-plot ANOVAs were conducted for bilateral conditions in which incorrect CF (ICF), correct-but-late CF (CBLCF) and aggregate CF deficit (ACF [ICF + CBLCF]) scores were examined separately as a function of Group and Time. The Group effect was significant in two of the three analyses performed (see Table 12). Dyslexics made significantly more ICF identifications than normal readers, $F(1, 18) = 6.28$, $p < .03$, and dyslexic ACF performance was inferior to that of normal readers, $F(1, 18) = 4.44$, $p < .05$. Thus on bilateral conditions dyslexics experienced significantly greater difficulty in correctly identifying rapidly presented CF stimuli.

Analysis VI:

An analogous series of three split-plot ANOVAs were conducted for unilateral conditions in which ICF, CBLCF and ACF scores were examined

²⁶The Rate factor was excluded from this analysis since, on unilateral conditions, the number of CBLVF scores for either reader group was too small to be considered reliable within interval rates.

Table 12

SPLIT-PLOT ANOVAS OF INCORRECT CF IDENTIFICATIONS, CORRECT-BUT-LATE CF RESPONSES
AND AGGREGATE CF DEFICIT SCORES ON THE EXPERIMENTAL TASK AS A FUNCTION OF
GROUP (DYSLEXIC, NORMAL READER) AND TIME (BILATERAL 1, BILATERAL 2)

Source	Sum of Squares	Degrees of Freedom	Mean Square	<u>F</u>	Tail Prob.
MEAN	9610.00	1	9610.00	11.31	0.004
GROUP	5336.10	1	5336.10	6.28	0.022*
ERROR	15297.90	18	849.88		
TIME	36.10	1	36.10	1.15	0.298
TG	6.40	1	6.40	0.20	0.657
ERROR	565.50	18	31.42		
MEAN	29866.23	1	29866.23	4.22	0.055
GROUP	26780.63	1	26780.63	3.79	0.068
ERROR	127319.65	18	7073.31		
TIME	342.23	1	342.23	1.83	0.193
TG	265.23	1	265.23	1.42	0.249
ERROR	3365.05	18	186.95		
MEAN	73359.23	1	73359.23	5.82	0.027
GROUP	56025.23	1	56025.23	4.44	0.049*
ERROR	226877.05	18	12604.28		
TIME	156.03	1	156.03	1.03	0.323
TG	189.23	1	189.23	1.25	0.278
ERROR	2716.25	18	150.90		

* $p < .05$

separately as a function of Group and Field. In each analysis the Group effect was significant (see Table 13). Dyslexics made significantly more ICF identifications, $F(1, 18) = 6.39$, $p < .03$, and CBLCF responses, $F(1, 18) = 4.64$, $p < .05$, than normal readers with the result that dyslexic ACF performance was significantly inferior to that of the normal reader group, $F(1, 18) = 5.35$, $p < .04$. Thus on unilateral conditions dyslexics experienced significantly greater difficulty in correctly identifying and responding to rapidly presented CF stimuli.

Correlation Analysis of NTB and Relevant Experimental Task Measures

For each reader group degree of correlation within and between measures of the two investigative areas--NTB and relevant experimental task performances--were examined in terms of product-moment correlations. The variables entered into the correlation matrix included the 18 NTB scores and 3 CF experimental task scores.²⁷ These CF measures were the only ones in the experimental task which, across the four presentations, yielded strong and consistent differences between the dyslexic and normal reader groups. Independent analyses were conducted for the dyslexic group and the normal reader group. Correlation results for the NTB were analyzed in terms of the test subcategories examined previously. In each of these subcategory sections correlations among scores within a set, as well as significant correlations ($p < .05$) between these scores and those NTB scores outside the set, are noted

²⁷ CF measures for each reader group included the mean ICF score, the mean CBLCF score and the ACF score (ICF + CBLCF) for the four presentations of the experimental task.

Table 13

SPLIT-PLOT ANOVAS OF INCORRECT CF IDENTIFICATIONS, CORRECT-BUT-LATE CF RESPONSES
AND AGGREGATE CF DEFICIT SCORES ON THE EXPERIMENTAL TASK AS A FUNCTION .
OF GROUP (DYSLEXIC, NORMAL READER), AND FIELD (LEFT, RIGHT)

Source	Sum of Squares	Degrees of Freedom	Mean Square	F	Tail Prob.
MEAN	10989.23	1	10989.23	10.85	0.004
GROUP	6477.03	1	6477.03	6.39	0.021*
ERROR	18239.25	18	1013.29		
FIELD	50.63	1	50.63	0.25	0.621
FG	55.23	1	55.23	0.28	0.606
ERROR	3607.65	18	200.43		
MEAN	27457.60	1	27457.60	5.35	0.033
GROUP	23814.40	1	23814.40	4.64	0.045*
ERROR	92321.00	18	5128.94		
FIELD	1.60	1	1.60	0.01	0.933
FG	25.60	1	25.60	0.12	0.738
ERROR	3991.80	18	221.77		
MEAN	73188.03	1	73188.03	7.10	0.016
GROUP	55130.63	1	55130.63	5.35	0.033*
ERROR	185485.85	18	10304.77		
FIELD	70.23	1	70.23	0.11	0.741
FG	156.03	1	156.03	0.25	0.622
ERROR	11176.25	18	620.90		

* $p < .05$

and reviewed. Correlations among CF scores and significant correlations ($p < .05$) of CF scores with NTB scores are noted and reviewed in a separate subcategory section.

Reading Tests:

Correlations among reading scores and significant correlations between reading scores and other NTB scores are reported in Table 14 with separate matrices for each reader group. Both dyslexic and normal reader group performances demonstrated significant correlations among all reading scores with non-zero standard deviations; the degree of significance of these correlations, however, was greater in the dyslexic group. Significant correlations between reading scores and other NTB scores were present for verbal tests in both reader groups and for the arithmetic test in the dyslexic group. Dyslexics displayed a consistently strong relationship between reading and spelling performances and a relatively strong relationship between reading and arithmetic performances; both the spelling and arithmetic tests involve cognitive tasks which, like reading, require a decoding of symbolic stimuli (alphabetical or numerical). Normal readers, in contrast, demonstrated a consistent relationship between reading and VIQ performances, as well as a relatively strong relationship between the reading section of the WRAT and object naming, both cognitive tasks involving identification skills (words and objects).

Verbal (Excluding Reading) Tests:

Correlations among verbal scores and significant correlations between verbal scores and other NTB scores are reported in Table 15 with separate matrices for each reader group. Only one significant correla-

Table 14

CORRELATIONS AMONG READING TESTS AND SIGNIFICANT CORRELATIONS BETWEEN
READING SCORES AND OTHER NTB SCORES FOR DYSLEXIC AND
NORMAL READER GROUPS

	Dyslexics			Normal Readers		
	Woodcock	Gray Oral	WRAT Reading	Woodcock	Gray Oral ^a	WRAT Reading
WOODCOCK	-	.78**	.95***	-	.00	.67*
GRAY ORAL	-	-	.83**	-	-	.00
WAIS VIQ	.72*	-	-	.62*	-	.63*
WRAT SPELLING	.94***	.88***	.99***	-	-	.66*
BOSTON NAMING	-	-	-	-	-	.80**
WRAT ARITHMETIC	.79**	-	.66*	-	-	-

^aCorrelations could not be computed with the Gray Oral Reading Test as all normal reader subjects scored the maximum grade level (12.0) on this reading test and the standard deviation was zero.

* $p < .05$

** $p < .01$

*** $p < .001$

Table 15

CORRELATIONS AMONG VERBAL TESTS (EXCLUDING READING) AND SIGNIFICANT
CORRELATIONS BETWEEN VERBAL SCORES AND OTHER NTB SCORES
FOR DYSLEXIC AND NORMAL READER GROUPS

	Dyslexics				Normal Readers			
	WAIS VIQ	WRAT Spelling	Boston Naming	Sentence Repetition	WAIS VIQ	WRAT Spelling	Boston Naming	Sentence Repetition
WAIS VIQ	-	.57	.45	.54	-	.79**	.69*	.56
WRAT SPELLING	-	-	.44	.49	-	-	.66*	.55
BOSTON NAMING	-	-	-	.64*	-	-	-	.77**
WOODCOCK	.72*	.94***	-	-	.62*	-	-	-
GRAY ORAL	-	.88***	-	-	-	-	-	-
WRAT READING	-	.99***	-	-	.63*	.66*	.80**	-
WRAT ARITHMETIC	.85**	.68*	-	-	.68*	-	-	-
WAIS PIQ	-	-	-	-	.85**	-	-	-
RAVEN	-	-	-	-	.77**	.79**	-	-
REY-O COPYING	-	-	-	-	.72*	-	-	-
REY-O MEMORY	-	-	-	-	-	-	.71*	-
PURDUE ASSEMBLY	-	-	-	-	.75**	.80**	.66*	-
DSS ^a	-	-	-	-	-	.66*	-	-

^aThe DSS test was scored using number of deficit responses; all other NTB tests were scored using correct responses. Correlation signs for the DSS test have been inverted in this table; therefore a significant correlation implies a positive relationship between the verbal skill involved and proficiency in DSS performance.

* $p < .05$

** $p < .01$

*** $p < .001$

tion occurred among the six verbal scores in the dyslexic group while the normal reader group presented a more integrated verbal ability pattern with four of the six correlations among verbal scores significant. In the dyslexic group correlations of verbal scores with other NTB scores remained basically those reported in the reading section with two additional significant correlations of spelling and VIQ with arithmetic. In contrast, a substantial number of significant correlations occurred in the normal reader group between verbal scores and other NTB scores. Thus for normal readers verbal abilities tested were inter-related and, except for sentence repetition, these verbal abilities also paralleled functional abilities measured by standardized tests in a variety of other intellectual, cognitive, perceptual, sensory and motor areas. Dyslexics, however, demonstrated a relative lack of integration on verbal abilities tested. In addition, only two of the four verbal scores correlated with scores from other NTB subcategories; these relationships were further limited to the reading and arithmetic subcategories. Therefore with dyslexics significant correlations occurring in this subcategory involved essentially those NTB tests in which again a primary requirement was manipulation of symbolic stimuli.

Visual Tests:

Correlations among visual scores and significant correlations between visual scores and other NTB scores are reported in Table 16 with separate matrices for each reader group. Correlation results among the five visual tests for both reader groups indicated that visual abilities tested in this battery were relatively independent of each other. Both reader groups attained an expected significant cor-

Table 16

CORRELATIONS AMONG VISUAL TESTS AND SIGNIFICANT CORRELATIONS BETWEEN VISUAL SCORES
AND OTHER NTB SCORES FOR DYSPLEXIC AND NORMAL READER GROUPS

	Dyslexics					Normal Readers				
	WAIS PIQ	RAVEN	KNOX	REY-O COPYING	REY-O MEMORY	WAIS PIQ	RAVEN	KNOX	REY-O COPYING	REY-O MEMORY
WAIS PIQ	-	.64*	.72*	.20	-.20	-	.66*	.35	.56	.55
RAVEN	-	-	.45	.21	-.13	-	-	.02	.62	.26
KNOX	-	-	-	-.32	-.32	-	-	-	-.15	-.18
REY-O COPYING	-	-	-	-	.25	-	-	-	-	.42
WRAT ARITHMETIC	-	-	-	.81**	-	.85**	-	-	-	-
WAIS VIQ	-	-	-	-	-	.85**	.77**	-	.72*	-
WRAT SPELLING	-	-	-	-	-	-	.79**	-	-	-
BOSTON NAMING	-	-	-	-	-	-	-	-	-	.71*
DSS ^a	-	-	-	-	-	-	.74**	-	-	-

^aThe DSS test was scored using number of deficit responses; all other NTB tests were scored using correct responses. Correlation signs for the DSS test have been inverted in this table; therefore a significant correlation implies a positive relationship between the visual skill involved and proficiency in DSS performance.

* $p < .05$

** $p < .01$

relation between PIQ and Raven scores (tests purported to measure nonverbal "intelligence"); the dyslexic group achieved an additional significant relationship between PIQ and Knox (nonverbal sequencing task) test scores. Significant correlations of visual tests with other NTB tests occurred in only one instance with dyslexics. Normal readers, however, demonstrated a pattern of correlations with other NTB test scores suggesting that visual abilities (excluding those tested by the Knox) related to or paralleled functional abilities in a variety of other areas.

Sensory/Motor Tests:

Correlations among sensory/motor scores and significant correlations between sensory/motor scores and NTB scores are reported in Table 17 with separate matrices for each reader group. Significant correlations for both reader groups occurred only among the four motor conditions of the Purdue. Neither group demonstrated a relationship between right and left hand performance on the same motor task but in each reader group right and left hand performance did correlate with performance on the same motor task using both hands. A curious anomaly, however, occurred in that (although both reader groups were right handed) the degree of this significance in each group was greater for the left hand. Dyslexic scores on Purdue assembly (alternating, successive motor manipulation with both hands) also correlated with their scores on the Purdue "both hands" condition (simultaneous, successive motor manipulation with both hands). This relationship was not significant in normal readers and, in fact, scores on Purdue assembly in this reader group correlated with a cluster of NTB verbal scores. Normal readers also demonstrated significant relationships

Table 17

CORRELATIONS AMONG SENSORY/MOTOR TESTS AND SIGNIFICANT CORRELATIONS
BETWEEN SENSORY/MOTOR SCORES AND OTHER NTB SCORES FOR
DYSLEXIC AND NORMAL READER GROUPS

	Dyslexics					Normal Readers				
	Purdue Right	Purdue Left	Purdue Both	Purdue Assembly	DSS ^a	Purdue Right	Purdue Left	Purdue Both	Purdue Assembly	DSS ^a
PURDUE RIGHT	-	.55	.71*	.52	.32	-	.37	.63*	.28	.15
PURDUE LEFT	-	-	.89***	.60	.05	-	-	.81**	.11	.48
PURDUE BOTH	-	-	-	.70*	.03	-	-	-	.34	.26
PURDUE ASSEMBLY	-	-	-	-	.18	-	-	-	-	.42
WAIS VIQ	-	-	-	-	-	-	-	-	.75**	-
WRAT SPELLING	-	-	-	-	-	-	-	-	.80**	.66*
BOSTON NAMING	-	-	-	-	-	-	-	-	.66*	-
RAVEN	-	-	-	-	-	-	-	-	-	.75**

^aThe DSS test was scored using number of deficit responses; all other NTB tests were scored using correct responses. Correlation signs for the DSS test have been inverted in this table; therefore a significant correlation implies a positive relationship between the NTB skill involved and proficiency in DSS performance.

* $p < .05$

** $p < .01$

*** $p < .001$

between sensory performance and performance on visual and verbal NTB tests. Dyslexics attained no significant correlations between sensory or motor test scores and scores from other NTB subcategories.

CF Experimental Task Measures:

Correlations among CF scores and significant correlations between CF scores and NTB scores are reported in Table 18 with separate matrices for each reader group. Near perfect correlations occurred among the three CF scores in the dyslexic group indicating that the two independently scored CF measures--ICF and CBLCF--appear to be testing the same aspect of some performance ability (further confirmed by each of these scores' similarly strong correlation with the ACF score). Normal readers, in contrast, demonstrated no significant relationship between the two independently scored CF performance measures; a greater degree of significance, however, did occur between ICF and ACF scores than between CBLCF and ACF scores. Correlations of these CF scores with NTB scores also demonstrated quite different patterns for the two reader groups. In the dyslexic group each of the CF measures consistently correlated with scores from two relatively distinct NTB test areas: reading (specifically those two reading tests relying primarily on decoding the written word rather than on aggregate reading skills) and DSS (a sensory test in which deficit scores are suggestive of brain damage).²⁸ In the normal reader group no significant correlations occurred between the CBLCF score and scores on the NTB; significant correlations did occur, however, between the ICF score and test scores from various NTB subcategories and between the ACF score and verbal

²⁸Performance on this test, however, for the two reader groups was not significantly different.

Table 18

CORRELATIONS AMONG CF SCORES OF THE EXPERIMENTAL TASK AND
SIGNIFICANT CORRELATIONS BETWEEN CF SCORES AND
NTB SCORES FOR DYSLEXIC AND NORMAL READER GROUPS

	Dyslexics			Normal Readers		
	ICF ^a	CBLCF ^a	ACF ^a	ICF ^a	CBLCF ^a	ACF ^a
ICF	-	.96***	.98***	-	.53	.90***
LCF	-	-	1.00***	-	-	.64*
WOODCOCK	-	-	-	.67*	-	-
GRAY ORAL	.65*	-	.63*	-	-	-
WRAT READING	.66*	.65*	.66*	-	-	-
WAIS VIQ	-	-	-	.80**	-	.63*
BOSTON NAMING	-	-	-	-	-	.71*
SENTENCE REPETITION	-	-	-	.64*	-	.77**
WRAT ARITHMETIC	-	-	-	.77**	-	-
WAIS PIQ	-	-	-	.73*	-	-
DSS	.89***	.83**	.85**	-	-	-

^aExperimental task measures (ICF, CBLCF, ACF) and the DSS test were scored using number of deficit responses; all other NTB tests were scored using correct responses. Correlation signs for NTB tests (excluding the DSS test) that correlate with CF measures have therefore been inverted in this table and a significant correlation implies a positive relationship between the NTB test skill involved and proficiency in CF performance on the experimental task.

* $p < .05$

** $p < .01$

*** $p < .001$

scores. Thus in the dyslexic group reading and particularly DSS test performances appeared to be independent²⁹ yet consistent predictors of all three measures of CF performance in the experimental task. In the normal reader group performances in several subcategories (reading, visual, arithmetic and particularly verbal) of the NTB appeared to predict ICF performance in the experimental task; CBLCF performance, however, failed to correlate with any performance on the NTB.

In this correlation analysis dyslexics, in general, demonstrated a less integrated and more fragmented test performance profile than that of the normal reader group. When correlations did occur between NTB subcategories in the dyslexic group, however, these correlations tended to tight clusterings. These clusters involved those NTB tests (except for DSS) and the CF component of the experimental task on which dyslexics had demonstrated, in earlier analyses, significantly inferior performance. Especially relevant was the consistent and persistent relationship in the dyslexic group between reading scores and CF performances on the experimental task. The normal reader group, in contrast, exhibited a more consistently related performance pattern across all NTB subcategories as well as demonstrated an ability--which persisted throughout NTB testing--to maintain performance level while processing varied stimuli input, perceptual modes, sensory modalities and/or cognitive procedures. this functional ability component, characteristic of the normal reader group, extended to their CF performance on the experimental task.

²⁹The highest correlation between a reading test score and the DSS test score occurred with the reading section of the WRAT ($r = .56$) and was nonsignificant ($p < .09$).

Addendum

NTB results within the dyslexic group confirmed a relative homogeneity of cognitive and psychological functioning in which the only substantive distinction ascertained for possible subgrouping of subjects involved degree of impairment. The limited number of subjects as well as this within-group test performance consistency made analysis of the data drawn from this investigation by subgroup classification unfeasible. Examination of dyslexic within-group performance for VF and particularly CF processing on the experimental task resulted in the delineation of various subject clusters based on differential performance. Attempts to compare these experimental subject clusters with respective NTB test performance profiles and thus establish possible parallels, however, failed to yield salient correspondences.

V. Discussion

The research completed here does indicate that psychological and achievement test performance profiles of adult developmental dyslexics demonstrate comparable deficit patterns found in clinical study of child developmental dyslexics (Benton, 1975; Critchley, 1970; Critchley & Critchley, 1978; Money, 1962; Naidoo, 1972; Petrauskas & Rourke, 1979; Rutter, 1978; Symmes, 1972; Symmes & Rapoport, 1972). Although few of these child studies offer extensive summaries of actual psychometric test data, their basic assumptions and conclusions pertaining to the nature and identification of the dyslexia syndrome are relatively circumscribed. Conforming to the conceptual overview of dyslexia postulated earlier, test profiles found in this study of adult dyslexic subjects, which parallel those ascribed to child developmental dyslexics, necessitate a discussion of specific qualitative and quantitative performance results on the NTB.

Dyslexic performance was significantly inferior ($p < .001$) to that of normal readers on all three reading tests and correlations among these tests were more highly significant in dyslexics. Corroborating the assignment of specific decoding problems as a major characteristic of developmental dyslexia, dyslexic subjects in this study exhibited more impaired word decoding and identification skills than reading comprehension problems. On the three tests which examined varied cognitive components of reading, these adult dyslexics experienced their greatest difficulty in decoding of nonsense words, then real words in paragraph form (timed) and finally isolated real words (untimed);

although still well below grade level, their performance on letter identification and reading comprehension was considerably better. When each dyslexic subject reached his reading comprehension limit (inability to identify a sufficient number of words for comprehension of a passage or analogy), remaining test material was presented orally and, using this procedure, these subjects easily exceeded their scored reading level. Such findings tend to confirm Vellutino's (1979) primary contention that the child dyslexic "has unusual deficiencies at the level of individual word decoding . . . [as well as] in analyzing their component sounds" (p. 24); these observations also indicate that compensatory strategies in contextual processing provide some improvement in general reading ability.

Critchley and Critchley's (1978) observation that child, adolescent and adult dyslexics exhibit an erratic inconsistency in spelling performance with frequent phonetic substitutions, rotations and reversals, omissions, confused serialization and, on occasion, a superfluity of letters, appropriately describes the spelling-error patterns characterizing this particular group of adult dyslexics. In fact, their ability to spell individual words proved somewhat inferior to their ability to read comparable words³⁰--a differential distinguishing dyslexic spelling and reading skills, confirmed in systematic cross-sectional and longitudinal studies of dyslexic children (Frauenheim, 1978; Naidoo, 1972; Rutter, Tizard & Whitmore, 1970; Traites & Fiedorowicz, 1976). A critical

³⁰ Despite a large significant group difference ($p < .001$), the normal reader group also revealed spelling problems (but not conforming to those discernable dyslexic patterns), probably attributable to a general lapse in contemporary academic and professional standards (Critchley & Critchley, 1978).

qualitative assessment of writing skills in this adult dyslexic group also indicated severe retardation. All subjects in this study were asked to write a paragraph on any theme of their choosing; slow, labored efforts by dyslexics on this task resulted in relatively short, disjointed sentences displaying minimal vocabulary, marked spelling and punctuation errors, and often no sense of structure or continuity. When requested to relate the composed text orally, their response was invariably livelier, more structured and coherent, semantically expressive, and it contained nuances conspicuously absent from their written efforts. This contrast between written and oral expression among dyslexics appears to widen as maturing dyslexics increase their analytical capacities and means of oral expression while their reading and writing levels remain relatively static.

Although dyslexic performance was significantly inferior ($p < .02$) to that of normal readers on the arithmetic section of the WRAT, normal reader mean performance was also well below grade level--attributable to any number of reasons including a general decline in academic standards. Deficit dyslexic arithmetic performance contrasted with that of normal readers in kinds of error committed (i.e., number reversals, omissions and inaccurate arithmetic punctuation) while the normal reader group's errors were usually the result of simple miscalculation.³¹ Some clinical research on the dyslexia syndrome (see Critchley, 1970; Critchley & Critchley, 1978) has proposed a "paradoxical" condition in which dyslexics often demonstrate average (or even superior) arithmetical skills despite severe reading impairment. Recent research cross-

³¹The time factor on the arithmetic section of the WRAT induced a degree of anxiety in both reader groups which conceivably negatively affected their respective performances.

sectional (Fisk & Rourke, 1979) and longitudinal studies (Frauenheim, 1978; Traites & Fiedorowicz, 1976) as well as this investigation, has found, however, that dyslexics display significant deficits in arithmetic performance (particularly with advancing age) comparable in degree to their reading dysfunction. In addition, contrary to normal readers in this study, consistently large significant correlations occurred among arithmetic, reading and spelling performances in dyslexics (also see Traites & Fiedorowicz, 1976). Although other explanations may also be applicable (see Critchley, 1970; Critchley & Critchley, 1978), such findings support the hypothesis that dyslexics experience a specific dysfunction in the decoding, sequencing and manipulation of symbolic stimuli (numbers as well as letters)--the underlying component common to all three achievement areas.

Considerable research has been conducted using WISC subtest profiles in the identification and analysis of reading deficits among children (Altus, 1956; Belmont & Birch, 1966; Burks & Bruce, 1955; Fisk & Rourke, 1979; Huelman, 1970; Klasen, 1972; McLeod, 1965; Naidoo, 1972; Neville, 1961; Petrauskas & Rourke, 1979); unfortunately, subject groupings (usually "poor" and "good" readers as well as "dyslexics" diagnosed with less than stringent criteria) and integration of WISC subtests with other psychometric tests negate the applicability of these studies' findings to this investigation. Particularly relevant is Symmes & Rapoport's (1972; also see Symmes, 1972) cross-sectional study of child dyslexics, examining performances on the WISC as well as on a variety of visual psychometric tests, which applied the same stringent diagnostic criteria as this investigation for its selection of dyslexic subjects (age 7 to 13); this shared criteria validates a comparison of develop-

mental dyslexic child and adult test profiles. In their study child dyslexics obtained a higher PIQ than VIQ mean score and, within subtests on the WISC, mean scores were poorest on Digit Span, Coding and Arithmetic.³² Highest mean scores (well above average) occurred on Comprehension and Similarities within verbal subtests and on Picture Completion and Object Assembly within performance subtests. These dyslexic children also demonstrated average or above average performance levels on the visual test battery which surveyed a variety of nonverbal skills. Finally, Symmes and Rapoport found that test scores, across age groups, maintained a remarkably similar profile configuration with the exception that the oldest dyslexic subgroup tested (age 11 to 13) showed a decline in performance on Information and Vocabulary subtests.

In this investigation of adult dyslexics, test performances were examined, as well as compared with those of a normal reader control group. Within the dyslexic group, conforming to Symmes and Rapoport's study of children, the PIQ mean score was considerably higher than that of the VIQ; this differential did not occur within the normal reader group. In fact, although FSIQ and VIQ mean scores of dyslexics were significantly inferior to those of normal readers, no significant group difference occurred on PIQ. Adult dyslexic WAIS subtest results closely paralleled WISC subtest results achieved by Symmes and Rapoport's child dyslexics, both quantitatively and qualitatively. In addition, their suggested trend of diminished performance, with advancing age, on Information and Vocabulary subtests was confirmed in this investigation; adult dyslexics performed poorly on both these subtests. Examination of mean

³²Factor analysis indicated that the underlying component in these three tasks (whether visual or verbal) was the sequencing of symbols.

score differences between adult dyslexic and normal reader groups yielded a strong correspondence to this variable WAIS subtest performance pattern: no significant group differences occurred for subtests on which dyslexics did well, but significant group differences did occur for subtests on which dyslexics scored poorly. Thus common to both child and adult dyslexics (using stringent diagnostic criteria) Wechsler IQ subtest profiles demonstrated proficiency in visual and verbal analytic processing, dysfunction in visual and verbal sequencing and finally slow but steady decline with advancing age in "factual information known." Adult dyslexic performances on psychometric tests examining visual abilities were average and not significantly different from those of normal readers--a finding also commensurate with results in the Symmes and Rapoport study of dyslexic children using a different but analogous battery of visual tests.

On the two remaining verbal tests given in this NTB--Benton Sentence Repetition and Boston Naming--dyslexic performance was significantly inferior to that of normal readers only on the Boston Naming test. This deficit in the naming of object drawings had been observed by Denckla and Rudel (1976a) in child dyslexics. No significant correlations between visual and verbal tests examined on the NTB were found in the dyslexic group while the normal reader group, as would be expected, demonstrated numerous significant correlations between these two areas. Thus in dyslexics visual and verbal cognitive processes appear to reside in separate realms, with visual skills superior to verbal skills; in normal readers cognitive processing seems to function as an integration of visual and verbal abilities, with visual skills equivalent to verbal skills.

Motor (manual) and sensory performances were comparable in dyslexics and normal readers. Anomalously, despite established right-handedness (assessed by two independent laterality questionnaires), each group exhibited a nonsignificantly higher mean left hand advantage as well as a more highly significant correlation of left hand performance to "both hands" performance (right hand performance significantly correlated to "both hands" performance but to a lesser degree).

Analysis of NTB data in adult dyslexics relative to normal readers and comparison of adult dyslexic test performance profiles to those of child developmental dyslexics provide strong evidence for persistence of deficits (in kind) in reading, spelling and writing. Specific psychological dysfunctions characteristic of child developmental dyslexics also remain impaired in their adult counterparts. Thus despite scholastic achievement--attributable to compensatory strategies and/or some positive response to remediation efforts--the dyslexia syndrome continues to manifest itself in these adult dyslexic subjects with many of the characteristics symptomatic of the problem in dyslexic children.

The conception of the dyslexic eventually "outgrowing" his disability and/or significantly meliorating the dysfunction through remediation (Critchley & Critchley, 1978; Orton, 1937) is provided minimal support based on the NTB results of this investigation. Thus this research fails to support the "maturational lag" theory (de Hirsch, Jansky & Langford, 1966; Koppitz, 1971; Satz, Friel & Goebel, 1975; Satz, Friel & Rudegear, 1974; Satz & Van Nostrand, 1973) as one explanation of dyslexia etiology. In fact, results derived tend to refute the hypothesis that dyslexia may be attributed to a specific maturational

defect; adult dyslexic NTB performance profiles (including reading) do not qualitatively conform to those of young normal readers but actually, with some degree of variation, resemble those performance patterns of child dyslexics (see Symmes, 1972; Symmes & Rapoport, 1972). This view of developmental dyslexia as the result of a delay in the maturation process of the cerebral cortex (with the corollary that this condition can eventually correct itself) is contradicted by the persistence of deficits into adulthood similar to those found in children (also see Frauenheim, 1978; Trites & Fiedorowicz, 1976). The model for dyslexia etiology positing a cerebral deficit either in the form of a "focal maldevelopment" (Geschwind, 1965) or in the overall organization of cerebral function (Gazzaniga, 1974; Orton, 1925, 1937) is neither supported nor refuted in this investigation; NTB findings for this particular group of adult dyslexics concur with this hypothesis only to the extent that reading as well as other test performance profile patterns indicative of the dyslexia syndrome remain intact.

Examination of correct-but-late VF (CBLVF) response performance (scored using a gross but accurate reaction-time criterion) in the experimental task revealed a significant group difference across bilateral mode conditions but not across unilateral mode conditions. Dyslexics experienced significantly greater difficulty than normal readers, particularly in the RVF, in responding quickly to single-digit stimuli flashed irregularly, counterbalanced in the two VF. Significantly slower response latencies were reported for dyslexics in verbal reaction-time studies involving VF identification of isolated letters (Bouma & Legein, 1980; McKeever & VanDeventer, 1975) as well as embedded letters and words (Bouma & Legein, 1980). The significant

CBLVF result in the present experiment conforms to these findings: the small number of subjects in each group (10), the occurrence of ceiling effects in 8 normal readers and 5 dyslexics for CBLVF scores across bilateral mode conditions, and the lack of absolute VF verbal reaction-time data, however, all affect the reliability of this finding and preclude the possibility of positing any conclusive interpretation of this CBLVF group difference. Despite the indication of slower VF response latencies in dyslexics, overall incorrect VF (IVF) identification and aggregate VF (AVF) deficit scores were comparable for dyslexics and normal readers across both unilateral and bilateral mode conditions. Equivalent average VF performance levels had also been found for dyslexics and normal readers in studies requiring verbal identification of isolated letters (Bouma & Legein, 1977, 1980) and words (Kershner, 1977; Pirozzolo & Rayner, 1979). In contrast to these findings dyslexics were found to exhibit impaired overall VF performance levels relative to that of normal readers in verbal identification of embedded letters (Bouma & Legein, 1977, 1980) as well as words³³ (Bouma & Legein, 1977, 1980; McKeever & VanDeventer, 1975).

Analysis of IVF identifications across the bilateral mode conditions (administered within the same test session but with two intervening unilateral mode conditions) indicated that LVF/RVF performance across time was variable and not consistent within either reader group. In fact, the differences between LVF and RVF incorrect identifications reversed for each reader group (but in opposite directions) from the first to the second bilateral presentation of this task (see Figure 1 in Results

³³The inconsistency and inherent contradiction of findings between the Kershner (1977) and Pirozzolo and Rayner (1979) studies and the Bouma and Legein (1977), 1980) and McKeever and VanDeventer (1975) studies for word identification have been noted but remain unresolved.

section). This inconsistent and even erratic VF performance in each reader group is consonant with Zaidel's (1979) observation on the low reliability factor in testing for perceptual asymmetry. The sensitivity of subjects to stimulus and task parameters--materials, sensory modalities, response modes as well as other extrinsic or tangential elements--has been found to influence perceptual asymmetries. Hines and Satz (1974) examined test-retest correlations and discovered a reliability of only 0.46 in testing (tachistoscopic presentation) VF recall of digit stimuli. Particularly salient to the variable VF findings across time in the present experiment are the results derived from the research of Hakes, Evans and Studdert-Kennedy (1974) examining speech perception; a disappearance of REA or a shift from REA to LEA on a dichotic listening task was shown to occur within a single test session. In addition, because single-digit stimuli have been shown to be less lateralized than other verbal stimuli, particularly in identification tasks (Drozdenko, 1979; Kimura, 1966; White, 1969), VF performance in the present investigation was perhaps vulnerable to or influenced by subtle changes in such intrinsic subject factors as hemisphere attentional focus, perceptual/cognitive strategies and/or fatigue effects. Variability in VF performance across time, attributable to any of these or other possible intrinsic or extrinsic influences, appeared to a greater degree within the dyslexic group; differences between the number of LVF and RVF incorrect identifications over time was significant only within the dyslexic group. Dyslexics also made significantly more LVF than RVF incorrect identifications in the first bilateral presentation, providing evidence of greater VF asymmetry during the initial stage of the test session; significant lateralized performance did not occur for either bilateral mode condition within the

normal reader group. This last finding contrasts with previous dyslexia research which tested verbal identification of word stimuli (in unilateral presentations which correspond to the bilateral mode condition in the present experiment); these studies have indicated less lateralization in dyslexics than in normal readers (Kershner, 1977; McKeever & VanDeventer, 1975; Pirozzolo & Rayner, 1979). Significantly depressed LVF word identification performance in dyslexics, nevertheless, was shown in McKeever and VanDeventer's (1975) study during bilateral (simultaneous) VF presentation; this differential VF effect was eliminated, however, during unilateral (successive) VF presentation in the same study.³⁴

Rate (four VF trial intervals) was significant across both unilateral and bilateral mode conditions, although this effect could not be explained for either condition with pair wise comparisons. But, across the unilateral mode conditions, a significant Rate X Field interaction occurred in the normal reader group, attributable to high performance variability among the rate intervals, as well as inconstant differential VF performance among these rate intervals. For rate 3 (shortest VF trial interval), in fact, normal readers made significantly more IVF identifications in the unilateral mode condition which examined only LVF performance (relative to that unilateral mode condition which examined only RVF performance). Unlike that of the normal reader group, dyslexic performance among the rate intervals and within VF across rate intervals was essentially stable and revealed no significant findings (see Figure 3 in Results section). These relative intragroup and intergroup rate patterns could conceivably be the result of hemisphere vigilance

³⁴Since a bilateral (simultaneous) VF presentation procedure was not employed in the present testing, parallels cannot be drawn.

performance distinctions but, because markedly different methods have been used in previous hemisphere vigilance research (Dimond, 1976, 1977, 1979; Dimond & Beaumont, 1971b, 1973; Ellenberg & Sperry, 1979, 1980; Heilman & Van Den Abell, 1979, 1980; Warm, Schumsky & Hawley, 1976), none of which examined a dyslexic population, extrapolation from these studies and comparison of their results with this experiment are not applicable. In addition, the demanding performance requirements for subjects inherent in this experimental design--rapid verbal identification of 620 single digits within each task presentation--of necessity limited test duration to only 10 minutes for each presentation. This time constraint contrasts with previous vigilance experiments which, by definition, usually require sustained attention over a much longer test period. Although inconclusive, VF data derived from this experiment does offer evidence of significantly different left/right hemisphere attentional patterns in normal readers and differences (nonsignificant) in hemisphere attentional patterns between dyslexics and normal readers--these observations meriting further investigation. Utilizing the innovative instrumentation employed in this experiment but altering the experimental task design (retention of only VF components examining hemisphere vigilance performance as well as a substantial increase in task duration) could possibly yield relevant insights into the nature of hemisphere vigilance for both dyslexics and normal readers.

In the present experiment, although VF performance failed to reveal major distinctions between the dyslexic and normal reader groups, significant group differences were manifest in CF performance across both unilateral and bilateral mode conditions. Dyslexic performance (analyzed separately for incorrect CF [ICF] identifications, correct-but-late CF

[CBLCF] responses and aggregate CF [ACF] deficit scores across each of the two conditions), despite large within-group variability, was significantly inferior to that of normal readers on all CF measures except CBLCF performance in the bilateral mode conditions. Dyslexics experienced considerably greater difficulty in the verbal identification of and response to single-digit stimuli flashed regularly, successively and rapidly for short durations in the CF. Separate discriminant analyses of mean CBLCF and ICF scores provide dramatic contrasts distinguishing performance levels between the two groups; on each independently scored CF measure, performance of 9 dyslexic subjects could be clearly discriminated from that of normal reader subjects.³⁵ In effect this relative absence of any overlap in score distribution between the two reader groups further demonstrates the severity of dyslexic impairment in CF performance on this task. In addition, averaged across the four presentations, dyslexic CBLCF responses (8.95%) were considerably higher than ICF identifications (4.86%) while in normal reader testing, ceiling effects occurred and CBLCF responses (0.28%) were lower than ICF identifications (0.67%).³⁶ The degree of dyslexic impairment in CF performance inherent in these findings may be attributed to problems in several areas: attentional lapses, slowed verbal retrieval, higher visual thresholds, impaired rate of perceptual processing and/or dysfunctional rudimentary foveal perception.

The experimental design and the particular task demands preclude

³⁵The dyslexic not discriminated in the two CF measures was a different subject for each.

³⁶The extremely high level of normal reader CF performance, however, would appear to negate the importance of the relative CBLCF and ICF differential.

specific identification of subtle lapses or shifts in attention as a factor influencing dyslexic CF deficit performance.³⁷ Dyslexic "visual-speech recoding" difficulty was encountered by Bouma and Legein (1980) in their tachistoscopic experiment testing CF and VF reaction time for identification of verbal stimuli. This form of slowed verbal retrieval, according to Bouma and Legein, explained significantly slower dyslexic verbal response latencies (120 msec in both CF and VF for isolated letters). Similar dyslexic decrements in speed of reporting both CF and VF single-letter stimuli (comparable in magnitude to those found in the Bouma and Legein [1980] study) had also occurred in the McKeever and VanDeventer (1975) investigation. Attributing dyslexic slower verbal response latencies to verbal retrieval difficulties or visual-speech decoding dysfunction, however, does not appear viable in explaining dyslexic CBLCF and/or ICF performance in the present experiment. Superiority of dyslexic VF to CF performance (the former comparable to normal readers), identifying the same single-digit stimuli, is inconsistent with such an attribution. If either verbal retrieval or visual-speech recoding impairment were responsible for the dyslexic CF deficit profile in this experimental task, significant group differences (and/or similar dyslexic deficit patterns) should have been revealed in both VF and CF performance. Gross, Rothenberg, Schottenfeld and Drake's (1978) study found that dyslexics displayed significantly higher thresholds for VF identification of single letters; the attribution of higher thresholds to explain dyslexic deficient CF results in the present experiment also

³⁷ Corcoran's (1963) CF vigilance experiment, testing sleep-deprived subjects, offers a possible paradigm for investigating attentional behavior in dyslexic CF performance.

appears lacking in consistency. Performance with identical CF and VF stimulus parameters (single digits and stimulus duration) tested here yielded contrasting CF/VF results (nonsignificant group differences in VF performance and significant group differences in CF performance) which prohibits the assignment of higher visual thresholds as the factor responsible for dyslexic CF performance difficulties in this task. In addition, with foveal to parafoveal visual acuity decreasing dramatically (50% at only 1° off center), relative dyslexic VF performance (if visual thresholds were too high) should have been at least as poor as CF performance.

The single element which distinguishes CF from VF performance demands (aside from retinal locus of stimuli) in this experiment is the rate at which stimuli were presented for perceptual processing. Since VF performance was comparable for dyslexics and normal readers and CF performance severely impaired in the dyslexic group, dyslexics appear to experience considerably greater difficulty on a visual task (not involving a memory component) necessitating the processing of verbal stimuli (of short duration) presented regularly, successively and rapidly. Although dyslexic temporal order and/or sequential perceptual dysfunction was observed by Orton (1937) in his pioneer study of the dyslexia syndrome and has since been confirmed in empirical research (Bakker, 1970, 1972; Corkin, 1974; Rudel & Denckla, 1976a, 1976b; Sterritt & Rudnick, 1966; Zurif & Carson, 1970) with tasks requiring both retention and perception, dyslexic primary temporal perceptual processing, involving no memory component, remains relatively unexplored. Relevant is Denckla and Rudel's (1976b) study examining rapid automatized naming (R.A.N.). In their experimental design, which eliminated the memory

factor from testing of temporal perception, results derived still differentiated dyslexic child performance from non-dyslexic learning-disabled child performance as well as from that of normal readers; deficits in dyslexic performance which demanded "automatization" of verbal responses to visually presented stimuli (objects, colors, letters and numerals, cited in descending order of difficulty) were manifested in a significantly slower rate of response to the stimuli in series. The methods utilized to test rate of perceptual processing in their study, however, differs from and therefore precludes direct comparison with the present experiment; the rate factor in the Denckla and Rudel task was "subject-paced" with constant stimulus presentation of extended duration (present throughout testing) rather than "experimenter-paced" in the form of regular, rapid stimulus presentation of short duration.

Tallal's (1980) "experimenter-paced" nonverbal auditory study which tested primary aspects of temporal perceptual processing in reading-impaired children is of particular interest to the present investigation. Tallal found that a "subgroup" of reading-impaired children³⁸ experienced significant difficulty on both sequencing (temporal order requisite) and discrimination (no temporal order requisite) tasks only when the inter-stimulus timing was shortened. Dysfunctional temporal perception in the subgroup of reading-impaired children therefore was attributed by

³⁸Tallal's subject population contained some severely reading-impaired children (possibly dyslexic?) and some children only slightly reading-impaired. Her failure to identify the reading-impaired subgroup and to analyze this subgroup data relative to the other reading-impaired subjects, as well as the normal reader group, limits the specific application of her conclusions to an analysis of the dyslexia syndrome.

Tallal to a primary deficit which influenced the perceptual rate at which information could be processed. Tallal's approach--nonverbal stimuli, auditory mode, manual response, short stimulus trials as well as manipulation of interstimulus timing--is in marked contrast to the methodology employed in the present experiment. Nevertheless, her attribution of a primary deficit in rate of perceptual processing appears a valid rationale to explain the significant dyslexic performance decrement in that aspect of this experimental task--CF identification--which specifically tested continuous, rapid stimulus presentation. Tachistoscopic studies which have examined verbal identification of single letters presented CF (Bouma & Legein, 1977, 1980; Kershner, 1975), in contrast to the present investigation, revealed no significant dyslexic CF performance decrements relative to normal readers. The disparity between their results and those elicited here seems clearly attributable to differences in rate of perceptual processing; in their studies stimulus onset was of longer duration, and more important, CF interstimulus timing was relatively nonrapid (as well as irregular) and therefore did not specifically investigate temporal perception. Thus findings of Tallal (1980) and of the present investigation concur in the assignment of rate as a crucial factor in the perceptual-informational process of reading, affecting both reading-impaired children and adult dyslexics. Since continuous visual-verbal temporal processing is a major component in reading, this experimental task could be modified to more specifically ascertain a contributory relationship between rate of

visual-verbal perceptual processing and reading impairment in dyslexics.³⁹

The Parsons, Majumder and Chandler (1967, 1968) research on "visual temporal acuity" (flicker detection) deficits (in brain-damaged subjects) posited a possible defect in interhemispheric articulation as the underlying dysfunctional mechanism; this finding and subsequent extrapolation conceivably offer a different but related approach to investigating that rate processing deficit characterizing dyslexic CF performance in the present experiment. Dyslexics had been shown, in two separate EEG studies (Leisman & Ashkenazi, 1980; Sklar, Hanley & Simmons, 1972), to demonstrate greater intrahemisphere than interhemisphere (between homologous regions) coherence values than controls both at rest and during cognitive task performance (including reading); therefore dyslexics may be said to manifest less synchrony between hemispheres relative to that of normal readers. Physiological research (with cats), conducted by Whitteridge (1966) and Choudury, Whitteridge and Wilson (1965), had earlier suggested that, under normal conditions, vertical meridian callosal fiber connections are required during the initial phase of the perceptual process; nevertheless, the speed and efficiency of transmission across this callosal linkage, functioning with no identifiable problems in man, has not been the subject of behavioral research. Additional physiological study by Hubel and Weisel (1965, 1967), examining

³⁹Instrumentation utilized in this experiment could easily be adapted to concentrate on CF identification performance in which interstimulus timing is varied with letter stimuli substituted, thus focusing on this process.

A similar experimental task, substituting light flashes for verbal stimuli and manual response for verbal response might also be conducted to eliminate verbal components and concentrate only on primary visual temporal perceptual processing in dyslexics.

these interhemispheric connections utilizing a similar methodology, led them to conclude that transmission by the corpus callosum of elemental visual material, again under normal circumstances, both extends the visual fields across the midline and also serves in the creation of more complex visual fields involved in the establishment of neural integrity for rudimentary visual perception.

This physiological research and those studies investigating visual temporal acuity and dyslexic EEGs taken together provide background for a tentative hypothesis to explain the CF identification difficulty experienced by dyslexics in this experimental task. That the dyslexic impairment may be related to rudimentary visual perception dysfunction--resulting from poorly developed or even abnormal organization of vertical meridian callosal fiber connections--is, in terms of the present experiment, highly conjectural. Nevertheless the manifestation of marked dyslexic CF performance decrements when stimulus duration is brief and the rate of stimulus presentation rapid may be understood in terms of temporal perceptual processing difficulties symptomatic of a subtle dysfunction in rudimentary foveal perception.

Separate correlation analyses conducted in this investigation revealed a highly significant positive correlation between the two independently scored CF measures (ICF and CBLCF) in the dyslexic group; no significant relationship, however, occurred between these two CF measures in the normal reader group. In dyslexics these two CF measures appear to be dependent on a shared component influencing performance--possibly temporal perceptual processing. In addition, for dyslexics the only NTB tests (with the exception of DSS) which correlated significantly with these two CF measures were those examining reading

skills; the reading tests involved (WRAT and Gray Oral) required specific decoding of the written word rather than reliance on aggregate reading skills (Woodcock). Thus in dyslexics the two independently scored CF measures in the experimental task appear to be consistent predictors of reading and particularly DSS performances, although the two NTB test areas are unrelated.⁴⁰ In contrast, normal readers attained no significant correlations between the CBLCF measure and any NTB score but significant correlations were shown between the ICF measure and NTB scores in numerous test subcategories, including reading as well as visual, verbal and arithmetic. These independent correlation profiles, particularly within the dyslexic group, demonstrate a positive relationship between CF performance ability and reading skills; this relationship, consistent and circumscribed in dyslexics, suggests a common yet subtle perceptual and/or temporal mechanism, integral to both CF performance and reading process demands.

⁴⁰ Even though consistent high correlations between both CF measures and the DSS test were found, the highest correlation between a reading test (WRAT) and the DSS test was $\underline{r} = .56$, which was nonsignificant ($\underline{p} = .09$).

APPENDIX A
INDIVIDUAL DYSLEXIC AND NORMAL READER
NTB PROFILES

Subject: CH Dyslexic: x Normal Reader:

Age: 26 Education: College Freshman

Laterality Preference

Hand: R Foot: R Eye: R Ear: R

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>12</u>	<u>75</u>	
Comprehension	<u>17</u>	<u>99</u>	
Arithmetic	<u>7</u>	<u>16</u>	
Similarities	<u>11</u>	<u>63</u>	
Digit Span	<u>10</u>	<u>50</u>	
Vocabulary	<u>13</u>	<u>84</u>	
Verbal Score	<u>70</u>		<u>+0.63</u>
[Verbal IQ <u>109</u>]		<u>73</u>	
Digit Symbol	<u>10</u>	<u>50</u>	
Picture Completion	<u>14</u>	<u>91</u>	
Block Design	<u>11</u>	<u>63</u>	
Picture Arrangement	<u>15</u>	<u>95</u>	
Object Assembly	<u>13</u>	<u>84</u>	
Performance Score	<u>63</u>		<u>+1.15</u>
[Performance IQ <u>118</u>]		<u>89</u>	
Full Scale Score	<u>133</u>		<u>+0.91</u>
[Full Scale IQ <u>113</u>]		<u>80</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>12.9</u>	<u>58</u>	<u>78</u>	<u>+0.8</u>
Word Identification	<u>8.6</u>	<u>38</u>	<u>11</u>	<u>-1.2</u>
Word Attack	<u>3.9</u>	<u>38</u>	<u>11</u>	<u>-1.2</u>
Word Comprehension	<u>12.9</u>	<u>52</u>	<u>57</u>	<u>+0.2</u>
Passage Comprehension	<u>11.6</u>	<u>49</u>	<u>45</u>	<u>-0.1</u>
Total Reading	<u>9.6</u>	<u>44</u>	<u>29</u>	<u>-0.6</u>

Gray Oral

Total Score 52 Grade Equivalent 6.8 Comprehension Score 25.5

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>7.9</u>	<u>94</u>	<u>34</u>	<u>-0.4</u>
Spelling	<u>6.7</u>	<u>88</u>	<u>21</u>	<u>-0.8</u>
Arithmetic	<u>7.1</u>	<u>90</u>	<u>25</u>	<u>-0.67</u>

Sentence Repetition (NCCEA)

#Correct 17 Percentile 70

Boston Naming Test

#Correct Without Cue 71 #Category Cues Given 14
 #Correct Following Category Cue 3 #Phonemic Cues Given 11
 #Correct 5 and Incorrect 6 Following Phonemic Cue
 (Age Level 25
 Total Score 74 Z-Score -0.31 Percentile {Educ. Level 25

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>12</u>	<u>12</u>
B	<u>12</u>	<u>11</u>
C	<u>11</u>	<u>10</u>
D	<u>10</u>	<u>10</u>
E	<u>6</u>	<u>7</u>

Total Score 51 Percentile 81

Rey-Osterrieth Complex Figure

Copy Score 35 Percentile 87.5 Memory Score 19 Percentile 31.25

Knox Cube

Trial I 11 Trial II 13 Average Score 12 CA 14.5/15.5

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>43</u>	<u>41</u>	<u>34</u>	
1-Trial Score				<u>34</u>
Percentile	<u>17.5</u>	<u>22</u>	<u>24</u>	

Double Simultaneous Stimulation (A. Smith)

Right Sensory 0 Left Sensory 1 Bilateral 0 Errors

Subject: RI Dyslexic: x Normal Reader:

Age: 19 Education: College Freshman

Laterality Preference

Hand: R Foot: R(L) Eye: R(L) Ear: L

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>6</u>	<u>9</u>	
Comprehension	<u>12</u>	<u>75</u>	
Arithmetic	<u>8</u>	<u>24</u>	
Similarities	<u>11</u>	<u>63</u>	
Digit Span	<u>10</u>	<u>50</u>	
Vocabulary	<u>8</u>	<u>24</u>	
Verbal Score	<u>55</u>		<u>-0.16</u>
[Verbal IQ <u>98</u>]		<u>45</u>	
Digit Symbol	<u>8</u>	<u>24</u>	
Picture Completion	<u>18</u>	<u>99+</u>	
Block Design	<u>9</u>	<u>37</u>	
Picture Arrangement	<u>11</u>	<u>63</u>	
Object Assembly	<u>10</u>	<u>50</u>	
Performance Score	<u>56</u>		<u>+0.56</u>
[Performance IQ <u>109</u>]		<u>73</u>	
Full Scale Score	<u>111</u>		<u>+0.17</u>
[Full Scale IQ <u>103</u>]		<u>58</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter				
Identification	<u>4.3</u>	<u>43</u>	<u>23</u>	<u>-0.7</u>
Word				
Identification	<u>5.7</u>	<u>34</u>	<u>5</u>	<u>-1.6</u>
Word				
Attack	<u>3.1</u>	<u>34</u>	<u>5</u>	<u>-1.6</u>
Word				
Comprehension	<u>5.1</u>	<u>37</u>	<u>10</u>	<u>-1.3</u>
Passage				
Comprehension	<u>6.7</u>	<u>40</u>	<u>16</u>	<u>-1.0</u>
Total Reading	<u>4.7</u>	<u>32</u>	<u>4</u>	<u>-1.8</u>

Gray Oral

Total Score 42 Grade Equivalent 5.0 Comprehension Score 27

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>6.2</u>	<u>82</u>	<u>12</u>	<u>-1.2</u>
Spelling	<u>5.2</u>	<u>77</u>	<u>6</u>	<u>-1.53</u>
Arithmetic	<u>4.9</u>	<u>75</u>	<u>5</u>	<u>-1.66</u>

Sentence Repetition (NCCEA)

#Correct 13 Percentile 4

Boston Naming Test

#Correct Without Cue 67 #Category Cues Given 18
 #Correct Following Category Cue 1 #Phonemic Cues Given 17
 #Correct 9 and Incorrect 8 Following Phonemic Cue
 (Age Level 14.29
 Total Score 68 Z-Score -1.25 Percentile 16.18 Educ. Level 16.18

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>11</u>	<u>10</u>
B	<u>10</u>	<u>9</u>
C	<u>8</u>	<u>8</u>
D	<u>9</u>	<u>9</u>
E	<u>3</u>	<u>4</u>

Total Score 41 Percentile 39.28

Rey-Osterrieth Complex Figure

Copy Score 31 Percentile 25 Memory Score 28 Percentile 79.2

Knox Cube

Trial I 14 Trial II 13 Average Score 13.5 CA 15.5+

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>47</u>	<u>41</u>	<u>35</u>	
1-Trial Score				<u>43</u>
Percentile	<u>37</u>	<u>22</u>	<u>31</u>	<u>96</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 1 Left Sensory 0 Bilateral 0 Errors

Subject: TE Dyslexic: X Normal Reader:

Age: 18 Education: High School Graduate

Laterality Preference

Hand: R Foot: R Eye: R Ear: L(R)

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>9</u>	<u>37</u>	
Comprehension	<u>9</u>	<u>37</u>	
Arithmetic	<u>10</u>	<u>50</u>	
Similarities	<u>13</u>	<u>84</u>	
Digit Span	<u>7</u>	<u>16</u>	
Vocabulary	<u>7</u>	<u>16</u>	
Verbal Score	<u>55</u>		<u>-0.83</u>
[Verbal IQ <u>98</u>]		<u>45</u>	
Digit Symbol	<u>8</u>	<u>24</u>	
Picture Completion	<u>14</u>	<u>91</u>	
Block Design	<u>15</u>	<u>95</u>	
Picture Arrangement	<u>13</u>	<u>84</u>	
Object Assembly	<u>12</u>	<u>75</u>	
Performance Score	<u>62</u>		<u>+1.06</u>
[Performance IQ <u>116</u>]		<u>86</u>	
Full Scale Score	<u>117</u>		<u>+0.41</u>
[Full Scale IQ <u>106</u>]		<u>66</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>6.2</u>	<u>50</u>	<u>50</u>	<u>0.0</u>
Word Identification	<u>2.4</u>	<u>off scale</u>	<u>off scale</u>	<u>off scale</u>
Word Attack	<u>3.5</u>	<u>36</u>	<u>8</u>	<u>-1.4</u>
Word Comprehension	<u>6.3</u>	<u>39</u>	<u>14</u>	<u>-1.1</u>
Passage Comprehension	<u>3.1</u>	<u>off scale</u>	<u>off scale</u>	<u>off scale</u>
Total Reading	<u>3.4</u>	<u>off scale</u>	<u>off scale</u>	<u>off scale</u>

Gray Oral

Total Score 6 Grade Equivalent 1.4 Comprehension Score 23.5

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>4.2</u>	<u>72</u>	<u>3</u>	<u>-1.87</u>
Spelling	<u>3.0</u>	<u>66</u>	<u>1</u>	<u>-2.27</u>
Arithmetic	<u>5.7</u>	<u>79</u>	<u>8</u>	<u>-1.4</u>

Sentence Repetition (NCCEA)

#Correct 14 Percentile 14

Boston Naming Test

#Correct Without Cue 73 #Category Cues Given 12
 #Correct Following Category Cue 5 #Phonemic Cues Given 7
 #Correct 3 and Incorrect 4 Following Phonemic Cue
 (Age Level 50
 Total Score 78 Z-Score +0.31 Percentile {Educ. Level 70

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>12</u>	<u>11</u>
B	<u>8</u>	<u>9</u>
C	<u>8</u>	<u>8</u>
D	<u>6</u>	<u>8</u>
E	<u>5</u>	<u>3</u>

Total Score 39 Percentile 32

Rey-Osterrieth Complex Figure

Copy Score 32 Percentile 50 Memory Score 12 Percentile 16.67

Knox Cube

Trial I 12 Trial II 13 Average Score 12.5 CA 15.5+

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>48</u>	<u>47</u>	<u>36</u>	
1-Trial Score				<u>44</u>
Percentile	<u>43</u>	<u>56</u>	<u>37</u>	<u>97.5</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 2 Left Sensory 2 Bilateral 0 Errors

Subject: LE Dyslexic: X Normal Reader:

Age: 18 Education: College Freshman

Laterality Preference

Hand: R Foot: R Eye: R Ear: R

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>8</u>	<u>24</u>	
Comprehension	<u>11</u>	<u>63</u>	
Arithmetic	<u>9</u>	<u>37</u>	
Similarities	<u>14</u>	<u>91</u>	
Digit Span	<u>10</u>	<u>50</u>	
Vocabulary	<u>9</u>	<u>37</u>	
Verbal Score	<u>61</u>		<u>+0.25</u>
[Verbal IQ <u>104</u>]		<u>60</u>	
Digit Symbol	<u>11</u>	<u>63</u>	
Picture Completion	<u>18</u>	<u>99</u>	
Block Design	<u>16</u>	<u>98</u>	
Picture Arrangement	<u>13</u>	<u>84</u>	
Object Assembly	<u>17</u>	<u>99</u>	
Performance Score	<u>75</u>		<u>+2.16</u>
[Performance IQ <u>133</u>]		<u>99</u>	
Full Scale Score	<u>136</u>		<u>+1.16</u>
[Full Scale IQ <u>117</u>]		<u>87</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>12.9</u>	<u>58</u>	<u>78</u>	<u>+0.8</u>
Word Identification	<u>5.7</u>	<u>34</u>	<u>5</u>	<u>-1.6</u>
Word Attack	<u>4.5</u>	<u>40</u>	<u>16</u>	<u>-1.0</u>
Word Comprehension	<u>10.3</u>	<u>45</u>	<u>31</u>	<u>-0.5</u>
Passage Comprehension	<u>8.4</u>	<u>44</u>	<u>27</u>	<u>-0.6</u>
Total Reading	<u>6.8</u>	<u>39</u>	<u>14</u>	<u>-1.1</u>

Gray Oral

Total Score 31 Grade Equivalent 3.4 Comprehension Score 24.5

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>7.1</u>	<u>86</u>	<u>18</u>	<u>-0.93</u>
Spelling	<u>5.8</u>	<u>80</u>	<u>9</u>	<u>-1.33</u>
Arithmetic	<u>7.1</u>	<u>86</u>	<u>18</u>	<u>-0.93</u>

Sentence Repetition (NCCEA)

#Correct 14 Percentile 14

Boston Naming Test

#Correct Without Cue 65 #Category Cues Given 20
 #Correct Following Category Cue 5 #Phonemic Cues Given 15
 #Correct 10 and Incorrect 5 Following Phonemic Cue
 (Age Level 17.85
 Total Score 70 Z-Score -0.94 Percentile {Educ. Level 19.11

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>12</u>	<u>12</u>
B	<u>12</u>	<u>11</u>
C	<u>10</u>	<u>10</u>
D	<u>10</u>	<u>10</u>
E	<u>4</u>	<u>7</u>

Total Score 48 Percentile 70

Key-Osterrieth Complex Figure

Copy Score 34 Percentile 75 Memory Score 17 Percentile 23.61

Knox Cube

Trial I 12 Trial II 16 Average Score 14 CA 15.5+

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>37</u>	<u>40</u>	<u>32</u>	
1-Trial Score				<u>32</u>
Percentile	<u>4</u>	<u>16</u>	<u>14</u>	<u>50</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 1 Left Sensory 0 Bilateral 0 Errors

Subject: MM Dyslexic: x Normal Reader:

Age: 20 Education: High School Graduate

Laterality Preference

Hand: R Foot: R Eye: L Ear: R

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>7</u>	<u>16</u>	
Comprehension	<u>12</u>	<u>75</u>	
Arithmetic	<u>7</u>	<u>16</u>	
Similarities	<u>9</u>	<u>37</u>	
Digit Span	<u>6</u>	<u>9</u>	
Vocabulary	<u>8</u>	<u>24</u>	
Verbal Score	<u>49</u>		<u>-0.69</u>
[Verbal IQ <u>90</u>]		<u>25</u>	
Digit Symbol	<u>9</u>	<u>37</u>	
Picture Completion	<u>11</u>	<u>63</u>	
Block Design	<u>6</u>	<u>9</u>	
Picture Arrangement	<u>11</u>	<u>63</u>	
Object Assembly	<u>10</u>	<u>50</u>	
Performance Score	<u>47</u>		<u>-0.30</u>
[Performance IQ <u>95</u>]		<u>37</u>	
Full Scale Score	<u>96</u>		<u>-0.55</u>
[Full Scale IQ <u>92</u>]		<u>30</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter				
Identification	<u>3.2</u>	<u>off scale</u>	<u>off scale</u>	<u>off scale</u>
Word				
Identification	<u>2.8</u>	<u>off scale</u>	<u>off scale</u>	<u>off scale</u>
Word				
Attack	<u>2.0</u>	<u>off scale</u>	<u>off scale</u>	<u>off scale</u>
Word				
Comprehension	<u>4.2</u>	<u>34</u>	<u>6</u>	<u>-1.6</u>
Passage				
Comprehension	<u>3.1</u>	<u>off scale</u>	<u>off scale</u>	<u>off scale</u>
Total Reading	<u>3.0</u>	<u>off scale</u>	<u>off scale</u>	<u>off scale</u>

Gray Oral

Total Score 16 Grade Equivalent 1.9 Comprehension Score 12.5

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>3.5</u>	<u>67</u>	<u>1</u>	<u>-2.20</u>
Spelling	<u>2.6</u>	<u>63</u>	<u>1</u>	<u>-2.47</u>
Arithmetic	<u>4.9</u>	<u>74</u>	<u>4</u>	<u>-1.73</u>

Sentence Repetition (NCCEA)

#Correct 12 Percentile off scale (70% on aphasic scale)

Boston Naming Test

#Correct Without Cue 66 #Category Cues Given 19
 #Correct Following Category Cue 2 #Phonemic Cues Given 17
 #Correct 9 and Incorrect 8 Following Phonemic Cue
 (Age Level 14.29
 Total Score 68 Z-Score -0.47 Percentile {Educ. Level 24.52

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>11</u>	<u>10</u>
B	<u>9</u>	<u>9</u>
C	<u>7</u>	<u>8</u>
D	<u>9</u>	<u>9</u>
E	<u>4</u>	<u>4</u>

Total Score 40 Percentile 36

Rey-Osterrieth Complex Figure

Copy Score 32 Percentile 50 Memory Score 17 Percentile 23.61

Knox Cube

Trial I 11 Trial II 11 Average Score 11 CA 12.5

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>43</u>	<u>47</u>	<u>35</u>	
1-Trial Score				<u>30</u>
Percentile	<u>17</u>	<u>56</u>	<u>32</u>	<u>45</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 1 Left Sensory 0 Bilateral 0 Errors

Subject: SC Dyslexic: x Normal Reader:

Age: 21 Education: College Senior

Laterality Preference

Hand: R Foot: R Eye: R Ear: R/L

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>10</u>	<u>50</u>	
Comprehension	<u>13</u>	<u>84</u>	
Arithmetic	<u>10</u>	<u>50</u>	
Similarities	<u>13</u>	<u>84</u>	
Digit Span	<u>7</u>	<u>16</u>	
Vocabulary	<u>11</u>	<u>63</u>	
Verbal Score	<u>64</u>		<u>+0.30</u>
[Verbal IQ <u>104</u>]		<u>60</u>	
Digit Symbol	<u>10</u>	<u>50</u>	
Picture Completion	<u>12</u>	<u>75</u>	
Block Design	<u>16</u>	<u>93</u>	
Picture Arrangement	<u>12</u>	<u>75</u>	
Object Assembly	<u>13</u>	<u>84</u>	
Performance Score	<u>63</u>		<u>+1.03</u>
[Performance IQ <u>116</u>]		<u>86</u>	
Full Scale Score	<u>127</u>		<u>+0.66</u>
[Full Scale IQ <u>110</u>]		<u>75</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>12.9</u>	<u>58</u>	<u>78</u>	<u>+0.8</u>
Word Identification	<u>7.4</u>	<u>40</u>	<u>15</u>	<u>-1.0</u>
Word Attack	<u>4.1</u>	<u>38</u>	<u>12</u>	<u>-1.2</u>
Word Comprehension	<u>12.9</u>	<u>48</u>	<u>43</u>	<u>-0.2</u>
Passage Comprehension	<u>8.7</u>	<u>44</u>	<u>28</u>	<u>-0.6</u>
Total Reading	<u>8.6</u>	<u>43</u>	<u>24</u>	<u>-0.7</u>

Gray Oral

Total Score 42 Grade Equivalent 5.0 Comprehension Score 25

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>8.1</u>	<u>90</u>	<u>25</u>	<u>-0.67</u>
Spelling	<u>6.7</u>	<u>83</u>	<u>13</u>	<u>-1.13</u>
Arithmetic	<u>6.3</u>	<u>81</u>	<u>10</u>	<u>-1.27</u>

Sentence Repetition (NCCEA)

#Correct 11 Percentile off scale (62% on aphasic scale)

Boston Naming Test

#Correct Without Cue 72 #Category Cues Given 13
 #Correct Following Category Cue 2 #Phonemic Cues Given 11
 #Correct 7 and Incorrect 4 Following Phonemic Cue
 Total Score 74 Z-Score -0.31 Percentile { Age Level 25
 Educ. Level 25

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>12</u>	<u>12</u>
B	<u>11</u>	<u>11</u>
C	<u>11</u>	<u>10</u>
D	<u>10</u>	<u>10</u>
E	<u>7</u>	<u>7</u>

Total Score 51 Percentile 81

Rey-Osterrieth Complex Figure

Copy Score 32 Percentile 50 Memory Score 6 Percentile 8.33

Knox Cube

Trial I 14 Trial II 15 Average Score 14.5 CA 15.5+

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>44</u>	<u>40</u>	<u>31</u>	
1-Trial Score				<u>35</u>
Percentile	<u>23</u>	<u>16</u>	<u>11.5</u>	<u>67</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 2 Left Sensory 1 Bilateral 0 Errors

Subject: TC Dyslexic: X Normal Reader:

Age: 19 Education: College Sophomore

Laterality Preference

Hand: R Foot: R(L) Eye: R Ear: L/R

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>11</u>	<u>63</u>	
Comprehension	<u>16</u>	<u>98</u>	
Arithmetic	<u>12</u>	<u>75</u>	
Similarities	<u>13</u>	<u>84</u>	
Digit Span	<u>9</u>	<u>37</u>	
Vocabulary	<u>12</u>	<u>75</u>	
Verbal Score	<u>73</u>		<u>+1.05</u>
[Verbal IQ <u>115</u>]		<u>84</u>	
Digit Symbol	<u>10</u>	<u>50</u>	
Picture Completion	<u>13</u>	<u>84</u>	
Block Design	<u>11</u>	<u>63</u>	
Picture Arrangement	<u>11</u>	<u>63</u>	
Object Assembly	<u>13</u>	<u>84</u>	
Performance Score	<u>58</u>		<u>+0.73</u>
[Performance IQ <u>111</u>]		<u>77</u>	
Full Scale Score	<u>131</u>		<u>+0.96</u>
[Full Scale IQ <u>114</u>]		<u>82</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>12.9</u>	<u>58</u>	<u>78</u>	<u>+0.8</u>
Word Identification	<u>9.9</u>	<u>45</u>	<u>30</u>	<u>-0.5</u>
Word Attack	<u>4.9</u>	<u>41</u>	<u>19</u>	<u>-0.9</u>
Word Comprehension	<u>12.9</u>	<u>57</u>	<u>77</u>	<u>+0.7</u>
Passage Comprehension	<u>12.2</u>	<u>49</u>	<u>47</u>	<u>-0.1</u>
Total Reading	<u>11.3</u>	<u>48</u>	<u>42</u>	<u>-0.2</u>

Gray Oral

Total Score 52 Grade Equivalent 6.8 Comprehension Score 33.5

<u>WRAT</u>	grade level	standard score	percentile	z-score
Reading	<u>9.1</u>	<u>96</u>	<u>39</u>	<u>-0.27</u>
Spelling	<u>7.8</u>	<u>90</u>	<u>25</u>	<u>-0.67</u>
Arithmetic	<u>9.0</u>	<u>95</u>	<u>37</u>	<u>-0.33</u>

Sentence Repetition (NCCEA)

#Correct 16 Percentile 57

Boston Naming Test

#Correct Without Cue 79 #Category Cues Given 6
 #Correct Following Category Cue 2 #Phonemic Cues Given 4
 #Correct 3 and Incorrect 1 Following Phonemic Cue
 (Age Level 77.78
 Total Score 81 Z-Score +0.78 Percentile {Educ. Level 75

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>12</u>	<u>11</u>
B	<u>10</u>	<u>10</u>
C	<u>10</u>	<u>10</u>
D	<u>11</u>	<u>9</u>
E	<u>2</u>	<u>5</u>

Total Score 45 Percentile 55

Rey-Osterrieth Complex Figure

Copy Score 35 Percentile 87.5 Memory Score 25 Percentile 65

Knox Cube

Trial I 12 Trial II 12 Average Score 12 CA 14.5/15.5

<u>Purdue Pegboard</u>	Right	Left	Both	Assembly
3-Trial Score	<u>55</u>	<u>50</u>	<u>43</u>	
1-Trial Score				<u>48</u>
Percentile	<u>84</u>	<u>73</u>	<u>87</u>	<u>99+</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 0 Left Sensory 0 Bilateral 0 Errors

Subject: LI Dyslexic: x Normal Reader:

Age: 18 Education: College Freshman

Laterality Preference

Hand: R Foot: R(L) Eye: L(R) Ear: L

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>7</u>	<u>16</u>	
Comprehension	<u>10</u>	<u>50</u>	
Arithmetic	<u>7</u>	<u>16</u>	
Similarities	<u>12</u>	<u>75</u>	
Digit Span	<u>6</u>	<u>9</u>	
Vocabulary	<u>9</u>	<u>37</u>	
Verbal Score	<u>51</u>		<u>-0.26</u>
[Verbal IQ <u>96</u>]		<u>40</u>	
Digit Symbol	<u>10</u>	<u>50</u>	
Picture Completion	<u>12</u>	<u>75</u>	
Block Design	<u>7</u>	<u>16</u>	
Picture Arrangement	<u>9</u>	<u>37</u>	
Object Assembly	<u>9</u>	<u>37</u>	
Performance Score	<u>47</u>		<u>-0.16</u>
[Performance IQ <u>98</u>]		<u>45</u>	
Full Scale Score	<u>98</u>		<u>-0.23</u>
[Full Scale IQ <u>97</u>]		<u>42</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>12.9</u>	<u>58</u>	<u>78</u>	<u>+0.8</u>
Word Identification	<u>6.4</u>	<u>36</u>	<u>8</u>	<u>-1.4</u>
Word Attack	<u>3.7</u>	<u>37</u>	<u>9</u>	<u>-1.3</u>
Word Comprehension	<u>7.1</u>	<u>40</u>	<u>17</u>	<u>-1.0</u>
Passage Comprehension	<u>9.2</u>	<u>45</u>	<u>32</u>	<u>-0.5</u>
Total Reading	<u>6.6</u>	<u>39</u>	<u>13</u>	<u>-1.1</u>

Gray Oral

Total Score 45 Grade Equivalent 5.6 Comprehension Score 36.5

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>7.5</u>	<u>89</u>	<u>23</u>	<u>-0.73</u>
Spelling	<u>6.3</u>	<u>83</u>	<u>13</u>	<u>-1.13</u>
Arithmetic	<u>6.3</u>	<u>83</u>	<u>13</u>	<u>-1.13</u>

Sentence Repetition (NCCEA)

#Correct 13 Percentile 4

Boston Naming Test

#Correct Without Cue 64 #Category Cues Given 21
 #Correct Following Category Cue 3 #Phonemic Cues Given 18
 #Correct 8 and Incorrect 10 Following Phonemic Cue
 Total Score 67 Z-Score -1.41 Percentile 12.5 (Age Level)
 Educ. Level 10.3

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>11</u>	<u>10</u>
B	<u>9</u>	<u>8</u>
C	<u>5</u>	<u>6</u>
D	<u>5</u>	<u>4</u>
E	<u>0</u>	<u>2</u>

Total Score 30 Percentile 13.33

Rey-Osterrieth Complex Figure

Copy Score 34 Percentile 75 Memory Score 17 Percentile 13.6

Knox Cube

Trial I 11 Trial II 12 Average Score 11.5 CA 13.5

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>38</u>	<u>32</u>	<u>27</u>	
1-Trial Score				<u>27</u>
Percentile	<u>5.5</u>	<u>2</u>	<u>2.5</u>	<u>19</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 1 Left Sensory 0 Bilateral 0 Errors

Subject: FR Dyslexic: X Normal Reader:

Age: 19 Education: College Freshman

Laterality Preference

Hand: R Foot: R Eye: R/L Ear: R

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>10</u>	<u>50</u>	
Comprehension	<u>12</u>	<u>75</u>	
Arithmetic	<u>12</u>	<u>75</u>	
Similarities	<u>12</u>	<u>75</u>	
Digit Span	<u>7</u>	<u>16</u>	
Vocabulary	<u>8</u>	<u>24</u>	
Verbal Score	<u>61</u>		<u>+0.25</u>
[Verbal IQ <u>104</u>]		<u>60</u>	
Digit Symbol	<u>10</u>	<u>50</u>	
Picture Completion	<u>12</u>	<u>75</u>	
Block Design	<u>13</u>	<u>84</u>	
Picture Arrangement	<u>12</u>	<u>75</u>	
Object Assembly	<u>12</u>	<u>75</u>	
Performance Score	<u>59</u>		<u>+0.81</u>
[Performance IQ <u>113</u>]		<u>81</u>	
Full Scale Score	<u>120</u>		<u>+0.53</u>
[Full Scale IQ <u>108</u>]		<u>70</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>6.2</u>	<u>50</u>	<u>50</u>	<u>0.0</u>
Word Identification	<u>2.5</u>	<u>off scale</u>	<u>off scale</u>	<u>off scale</u>
Word Attack	<u>1.8</u>	<u>off scale</u>	<u>off scale</u>	<u>off scale</u>
Word Comprehension	<u>3.8</u>	<u>34</u>	<u>5</u>	<u>-1.6</u>
Passage Comprehension	<u>2.4</u>	<u>off scale</u>	<u>off scale</u>	<u>off scale</u>
Total Reading	<u>2.9</u>	<u>off scale</u>	<u>off scale</u>	<u>off scale</u>

Gray Oral

Total Score 10 Grade Equivalent 1.6 Comprehension Score 22.5

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>3.5</u>	<u>68</u>	<u>2</u>	<u>-2.13</u>
Spelling	<u>3.3</u>	<u>67</u>	<u>1</u>	<u>-2.20</u>
Arithmetic	<u>6.5</u>	<u>83</u>	<u>13</u>	<u>-1.13</u>

Sentence Repetition (NCCEA)

#Correct 11 Percentile off scale (62% on aphasic scale)

Boston Naming Test

#Correct Without Cue 54 #Category Cues Given 31
 #Correct Following Category Cue 6 #Phonemic Cues Given 25
 #Correct 13 and Incorrect 12 Following Phonemic Cue
 (Age Level 0.0
 Total Score 60 Z-Score -2.5 Percentile Educ. Level 4.4

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>11</u>	<u>11</u>
B	<u>11</u>	<u>10</u>
C	<u>7</u>	<u>10</u>
D	<u>9</u>	<u>9</u>
E	<u>6</u>	<u>5</u>

Total Score 44 Percentile 50

Rey-Osterrieth Complex Figure

Copy Score 34 Percentile 75 Memory Score 22 Percentile 50

Knox Cube

Trial I 13 Trial II 13 Average Score 13 CA 15.5+

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>42</u>	<u>46</u>	<u>38</u>	
1-Trial Score				<u>39</u>
Percentile	<u>18</u>	<u>48</u>	<u>52</u>	<u>79</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 7 Left Sensory 2 Bilateral 0 Errors

Subject: AE Dyslexic: X Normal Reader:

Age: 21 Education: College Freshman

Laterality Preference

Hand: R Foot: L/R Eye: R Ear: R

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>10</u>	<u>50</u>	
Comprehension	<u>10</u>	<u>50</u>	
Arithmetic	<u>7</u>	<u>16</u>	
Similarities	<u>10</u>	<u>50</u>	
Digit Span	<u>7</u>	<u>16</u>	
Vocabulary	<u>7</u>	<u>16</u>	
Verbal Score	<u>51</u>		<u>-0.56</u>
[Verbal IQ <u>92</u>]		<u>30</u>	
Digit Symbol	<u>10</u>	<u>50</u>	
Picture Completion	<u>13</u>	<u>84</u>	
Block Design	<u>11</u>	<u>63</u>	
Picture Arrangement	<u>9</u>	<u>37</u>	
Object Assembly	<u>11</u>	<u>63</u>	
Performance Score	<u>54</u>		<u>+0.28</u>
[Performance IQ <u>104</u>]		<u>60</u>	
Full Scale Score	<u>105</u>		<u>-0.20</u>
[Full Scale IQ <u>97</u>]		<u>42</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>12.9</u>	<u>58</u>	<u>78</u>	<u>+0.8</u>
Word Identification	<u>6.4</u>	<u>35</u>	<u>7</u>	<u>-1.5</u>
Word Attack	<u>3.9</u>	<u>38</u>	<u>11</u>	<u>-1.2</u>
Word Comprehension	<u>5.8</u>	<u>38</u>	<u>12</u>	<u>-1.2</u>
Passage Comprehension	<u>8.4</u>	<u>44</u>	<u>27</u>	<u>-0.6</u>
Total Reading	<u>6.3</u>	<u>38</u>	<u>11</u>	<u>-1.2</u>

Gray Oral

Total Score 55 Grade Equivalent 7.5 Comprehension Score 29.5

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>6.6</u>	<u>82</u>	<u>12</u>	<u>-1.2</u>
Spelling	<u>6.1</u>	<u>80</u>	<u>9</u>	<u>-1.33</u>
Arithmetic	<u>6.1</u>	<u>80</u>	<u>9</u>	<u>-1.33</u>

Sentence Repetition (NCCEA)

#Correct 13 Percentile 4

Boston Naming Test

#Correct Without Cue 67 #Category Cues Given 18
 #Correct Following Category Cue 3 #Phonemic Cues Given 15
 #Correct 8 and Incorrect 7 Following Phonemic Cue
 (Age Level 17.86
 Total Score 70 Z-Score -0.94 Percentile 19.11 Educ. Level 19.11

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>11</u>	<u>11</u>
B	<u>11</u>	<u>10</u>
C	<u>8</u>	<u>10</u>
D	<u>10</u>	<u>9</u>
E	<u>5</u>	<u>5</u>

Total Score 45 Percentile 58.33

Rey-Osterrieth Complex Figure

Copy Score 34 Percentile 75 Memory Score 23 Percentile 55

Knox Cube

Trial I 10 Trial II 12 Average Score 11 CA 12.5

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>43</u>	<u>37</u>	<u>30</u>	
1-Trial Score				<u>34</u>
Percentile	<u>15</u>	<u>7.5</u>	<u>9</u>	<u>57</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 2 Left Sensory 3 Bilateral 0 Errors

Subject: AR Dyslexic: Normal Reader: X

Age: 18 Education: College Sophomore

Laterality Preference

Hand: R Foot: R Eye: R(L) Ear: L(R)

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>12</u>	<u>75</u>	
Comprehension	<u>14</u>	<u>91</u>	
Arithmetic	<u>17</u>	<u>99</u>	
Similarities	<u>14</u>	<u>91</u>	
Digit Span	<u>19</u>	<u>99</u>	
Vocabulary	<u>10</u>	<u>50</u>	
Verbal Score	<u>86</u>		<u>+1.93</u>
[Verbal IQ <u>128</u>]		<u>97</u>	
Digit Symbol	<u>17</u>	<u>99</u>	
Picture Completion	<u>16</u>	<u>98</u>	
Block Design	<u>17</u>	<u>99</u>	
Picture Arrangement	<u>15</u>	<u>95</u>	
Object Assembly	<u>16</u>	<u>98</u>	
Performance Score	<u>81</u>		<u>+2.67</u>
[Performance IQ <u>141</u>]		<u>99.7</u>	
Full Scale Score	<u>167</u>		<u>+2.40</u>
[Full Scale IQ <u>136</u>]		<u>99.2</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter				
Identification	<u>12.9</u>	<u>58</u>	<u>78</u>	<u>+0.8</u>
Word				
Identification	<u>12.9</u>	<u>57</u>	<u>75</u>	<u>+0.7</u>
Word				
Attack	<u>12.9</u>	<u>56</u>	<u>74</u>	<u>+0.6</u>
Word				
Comprehension	<u>12.9</u>	<u>65</u>	<u>93</u>	<u>+1.5</u>
Passage				
Comprehension	<u>12.9</u>	<u>60</u>	<u>85</u>	<u>+1.0</u>
Total Reading	<u>12.9+</u>	<u>64</u>	<u>92</u>	<u>+1.4</u>

Gray Oral

Total Score 84 Grade Equivalent 12.0+ Comprehension Score 41.5

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>13.0</u>	<u>115</u>	<u>84</u>	<u>+1.0</u>
Spelling	<u>11.6</u>	<u>108</u>	<u>70</u>	<u>+0.5</u>
Arithmetic	<u>14.9</u>	<u>124</u>	<u>95</u>	<u>+1.6</u>

Sentence Repetition (NCCEA)

#Correct 17 Percentile 71

Boston Naming Test

#Correct Without Cue 74 #Category Cues Given 11
 #Correct Following Category Cue 3 #Phonemic Cues Given 8
 #Correct 4 and Incorrect 4 Following Phonemic Cue
 (Age Level 43.75
 Total Score 77 Z-Score +0.16 Percentile } Educ. Level 54.17

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>12</u>	<u>12</u>
B	<u>12</u>	<u>11</u>
C	<u>10</u>	<u>11</u>
D	<u>12</u>	<u>11</u>
E	<u>9</u>	<u>10</u>

Total Score 55 Percentile 95

Rey-Osterrieth Complex Figure

Copy Score 35 Percentile 87.5 Memory Score 25.5 Percentile 67.5

Knox Cube

Trial I 16 Trial II 17 Average Score 16.5 CA 15.5+

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>47</u>	<u>43</u>	<u>36</u>	
1-Trial Score				<u>38</u>
Percentile	<u>38</u>	<u>32</u>	<u>38</u>	<u>81</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 0 Left Sensory 0 Bilateral 0 Errors

Subject: EF Dyslexic: Normal Reader: x

Age: 18 Education: High School Graduate

Laterality Preference

Hand: R Foot: R Eye: R Ear: R

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>10</u>	<u>50</u>	
Comprehension	<u>14</u>	<u>91</u>	
Arithmetic	<u>9</u>	<u>37</u>	
Similarities	<u>13</u>	<u>84</u>	
Digit Span	<u>10</u>	<u>50</u>	
Vocabulary	<u>12</u>	<u>75</u>	
Verbal Score	<u>68</u>		<u>+0.72</u>
[Verbal IQ <u>111</u>]		<u>77</u>	
Digit Symbol	<u>11</u>	<u>63</u>	
Picture Completion	<u>14</u>	<u>91</u>	
Block Design	<u>11</u>	<u>63</u>	
Picture Arrangement	<u>8</u>	<u>24</u>	
Object Assembly	<u>14</u>	<u>91</u>	
Performance Score	<u>58</u>		<u>+0.72</u>
[Performance IQ <u>111</u>]		<u>77</u>	
Full Scale Score	<u>126</u>		<u>+0.77</u>
[Full Scale IQ <u>111</u>]		<u>77</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>6.2</u>	<u>50</u>	<u>50</u>	<u>0.0</u>
Word Identification	<u>12.9</u>	<u>57</u>	<u>75</u>	<u>+0.7</u>
Word Attack	<u>12.3</u>	<u>50</u>	<u>50</u>	<u>0.0</u>
Word Comprehension	<u>11.9</u>	<u>48</u>	<u>43</u>	<u>-0.20</u>
Passage Comprehension	<u>12.9</u>	<u>55</u>	<u>68</u>	<u>+0.5</u>
Total Reading	<u>12.9+</u>	<u>51</u>	<u>54</u>	<u>+0.1</u>

Gray Oral

Total Score 81 Grade Equivalent 12+ Comprehension Score 39.5

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>15.3</u>	<u>126</u>	<u>96</u>	<u>+1.73</u>
Spelling	<u>8.1</u>	<u>91</u>	<u>27</u>	<u>-0.60</u>
Arithmetic	<u>6.3</u>	<u>82</u>	<u>12</u>	<u>-1.20</u>

Sentence Repetition (NCCEA)

#Correct 17 Percentile 70

Boston Naming Test

#Correct Without Cue 79 #Category Cues Given 6
 #Correct Following Category Cue 2 #Phonemic Cues Given 4
 #Correct 3 and Incorrect 1 Following Phonemic Cue
 (Age Level 77.8
 Total Score 81 Z-Score +0.55 Percentile {Educ. Level 87.5

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>11</u>	<u>10</u>
B	<u>8</u>	<u>9</u>
C	<u>9</u>	<u>8</u>
D	<u>9</u>	<u>9</u>
E	<u>2</u>	<u>4</u>

Total Score 39 Percentile 32

Rey-Osterrieth Complex Figure

Copy Score 33 Percentile 62.5 Memory Score 23 Percentile 55

Knox Cube

Trial I 16 Trial II 15 Average Score 15.5 CA 15.5+

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>48</u>	<u>50</u>	<u>38</u>	
1-Trial Score				<u>36</u>
Percentile	<u>43</u>	<u>73</u>	<u>52</u>	<u>72</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 4 Left Sensory 3 Bilateral 0 Errors

Subject: AP Dyslexic: Normal Reader: x

Age: 19 Education: College Sophomore

Laterality Preference

Hand: R Foot: R(L) Eye: R Ear: L

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>15</u>	<u>95</u>	
Comprehension	<u>15</u>	<u>95</u>	
Arithmetic	<u>17</u>	<u>99</u>	
Similarities	<u>14</u>	<u>91</u>	
Digit Span	<u>14</u>	<u>91</u>	
Vocabulary	<u>14</u>	<u>91</u>	
Verbal Score	<u>89</u>		<u>+2.13</u>
[Verbal IQ <u>131</u>]		<u>98</u>	
Digit Symbol	<u>15</u>	<u>95</u>	
Picture Completion	<u>14</u>	<u>91</u>	
Block Design	<u>13</u>	<u>84</u>	
Picture Arrangement	<u>13</u>	<u>84</u>	
Object Assembly	<u>16</u>	<u>98</u>	
Performance Score	<u>71</u>		<u>+1.82</u>
[Performance IQ <u>128</u>]		<u>97</u>	
Full Scale Score	<u>160</u>		<u>+2.15</u>
[Full Scale IQ <u>132</u>]		<u>98</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter				
Identification	<u>12.9</u>	<u>58</u>	<u>78</u>	<u>+0.8</u>
Word				
Identification	<u>12.9</u>	<u>69</u>	<u>97</u>	<u>+1.9</u>
Word				
Attack	<u>12.9</u>	<u>60</u>	<u>83</u>	<u>+1.0</u>
Word				
Comprehension	<u>12.9</u>	<u>62</u>	<u>89</u>	<u>+1.2</u>
Passage				
Comprehension	<u>12.9</u>	<u>57</u>	<u>77</u>	<u>+0.7</u>
Total Reading	<u>12.9+</u>	<u>71</u>	<u>98</u>	<u>+2.1</u>

Gray Oral

Total Score 100 Grade Equivalent 12.0+ Comprehension Score 41

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>16.5</u>	<u>132</u>	<u>98</u>	<u>+2.13</u>
Spelling	<u>14.0</u>	<u>120</u>	<u>91</u>	<u>+1.33</u>
Arithmetic	<u>14.4</u>	<u>124</u>	<u>95</u>	<u>+1.60</u>

Sentence Repetition (NCCEA)

#Correct 16 Percentile 56

Boston Naming Test

#Correct Without Cue 79 #Category Cues Given 6
 #Correct Following Category Cue 4 #Phonemic Cues Given 2
 #Correct 1 and Incorrect 1 Following Phonemic Cue

Total Score 83 Z-Score +1.09 Percentile (Age Level 88.9 Educ. Level 87.5)

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>12</u>	<u>12</u>
B	<u>11</u>	<u>11</u>
C	<u>10</u>	<u>11</u>
D	<u>11</u>	<u>11</u>
E	<u>9</u>	<u>10</u>

Total Score 53 Percentile 87

Rey-Osterrieth Complex Figure

Copy Score 35 Percentile 87.5 Memory Score 21.5 Percentile 46.88

Knox Cube

Trial I 15 Trial II 16 Average Score 15.5 CA 15.5+

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>43</u>	<u>43</u>	<u>35</u>	
1-Trial Score				<u>41</u>
Percentile	<u>17</u>	<u>32</u>	<u>31</u>	<u>91</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 0 Left Sensory 0 Bilateral 0 Errors

Subject: HL Dyslexic: Normal Reader: x

Age: 19 Education: College Sophomore

Laterality Preference

Hand: R Foot: R Eye: R Ear: R

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>14</u>	<u>91</u>	
Comprehension	<u>12</u>	<u>75</u>	
Arithmetic	<u>13</u>	<u>84</u>	
Similarities	<u>12</u>	<u>75</u>	
Digit Span	<u>14</u>	<u>91</u>	
Vocabulary	<u>12</u>	<u>75</u>	
Verbal Score	<u>77</u>		<u>+1.32</u>
[Verbal IQ <u>119</u>]		<u>90</u>	
Digit Symbol	<u>14</u>	<u>91</u>	
Picture Completion	<u>16</u>	<u>98</u>	
Block Design	<u>14</u>	<u>91</u>	
Picture Arrangement	<u>12</u>	<u>75</u>	
Object Assembly	<u>10</u>	<u>50</u>	
Performance Score	<u>66</u>		<u>+2.08</u>
[Performance IQ <u>122</u>]		<u>93</u>	
Full Scale Score	<u>143</u>		<u>+1.44</u>
[Full Scale IQ <u>122</u>]		<u>93</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>12.9</u>	<u>58</u>	<u>78</u>	<u>+0.8</u>
Word Identification	<u>12.9</u>	<u>55</u>	<u>70</u>	<u>+0.5</u>
Word Attack	<u>12.9</u>	<u>50</u>	<u>50</u>	<u>0.0</u>
Word Comprehension	<u>12.9</u>	<u>59</u>	<u>82</u>	<u>+0.9</u>
Passage Comprehension	<u>12.2</u>	<u>49</u>	<u>47</u>	<u>-0.1</u>
Total Reading	<u>12.9+</u>	<u>55</u>	<u>68</u>	<u>+0.5</u>

Gray Oral

Total Score 85 Grade Equivalent 12.0+ Comprehension Score 37.5

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>13.0</u>	<u>115</u>	<u>84</u>	<u>+1.0</u>
Spelling	<u>10.2</u>	<u>101</u>	<u>53</u>	<u>+0.7</u>
Arithmetic	<u>8.0</u>	<u>91</u>	<u>27</u>	<u>-0.6</u>

Sentence Repetition (NCCEA)

#Correct 13 Percentile 4

Boston Naming Test

#Correct Without Cue 71 #Category Cues Given 14
 #Correct Following Category Cue 3 #Phonemic Cues Given 11
 #Correct 7 and Incorrect 4 Following Phonemic Cue
 (Age Level 25
 Total Score 74 Z-Score -0.31 Percentile 25 Educ. Level 25

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>12</u>	<u>12</u>
B	<u>12</u>	<u>11</u>
C	<u>11</u>	<u>11</u>
D	<u>10</u>	<u>11</u>
E	<u>8</u>	<u>10</u>

Total Score 53 Percentile 87

Rey-Osterrieth Complex Figure

Copy Score 34 Percentile 75 Memory Score 12 Percentile 8.64

Knox Cube

Trial I 14 Trial II 16 Average Score 15 CA 15.5+

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>49</u>	<u>48</u>	<u>35</u>	
1-Trial Score				<u>38</u>
Percentile	<u>47</u>	<u>62</u>	<u>31</u>	<u>82</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 1 Left Sensory 0 Bilateral 0 Errors

Subject: HW Dyslexic: Normal Reader: x

Age: 26 Education: College Graduate

Laterality Preference

Hand: R Foot: R Eye: R Ear: R

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>12</u>	<u>75</u>	
Comprehension	<u>13</u>	<u>84</u>	
Arithmetic	<u>13</u>	<u>84</u>	
Similarities	<u>11</u>	<u>63</u>	
Digit Span	<u>7</u>	<u>16</u>	
Vocabulary	<u>10</u>	<u>50</u>	
Verbal Score	<u>66</u>		<u>+0.35</u>
[Verbal IQ <u>105</u>]		<u>63</u>	
Digit Symbol	<u>14</u>	<u>91</u>	
Picture Completion	<u>9</u>	<u>37</u>	
Block Design	<u>9</u>	<u>37</u>	
Picture Arrangement	<u>12</u>	<u>75</u>	
Object Assembly	<u>7</u>	<u>16</u>	
Performance Score	<u>51</u>		<u>+0.12</u>
[Performance IQ <u>102</u>]		<u>55</u>	
Full Scale Score	<u>117</u>		<u>+0.27</u>
[Full Scale IQ <u>104</u>]		<u>60</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>12.9</u>	<u>58</u>	<u>78</u>	<u>+0.8</u>
Word Identification	<u>12.9</u>	<u>55</u>	<u>70</u>	<u>+0.5</u>
Word Attack	<u>10.1</u>	<u>48</u>	<u>43</u>	<u>-0.2</u>
Word Comprehension	<u>12.9</u>	<u>57</u>	<u>77</u>	<u>+0.7</u>
Passage Comprehension	<u>12.9</u>	<u>51</u>	<u>55</u>	<u>+0.1</u>
Total Reading	<u>12.9+</u>	<u>55</u>	<u>68</u>	<u>+0.5</u>

Gray Oral

Total Score 81 Grade Equivalent 12.0 Comprehension Score 28.5

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>14.7</u>	<u>129</u>	<u>97</u>	<u>+1.93</u>
Spelling	<u>10.8</u>	<u>109</u>	<u>73</u>	<u>+0.60</u>
Arithmetic	<u>6.7</u>	<u>88</u>	<u>21</u>	<u>-0.80</u>

Sentence Repetition (NCCEA)

#Correct 15 Percentile 34

Boston Naming Test

#Correct Without Cue 71 #Category Cues Given 14
 #Correct Following Category Cue 5 #Phonemic Cues Given 9
 #Correct 3 and Incorrect 6 Following Phonemic Cue
 (Age Level 37.5
 Total Score 76 Z-Score 0.0 Percentile 50 Educ. Level 50

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>11</u>	<u>12</u>
B	<u>12</u>	<u>11</u>
C	<u>10</u>	<u>10</u>
D	<u>11</u>	<u>10</u>
E	<u>7</u>	<u>7</u>

Total Score 51 Percentile 81

Rey-Osterrieth Complex Figure

Copy Score 33 Percentile 62.5 Memory Score 17 Percentile 23.61

Knox Cube

Trial I 12 Trial II 11 Average Score 11.5 CA 13.5

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>43</u>	<u>44</u>	<u>35</u>	
1-Trial Score				<u>34</u>
Percentile	<u>23</u>	<u>38</u>	<u>31</u>	<u>61</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 1 Left Sensory 0 Bilateral 0 Errors

Subject: GY Dyslexic: Normal Reader: X

Age: 18 Education: High School Graduate

Laterality Preference

Hand: R Foot: R Eye: R Ear: R(L)

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>10</u>	<u>50</u>	
Comprehension	<u>10</u>	<u>50</u>	
Arithmetic	<u>8</u>	<u>24</u>	
Similarities	<u>9</u>	<u>37</u>	
Digit Span	<u>10</u>	<u>50</u>	
Vocabulary	<u>10</u>	<u>50</u>	
Verbal Score	<u>57</u>		<u>+0.02</u>
[Verbal IQ <u>100</u>]		<u>50</u>	
Digit Symbol	<u>10</u>	<u>50</u>	
Picture Completion	<u>11</u>	<u>63</u>	
Block Design	<u>12</u>	<u>75</u>	
Picture Arrangement	<u>11</u>	<u>63</u>	
Object Assembly	<u>11</u>	<u>63</u>	
Performance Score	<u>55</u>		<u>+0.47</u>
[Performance IQ <u>107</u>]		<u>68</u>	
Full Scale Score	<u>112</u>		<u>+0.21</u>
[Full Scale IQ <u>103</u>]		<u>58</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>6.2</u>	<u>50</u>	<u>50</u>	<u>0.0</u>
Word Identification	<u>12.9</u>	<u>50</u>	<u>50</u>	<u>0.0</u>
Word Attack	<u>12.9</u>	<u>51</u>	<u>55</u>	<u>+0.1</u>
Word Comprehension	<u>12.9</u>	<u>52</u>	<u>57</u>	<u>+0.2</u>
Passage Comprehension	<u>12.9</u>	<u>53</u>	<u>62</u>	<u>+0.3</u>
Total Reading	<u>12.3</u>	<u>50</u>	<u>50</u>	<u>0.0</u>

Gray Oral

Total Score 83 Grade Equivalent 12.0 + Comprehension Score 36

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>10.8</u>	<u>106</u>	<u>61</u>	<u>+0.40</u>
Spelling	<u>8.7</u>	<u>95</u>	<u>34</u>	<u>-0.33</u>
Arithmetic	<u>6.3</u>	<u>82</u>	<u>12</u>	<u>-1.20</u>

Sentence Repetition (NCCEA)

#Correct 13 Percentile 4

Boston Naming Test

#Correct Without Cue 72 #Category Cues Given 13
 #Correct Following Category Cue 3 #Phonemic Cues Given 9
 #Correct 6 and Incorrect 3 Following Phonemic Cue
 (Age Level 81)
 Total Score 75 Z-Score +0.08 Percentile {Educ. Level 55

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>12</u>	<u>10</u>
B	<u>9</u>	<u>9</u>
C	<u>9</u>	<u>8</u>
D	<u>8</u>	<u>9</u>
E	<u>2</u>	<u>4</u>

Total Score 40 Percentile 36

Rey-Osterrieth Complex Figure

Copy Score 34 Percentile 75 Memory Score 19 Percentile 31.25

Knox Cube

Trial I 13 Trial II 13 Average Score 13 CA 15.5+

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>46</u>	<u>48</u>	<u>36</u>	
1-Trial Score				<u>36</u>
Percentile	<u>32</u>	<u>62</u>	<u>37</u>	<u>72</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 1 Left Sensory 0 Bilateral 0 Errors

Subject: TR Dyslexic: Normal Reader: x

Age: 22 Education: College Senior

Laterality Preference

Hand: R Foot: R(L) Eye: R(L) Ear: R(L)

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>14</u>	<u>91</u>	
Comprehension	<u>16</u>	<u>98</u>	
Arithmetic	<u>17</u>	<u>99</u>	
Similarities	<u>15</u>	<u>95</u>	
Digit Span	<u>15</u>	<u>95</u>	
Vocabulary	<u>16</u>	<u>98</u>	
Verbal Score	<u>93</u>		<u>+2.20</u>
[Verbal IQ <u>133</u>]		<u>99</u>	
Digit Symbol	<u>13</u>	<u>84</u>	
Picture Completion	<u>14</u>	<u>91</u>	
Block Design	<u>17</u>	<u>99</u>	
Picture Arrangement	<u>14</u>	<u>91</u>	
Object Assembly	<u>11</u>	<u>63</u>	
Performance Score	<u>69</u>		<u>+1.53</u>
[Performance IQ <u>124</u>]		<u>95</u>	
Full Scale Score	<u>162</u>		<u>+2.02</u>
[Full Scale IQ <u>131</u>]		<u>98</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>12.9</u>	<u>58</u>	<u>78</u>	<u>+0.8</u>
Word Identification	<u>12.9</u>	<u>57</u>	<u>75</u>	<u>+0.7</u>
Word Attack	<u>12.9</u>	<u>56</u>	<u>74</u>	<u>+0.6</u>
Word Comprehension	<u>12.9</u>	<u>73</u>	<u>99</u>	<u>+2.3</u>
Passage Comprehension	<u>12.9</u>	<u>69</u>	<u>97</u>	<u>+1.9</u>
Total Reading	<u>12.9+</u>	<u>73</u>	<u>99+</u>	<u>+2.3</u>

Gray Oral

Total Score 87 Grade Equivalent 12.0 + Comprehension Score 41.5

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>17.1</u>	<u>134</u>	<u>99</u>	<u>+2.27</u>
Spelling	<u>12.4</u>	<u>111</u>	<u>77</u>	<u>+0.73</u>
Arithmetic	<u>7.7</u>	<u>88</u>	<u>21</u>	<u>-0.80</u>

Sentence Repetition (NCCEA)

#Correct 16 Percentile 56

Boston Naming Test

#Correct Without Cue 81 #Category Cues Given 4
 #Correct Following Category Cue 3 #Phonemic Cues Given 1
 #Correct 1 and Incorrect 0 Following Phonemic Cue
 (Age Level 93.75
 Total Score 84 Z-Score +1.25 Percentile 94.44 Educ. Level 94.44

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>12</u>	<u>12</u>
B	<u>11</u>	<u>11</u>
C	<u>11</u>	<u>11</u>
D	<u>11</u>	<u>11</u>
E	<u>9</u>	<u>10</u>

Total Score 54 Percentile 90

Rey-Osterrieth Complex Figure

Copy Score 36 Percentile 100 Memory Score 29.5 Percentile 82.81

Knox Cube

Trial I 8 Trial II 10 Average Score 9 CA 7.5

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>51</u>	<u>43</u>	<u>37</u>	
1-Trial Score				<u>41</u>
Percentile	<u>61</u>	<u>32</u>	<u>46</u>	<u>81</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 1 Left Sensory 0 Bilateral 0 Errors

Subject: BR Dyslexic: Normal Reader: X

Age: 19 Education: College Sophomore

Laterality Preference

Hand: R Foot: R Eye: R Ear: R/L

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>13</u>	<u>84</u>	
Comprehension	<u>19</u>	<u>99+</u>	
Arithmetic	<u>9</u>	<u>37</u>	
Similarities	<u>14</u>	<u>91</u>	
Digit Span	<u>14</u>	<u>91</u>	
Vocabulary	<u>12</u>	<u>75</u>	
Verbal Score	<u>81</u>		<u>+1.59</u>
[Verbal IQ <u>123</u>]		<u>94</u>	
Digit Symbol	<u>15</u>	<u>95</u>	
Picture Completion	<u>13</u>	<u>84</u>	
Block Design	<u>10</u>	<u>50</u>	
Picture Arrangement	<u>12</u>	<u>75</u>	
Object Assembly	<u>11</u>	<u>63</u>	
Performance Score	<u>61</u>		<u>+0.98</u>
[Performance IQ <u>115</u>]		<u>84</u>	
Full Scale Score	<u>142</u>		<u>+1.40</u>
[Full Scale IQ <u>121</u>]		<u>92</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>12.9</u>	<u>58</u>	<u>78</u>	<u>+0.8</u>
Word Identification	<u>12.9</u>	<u>59</u>	<u>82</u>	<u>+0.9</u>
Word Attack	<u>12.3</u>	<u>50</u>	<u>50</u>	<u>0.0</u>
Word Comprehension	<u>12.9</u>	<u>62</u>	<u>89</u>	<u>+1.2</u>
Passage Comprehension	<u>12.9</u>	<u>55</u>	<u>68</u>	<u>+0.5</u>
Total Reading	<u>12.9+</u>	<u>60</u>	<u>83</u>	<u>+1.0</u>

Gray Oral

Total Score 82 Grade Equivalent 12.0 + Comprehension Score 39.5

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>16.2</u>	<u>131</u>	<u>98</u>	<u>+2.07</u>
Spelling	<u>14.0</u>	<u>120</u>	<u>91</u>	<u>+1.33</u>
Arithmetic	<u>7.1</u>	<u>86</u>	<u>18</u>	<u>-0.93</u>

Sentence Repetition (NCCEA)

#Correct 18 Percentile 80

Boston Naming Test

#Correct Without Cue 80 #Category Cues Given 5
 #Correct Following Category Cue 2 #Phonemic Cues Given 3
 #Correct 3 and Incorrect 0 Following Phonemic Cue
 (Age Level 83.33
 Total Score 82 Z-Score +0.94 Percentile 81.25 Educ. Level 81.25

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>12</u>	<u>12</u>
B	<u>12</u>	<u>11</u>
C	<u>8</u>	<u>10</u>
D	<u>11</u>	<u>10</u>
E	<u>9</u>	<u>7</u>

Total Score 52 Percentile 84

Rey-Osterrieth Complex Figure

Copy Score 35 Percentile 87.5 Memory Score 17 Percentile 23.61

Knox Cube

Trial I 14 Trial II 16 Average Score 15 CA 15.5+

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>50</u>	<u>56</u>	<u>43</u>	
1-Trial Score				<u>54</u>
Percentile	<u>54</u>	<u>95.5</u>	<u>83</u>	<u>99+</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 1 Left Sensory 0 Bilateral 0 Errors

Subject: WN Dyslexic: Normal Reader: X

Age: 18 Education: College Freshman

Laterality Preference

Hand: R Foot: R Eye: R Ear: R

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>14</u>	<u>91</u>	
Comprehension	<u>16</u>	<u>98</u>	
Arithmetic	<u>16</u>	<u>98</u>	
Similarities	<u>12</u>	<u>75</u>	
Digit Span	<u>14</u>	<u>91</u>	
Vocabulary	<u>13</u>	<u>84</u>	
Verbal Score	<u>85</u>		<u>+1.86</u>
[Verbal IQ <u>127</u>]		<u>96</u>	
Digit Symbol	<u>17</u>	<u>99</u>	
Picture Completion	<u>14</u>	<u>91</u>	
Block Design	<u>13</u>	<u>84</u>	
Picture Arrangement	<u>11</u>	<u>63</u>	
Object Assembly	<u>14</u>	<u>91</u>	
Performance Score	<u>69</u>		<u>+1.65</u>
[Performance IQ <u>126</u>]		<u>96</u>	
Full Scale Score	<u>154</u>		<u>+1.88</u>
[Full Scale IQ <u>128</u>]		<u>97</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>12.9</u>	<u>58</u>	<u>78</u>	<u>+0.8</u>
Word Identification	<u>12.9</u>	<u>63</u>	<u>90</u>	<u>+1.3</u>
Word Attack	<u>12.9</u>	<u>51</u>	<u>55</u>	<u>+0.1</u>
Word Comprehension	<u>12.9</u>	<u>65</u>	<u>93</u>	<u>+1.5</u>
Passage Comprehension	<u>12.9</u>	<u>60</u>	<u>85</u>	<u>+1.0</u>
Total Reading	<u>12.9+</u>	<u>66</u>	<u>94</u>	<u>+1.6</u>

Gray Oral

Total Score 96 Grade Equivalent 12.0+ Comprehension Score 37.5

<u>WRAT</u>	<u>grade level</u>	<u>standard score</u>	<u>percentile</u>	<u>z-score</u>
Reading	<u>14.1</u>	<u>121</u>	<u>92</u>	<u>+1.40</u>
Spelling	<u>12.8</u>	<u>114</u>	<u>82</u>	<u>+0.93</u>
Arithmetic	<u>12.3</u>	<u>112</u>	<u>79</u>	<u>+0.80</u>

Sentence Repetition (NCCEA)

#Correct 18 Percentile 80

Boston Naming Test

#Correct Without Cue 80 #Category Cues Given 5
 #Correct Following Category Cue 4 #Phonemic Cues Given 1
 #Correct 0 and Incorrect 1 Following Phonemic Cue
 Total Score 84 Z-Score +1.25 Percentile 97.22 (Age Level 97.22 Educ. Level 93.97)

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>12</u>	<u>12</u>
B	<u>11</u>	<u>11</u>
C	<u>12</u>	<u>11</u>
D	<u>11</u>	<u>11</u>
E	<u>11</u>	<u>10</u>

Total Score 57 Percentile 97

Rey-Osterrieth Complex Figure

Copy Score 36 Percentile 100 Memory Score 25.5 Percentile 67.5

Knox Cube

Trial I 16 Trial II 15 Average Score 15.5 CA 15.5+

<u>Purdue Pegboard</u>	<u>Right</u>	<u>Left</u>	<u>Both</u>	<u>Assembly</u>
3-Trial Score	<u>45</u>	<u>43</u>	<u>34</u>	
1-Trial Score				<u>43</u>
Percentile	<u>28</u>	<u>32</u>	<u>23</u>	<u>96</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 0 Left Sensory 0 Bilateral 0 Errors

Subject: ST Dyslexic: Normal Reader: x

Age: 19 Education: College Junior

Laterality Preference

Hand: R Foot: R(L) Eye: L(R) Ear: R

<u>WAIS</u>	scaled score	percentile	z-score
Information	<u>14</u>	<u>91</u>	
Comprehension	<u>16</u>	<u>98</u>	
Arithmetic	<u>14</u>	<u>91</u>	
Similarities	<u>13</u>	<u>84</u>	
Digit Span	<u>16</u>	<u>98</u>	
Vocabulary	<u>16</u>	<u>98</u>	
Verbal Score	<u>89</u>		<u>+2.13</u>
[Verbal IQ <u>131</u>]		<u>98</u>	
Digit Symbol	<u>14</u>	<u>91</u>	
Picture Completion	<u>18</u>	<u>99</u>	
Block Design	<u>16</u>	<u>98</u>	
Picture Arrangement	<u>9</u>	<u>37</u>	
Object Assembly	<u>18</u>	<u>99</u>	
Performance Score	<u>75</u>		<u>+2.16</u>
[Performance IQ <u>133</u>]		<u>99</u>	
Full Scale Score	<u>164</u>		<u>+2.28</u>
[Full Scale IQ <u>134</u>]		<u>99</u>	

<u>Woodcock</u>	grade level	standard score	percentile	z-score
Letter Identification	<u>6.2</u>	<u>50</u>	<u>50</u>	<u>0.0</u>
Word Identification	<u>12.9</u>	<u>59</u>	<u>82</u>	<u>+0.9</u>
Word Attack	<u>12.9</u>	<u>66</u>	<u>94</u>	<u>+1.6</u>
Word Comprehension	<u>12.9</u>	<u>69</u>	<u>97</u>	<u>+1.9</u>
Passage Comprehension	<u>12.9</u>	<u>69</u>	<u>97</u>	<u>+1.9</u>
Total Reading	<u>12.9+</u>	<u>73</u>	<u>99</u>	<u>+2.3</u>

Gray Oral

Total Score 99 Grade Equivalent 12.0 + Comprehension Score 40

<u>WRAT</u>	grade level	standard score	percentile	z-score
Reading	<u>17.1</u>	<u>135</u>	<u>99</u>	<u>+2.33</u>
Spelling	<u>14.4</u>	<u>122</u>	<u>93</u>	<u>+1.47</u>
Arithmetic	<u>11.3</u>	<u>107</u>	<u>68</u>	<u>+0.47</u>

Sentence Repetition (NCCEA)

#Correct 17 Percentile 70

Boston Naming Test

#Correct Without Cue 82 #Category Cues Given 3
 #Correct Following Category Cue 2 #Phonemic Cues Given 1
 #Correct 1 and Incorrect 0 Following Phonemic Cue

Total Score 84 Z-Score +1.25 Percentile (Age Level 97.22)
 Educ. Level 93.75

Raven Standard Progressive Matrices

set #correct expected score given total score

A	<u>12</u>	<u>12</u>
B	<u>12</u>	<u>11</u>
C	<u>11</u>	<u>11</u>
D	<u>12</u>	<u>11</u>
E	<u>8</u>	<u>10</u>

Total Score 55 Percentile 95

Rey-Osterrieth Complex Figure

Copy Score 34 Percentile 75 Memory Score 30 Percentile 84.38

Knox Cube

Trial I 16 Trial II 14 Average Score 15 CA 15.5+

<u>Purdue Pegboard</u>	Right	Left	Both	Assembly
3-Trial Score	<u>50</u>	<u>46</u>	<u>38</u>	
1-Trial Score				<u>45</u>
Percentile	<u>54</u>	<u>48</u>	<u>52</u>	<u>98.2</u>

Double Simultaneous Stimulation (A. Smith)

Right Sensory 0 Left Sensory 0 Bilateral 0 Errors

APPENDIX B
COMPUTER PROGRAM PRINTOUT
OF EXPERIMENTAL TASK

Computer program written and executed by

John Chester
Director
Chaos Audio

PROGRAM DESCRIPTION - SUSAN KENNIFF'S VISUAL EXPERIMENT PROGRAM
WRITTEN BY JOHN K. CHESTER FEBRUARY 5, 1980

-----DATA STRUCTURE-----

DISPLAY DATA:
LEFT CENTER AND RIGHT DIGITS IN ORDER, 580 SETS MAX. -
0 WILL BE DISPLAYED WHEN PRESENT IN CENTER DIGIT
BUT WILL NOT BE DISPLAYED WHEN PRESENT IN LEFT OR RIGHT

RESULT DATA:
TRIAL NO., RESPONSE 1, RESPONSE 2 IN ORDER, 75 SETS MAX.

VARIABLES:

L (INTEGER)	LEFT DIGIT
C (INTEGER)	CENTER DIGIT
R (INTEGER)	RIGHT DIGIT
T1 (INTEGER)	ON TIME OF DISPLAY (MILLISECONDS)
T2 (INTEGER)	TOTAL TIME FOR SINGLE DIGIT TRIAL (MILLISECONDS)
T3 (INTEGER)	TOTAL TIME FOR DOUBLE DIGIT TRIAL (MILLISECONDS)
T4 (INTEGER)	TIME FROM START OF TRIAL UNTIL VOX IS FIRST CHECKED
A (INTEGER)	NUMBER OF CURRENT TRIAL (LOOP CONTROL VARIABLE)
NT (INTEGER)	NUMBER OF TRIALS (ALSO NUMBER OF DATA SETS)
PM (STRING)	PRESENTATION MODE (L FOR LEFT, C FOR CENTER, R FOR RIGHT)

-----PROGRAM STRUCTURE-----

BASIC PROGRAM:

SET T1, T2, T3, T4
SET NT (580 MAXIMUM)

ASK WHETHER PRESENTATION SHOULD BE BILATERAL, LEFT ONLY, OR RIGHT ONLY

FOR DATA SETS FROM 1 TO NT
READ LEFT, CENTER, AND RIGHT DIGITS
STORE CENTER DIGIT IN DATA ARRAY
IF PRESENTATION MODE FLAG IS LEFT ONLY
IF LEFT DIGIT IS 0
THEN LEFT DIGIT = RIGHT DIGIT
RIGHT DIGIT = 0
IF PRESENTATION MODE FLAG IS RIGHT ONLY
IF RIGHT DIGIT IS 0
THEN RIGHT DIGIT = LEFT DIGIT
LEFT DIGIT = 0
IF LEFT DIGIT IS 0
THEN STORE IT AS 15 IN DATA ARRAY
ELSE STORE ACTUAL DIGIT
IF RIGHT DIGIT IS 0
THEN STORE IT AS 15 IN DATA ARRAY
ELSE STORE ACTUAL DIGIT

DIVIDE T1 BY 5 (CONVERT FROM MILLISECONDS TO 5'S OF MILLISECONDS)
 DIVIDE T2, T3, AND T4 BY 10 (CONVERT TO 10'S OF MILLISECONDS)
 STORE T1, T2, T3, T4, NT IN TIMER PROGRAM AREA

JUMP TO ASSEMBLY LANGUAGE PROGRAM

ASSEMBLY LANGUAGE PROGRAM:

 GET PARAMETERS FROM BASIC PROGRAM
 CLEAR VARIABLES
 OUTPUT FIRST SET OF DIGITS TO DISPLAY LATCHES
 FOR TRIAL NUMBERS FROM 1 TO NT
 WAIT FOR RISING EDGE OF TIMEBASE
 TURN ON DISPLAY
 SET DISPLAY TIMER TO 0
 SET TRIAL TIMER TO 0
 INCREMENT TRIAL NUMBER
 IF LEFT OR RIGHT DIGIT IS NOT 15
 THEN SET MAXIMUM TIME TO T3
 SET DOUBLE DIGIT TRIAL FLAG
 ELSE SET MAXIMUM TIME TO T2
 CLEAR DOUBLE DIGIT TRIAL FLAG
 UNTIL DISPLAY TIMER = T1
 UPDATE TIMERS
 TURN OFF DISPLAY
 SET PREVIOUS VOX = 0
 OUTPUT NEXT SET OF DIGITS TO DISPLAY LATCHES
 UNTIL TRIAL TIMER = MAXIMUM TIME -1
 IF DOUBLE DIGIT TRIAL FLAG IS SET
 THEN CHECK VOX
 UPDATE TIMERS
 IF DOUBLE DIGIT TRIAL FLAG IS SET
 THEN INCREMENT RESULT NUMBER
 PASS NUMBER OF RESULTS BACK TO BASIC PROGRAM
 RETURN TO BASIC PROGRAM

UPDATE TIMERS:

INCREMENT DISPLAY TIMER
 IF TIMEBASE = 0
 THEN INCREMENT TRIAL TIMER
 SAVE CURRENT TIMEBASE VALUE
 WAIT UNTIL TIMEBASE = PREVIOUS TIMEBASE
 RETURN

CHECK VOX:

PUT CURRENT TRIAL NUMBER IN RESULT ARRAY

IF TRIAL TIMER > T4

THEN IF PREVIOUS VOX <> CURRENT VOX

THEN IF VOX = 1

THEN IF TIME 1 IN RESULT ARRAY = 0

THEN SET TIME 1 = TRIAL TIMER

ELSE IF TIME 2 IN RESULT ARRAY = 0

THEN SET TIME 2 = TRIAL TIMER

SET PREVIOUS VOX = VOX

RETURN

BASIC PROGRAM:

PRINT HEADING

ZERO LINE COUNTER

FOR N = 1 TO NUMBER OF RESULTS

IF LINE COUNTER > 9

THEN STOP AND WAIT FOR OPERATOR RESPONSE

PRINT HEADING

ZERO LINE COUNTER

PRINT LINE OF DATA (TRIAL NO., RESPONSE 1, RESPONSE 2, STIMULUS)

ASK IF RESULTS SHOULD BE DISPLAYED AGAIN

IF RESPONSE IS YES

THEN GO BACK TO "PRINT HEADING"

ASK IF SAME PRESENTATION SHOULD BE RUN AGAIN

IF RESPONSE IS YES

THEN REENTER MACHINE LANGUAGE PROGRAM

ELSE GO BACK TO BEGINNING OF PROGRAM

```

1 REM SUSAN KENNIFF'S VISUAL PERCEPTION EXPERIMENT
2 REM PROGRAM WRITTEN BY JOHN K. CHESTER      FEBRUARY 5, 1980
3 REM THIS PROGRAM MUST BE USED WITH A MACHINE LANGUAGE PROGRAM WHICH CONTROLS DATA DISPLAY AND RECORDS RESULTS.
9 REM
10 OUT 15,0 : 'CLEAR AND BLANK DISPLAY
12 DEFINT L,C,R,T,A,N
14 DEFSTR P
16 RESTORE
18 CLS : PRINT "SELECT PRESENTATION MODE"
20 INPUT "B = BILATERAL, L = LEFT ONLY, R = RIGHT ONLY ";PM
29 REM ALL TIMES ARE IN MILLISECONDS.
30 REM T1 IS LENGTH OF TIME DISPLAY IS ON.
31 T1=15
32 REM T2 IS THE TOTAL LENGTH OF A SINGLE DIGIT TRIAL.
33 T2=1000
34 REM T3 IS THE TOTAL LENGTH OF A DOUBLE DIGIT TRIAL.
35 T3=1500
36 REM T4 IS THE TIME FROM THE BEGINNING OF A TRIAL UNTIL THE VOX IS FIRST CHECKED.
37 T4=250
38 REM NT IS THE NUMBER OF TRIALS.
39 NT=580

```

50 DATA 0,1,0,0,2,0,0,3,0,0,6,0,0,9,0,0,8,0,0,7,0,0,4,0,0,5,0,6,0,0,8,0,0,4,0,0,9,0,0,7,0,0,6,0,0,3,0,0,7,0,0,2,0,0,5,0,0,9,0,0,5,0,0,
0,4,0,8,0,0,1,0,0,7,0,0,9,0,0,3,0,0,6,0,0,9,0,0,2,0,0,7,0,0,8,0,5,0,6,0,0,7,0,0,6,0,0,7,0,0,8,0,0,2,0,0,4,0,0,9,0,0,3,0,0,5,0,0,4,0,0,7
55 DATA 0,1,0,0,2,0,0,9,0,0,6,0,0,8,0,0,3,0,0,2,0,0,3,0,0,7,0,0,8,0,0,1,0,0,9,0,0,6,0,0,5,0,0,4,0,0,5,0,0
56 DATA 0,9,0,0,6,0,9,0,0,5,0,0,2,0,0,1,0,0,3,0,0,8,0,0,4,0,0,7,0,0,8,0,0,1,0,0,7,0,0,3,0,0,4,0,0,8,0,0,5,0,0,9,0,0,2,0,0,8,0,0,
0,2,0,0,5,0,0,6,0,0,9,0,0,4,0,0,7,0,0,1,0,0,3,0,0,2,0,0,8,0,0,3,0,0,4,0,0,6,0,0,1,0,0,7,0,0,9,0,0,8,0,0,5,0,0,7,0,0,0,3,0,6,0,0,5
57 DATA 0,0,8,0,0,1,0,0,4,0,0,3,0,0,9,0,0,2,0,0,1,0,0,4,0,0,9,0,0,2,0,0,6,0,0,7,0,0,5,0,0,3,0,0,8,0,6,0,0
58 DATA 0,5,0,0,8,0,0,1,0,0,9,0,0,3,0,0,2,0,0,4,0,0,6,0,0,7,0,0,5,0,0,9,0,0,2,0,0,6,0,0,1,0,0,4,0,0,8,0,0,3,0,0,7,0,0,5,0,0,2,0,0,
0,1,0,8,0,0,7,0,0,9,0,0,3,0,0,4,0,0,9,0,0,6,0,0,4,0,0,7,0,0,8,0,0,1,0,0,3,0,0,9,0,0,2,0,0,6,0,0,5,0,0,7,0,0,2,0,0,9,0,0,5,0,0,4
59 DATA 0,0,1,0,6,0,0,8,0,0,3,0,0,8,0,0,3,0,0,8,0,0,3,0,0,6,0,0,7,0,0,4,0,0,2,0,0,9,0,0,5,0,0,1,0,0,8,0,0,3,0,0,5,0,0
60 DATA 0,4,0,0,1,0,0,9,0,0,2,0,0,8,0,0,9,0,0,7,0,0,5,0,0,6,0,0,9,0,0,6,0,0,8,0,0,3,0,0,5,0,0,8,0,0,7,0,0,5,0,0,4,0,0,9,0,0,1,0,0,2,0,0,3,0,0,
0,5,0,0,8,0,0,9,0,0,7,0,0,4,0,0,6,0,0,3,0,0,1,0,0,2,0,0,4,0,0,8,0,0,3,0,0,5,0,0,8,0,0,7,0,0,2,0,0,9,0,0,6,0,0,1,0
61 DATA 0,3,0,0,2,0,0,7,0,0,5,0,0,9,0,0,6,0,0,4,0,0,1,0,0,8,0,0,1,0,0,6,0,0,8,0,0,4,0,0,2,0,0,7,0,0,3,0,0,5,0,0,9,0,0,0,5
62 DATA 0,3,0,0,7,0,0,8,0,0,6,0,0,4,0,0,9,0,0,5,0,0,2,0,0,1,0,0,4,0,0,6,0,0,3,0,0,2,0,0,8,0,0,9,0,0,1,0,0,5,0,0,4,0,0,6,0,0,7,0,0,8,0,0,
5,0,0,0,3,0,0,5,0,0,6,0,0,1,0,0,7,0,0,9,0,0,2,0,0,4,0,0,2,0,0,5,0,0,8,0,0,6,0,0,3,0,0,9,0,0,7,0,0,4,0,0,1,0,0,9,0
63 DATA 0,5,0,0,4,0,0,1,0,0,3,0,0,2,0,0,7,0,0,8,0,0,6,0,0,3,0,0,8,0,0,6,0,0,9,0,0,4,0,0,1,0,0,7,0,0,5,0,0,3,0,0,2,0,0,8
70 DATA 0,3,0,0,4,0,0,1,0,0,8,0,0,9,0,0,2,0,0,5,0,0,7,0,0,6,0,0,4,0,0,9,0,0,5,0,0,1,0,0,8,0,0,3,0,0,2,0,0,7,0,0,6,0,0,1,0,0,7,0,0,
0,2,0,0,5,0,0,8,0,0,4,0,0,3,0,0,9,0,0,3,0,0,6,0,0,4,0,0,6,0,0,1,0,0,7,0,0,3,0,0,6,0,0,5,0,0,8,0,0,9,0,0,2,0,0,1,0,8,0,0,6,0
71 DATA 0,3,0,0,9,0,0,4,0,0,8,0,0,7,0,0,5,0,0,2,0,0,5,0,0,7,0,0,9,0,0,2,0,0,8,0,0,7,0,0,6,0,0,1,0,0,3,0,0,5,0,0
72 DATA 0,7,0,0,5,0,0,8,0,0,4,0,0,9,0,0,6,0,0,1,0,0,3,0,0,2,0,0,6,0,0,6,0,0,5,0,0,9,0,0,1,0,0,3,0,0,4,0,0,7,0,0,6,0,0,2,0,0,8,0,0,6,0,0,
0,4,0,0,1,0,0,7,0,0,8,0,0,3,0,0,5,0,0,2,0,0,9,0,0,2,0,0,7,0,0,3,0,0,8,0,0,6,0,0,5,0,0,4,0,0,1,0,0,3,0,0,9,0,0,2,0,0,9,0,0,8,0,0,4
73 DATA 0,0,5,0,0,3,0,0,6,0,0,7,0,0,1,0,0,6,0,0,4,0,0,2,0,0,7,0,0,5,0,0,9,0,0,1,0,0,9,0,0,8,0,0,3,0,0,0,5
80 DATA 0,9,0,0,3,0,0,1,0,0,6,0,0,7,0,0,5,0,0,8,0,0,2,0,0,4,0,0,3,0,0,6,0,0,7,0,0,4,0,0,1,0,0,9,0,0,5,0,0,8,0,0,0,3,0,0,2,0,0,6,0,0,
0,5,0,0,4,0,0,2,0,0,9,0,0,3,0,0,1,0,0,7,0,0,9,0,0,8,0,0,5,0,0,2,0,0,3,0,0,9,0,0,1,0,0,7,0,0,4,0,0,8,0,0,6,0,0,4
81 DATA 0,0,1,0,8,0,0,3,0,0,7,0,0,6,0,0,9,0,0,2,0,0,5,0,0,8,0,0,1,0,0,9,0,0,5,0,0,4,0,0,2,0,0,6,0,0,5,0,0,3,0,0,7,0,8,0,0
90 DATA 0,4,0,0,8,0,0,2,0,0,5,0,0,9,0,0,3,0,0,1,0,0,7,0,0,6,0,0,7,0,0,9,0,0,6,0,0,8,0,0,5,0,0,2,0,0,1,0,0,3,0,0,4,0,0,2,0,0,1,0,
0,7,0,9,0,0,5,0,0,4,0,0,6,0,0,3,0,0,6,0,0,8,0,0,2,0,0,9,0,0,6,0,0,8,0,0,3,0,0,5,0,0,7,0,0,1,0,0,0,3,0,4,0,0,1
91 DATA 0,0,9,0,0,3,0,0,7,0,0,4,0,0,2,0,0,6,0,0,5,0,0,9,0,0,8,0,0,4,0,0,6,0,0,3,0,0,8,0,0,2,0,0,9,0,0,1,0,0,5,0,0,7,0,8,0,0
95 DATA 0,6,0,0,2,0,0,3,0,0,7,0,0,5,0,0,3,0,0,9,0,0,8,0,0,4,0,0,1,0,0,8,0,0,7,0,0,3,0,0,6,0,0,5,0,0,9,0,0,2,0,0,1,0,0,4,0,0,6,0,0,
0,0,5,0,7,0,0,2,0,0,5,0,0,3,0,0,9,0,0,4,0,0,8,0,0,1,0,0,5,0,0,9,0,0,2,0,0,1,0,0,6,0,0,4,0,0,3,0,0,7,0,0,8,0,0,4
96 DATA 0,0,7,0,0,6,0,0,3,0,0,9,0,0,2,0,0,5,0,0,1,0,0,8,0,0,9,0,0,4,0,0,6,0,0,7,0,0,1,0,0,8,0,0,9,0,0,2,0,0,3,0,0,1,0,6,0,0

```

200 REM  LOAD DATA INTO MEMORY
205 PRINT "LOADING DATA"
215 FOR A=1 TO NT
220 READ L,C,R
225 POKE 29694+A*3,C
230 IF PM="L" THEN 235 ELSE 240
235 IF L>0 THEN 240 ELSE L=R : R=0
240 IF PM="R" THEN 245 ELSE 250
245 IF R>0 THEN 250 ELSE R=L : L=0
250 IF L>0 THEN 255 ELSE L=15
255 IF R>0 THEN 260 ELSE R=15
260 POKE 29693+A*3,L : POKE 29695+A*3,R
265 NEXT A
270 T5=CINT(T1/5+.5) : T6=CINT(T2/10+.5) : T7=CINT(T3/10+.5) : T8=CINT(T4/10+.5)
275 POKE 31744,T5 : POKE 31746,T6 : POKE 31748,T7 : POKE 31754,T8
280 FOR N=1 TO 304 : POKE 31439+N,0 : NEXT N
290 CLS : PRINT "PRESENTATION RUNNING"
299 REM
300 REM STARTING ADDRESS OF MACHINE LANGUAGE PROGRAM 31808 DECIMAL, 7C40 HEX
305 POKE 16526,64 : POKE 16527,124
310 NR=USR(NT) : 'JUMP TO MACHINE LANGUAGE PROGRAM
399 REM
400 REM      DATA DISPLAY
410 GOSUB 600
420 FOR N=1 TO NR
430 IF NL>9 THEN 450 ELSE GOSUB 500 : NL=NL+1
440 NEXT N : GOTO 800
450 INPUT "MORE DATA ";P : GOSUB 600 : GOTO 430
499 REM
500 REM      PRINT LINE OF DATA
510 N1=N*4
520 TN=PEEK(31437+N1)*256+PEEK(31436+N1)
530 T5=PEEK(31438+N1)*10 : T6=PEEK(31439+N1)*10
540 TS=PEEK(29693+TN*3)
550 IF TS=15 THEN TS=PEEK(29695+TN*3)
560 PRINT TN, T5, T6, TS
570 RETURN
599 REM
600 REM      PRINT HEADING
610 CLS : PRINT "TIME IN MILLISECONDS"
620 PRINT "TRIAL NO.", "RESPONSE 1", "RESPONSE 2", "STIMULUS"
630 NL=0 : RETURN
699 REM
800 REM  WHAT DO WE DO NOW?
810 INPUT "REVIEW DATA (Y OR N) ";P
820 IF P="Y" THEN 400
830 INPUT "RERUN SAME PRESENTATION ";P
840 IF P="Y" THEN 270 ELSE 1
850 END

```

```

00100 ;DISPLAY CONTROL AND TIMER PROGRAM FOR VISUAL PERCEPTION EXPERIMENT
00102 ;RUNS ON TRS80 LEVEL 2 16K IN CONJUNCTION WITH BASIC PROGRAM TO LOAD DATA AND DISPLAY RESULTS.
00110 ;VERSION 1.0      FEBRUARY 5, 1980
00120 ;WRITTEN BY JOHN K. CHESTER
00130 ;THIS PROGRAM DRIVES A THREE DIGIT DISPLAY VIA A CUSTOM INTERFACE CARD.
00140 ;PORTS 0-15 ARE USED FOR OUTPUTS; ADDRESS BIT 0 ACCESSES DISPLAY BLANKING
00150 ;AND BITS 1 TO 3 ACCESS THE THREE DISPLAY DIGITS.
00160 ;THESE ADDRESSES ARE NOT FULLY DECODED - THUS, ALL FOUR FUNCTIONS MAY BE ACCESSED VIA PORT 15.
00170 ;INPUT PORT 1 HAS A 100 HZ. SQUARE WAVE AT BIT 1 AND THE STATUS OF THE VOX AT BIT 4.
00180 ;INPUT DATA FOR THIS PROGRAM IS LOADED INTO THE ARRAY DISDAT, STARTING ADDRESS 7400 HEX, 29696 DECIMAL.
00190 ;EACH SET OF DATA CONSISTS OF THREE INTEGERS, ONE EACH FOR THE LEFT, CENTER, AND RIGHT DISPLAY DIGITS.
00200 ;VALUES FROM 0 TO 9 WILL BE DISPLAYED AS SUCH; A VALUE OF 15 WILL CAUSE THE DISPLAY TO REMAIN BLANK.
00210 ;THE ARRAY WILL ACCOMODATE UP TO 581 SETS OF DATA.
00220 ;RESULTS ARE PUT IN THE ARRAY RESDAT, STARTING ADDRESS 7AD0 HEX, 31440 DECIMAL.
00230 ;DATA WILL BE RECORDED FOR EACH TRIAL IN WHICH EITHER THE LEFT OR RIGHT DISPLAY DIGIT IS NOT BLANK.
00240 ;THE FIRST TWO BYTES OF EACH DATA SET CONTAIN THE TRIAL NUMBER.
00250 ;THE THIRD AND FOURTH BYTES CONTAIN THE TIMES OF THE FIRST AND SECOND VOX OCCURRENCES.
00260 ;UP TO 76 SETS OF DATA MAY BE PUT IN THIS ARRAY.
00270 ;REGISTER ASSIGNMENTS:
00280 ;      B - DISPLAY TIMER          C - TRIAL TIMER
00290 ;      D - DISPLAY ON TIME       E - TRIAL TIME
00300 ;      HL - TRIAL NUMBER
00310 ;      IX - INPUT DATA ADDRESS POINTER
00320 ;      IY - RESULT DATA ADDRESS POINTER
00330 ;      B'C' - INPUT DATA POINTER INCREMENT
00340 ;      D'E' - RESULT DATA POINTER INCREMENT
00350 ;      H'L' - UPTIM AND VOX SCRATCHPAD
00360 ;THE ENTRY ADDRESS OF THE PROGRAM IS 7C40 HEX, 31808 DECIMAL..
7400      00370      ORG 7400H
06D0      00380 DISDAT  DEFS 6D0H      ;DISPLAY DATA TABLE
0130      00390 RESDAT  DEFS 130H      ;RESULT DATA TABLE
7C00 0000      00400 T1      DEFW 00      ;DISPLAY ON TIME
7C02 0000      00410 T2      DEFW 00      ;SINGLE DIGIT TRIAL TIME
7C04 0000      00420 T3      DEFW 00      ;DOUBLE DIGIT TRIAL TIME
7C06 0000      00430 NT      DEFW 00      ;NUMBER OF TRIALS
7C08 0000      00440 NUMRES DEFW 00      ;NUMBER OF RESULTS
7C0A 00      00450 VOXEN  DEFB 0      ;VOX ENABLE TIME
7C0B 00      00460 OLDTIM DEFB 0      ;PREVIOUS VALUE OF TIMEBASE
7C0C 00      00470 OLDVOX DEFB 0      ;PREVIOUS VALUE OF VOX
7C0D 00      00480 DDFLG  DEFB 0      ;DOUBLE DIGIT TRIAL FLAG
7C40      00490      ORG 7C40H      ;START OF PROGRAM
7C40 CD7F0A      00500 BEGIN  CALL 0A7FH      ;GET PARAMETER FROM BASIC
7C43 22067C      00510      LD      (NT),HL      ;STORE IT IN NT
7C46 DD210074      00520      LD      IX,DISDAT      ;PUT START ADDRESS OF DATA TABLE IN IX
7C4A FD21007A      00530      LD      IY,RESDAT      ;PUT START OF RESULT DATA TABLE IN IY
7C4E D9      00540      EXX      ;EXCHANGE REGISTERS
7C4F 010300      00550      LD      BC,3H      ;PUT DISPLAY DATA INCREMENT IN BC
7C52 110400      00560      LD      DE,4H      ;PUT RESULT DATA INCREMENT IN DE
7C55 D9      00570      EXX      ;EXCHANGE REGISTERS
7C56 3E00      00580      LD      A,0
7C58 320C7C      00590      LD      (OLDVOX),A      ;CLEAR VARIABLES
7C5B 320D7C      00600      LD      (DDFLG),A
7C5E 320B7C      00610      LD      (OLDTIM),A
7C61 320B7C      00620      LD      (NUMRES),A
7C64 32097C      00630      LD      (NUMRES+1),A
7C67 210000      00640      LD      HL,00      ;CLEAR TRIAL COUNTER
7C6A DD7E00      00650      LD      A,(IX+0)      ;GET LEFT DIGIT

```

7C6D D302	00660	OUT	(2H),A	;PUT IT IN DISPLAY LATCH
7C6F DD7E01	00670	LD	A,(1X+1)	;GET CENTER DIGIT
7C72 D304	00680	OUT	(4H),A	;PUT IT IN DISPLAY LATCH
7C74 DD7E02	00690	LD	A,(1X+2)	;GET RIGHT DIGIT
7C77 D308	00700	OUT	(8H),A	;PUT IT IN DISPLAY LATCH
7C79 DB01	00710 WAIT0	IN	A,(1H)	;READ TIMEBASE
7C7B CB47	00720	BIT	0,A	;TEST TIME BIT
7C7D 20FA	00730	JR	NZ,WAIT0	;WAIT FOR 0
7C7F DB01	00740 WAIT1	IN	A,(1H)	;READ TIMEBASE
7C81 CB47	00750	BIT	0,A	;TEST TIME BIT
7C83 28FA	00760	JR	Z,WAIT1	;WAIT FOR 1
7C85 3E01	00770	LD	A,1H	
7C87 D301	00780 NEXT	OUT	(1H),A	;TURN ON DISPLAY
7C89 0600	00790	LD	B,0	;ZERO DISPLAY TIMER
7C8B 0E00	00800	LD	C,0	;ZERO TRIAL TIMER
7C8D 23	00810	INC	H,	;INCREMENT TRIAL NUMBER
7C8E 3E0F	00820	LD	A,15	
7C90 DDBE00	00830	CP	(1X+0)	;IS LEFT DIGIT 15
7C93 200F	00840	JR	NZ,DBLD	;NO
7C95 DDBE02	00850	CP	(1X+2)	;IS RIGHT DIGIT 15
7C98 200A	00860	JR	NZ,DBLD	;NO
7C9A 3E00	00870	LD	A,0	
7C9C 320D7C	00880	LD	(DDFLG),A	;CLEAR DOUBLE DIGIT FLAG
7C9F 3A027C	00890	LD	A,(T2)	;PUT T2 IN A
7CA2 1808	00900	JR	TIMDIS	
7CA4 3E01	00910 DBLD	LD	A,1	
7CA6 320D7C	00920	LD	(DDFLG),A	;SET DOUBLE DIGIT FLAG
7CA9 3A047C	00930	LD	A,(T3)	;PUT T3 IN A
7CAC 5F	00940 TIMDIS	LD	E,A	;PUT TRIAL TIME IN E
7CAD 1D	00950	DEC	E	;SET TO TERMINATE 1 COUNT EARLY
7CAE 3A007C	00960	LD	A,(T1)	;GET T1
7CB1 57	00970	LD	D,A	;PUT DISPLAY TIME IN D
7CB2 CD057D	00980 DISWT	CALL	UPTIM	;UPDATE TIMERS
7CB5 78	00990	LD	A,B	
7CB6 BA	01000	CP	D	;DISPLAY TIME FINISHED?
7CB7 20F9	01010	JR	NZ,DISWT	;NO
7CB9 3E00	01020	LD	A,0	
7CBB D301	01030	OUT	(1H),A	;YES, TURN OFF DISPLAY
7CBD 320C7C	01040	LD	(OLDVOX),A	;CLEAR OLDVOX
7CC0 D9	01050	EXX		;EXCHANGE REGISTERS
7CC1 DD09	01060	ADD	1X,BC	;ADD 3 TO DATA POINTER
7CC3 D9	01070	EXX		;EXCHANGE REGISTERS
7CC4 DD7E00	01080	LD	A,(1X+0)	;GET LEFT DIGIT
7CC7 D302	01090	OUT	(2H),A	;PUT IT IN DISPLAY LATCH
7CC9 DD7E01	01100	LD	A,(1X+1)	;GET CENTER DIGIT
7CCC D304	01110	OUT	(4H),A	;PUT IT IN DISPLAY LATCH
7CCE DD7E02	01120	LD	A,(1X+2)	;GET RIGHT DIGIT
7CD1 D308	01130	OUT	(8H),A	;PUT IT IN DISPLAY LATCH
7CD3 3A0D7C	01140 TRIWT	LD	A,(DDFLG)	;GET DOUBLE DIGIT FLAG
7CD6 FE01	01150	CP	1	;IS IT SET?
7CD8 2003	01160	JR	NZ,TIMTRI	;NO
7CDA CD207D	01170	CALL	VOX	;YES, CHECK VOX STATUS
7CDD CD057D	01180 TIMTRI	CALL	UPTIM	;UPDATE TIMERS
7CE0 79	01190	LD	A,C	
7CE1 BB	01200	CP	E	;TRIAL TIME FINISHED?
7CE2 20EF	01210	JR	NZ,TRIWT	;NO
7CE4 3A0D7C	01220	LD	A,(DDFLG)	;YES, GET DOUBLE DIGIT FLAG

7CE7	FE01	01230	CP	1	;IS IT SET?
7CE9	2008	01240	JR	NZ,NODD	;NO
7CEB	D9	01250	EXX		;EXCHANGE REGISTERS
7CEC	FD19	01260	ADD	1Y,DE	;ADD 4 TO RESULT DATA POINTER
7CEE	21087C	01270	LD	HL,NUMRES	
7CF1	34	01280	INC	(HL)	;INCREMENT RESULT COUNTER
7CF2	D9	01290	EXX		;EXCHANGE REGISTERS
7CF3	E5	01300	PUSH	HL	;SAVE TRIAL NUMBER
7CF4	ED5B067C	01310	LD	DE,(NT)	;PUT MAX TRIAL NUMBER IN DE
7CF8	B7	01320	OR	A	;CLEAR CARRY
7CF9	ED52	01330	SBC	HL,DE	;MORE TRIALS?
7CFB	E1	01340	POP	HL	;RESTORE TRIAL NUMBER
7CFC	C2797C	01350	JP	NZ,WAITO	;YES
7CFF	2A087C	01360	LD	HL,(NUMRES)	;PUT NUMBER OF RESULTS IN HL
7D02	C39A0A	01370	JP	0A9AH	;BACK TO BASIC
7D05	04	01380	INC	B	;INCREMENT DISPLAY TIMER
7D06	DB01	01390	IN	A,(1H)	;READ TIMEBASE
7D08	E601	01400	AND	01	;CLEAR BITS 1 THRU 7
7D0A	2001	01410	JR	NZ,NOINC	;JUMP IF TIMEBASE IS NOT 0
7D0C	0C	01420	INC	C	;INCREMENT TRIAL TIMER
7D0D	D9	01430	EXX		;EXCHANGE REGISTERS
7D0E	67	01440	LD	H,A	;SAVE TIMEBASE VALUE
7D0F	3A0B7C	01450	LD	A,(OLDTIM)	;GET PREVIOUS VALUE OF TIMEBASE
7D12	6F	01460	LD	L,A	;PUT IT IN L
7D13	7C	01470	LD	A,H	;GET TIMEBASE VALUE
7D14	320B7C	01480	LD	(OLDTIM),A	;PUT IT BACK IN MEMORY
7D17	DB01	01490	IN	A,(1H)	;READ TIMEBASE
7D19	E601	01500	AND	01	;CLEAR BITS 1 THRU 7
7D1B	AD	01510	XOR	L	;SAME AS OLD VALUE?
7D1C	20F9	01520	JR	NZ,TBCHK	;NO
7D1E	D9	01530	EXX		;YES, EXCHANGE REGISTERS
7D1F	C9	01540	RET		;RETURN
7D20	FD22257D	01550	LD	(TRISAV+1),1Y	;SET RESULT ADDRESS
7D24	22D07A	01560	LD	(RESDAT),HL	;STORE TRIAL NUMBER
7D27	79	01570	LD	A,C	;PUT TRIAL TIMER IN A
7D28	D9	01580	EXX		;EXCHANGE REGISTERS
7D29	210A7C	01590	LD	HL,VOXEN	
7D2C	BE	01600	CP	(HL)	;VOX ENABLED YET?
7D2D	382D	01610	JR	C,EXIT	;NO
7D2F	3A0C7C	01620	LD	A,(OLDVOX)	;YES, GET PREVIOUS VALUE OF VOX
7D32	6F	01630	LD	L,A	;PUT IT IN L
7D33	DB01	01640	IN	A,(1H)	;READ VOX STATUS
7D35	E608	01650	AND	8H	;CLEAR ALL BITS EXCEPT 3
7D37	67	01660	LD	H,A	;SAVE VOX VALUE
7D38	AD	01670	XOR	L	;SAME AS OLD VALUE?
7D39	2821	01680	JR	Z,EXIT	;YES
7D3B	7C	01690	LD	A,H	;NO, GET CURRENT VALUE
7D3C	320C7C	01700	LD	(OLDVOX),A	;PUT IT IN MEMORY
7D3F	FE08	01710	CP	8H	;IS VOX BIT 1?
7D41	2019	01720	JR	NZ,EXIT	;NO
7D43	D9	01730	EXX		;YES, EXCHANGE REGISTERS
7D44	3E00	01740	LD	A,0	
7D46	FDBE02	01750	CP	(1Y+2)	;DATA IN TIME 1?
7D49	2006	01760	JR	NZ,DATA2	;YES
7D4B	79	01770	LD	A,C	;NO, GET TIME
7D4C	FD7702	01780	LD	(1Y+2),A	;PUT IT IN RESULT TABLE
7D4F	180C	01790	JR	RETURN	

7D51	FDBE03	01800	DATA2	CP	(1Y+3)	;DATA IN TIME 2?
7D54	2007	01810		JR	NZ,RETURN	;YES
7D56	79	01820		LD	A,C	;NO, GET TIME
7D57	FD7703	01830		LD	(1Y+3),A	;PUT IT IN RESULT TABLE
7D5A	1801	01840		JR	RETURN	
7D5C	D9	01850	EXIT	EXX		;EXCHANGE REGISTERS
7D5D	C9	01860	RETURN	RET		;RETURN
0000		01870		END		
00000	TOTAL ERRORS					

RETURN	7D5D
DATA2	7D51
EXIT	7D5C
TRISAV	7D24
TBCHK	7D17
NOINC	7D0D
NODD	7CF3
VOX	7D20
TIMTRI	7CD0
TRIWT	7CD3
UPTIM	7D05
DISWT	7CB2
TIMDIS	7CAC
DBLD	7CA4
NEXT	7C87
WAIT1	7C7F
WAIT0	7C79
BEGIN	7C40
DDFLG	7C0D
OLDVOX	7C0C
OLDTJM	7C0B
VOXEN	7C0A
NUMRES	7C08
NT	7C06
T3	7C04
T2	7C02
T1	7C00
RESDAT	7AD0
DISDAT	7400

OPERATING INFORMATION FOR VISUAL EXPERIMENT PROGRAM

STARTUP PROCEDURE:

Apply power to interface box, then turn on TRS80.
 For "MEMORY SIZE" type 29600, hit ENTER.
 Put cassette in machine and find start of basic program.
 Type CLOAD "A", hit ENTER to load program.
 When program has loaded type SYSTEM, hit ENTER.
 To load machine language program type TIMER, hit ENTER.
 When program has loaded hit BREAK.
 To run program type RUN, hit ENTER.

TO RUN THE PROGRAM WITHOUT RELOADING DATA:

Type GOTO 270, hit ENTER.
 This should only be done after the program has run at least once.

DISPLAY INTENSITY CONTROL:

BRIGHTNESS	SWITCH 1	SWITCH 2	SWITCH 3	SWITCH 4
FULL	CLOSED	CLOSED	CLOSED	CLOSED
15/16	CLOSED	CLOSED	CLOSED	OPEN
14/16	CLOSED	CLOSED	OPEN	CLOSED
13/16	CLOSED	CLOSED	OPEN	OPEN
12/16	CLOSED	OPEN	CLOSED	CLOSED
11/16	CLOSED	OPEN	CLOSED	OPEN
10/16	CLOSED	OPEN	OPEN	CLOSED
9/16	CLOSED	OPEN	OPEN	OPEN
8/16	OPEN	CLOSED	CLOSED	CLOSED
7/16	OPEN	CLOSED	CLOSED	OPEN
6/16	OPEN	CLOSED	OPEN	CLOSED
5/16	OPEN	CLOSED	OPEN	OPEN
4/16	OPEN	OPEN	CLOSED	CLOSED
3/16	OPEN	OPEN	CLOSED	OPEN
2/16	OPEN	OPEN	OPEN	CLOSED
1/16	OPEN	OPEN	OPEN	OPEN

APPENDIX C
EQUIPMENT DESIGN DESCRIPTION

Instrumentation designed, constructed and
delineated by

John Chester
Director
Chaos Audio

MAJOR EQUIPMENT ITEMS

The electronic equipment for this experiment consisted of the following major items:

1. A TRS-80 computer with its associated video display and a cassette machine for data storage.
2. A box containing the buss interface for the TRS-80, the crystal time base, and the tone generator used to make the reference audio tape. This box was connected to the expansion port connector on the TRS-80 via a 40 conductor flat cable.
3. The display unit. This included the decoder-driver circuitry for the displays and the display temperature compensation circuit. It was connected to the buss interface via a 20 conductor flat cable.
4. The power supply for items 2 and 3. This supplied +9 volts to the buss interface, which included a voltage regulator to provide +5 volts for the logic circuitry. Both the +9 volts and the +5 volts were also used to power the display unit.

TRS-80 COMPUTER AND PROGRAMS

No modifications were made to the TRS-80. The BASIC language was the standard Level II BASIC stored in ROM. The machine language program was written on the TRS-80 using the Radio Shack editor-assembler. Both the BASIC program and the machine language program were stored on tape cassettes.

The program listings are thoroughly commented and should require no further explanation here. There is, however, a difference between the terminology in the listings and the terminology elsewhere:

display time = stimulus time
single digit trial time = inter-stimulus time,
nontarget
double digit trial time = inter-stimulus time, target

BUSS INTERFACE AND TIME BASE CIRCUITRY

The TRS-80 expansion port is connected to the buss interface card with a 6 inch long cable and the relevant signals are buffered with 8T98's for the address buss and an 8T26 for the data buss to prevent any interference with the computer's normal internal operations.

Input and output ports 0 to 15 are allocated to the equipment for this experiment. Address bits 4 to 7 are decoded by IC17A to ensure that the interface does not respond to port addresses above 15. Bits 0 to 3 are not

fully decoded; rather, each bit controls the clocking of data into one of the data latches IC2 to IC4. Setting an address bit will cause data from the data buss to be latched on the leading edge of the OUT strobe. This method requires less decoding logic and also has the advantage that all four latches can be cleared by a single output command when port 15 is addressed.

Only the lower 4 bits on the data buss are used. Port 1 drives the display blanking (only data bit 0 is used) and ports 2, 4, and 8 drive the left, center, and right displays. The displays can be blanked by sending them 15; however, if this method was used to turn on and off the displays two digits could not be turned on at exactly the same time. This method is used only to select which displays will be blanked for a particular trial. The program sets the blanking bit to turn off all displays, loads the display latches (displays which are to be blank during the upcoming trial are loaded with 15), then clears the blanking bit to turn on the displays.

All of the timing signals are derived from a 3.2768 MHz. crystal oscillator (IC30F). Its output is first divided by 16 by IC25. This signal goes to the display intensity control circuit and to IC15, which divides by 256 to produce an 800 Hz. output and by 2048 to produce a 100 Hz. output. The 800 Hz. signal goes to IC22D where it is gated by the display blanking signal. This provides an audio tone when any display is on so that reference marks can be put on a tape recording of the experiment.

The 100 Hz. output from IC15 is used by the computer to time both the presentation of stimuli and the results from the voice activated relay. The VOX and 100 Hz. signals are latched by IC9 when input port 1 is addressed and are put onto the data buss by IC1 for the duration of the IN strobe signal. The 100 Hz. signal is data bit 0 and the VOX is data bit 3. The 100 Hz. signal is a square wave (50 % duty cycle) and can therefore provide both 5 millisecond intervals for controlling the stimulus time and 10 millisecond intervals for all other timing.

The display intensity is controlled by IC's 24 and 19. IC24 is a 4 bit binary counter clocked by the 204.8 kHz. signal from IC25. IC19 is a 4 bit magnitude comparator whose two inputs are the 4 bits from the counter and 4 bits whose value can be set by a switch. The A<B output is ORed with the A=B output by IC27D to produce a 12.8 kHz. signal with a duty cycle of $(16-N)/16$ where N is the binary number set by the switch. When the display blanking bit from IC5 is cleared the displays will be turned on and off at the 12.8 kHz. rate; the ratio of on time to off time controls the intensity. When the display blanking bit from IC5 is set the displays are off.

DISPLAY DRIVERS AND TEMPERATURE COMPENSATION

The displays are driven by 7447 decoder-drivers. These take binary input codes of 0 to 9 and turn on the appropriate segments of the display to form digits 0 to 9. A binary input of 15 will cause all segments of the

display to remain off. The blanking inputs of all three drivers are connected to the blanking control line from the buss interface. The anodes of the display segments are connected to the output of the temperature compensation circuit which has an output voltage of 4.90 volts at 25 degrees C. The current through each display segment is limited by the 360 ohm resistor connected from the segment cathode to the driver output.

According to the manufacturer's specifications¹ the 5082-7730 displays have a change in luminous intensity with temperature which can be calculated by the following formula:

$$I_{VT_a} = I_{V25^{\circ}C} e^{[-.0188(T_a - 25^{\circ}C)]}$$

where I_V = luminous intensity

T_a = ambient temperature

This indicates that for a constant operating current the display intensity will change by +20.7% at 15 degrees C and by -17.1% at 35 degrees C. The voltage across the current limiting resistor at 25 degrees C is 4.90 volts less the display segment voltage of 1.55 volts and the saturation voltage of the driver output of .15 volts = 3.20 volts. To maintain constant display intensity this

1. Hewlett-Packard Co., Optoelectronics Designer's Catalog 1979, pp. 41-44

voltage should change by -20.7% or $-.662$ volts at 15 degrees C and by $+17.1\%$ or $+.547$ volts at 35 degrees C.

The compensation circuit uses 3 1N4148 silicon diodes as temperature sensors. These diodes have a temperature coefficient of forward voltage of -2.0 millivolts per degree C. Three diodes in series thus produce a change of -6.0 millivolts per degree C and this change is further amplified to produce a change of -60 millivolts per degree C at the compensation circuit output.

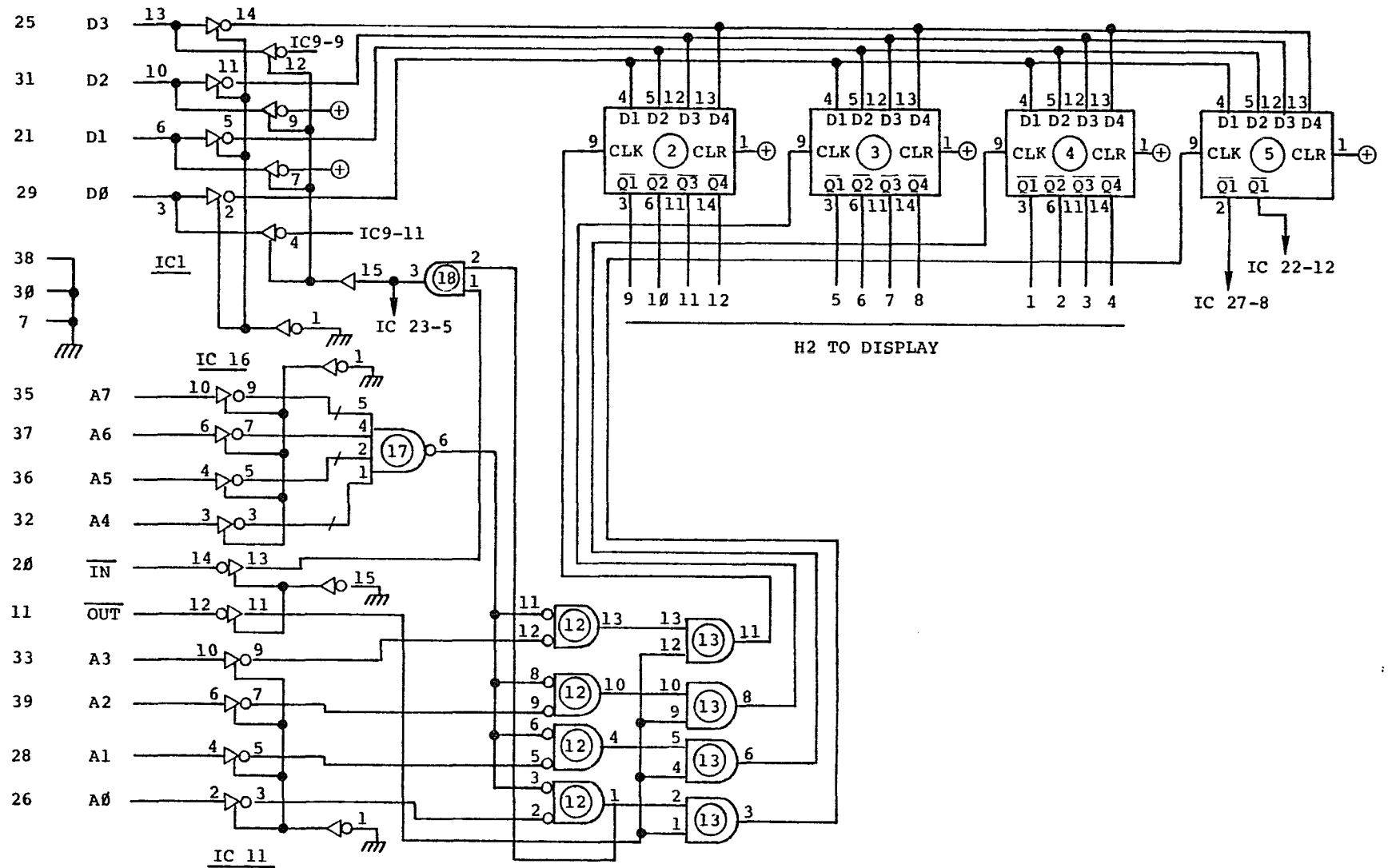
It should be noted that the change produced by this circuit is a straight-line change with temperature which does not perfectly match the intensity change of the displays; however, it is a sufficiently good match over a 20 degree C range to reduce the intensity change by nearly an order of magnitude. There are also other factors which have not been considered in the above analysis, the largest of which are the temperature coefficient of the display segment voltage of -2.0 millivolts per degree C and the temperature coefficient of the 5 volt power supply voltage of -1.1 millivolt per degree C. The power supply change will cause a change in the output voltage of the compensation circuit of about -4 millivolts per degree C. This will be partially offset by the change in the display voltage which will tend to increase the display current with temperature. Even considering these additional factors, the original compensation calculation would change by considerably less than 10% .

PERFORMANCE VERIFICATION

The timing accuracy of the experiment was checked in two ways: First, the display blanking waveform was observed on an oscilloscope for stimulus times ranging from 15 to 150 milliseconds and interstimulus times ranging from .5 to 1.5 seconds (non-target) and 1 to 2 seconds (target). The observed errors were less than 2%, which is on the same order as the calibration error of the oscilloscope itself. Second, the interstimulus times were set to values of 1 second (non-target) and 1.5 seconds (target) which should cause the total time for the experiment to be 10 minutes. This was measured with a stopwatch and errors of less than 1 second were consistently obtained.

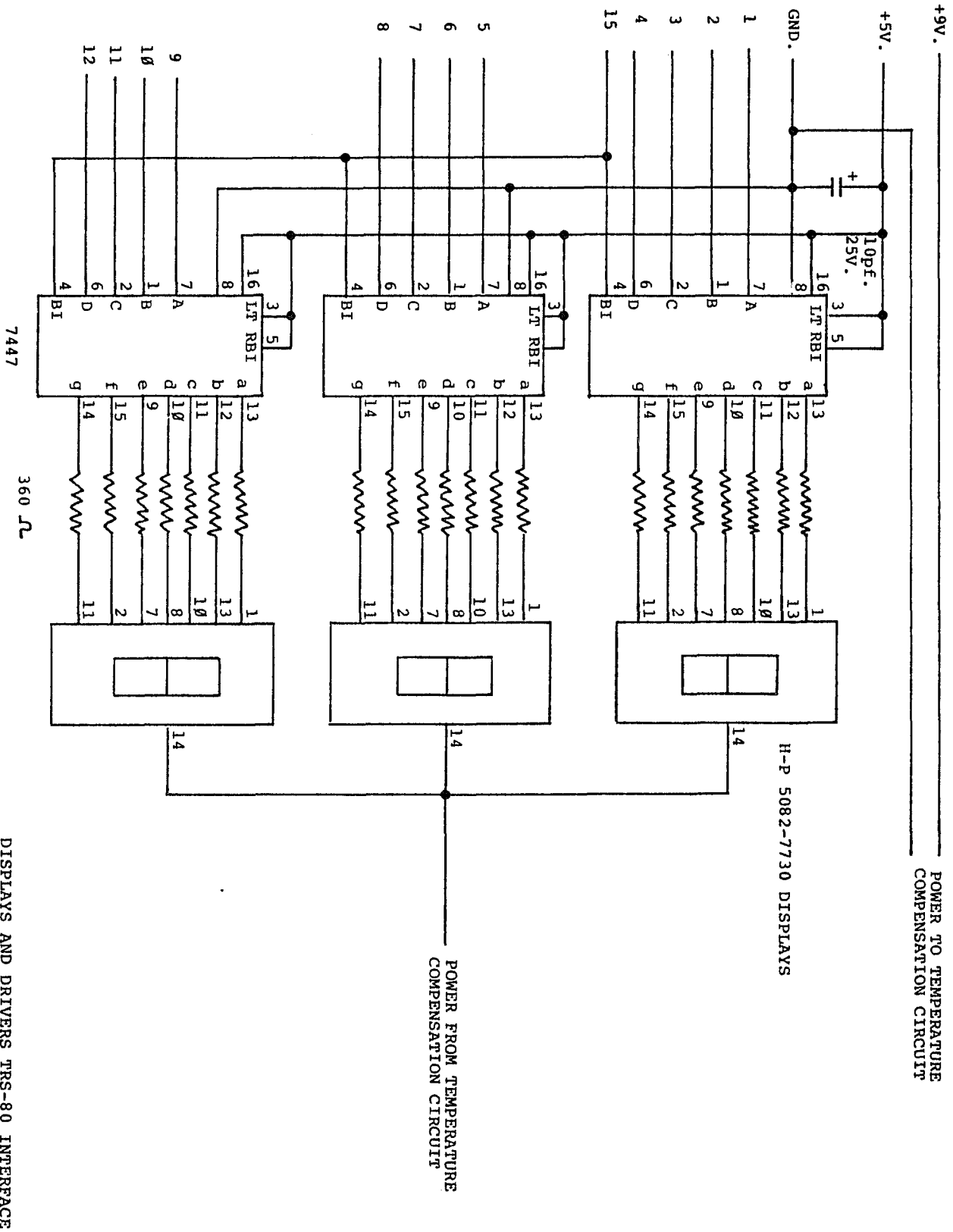
The digits appearing on the displays were checked against the data list for all three presentation modes and were found to be correct in all cases. These checks were made both with and without the VOX. The program was reloaded from tape in some cases and was rerun after displaying results in other cases in order to catch any program bugs; none were found.

TRS-80 BUSS -- NOTE: THESE NUMBERS ARE FLAT CABLE NO.'S NOT TRS-80 BUSS PIN NO.'S

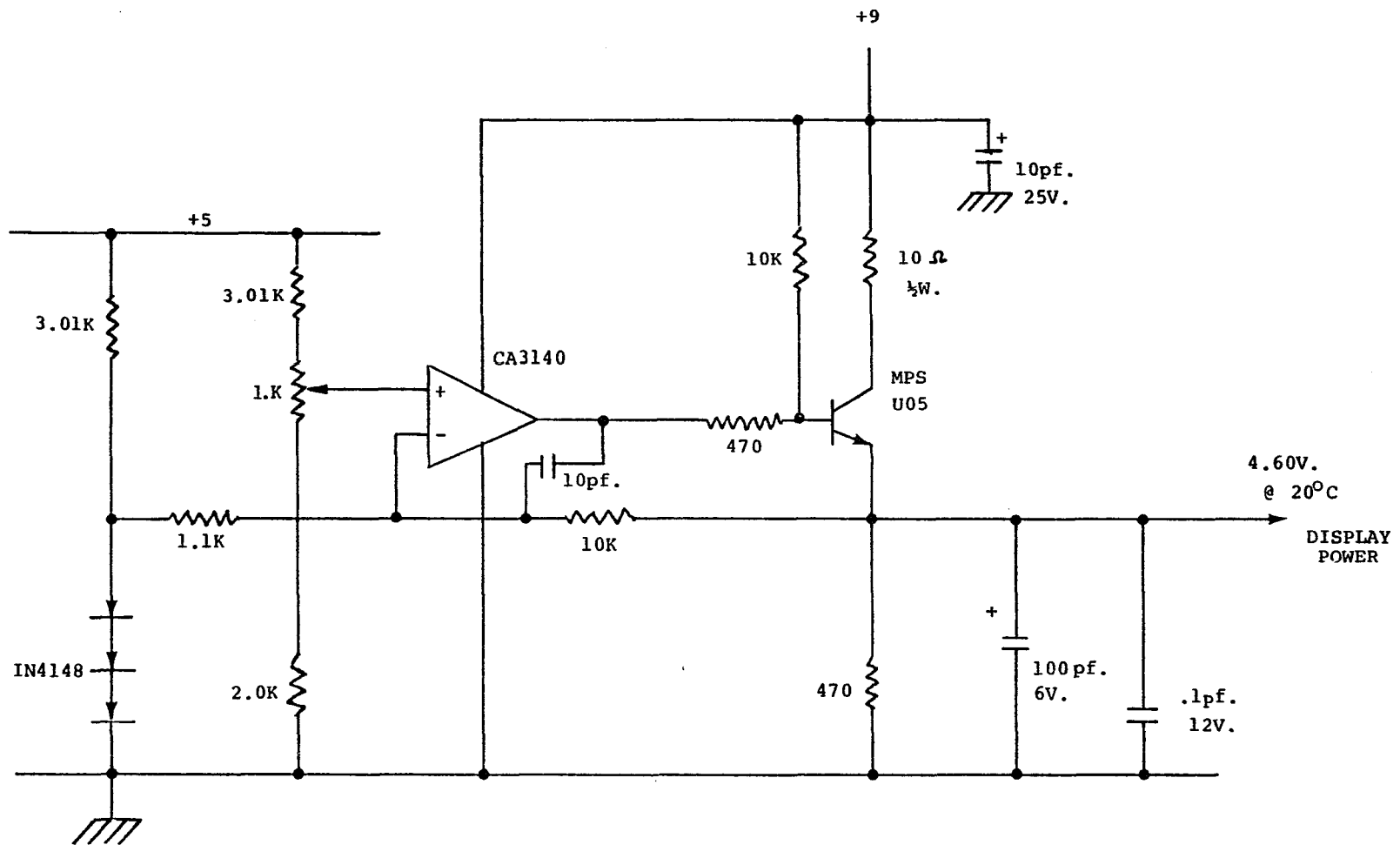


TRS-80 INTERFACE ADDRESS DECODERS AND DATA LATCHES

FLAT CABLE TO INTERFACE BOARD



DISPLAYS AND DRIVERS TRS-80 INTERFACE



TRS-80 INTERFACE DISPLAY TEMPERATURE COMPENSATION

APPENDIX D.
LUMINOSITY MEASUREMENT DATA

Display uniformity, luminous intensity,
spectral emission and thermal sensitivity
measurement

supervised by	Tom Maloney
	Manager Development Engineering
performed by	Ken Moyers
	Technician Specialist
	Burroughs OEM Corporation

TEST REPORT

DATE 9-3-80
REPORT NO. TCM-1

TEST REQUEST NO. _____
TEST REQUESTED Display Uniformity

PRODUCT TYPE 5082-7730 Series 7 Segment Display
MFR Hewlett Packard
LOT / CODE NO. _____ QUANTITY 3
REASON FOR TEST Special Request - Human Factors Study

TEST EQUIPMENT Gamma Scientific photometer, Model 2020-1, Serial No. 1155;
PM tube type 100, Serial No. 73756

TEST CONDITIONS / METHOD The test was performed using the Visual Experiment
Program Hardware with the filter removed and operating at a display intensity
control setting, as requested, at 3/16. The data from this experiment is
a relative reading to obtain the output uniformity between digits.

Data:

Digit Position	Char. Displayed	Run No. 1		
		Active Segments	Output Digit	Average Per Seg.
Right	7	3	990	330
Middle	4	4	1230	310
Left	3	5	1635	327

Run No. 2				
Right	7	3	995	332
Middle	4	4	1260	315
Left	3	5	1640	328

Avg. Seg. Variation = 324
+ 8 or +2%
- 14 -4%

PERFORMED BY K. Moyers
REVIEWED BY T. Maloney

TEST REPORT

DATE 9-3-80
REPORT NO. TCM-2

TEST REQUEST NO. _____
TEST REQUESTED Display Segment Luminous Intensity

PRODUCT TYPE 5082-7730 series seven segment display
MFR Hewlett Packard
LOT / CODE NO. _____ QUANTITY 3
REASON FOR TEST Special request - Human Factors Study

TEST EQUIPMENT Gamma Scientific Photometer Model 2020-1, Serial No. 1155;
PM tube 100, Serial No. 73756; Standard Lamp No. 626

TEST CONDITIONS / METHOD The test was performed using the Visual Experiment Program
Hardware with the filter removed and operating at a display intensity control setting,
as requested of 3/16. The data was taken using a microcandela probe mounted
vertically above the segment under test. The photometer and probe were calibrated
against a Gamma Scientific Standard Lamp Serial # 626.

<u>DATA:</u>	<u>Digit Position</u>	<u>Segment</u>	<u>Intensity Micro-Candelas</u>
	Right	g	38
		b	39
		c	40
	Middle	a	40
		b	41
		c	41
	Left	a	38
		b	40
		c	42

PERFORMED BY K. Movers
REVIEWED BY T. Maloney

TEST REPORT

DATE 9-3-30
REPORT NO. TCM - 3

TEST REQUEST NO. _____
TEST REQUESTED Spectral Emission

PRODUCT TYPE 5082-7730 Series Seven Segment Display
MFR Hewlett Packard
LOT / CODE NO. _____ QUANTITY _____
REASON FOR TEST Special request - Human Factors Study

TEST EQUIPMENT Jarrel Ash 0.5 meter Monochrometer

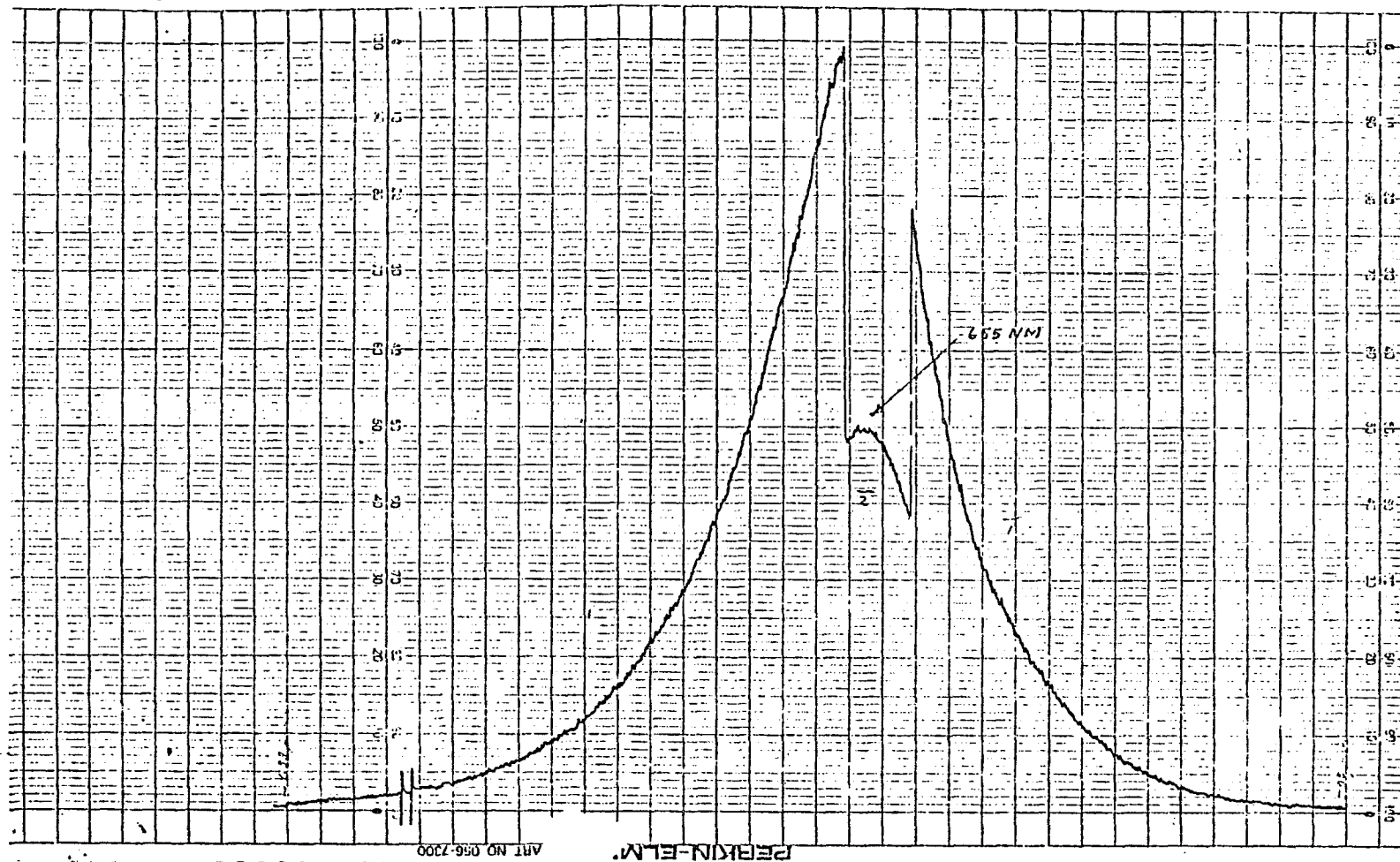
TEST CONDITIONS / METHOD The test was performed using the Visual Experiments Program Hardware with the filter removed and with the display operating at a display intensity control setting of 3/16.

Spectrometer Scanning Rate - 250 NM/Min.
PMT voltage setting - 547 volts
Chart speed - 80 nm/min.
Sensitivity - 10 mV/inch

The scale factor of the detector amplifier was reduced in half near the peak to keep the plot on scale.

The peak emission as shown on the attached plot was seen to peak at 655 nm.

PERFORMED BY K. Moyers
REVIEWED BY T. Maloney



Spectral Emission
Human Factors Study
LED Display

Test Report - TCM-3

TEST REPORT

DATE 9-3-80
REPORT NO. TCM-4

TEST REQUEST NO. _____
TEST REQUESTED Filter Transmission

PRODUCT TYPE Visual experiment program hardware filter
MFR _____
LOT / CODE NO. _____ QUANTITY 1
REASON FOR TEST Special request - Human Factors Study

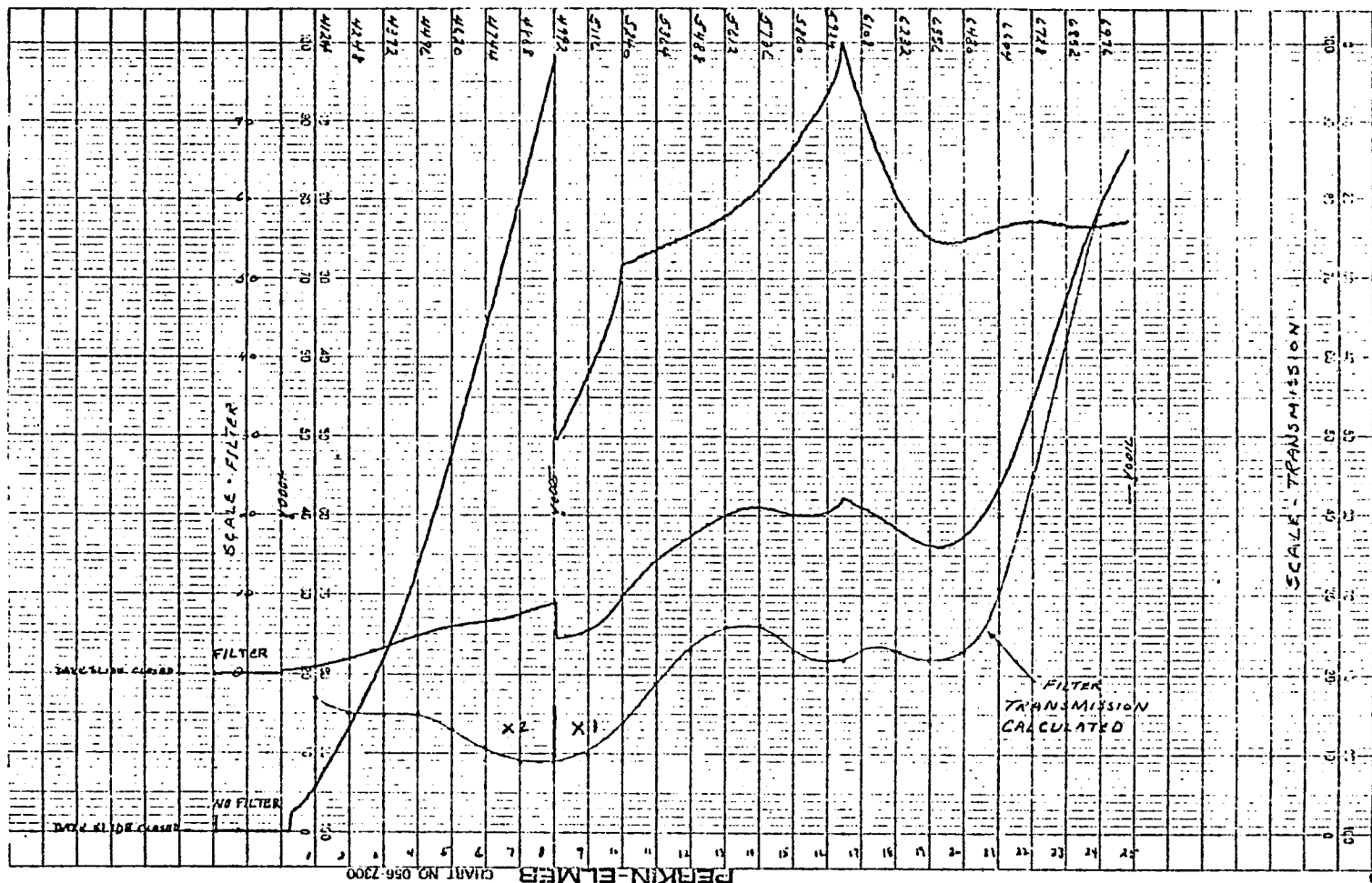
TEST EQUIPMENT Jarrel Ash 0.5 Meter Monochrometer, Gamma Scientific Standard
Light source No. 626

TEST CONDITIONS / METHOD The test was performed using the Jarrel Ash 0.5 meter spectrometer. Two scans were performed. The first was a scan of the standard lamp and then a second scan with the filter interposed between the lamp and the detector. The transmission is obtained by dividing the output of the second scan by the first at each wavelength of interest. The transmission is plotted on the attached chart.

Data:

<u>Chart</u> <u>Setting</u>	<u>Filter</u>	<u>No</u> <u>Filter</u>	<u>Trans.</u>	<u>Chart</u> <u>Setting</u>	<u>Filter</u>	<u>No</u> <u>Filter</u>	<u>Trans.</u>
1	1.0	5.8	.17	13	20.0	77.8	.26
2	2.0	13.5	.15	14	21.1	81.6	.26
3	3.3	22.0	.15	15	20.2	86.8	.23
4	4.9	33.6	.15	16	20.5	93.5	.22
5	6.1	47.8	.13	17	21.1	92.0	.23
6	6.7	63.7	.11	18	18.4	80.8	.23
7	7.5	80.7	.09	19	16.2	75.0	.22
8	4.5	49.5	.09	20	17.5	74.7	.23
9	5.6	58.0	.10	21	23.7	76.3	.31
10	10.0	71.8	.14	22	34.7	77.0	.45
11	14.3	73.5	.19	23	47.8	76.6	.62
12	17.4	75.6	.23	24	59.7	76.7	.78

PERFORMED BY K. Moyers
REVIEWED BY T. Maloney



Filter Spectral Transmission
Human Factors Study

TEST REPORT

DATE 12-12-80

REPORT NO. TCM-5

TEST REQUEST NO. _____

TEST REQUESTED Filter Transmission to LED Spectral Emission

PRODUCT TYPE Visual Experiment Program Hardware Filter

MFR _____

LOT / CODE NO. _____ QUANTITY 1

REASON FOR TEST Special Request - Human Factors Study

TEST EQUIPMENT Gamma Scientific Model DR-14 Photometer with a Model D-46A
photopically corrected PMT detector and a Model 700-10-37 Photomicroscope equipped
with a .006 inch diameter probe.

TEST CONDITIONS / METHOD The test was performed by focusing the microscope
probe on segment C of the middle digit with a display intensity control setting
of 3/16. With the filter removed the sensitivity of the detector amplifier was
adjusted to read 100. When the filter was placed on the display unit, the output
was reduced to a reading of 27 for an average LED emission filter transmission
of 27%.

PERFORMED BY K. Moyers

REVIEWED BY T. Maloney

TEST REPORT

DATE 12-12-80

REPORT NO. TCM-6

TEST REQUEST NO. _____

TEST REQUESTED Display Thermal Sensitivity

PRODUCT TYPE Visual Experiment Program Hardware

MFR _____

LOT / CODE NO. _____ QUANTITY 1

REASON FOR TEST Special Request - Human Factors Study

TEST EQUIPMENT Gamma Scientific Model DR-14 Photometer with a Model D-46A photopically corrected PMT detector and a Model 700-10-37 Photomicroscope equipped with a .006 inch diameter probe.

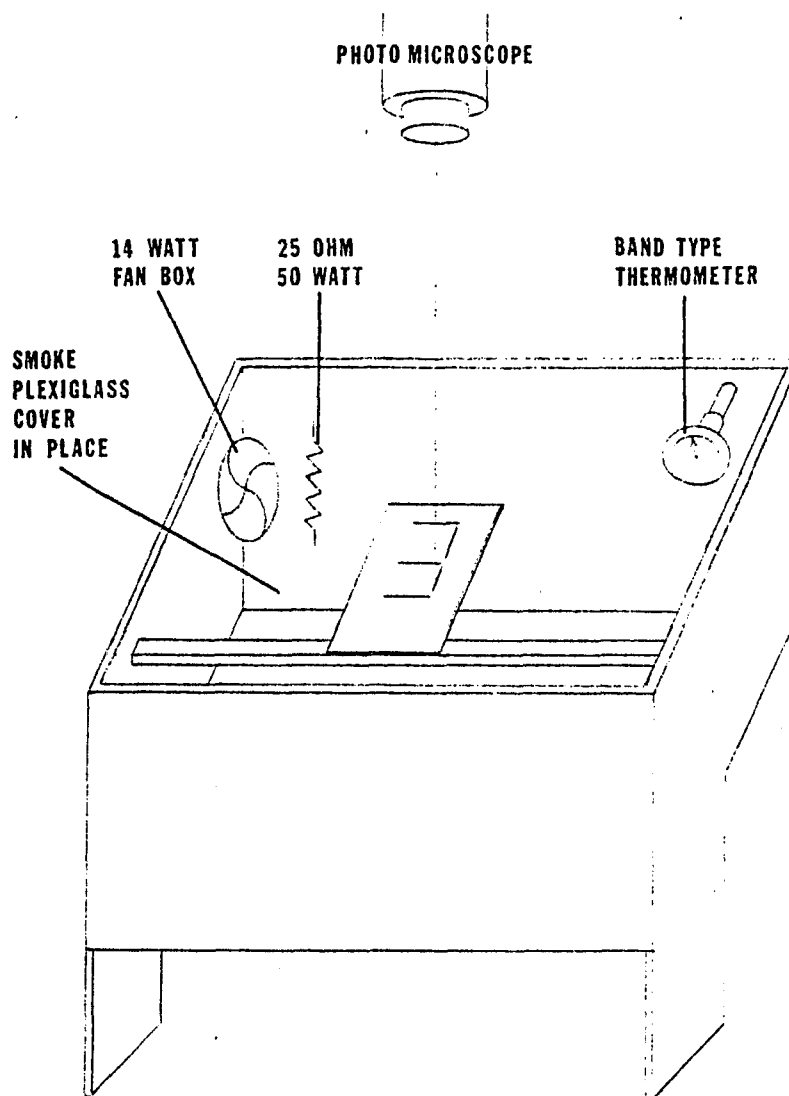
TEST CONDITIONS / METHOD The Hardware was modified slightly by adding a fan, an auto-transformer controlled resistor heater and a thermometer. The probe was focused on segment C of the middle digit with a display intensity control setting of 3/16. The temperature within the box was stabilized for five minutes at five temperatures from 20 - 41°C. The output was recorded at each temperature. Attached is a sketch of the apparatus and a plot of the data.

<u>Data:</u>	<u>Temp. °C</u>	<u>Output</u>
	20	100
	25	109
	31	120
	35	130
	41	133

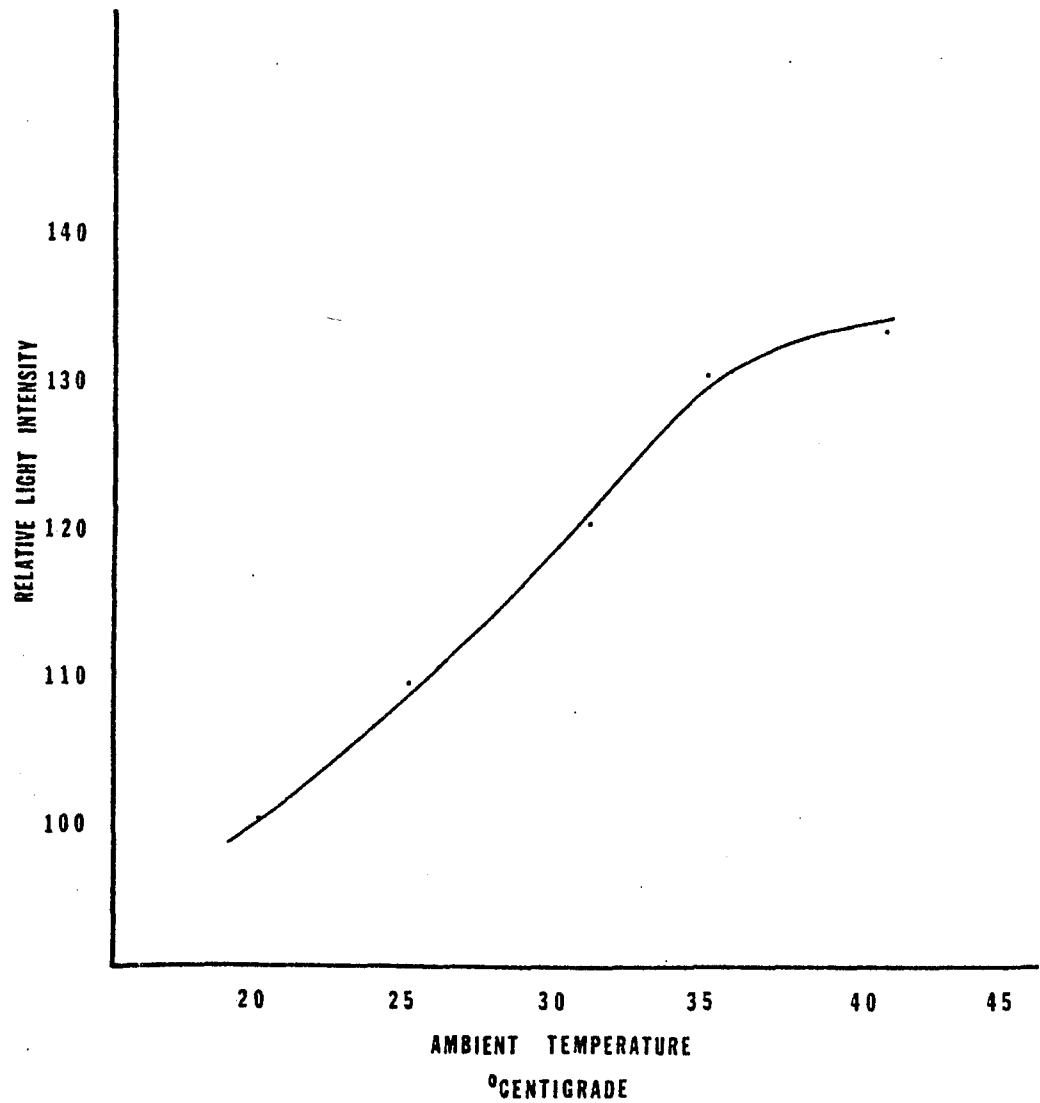
PERFORMED BY K. Moyers

REVIEWED BY T. Maloney

**THERMAL SENSITIVITY
TEST
VISUAL EXPERIMENT PROGRAM HARDWARE**



RELATIVE LIGHT INTENSITY
VS
AMBIENT TEMPERATURE



NEUROPSYCHOLOGICAL TEST BATTERY REFERENCES

- Arthur, G. A. Point Scale of Performance Tests. Rev. Form II. New York: Psychological Corporation, 1947.
- Borod, J. C., Goodglass, H., & Kaplan, E. Normative data on Boston Diagnostic Aphasia Examination, Parietal Lobe Battery and the Boston Naming Test. Journal of Clinical Neuropsychology, 1980, 2, 209-217.
- Centofanti, C. C., & Smith, A. Face-Hand Sensory Test. University of Michigan Neuropsychology Laboratory, Ann Arbor, Michigan, 1974.
- Coren, S., Porac, C., & Duncan, P. A behaviorally validated self-report inventory to assess four types of lateral preference. Journal of Clinical Neuropsychology, 1979, 1, 55-64.
- Gray, W. S. Gray Oral Reading Test. Form A. Indianapolis, Indiana: The Bobbs-Merrill Company, Inc., 1963.
- Jastak, J. F., & Jastak, S. R. The Wide Range Achievement Test Manual. Wilmington, Delaware: Guidance Associates, 1965.
- Kaplan, E., Goodglass, H., & Weintraub, S. Boston Naming Test. Experimental Edition. Boston Veterans Administration Hospital, Boston, Massachusetts, 1976.
- Lezak, M. D. Neuropsychological assessment. New York: Oxford University Press, 1976.
- Oldfield, R. C. The assessment and analysis of handedness: The Edinburgh inventory. Neuropsychologia, 1971, 9, 97-113.
- Osterrieth, P. A. Le test de copie d'une figure complexe. Archives de Psychologie, 1944, 30, 206-256.
- Raven, J. C. Guide to the Standard Progressive Matrices. London: H. K. Lewis, 1960.
- Smith, A. Neuropsychological testing in neurological disorders. In W. J. Friedlander (Ed.), Advances in neurology (Vol. 7). New York: Raven Press, 1975.
- Spreen, O., & Benton, A. Sentence Repetition Test: Administration, scoring and preliminary norms. University of Victoria Neuropsychology Laboratory, Victoria, British Columbia, 1963.
- Tiffin, J. Purdue Pegboard Examiner Manual. Lafayette, Indiana: Lafayette Instrument Co., 1948.

Wechsler, D. Wechsler Adult Intelligence Scale. New York:
Psychological Corporation, 1955.

Woodcock, R. W. Woodcock Reading Mastery Tests Manual. Form A.
Circle Pines, Minnesota: American Guidance Service, Inc., 1973.

REFERENCE LIST

- Ackerman, P. T., Dykman, R. A., & Peters, J. E. Teenage status of hyperactive and non-hyperactive learning disabled boys. American Journal of Orthopsychiatry, 1977, 47, 577-596.
- Ajax, E. T., Schenkenberg, T., & Kosteljanetz, M. Alexia without agraphia and the inferior splenium. Neurology, 1977, 27, 685-688.
- Altus, G. T. A WISC profile for retarded readers. Journal of Consulting Psychology, 1956, 20, 155-156.
- Asanuma, H., & Okuda, O. Effects of transcallosal volleys on pyramidal tract cell activity of cat. Journal of Neurophysiology, 1962, 25, 198-208.
- Bakker, D. J. Temporal order perception and reading retardation. In D. J. Bakker & P. Satz (Eds.), Specific reading disability: Advances in theory and method. Rotterdam: Rotterdam University Press, 1970.
- Bakker, D. J. Temporal order in disturbed reading development and neurological aspects in normal and reading-retarded children. Rotterdam: Rotterdam University Press, 1972.
- Bakker, D. J., Smink, T., & Reitsma, P. Ear dominance and reading ability. Cortex, 1973, 9, 301-312.
- Barlow, I. H. Lateral dominance characteristics and reading achievement in the first grade. Journal of Psychology, 1963, 55, 323-328.
- Bannatyne, A. D. The color phonics system. In J. Money & G. Schiffman (Eds.), The disabled reader: Education of the dyslexic child. Baltimore: The John Hopkins University Press, 1966.
- Barlow, J. S. Autocorrelation and cross-correlation technique in EEG analysis. Electroencephalography and Clinical Neurophysiology, 1961, Supplement, 20, 31-36.
- Basser, L. S. Hemiplegia of early onset and the faculty of speech with special reference to the effects of hemispherectomy. Brain, 1962, 85, 427-460.
- Bastian, H. C. A treatise on aphasia and other speech defects. New York: Appleton, 1898.
- Beaumont, J. G. Handedness and hemisphere function. In S. J. Dimond & J. G. Beaumont (Eds.), Hemisphere function in the human brain. New York: Halsted Press, 1974.

- Belmont, L., & Birch, H. G. Lateral dominance, lateral awareness and reading disability. Child Development, 1965, 36, 57-71.
- Belmont, L., & Birch, H. G. The intellectual profile of retarded readers. Perceptual and Motor Skills, 1966, 22, 787-816.
- Benson, D. & Geschwind, N. The alexias. In P. J. Vinken & G. W. Bruyn (Eds.), Handbook of clinical neurology (Vol. 4). New York: John Wiley & Sons, 1969.
- Benton, A. L. Developmental dyslexia: Neurological aspects. In W. J. Friedlander (Ed.), Advances in neurology (Vol. 7). New York: Raven Press, 1975.
- Benton, A. Some conclusions about dyslexia. In A. L. Benton & D. Pearl (Eds.), Dyslexia: An appraisal of current knowledge. New York: Oxford University Press, 1978.
- Berlucchi, G. Callosal activity in unrestrained, unanesthetized cats. Archives Italiennes de Biologie, 1965, 103, 623-634.
- Berlucchi, G. Electroencephalographic studies in "split-brain" cats. Electroencephalography and Clinical Neurophysiology, 1966, 20, 348-356.
- Berlucchi, G. Anatomical and physiological aspects of visual functions of corpus callosum. Brain Research, 1972, 37, 371-392.
- Berlucchi, G. Cerebral dominance and interhemispheric communication in normal man. In F. O. Schmitt & F. G. Worden (Eds.), The neurosciences: Third study program. Cambridge, Massachusetts: MIT Press, 1974.
- Berlucchi, G., Brizzolara, D., Marzi, C. A., Rizzolatti, G., & Umiltà, C. Can lateral asymmetries in attention explain interfield differences in visual perception? Cortex, 1974, 10, 177-185.
- Berlucchi, G., Gazzaniga, M. S., & Rizzolatti, G. Microelectrode analysis of transfer of visual information by the corpus callosum. Archives Italiennes de Biologie, 1967, 105, 583-596.
- Boder, E. Developmental dyslexia: A new diagnostic approach based on the identification of three subtypes. Journal of School Health, 1970, 40, 289-290.
- Boder, E. Developmental dyslexia: Prevailing diagnostic concepts and a new diagnostic approach. In H. Myklebust (Ed.), Progress in learning disabilities (Vol. 2). New York: Grune & Stratton, 1971.
- Boder, E. Developmental dyslexia: A diagnostic approach based on three atypical reading-spelling patterns. Developmental medicine and Child Neurology, 1973, 15, 663-687.

- Bogen, J. E. The callosal syndrome. In K. M. Heilman & E. Valenstein (Eds.), Clinical neuropsychology. New York: Oxford University Press, 1979.
- Bogen, J. E., & Bogen, G. M. The other side of the brain III: The corpus callosum and creativity. Bulletin of the Los Angeles Neurological Societies, 1969, 34, 191-208.
- Bouma, H., & Legein, CH. P. Foveal and parafoveal recognition of letters and words by dyslexics and by average readers. Neuropsychologia, 1977, 15, 69-80.
- Bouma, H., & Legein, CH. P. Dyslexia: A specific recoding deficit? An analysis of response latencies for letters and words in dyslexics and in average readers. Neuropsychologia, 1980, 18, 285-298.
- Brazier, M. A. B., & Barlow, J. S. Some applications of correlation analysis to clinical problems in electroencephalography. Electroencephalography and Clinical Neurophysiology, 1956, 8, 325-331.
- Brazier, M. A. B., & Casby, J. U. Cross-correlation and auto-correlation studies in electroencephalographic potentials. Electroencephalography and Clinical Neurophysiology, 1952, 4, 201-211.
- Bremer, F. Physiology of the corpus callosum. Research Publications of the Association for Research in Nervous and Mental Disease, 1958, 36, 424-448.
- Bremer, F. The corpus callosum in cerebral dynamics. Experientia, 1966, 22, 201-208.
- Bremer, F. An aspect of the physiology of corpus callosum. Electroencephalography and Clinical Neurophysiology, 1967, 22, 391. (abstract)
- Bremer, F., & Stoupel, N. Recherche d'une participation du corps calleux au mécanisme de la synergie bioélectrique des hémisphères cérébraux. Journal de Physiologie, 1957, 49, 66-67.
- Bryden, M. P. Tachistoscopic recognition, handedness and cerebral dominance. Neuropsychologia, 1965, 3, 1-8.
- Bryden, M. P. Laterality effects in dichotic listening: Relations with handedness and reading ability in children. Neuropsychologia, 1970, 8, 443-450.
- Bryden, M. P., Dick, A. O., & Mewhort, D. J. K. Tachistoscopic recognition of number sequences. Canadian Journal of Psychology, 1968, 22, 52-59.
- Burks, H. F., & Bruce, P. The characteristics of poor and good readers as disclosed by the Wechsler Intelligence Scale for Children. Journal of Educational Psychology, 1955, 46, 486-493.

- Burt, C. The backward child (3rd ed.). London: University of London Press, 1950.
- Butler, S. R., & Norrsell, U. Vocalization possibly initiated by the major hemisphere. Nature, 1968, 220, 793-794.
- Carlson, J., Netley, C., Hendrick, E. B., & Prichard, J. S. A reexamination of intellectual disabilities in hemispherectomized patients. Transactions of the American Neurological Association, 1968, 93, 198-201.
- Carmicael, A. E. The current status of hemispherectomy for infantile hemiplegia. Clinical Proceedings of the Children's Hospital, 1966, 22, 285-293.
- Choudhury, B. P., Whitteridge, D., & Wilson, M. E. The function of the callosal connections of the visual cortex. Quarterly Journal of Experimental Physiology, 1965, 50, 214-219.
- Claes, E. Contribution à l'étude physiologique de la fonction visuelle. I. Analyse oscillographique de l'activité spontanée et sensorielle de l'aire visuelle corticale chez le chat non anesthésié. Archives Internationales de Physiologie et de Biochimie, 1939, 48, 181-237.
- Cohen, J., & Cohen, P. Applied multiple regression/correlational analysis for the behavioral sciences. New York: John Wiley & Sons, 1975.
- Cohen, N. J., & Douglas, V. I. Characteristics of the orienting response in hyperactive and normal children. Psychophysiology, 1972, 9, 238-245.
- Coleman, R. I., & Deutsch, C. P. Lateral dominance and right-left discrimination: A comparison of normal and retarded readers. Perceptual and Motor Skills, 1964, 19, 43-50.
- Cooper, R., & Mundy-Castle, A. C. Spatial and temporal characteristics of the alpha rhythm: A toposcopic analysis. Electroencephalography and Clinical Neurophysiology, 1960, 12, 153-165.
- Corcoran, D. W. J. Doubling the rate of signal presentation in a vigilance task during sleep deprivation. Journal of Applied Psychology, 1963, 47, 412-415.
- Corkin, S. Serial-ordering deficits in inferior readers. Neuropsychologia, 1974, 12, 347-354.
- Cragg, B. G., & Ainsworth, A. The topography of the afferent projections in the circumstriate cortex of the monkey studied by the Nauta method. Vision Research, 1969, 9, 733-747.

- Crinella, F. M., Beck, F. W., & Robinson, J. W. Unilateral dominance is not related to neuropsychological integrity. Child Development, 1971, 42, 2033-2054.
- Critchley, M. Speech and speech-loss in relation to the duality of the brain. In V. B. Mountcastle (Ed.), Interhemispheric relations and cerebral dominance. Baltimore, Maryland: John Hopkins University Press, 1962.
- Critchley, M. The dyslexic child. Springfield, Illinois: Charles C. Thomas, 1970.
- Critchley, M. The parietal lobes. New York: Hafner Publishing Company, 1971. (Originally published, 1953.)
- Critchley, M., & Critchley, E. A. Dyslexia defined. Springfield, Illinois: Charles C. Thomas, 1978.
- Damasio, A. R. Varieties and significance of the alexias. Archives of Neurology, 1977, 34, 325-326.
- Davenport, H.A., & Ranson, S. W. Ratio of cells to fibers and of myelinated to unmyelinated fibers in spinal nerve roots. American Journal of Anatomy, 1931, 49, 193.
- Davis, R., & Schmidt, V. Timing and transfer of information between hemispheres in man. Acta Psychologica, 1971, 35, 335-346.
- Davis, R., & Schmidt, V. Visual and verbal coding in the interhemispheric transfer of information. Acta Psychologica, 1973, 37, 229-240.
- De Crinis, M. Aufbau und abbau der grosshirnleistungen und ihre anatomischen grundlagen. Berlin: Karger, 1934.
- De Hirsch, K., Jansky, J. J., & Langford, W. S. Predicting reading failure: A preliminary study. New York: Harper & Row, 1966.
- Dejerine, M. J. Sur un cas de cécité verbale avec agraphie, suivi d'autopsie. Comptes Rendus Hebdomadaires Séances et Mémoires de la Societe de Biologie, 1891, 3, 197-201.
- Dejerine, M. J. Différentes variétés de cécité verbale. Comptes Rendus Hebdomadaires Séances et Mémoires de la Societe de Biologie, 1892, 4, 61-90.
- Denckla, M. B. Minimal brain dysfunction and dyslexia: Beyond diagnosis by exclusion. In M. E. Blaw, I. Rapin & M. Kinsbourne (Eds.), Child neurology. New York: Spectrum Publications, 1977.
- Denckla, M. B. Childhood learning disabilities. In K. M. Heilman & E. Valenstein, Clinical neuropsychology. New York: Oxford University Press, 1979.

- Denckla, M. B., & Rudel, R. Naming of object-drawings by dyslexic and other learning disabled children. Brain and Language, 1976a, 3, 1-15.
- Denckla, M., & Rudel, R. Rapid "automatized" naming (R.A.N.): Dyslexia differentiated from other learning disabilities. Neuropsychologia, 1976b, 14, 471-479.
- Dennis, M., & Whitaker, H. A. Language acquisition following hemidecortication: Linguistic superiority of the left over the right hemisphere. Brain and Language, 1976, 3, 404-433.
- Dimond, S. J. Hemispheric refractoriness and control of reaction time. Quarterly Journal of Experimental Psychology, 1970, 22, 610-617.
- Dimond, S. J. Hemisphere function and word registration. Journal of Experimental Psychology, 1971, 87, 183-186.
- Dimond, S. J. The double brain. Edinburgh, Scotland: Churchill Livingstone, 1972.
- Dimond, S. J. Depletion of attentional capacity after total commissurotomy in man. Brain, 1976, 99, 347-356.
- Dimond, S. J. Vigilance and split-brain research. In R. R. Mackie (Ed.), Vigilance: Theory, operational performance and physiological correlates. New York: Plenum Press, 1977.
- Dimond, S. J. Performance by split-brain humans on lateralized vigilance tasks. Cortex, 1979, 15, 43-50.
- Dimond, S. J., & Beaumont, J. G. Use of the two hemispheres to increase brain capacity. Nature, 1971a, 232, 270-271.
- Dimond, S., & Beaumont, G. Hemisphere function and vigilance. Quarterly Journal of Experimental Psychology, 1971b, 23, 443-448.
- Dimond, S. J., & Beaumont, J. G. Hemispheric control of hand function in the human brain. Acta Psychologica, 1972a, 36, 32-36.
- Dimond, S. J., & Beaumont, J. G. Processing in perceptual integration between and within the cerebral hemispheres. British Journal of Psychology, 1972b, 63, 509-514.
- Dimond, S. J., & Beaumont, J. G. Difference in the vigilance performance of the right and left hemispheres. Cortex, 1973, 9, 259-265.
- Dimond, S. J., & Beaumont, J. G. Experimental studies of hemisphere function. In S. J. Dimond & J. G. Beaumont (Eds.), Hemisphere function in the human brain. New York: Halsted Press, 1974.
- Doehring, D. G. Patterns of impairment in specific reading disability. Bloomington, Indiana: Indiana University Press, 1968.

- Doty, R. W. Electrical stimulation of the brain in behavioral cortex. Annual Review of Psychology, 1969, 20, 289-320.
- Douglas, V. I. Perceptual and cognitive factors as determinants of learning disabilities: A review chapter with special emphasis on attentional factors. In R. M. Knights & D. J. Bakker (Eds.), The neuropsychology of learning disorders: Theoretical approaches. Baltimore, Maryland: University Park Press, 1976.
- Doyle, R. B., Anderson, R. P., & Halcomb, C. G. Attention deficits and the effects of visual distraction. Journal of Learning Disabilities, 1976, 9, 59-65.
- Drozdenki, R. G. The visual evoked potential and the process of selective attention under conditions of simultaneous bilateral stimulation. International Journal of Neuroscience, 1979, 8, 225-232.
- Duane, D. D. A neurologic afterview of specific language disability for the non-neurologist. Bulletin of the Orton Society, 1974, 24, 5-36.
- Dykman, R. A., Ackerman, P. T., Clements, S. D., & Peters, J. E. Specific learning disabilities: An attentional deficit syndrome. In H. R. Myklebust (Ed.), Progress in learning disabilities (Vol. 2). New York: Grune & Stratton, 1971.
- Dykman, R. A., Ackerman, M. A., & Oglesby, D. M. Selective and sustained attention in hyperactive, learning-disabled, and normal boys. The Journal of Nervous and Mental Disease, 1979, 167, 288-297.
- Eccles, J. C. The understanding of the brain. New York: McGraw-Hill, 1973.
- Efron, R. Temporal perception, aphasia, and déjà vu. Brain, 1963, 86, 403-424.
- Eidelberg, E. Callosal and non-callosal connexions between the sensorimotor cortices in cat and monkey. Electroencephalography and Clinical Neurophysiology, 1969, 26, 557-564.
- Eisenberg, L. Definitions of dyslexia: Their consequences for research and policy. In A. L. Benton & D. Pearl (Eds.), Dyslexia: An appraisal of current knowledge. New York: Oxford University Press, 1978.
- Eisenberg, L. Reading disorders: Strategies for recognition and management. Bulletin of the Orton Society, 1979, 29, 39-55.
- Elithorn, A., & Barnett, T. J. Apparent individual differences in channel capacity. Acta Psychologica, 1967, 27, 75-83.
- Ellenberg, L., & Sperry, R. W. Capacity for holding sustained attention following commissurotomy. Cortex, 1979, 15, 421-438.

- Ellenberg, L., & Sperry, R. W. Lateralized division of attention in the commissurotomed and intact brain. Neuropsychologia, 1980, 18, 411-418.
- Faglioni, P., Scott, G., & Spinnler, H. Impaired recognition of written letters following unilateral hemispheric damage. Cortex, 1969, 5, 120-133.
- Falconer, M. A. The treatment of encephalotrigeminal angiomas (Sturge-Weber disease) by hemispherectomy. Journal of Neurology and Psychiatry, 1960, 23, 81.
- Finucci, J. M., Guthrie, J. T., Childs, A. L., Abbey, H., & Childs, B. The genetics of specific reading disability. Annals of Human Genetics, 1976, 40, 1-23.
- Fischer, F. W., Liberman, I. S., Shankweiler, D. Reading reversals and developmental dyslexia: A further study. Cortex, 1978, 14, 496-510.
- Fisk, J. L., & Rourke, B. P. Identification of subtypes of learning-disabled children at three age levels: A neuropsychological multivariate approach. Journal of Clinical Neuropsychology, 1979, 1, 289-310.
- Flechsigs, P. Developmental (myelogenic) localisation of the cerebral cortex in the human subject. Lancet, 1901, 2, 1027-1029.
- Frauenheim, J. G. Academic achievement characteristics of adult males who were diagnosed as dyslexic in childhood. Journal of Learning Disabilities, 1978, 2, 476-483.
- Galaburda, A. M., LeMay, M., Kempler, T. L., & Geschwind, N. Right-left asymmetries in the brain: Structural differences between the hemispheres may underlie cerebral dominance. Science, 1978, 199, 852-856.
- Galaburda, A. M., Sanides, F., & Geschwind, N. Human brain: Cytoarchitectonic left-right asymmetries in the temporal speech region. Archives of Neurology, 1978, 35, 812-817.
- Galifret-Granjon, N., & Ajuriaguerra, J. Trouble de l'apprentissage de la lecture et dominance latérale. Encéphale, 1951, 3, 385-398.
- Galin, D. EEG studies of lateralization of verbal processes. In C. L. Ludlow & M. E. Doran-Quine (Eds.), The neurological bases of language disorders in children: Methods and directions for research (NINCDS Monograph No. 22, U. S. Department of Health, Education, and Welfare). Washington, D. C.: U. S. Government Printing Office, 1979.
- Galin, D., Diamond, R., & Herron, J. Development of crossed and uncrossed tactile localization on the fingers. Brain and Language, 1977, 4, 588-590.

- Galín, D., Johnstone, J., Nakell, L., & Herron, J. Development of the capacity for tactile information transfer between hemispheres in normal children. Science, 1979, 204, 1330-1332.
- Gardner, W. J., Karnosh, L. J., McClure, C. C., Jr., & Gardner, A. K. Residual function following hemispherectomy for tumor and for infantile hemiplegia. Brain, 1955, 78, 487-502.
- Garey, L. J., Jones, E. G., & Powell, T. P. S. Interrelationships of striate and extrastriate cortex with the primary relay sites of the visual pathway. Journal of Neurology, Neurosurgery and Psychiatry, 1968, 31, 135-157.
- Gazzaniga, M. S. The bisected brain. New York: Appleton-Century-Crofts, 1970.
- Gazzaniga, M. S. Cerebral dominance viewed as a decision system. In S. J. Dimond & J. G. Beaumont (Eds.), Hemisphere function in the human brain. New York: Halsted Press, 1974.
- Gazzaniga, M. S., Bogen, J. E., & Sperry, R. W. Observations on visual perception after disconnection of the cerebral hemispheres in man. Brain, 1965, 88, 221-236.
- Gazzaniga, M. S., Hillyard, S. A. Language and speech capacity of the right hemisphere. Neuropsychologia, 1971, 9, 273-280.
- Gazzaniga, M. S., & LeDoux, J. E. The integrated mind. New York: Plenum Press, 1978.
- Gazzaniga, M. S., Le Doux, J. E., & Wilson, D. H. Language, praxis, and the right hemisphere: Clues to some mechanisms of consciousness. Neurology, 1977, 27, 1144-1147.
- Gazzaniga, M. S., & Sperry, R. W. Language after section of the cerebral commissures. Brain, 1967, 90, 131-148.
- Geffen, G., Bradshaw, J. L., & Wallace, G. Interhemispheric effects on reaction time to verbal and nonverbal visual stimuli. Journal of Experimental Psychology, 1971, 87, 415-422.
- Geschwind, N. The anatomy of acquired disorders of reading. In J. Money (Ed.), Reading disability: Progress and research needs in dyslexia. Baltimore: John Hopkins Press, 1962.
- Geschwind, N. Disconnection syndromes in animals and man. Brain, 1965, 88, 237-294, 585-644.
- Geschwind, N. Asymmetries of the brain--New developments. Bulletin of the Orton Society, 1979, 29, 67-83.
- Geschwind, N., & Levitsky, W. Human brain: Left-right asymmetries in temporal speech region. Science, 1968, 161, 186-187.

- Heilman, K. M., & Van Den Abell, T. Right hemispheric dominance for mediating cerebral activation. Neuropsychologia, 1979, 17, 315-321.
- Heilman, K. M., & Van Den Abell, T. Right hemisphere dominance for attention: The mechanism underlying hemispheric asymmetries of inattention (neglect). Neurology, 1980, 30, 327-330.
- Heimer, L., Ebner, F. F., & Nauta, W. J. H. A note on the termination of commissural fibers in the neocortex. Brain Research, 1967, 5, 171-177.
- Hellige, J. B., & Webster, R. Right hemisphere superiority for initial stages of letter processing. Neuropsychologia, 1979, 17, 653-659.
- Heron, W. Perception as a function of retinal locus and attention. American Journal of Psychology, 1957, 70, 38-48.
- Hewitt, W. The development of the human corpus callosum. Journal of Anatomy, 1962, 96, 355-358.
- Hines, D., & Satz, P. Superiority of right visual half-fields in right-handers for recall of digits presented at varying rates. Neuropsychologia, 1971, 9, 21-25.
- Hines, D., & Satz, P. Cross-modal asymmetries in perception related to asymmetry in cerebral function. Neuropsychologia, 1974, 12, 239-247.
- Hines, D., Satz, P., Schell, B., & Schmidlin, S. Differential recall of digits in the left and right visual half-fields under free and fixed order of recall. Neuropsychologia, 1969, 7, 13-22.
- Hinshelwood, J. Congenital word-blindness. Lancet, 1900, 1, 1506-1508.
- Hinshelwood, J. Congenital word blindness. London: H. K. Lewis & Co., 1917.

- Hirsh, I. J. Auditory perception of temporal order. The Journal of the Acoustical Society of America, 1959, 31, 759-767.
- Hirsh, I. J., & Sherrick, C. E. Perceived order in different sense modalities. Journal of Experimental Psychology, 1961, 64, 1-19.
- Hodgson, W. R. Audiological report of a patient with left hemispherectomy. Journal of Speech and Hearing Disorders, 1967, 32, 39-45.
- Hoovey, Z. B., Heinemann, V., & Creutzfeldt, O. D. Inter-hemispheric "synchrony" of alpha waves. Electroencephalography and Clinical Neurophysiology, 1972, 32, 337-347.
- Hubel, D. H., & Wiesel, T. N. Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. Journal of Physiology, 1962, 160, 106-154.
- Hubel, D. H., & Wiesel, T. N. Receptive fields of cells in striate cortex of very young, visually inexperienced kittens. Journal of Neurophysiology, 1963, 26, 994-1002.
- Hubel, D. H., & Wiesel, T. N. Receptive fields and functional architecture in non-striate visual areas (18 and 19) of the cat. Journal of Neurophysiology, 1965, 28, 229-289.
- Hubel, D. H., & Weisel, T. N. Cortical and callosal connections concerned with the vertical meridian of visual fields in the cat. Journal of Neurophysiology, 1967, 30, 1561-1573.
- Huelsman, C. B. The WISC subtest syndrome for disabled readers. Perceptual and Motor Skills, 1970, 30, 535-550.
- Hughes, A., & Wilson, M. E. Callosal termination along the boundary between visual areas I and II in the rabbit. Brain Research, 1969, 12, 19-25.

- Goodglass, H., & Barton, M. Handedness and the differential perception of verbal stimuli in left and right visual fields. Perceptual and Motor Skills, 1963, 17, 851-854.
- Gordon, H. W., & Carmon, A. Transfer of dominance in speed of verbal response to visually presented stimuli from right to left hemisphere. Perceptual and Motor Skills, 1976, 42, 1091-1100.
- Gowers, W. R. Lectures on the diagnosis of diseases of the brain. London: Churchill, 1887.
- Greenblatt, S. H. Neurosurgery and the anatomy of reading: A practical review. Neurosurgery, 1977, 1, 6-15.
- Gross, K., Rothenberg, S., Schottenfield, S., & Drake, W. E. Duration thresholds for letter identification in left and right visual fields for normal and reading-disabled children. Neuropsychologia, 1978, 16, 709-715.
- Hakes, D. T., Evans, J. S., & Studdert-Kennedy, M. The right ear advantage in speech perception: Effects on monotic and dichotic presentation on identification latency and accuracy. Paper presented at the Fifteenth Annual Meeting of the Psychonomic Society, Boston, November 1974.
- Harcum, E. R. Effects of symmetry on the perception of tachistoscopic patterns. American Journal of Psychology, 1964, 77, 600-606.
- Harris, A. J. Lateral dominance, directional confusion and reading disability. Journal of Psychology, 1957, 44, 283-294.
- Hatta, T., & Dimond, S. J. Comparison of lateral differences for digit and random form recognition in Japanese and westerners. Journal of Experimental Psychology, 1980, 6, 368-374.
- Heath, C. J., & Jones, E. G. Connexions of area 17 and the lateral suprasylvian area of the visual cortex of the cat. Brain Research, 1970, 19, 302-305.
- Hecaen, H., & Albert, M. L. Human neuropsychology. New York: John Wiley & Sons, 1978.
- Heilman, K. M., Rothi, L., Campanella, D., & Wolfson, S. Wernicke's and global aphasia without alexia. Archives of Neurology, 1979, 36, 129-133.
- Heilman, K. M., & Van Den Abell, T. Right hemispheric dominance for mediating cerebral activation. Neuropsychologia, 1979, 17, 315-321.
- Heilman, K. M., & Van Den Abell, T. Right hemisphere dominance for attention: The mechanism underlying hemispheric asymmetries of inattention (neglect). Neurology, 1980, 30, 327-330.

- Heimer, L., Ebner, F. F., & Nauta, W. J. H. A note on the termination of commissural fibers in the neocortex. Brain Research, 1967, 5, 171-177.
- Hellige, J. B., & Webster, R. Right hemisphere superiority for initial stages of letter processing. Neuropsychologia, 1979, 17, 653-659.
- Heron, W. Perception as a function of retinal locus and attention. American Journal of Psychology, 1957, 70, 38-48.
- Hewitt, W. The development of the human corpus callosum. Journal of Anatomy, 1962, 96, 355-358.
- Hines, D., & Satz, P. Superiority of right visual half-fields in right-handers for recall of digits presented at varying rates. Neuropsychologia, 1971, 9, 21-25.
- Hines, D., & Satz, P. Cross-modal asymmetries in perception related to asymmetry in cerebral function. Neuropsychologia, 1974, 12, 239-247.
- Hines, D., Satz, P., Schell, B., & Schmidlin, S. Differential recall of digits in the left and right visual half-fields under free and fixed order of recall. Neuropsychologia, 1969, 7, 13-22.
- Hinshelwood, J. Congenital word-blindness. Lancet, 1900, 1, 1506-1508.
- Hinshelwood, J. Congenital word blindness. London: H. K. Lewis & Co., 1917.
- Hirsh, I. J. Auditory perception of temporal order. The Journal of the Acoustical Society of America, 1959, 31, 759-767.
- Hirsh, I. J., & Sherrick, C. E. perceived order in different sense modalities. Journal of Experimental Psychology, 1961, 64, 1-19.
- Hodgson, W. R. Audiological report of a patient with left hemispherectomy. Journal of Speech and Hearing Disorders, 1967, 32, 39-45.
- Hoovey, Z. B., Heinemann, V., & Creutzfeldt, O. D. Inter-hemispheric "synchrony" of alpha waves. Electroencephalography and Clinical Neurophysiology, 1972, 32, 337-347.
- Hubel, D. H., & Wiesel, T. N. Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. Journal of Physiology, 1962, 160, 106-154.
- Hubel, D. H., & Wiesel, T. N. Receptive fields of cells in striate cortex of very young, visually inexperienced kittens. Journal of Neurophysiology, 1963, 26, 994-1002.
- Hubel, D. H., & Wiesel, T. N. Receptive fields and functional architecture in non-striate visual areas (18 and 19) of the cat. Journal of Neurophysiology, 1965, 28, 229-289.

- Hubel, D. H., & Wiesel, T. N. Cortical and callosal connections concerned with the vertical meridian of visual fields in the cat. Journal of Neurophysiology, 1967, 30, 1561-1573.
- Huelsman, C. B. The WISC subtest syndrome for disabled readers. Perceptual and Motor Skills, 1970, 30, 535-550.
- Hughes, A., & Wilson, M. E. Callosal termination along the boundary between visual areas I and II in the rabbit. Brain Research, 1969, 12, 19-25.
- Hughes, J. R. Electroencephalographic and neurophysiological studies in dyslexia. In A. L. Benton, & D. Pearl (Eds.), Dyslexia: An appraisal of current knowledge. New York: Oxford University Press, 1978.
- Ingram, T. T. S. Specific developmental disorders of speech in children. Brain, 1959, 82, 450-467.
- Ingram, T. T. S. Pediatric aspects of specific developmental dysphasia, dyslexia and dysgraphia. Cerebral Palsy Bulletin, 1960, 2, 254-277.
- Ingram, T. T. S., Mason, A. W., & Blackburn, I. A. A retrospective study of 82 children with reading disability. Developmental Medicine and Child Neurology, 1970, 12, 271-281.
- Ironsides, R., & Guttmacher, M. Corpus callosum and its tumors. Brain, 1929, 52, 442-483.
- Jasper, H. H. Electroencephalography. In W. Penfield & H. H. Jasper (Eds.), Epilepsy and the functional anatomy of the human brain. Boston: Little Brown Co., 1954.
- Jerison, H. J. Vigilance: Biology, psychology, theory, and practice. In R. R. Mackie (Ed.), Vigilance: Theory, operational performance, and physiological correlates. New York: Plenum Press, 1977.
- Johnson, D., & Myklebust, H. Learning disabilities: Educational principles and practices. New York: Grune and Stratton, 1967.
- Johnson, O., & Harley, C. Handedness and sex differences in cognitive tests of brain laterality. Cortex, 1980, 16, 73-82.
- Jung, R. Summary of the conference. In V. V. Mountcastle (Ed.), Interhemispheric relations and cerebral dominance. Baltimore: John Hopkins Press, 1961.
- Kershner, J. R. Reading and laterality revisited. The Journal of Special Education, 1975, 9, 269-279.
- Kershner, J. R. Cerebral dominance in disabled readers, good readers, and gifted children: Search for a valid model. Child Development, 1977, 48, 61-67.

- Keynes, R. D. Ion channels in the nerve-cell membrane. Scientific American, 1979, 240(3), 126-132, 134-135.
- Kimura, D. Dual functional asymmetry of the brain in visual perception. Neuropsychologia, 1966, 4, 278-285.
- Kinsbourne, M. The cerebral basis of lateral asymmetries in attention. Acta Psychologica, 1970, 33, 193-201.
- Kinsbourne, M. Eye and head turning indicates cerebral localization. Science, 1972, 176, 539-541.
- Kinsbourne, M. Minimal brain dysfunction as a neurodevelopmental lag. Annals of the New York Academy of Science, 1972, 205, 268-273.
- Kinsbourne, M. Mechanisms of hemispheric interaction in man. In M. Kinsbourne & W. L. Smith (Eds.), Hemispheric disconnection and cerebral function. Springfield, Illinois: Charles C. Thomas, 1974a.
- Kinsbourne, M. Lateral interactions in the brain. In M. Kinsbourne & W. L. Smith (Eds.), Hemisphere disconnection and cerebral function. Springfield, Illinois: Charles C. Thomas, 1974b.
- Kinsbourne, M., & Warrington, E. K. Developmental factors in reading and writing backwardness. British Journal of Psychology, 1963, 54, 145-156.
- Klasen, E. The syndrome of specific dyslexia. Baltimore: University Park Press, 1972.
- Koppitz, E. M. Children with learning disabilities: A five year follow-up study. New York: Grune & Stratton, 1971.
- Krashen, S. Language and the left hemisphere. Working Papers in Phonetics, 1972, No. 24.
- Krashen, S. Lateralization, language, learning, and the critical period: Some new evidence. Language Learning, 1973, 23, 63-74.
- Krashen, S., & Harshman, R. Lateralization and the critical period. Working Papers in Phonetics, 1972, No. 22.
- Leisman, G. Conditioning variables in attentional handicaps. Neuropsychologia, 1973, 11, 199-205.
- Leisman, G. The relationship between saccadic eye movements and the alpha rhythm in attentionally handicapped patients. Neuropsychologia, 1974, 12, 201-210.
- Leisman, G., & Ashkenazi, M. Aetiological factors in dyslexia: IV. Cerebral hemispheres are functionally equivalent. International Journal of Neuroscience, 1980, 11, 157-164.

- Lenneberg, E. H. Biological foundations of language. New York: John Wiley, 1967.
- Lenneberg, E. H. Brain correlates of language. In F. O. Schmitt (Ed.), The neurosciences: Second study program. New York: The Rockefeller University Press, 1970.
- Lenneberg, E. H. Epilogue: Language, speech, and speakers, Neurosciences Research Program Bulletin, 1974, 12, 619-636.
- Levy-Agresti, J. Ipsilateral projection systems and minor hemisphere function in man after neocommissurotomy. Anatomical Record, 1968, 160, 384.
- Levy, J. Psychobiological implications of bilateral asymmetry. In S. J. Dimond & J. G. Beaumont (Eds.), Hemisphere function in the human brain. New York: Halsted Press, 1974.
- Levy, J. Manifestations and implications of shifting hemi-inattention in commissurotomy patients. In E. A. Weinstein & R. P. Friedland (Eds.), Advances in neurology (Vol. 18). New York: Raven Press, 1977.
- Levy, J., Nebes, R. D., & Sperry, R. W. Expressive language in the surgically separated minor hemisphere. Cortex, 1971, 7, 49-58.
- Levy, J., & Trevarthen, C. Perceptual, semantic and phonetic aspects of elementary language processes in split-brain patients. Brain, 1977, 100, 105-118.
- Levy, J., Trevarthen, C., & Sperry, R. W. Perception of bilateral chimeric figures following hemispheric disconnection. Brain, 1972, 95, 61-78.
- Liske, E., Hughes, H. M., & Stowe, D. E. Cross-correlation of human alpha activity: Normative data. Electroencephalography and Clinical Neurophysiology, 1967, 22, 429-436.
- Locke, S., & Yakovlev, P. I. Transcallosal connections of the cingulum of man. Archives of Neurology, 1965, 13, 471-476.
- Luttenberg, J. Contribution to the fetal ontogenesis of the corpus callosum in man. II. Folia Morphologica (Prague), 1965, 13, 136-144.
- Luttenberg, J. Contribution to the fetal ontogenesis of the corpus callosum in man. III. Myelination in the corpus callosum. Folia Morphologica (Prague), 1966, 14, 192-199.
- Lynn, J. G. Mimetic smiledness as related to handedness: An indicator of basic modes of human adaption. Archives of Neurology and Psychiatry, 1942, 47, 1060-1065.

- Macmeeken, A. M. Ocular dominance in relation to developmental aphasia. London: University of London Press, 1939.
- Magni, F., Melzack, R., & Smith, L. J. A stereotaxic method for sectioning the corpus callosum of the cat. Electroencephalography and Clinical Neurophysiology, 1960, 12, 517-518.
- Makarov, F. N. Commissural connections of the cat visual cortex (Areas 17, 18 and 19). Neuroscience Translations, 1968-69, 7, 785-790.
- Mancuso, R. P., Lawrence, A. F., Hintz, R. W., & White, C. T. Effect of altered central and peripheral visual field stimulation on correct recognition and visual evoked response. International Journal of Neuroscience, 1979, 9, 113-122.
- Marcel, T., Katz, L., & Smith, M. Laterality and reading proficiency. Neuropsychologia, 1974, 12, 131-139.
- Margolis, J. Academic correlates of sustained attention. Unpublished doctoral dissertation. University of California, Los Angeles, 1972.
- Martinius, J. W., & Hoovey, Z. B. Bilateral synchrony of occipital alpha waves, oculomotor activity and "attention" in children. Electroencephalography and Clinical Neurophysiology, 1972, 32, 349-356.
- Mattis, S. Dyslexia syndromes: A working hypothesis that works. In A. L. Benton & D. Pearl (Eds.), Dyslexia: An appraisal of current knowledge. New York: Oxford University Press, 1978.
- Mattis, S., French, J. H., & Rapin, I. Dyslexia in children and young adults: Three independent neuropsychological syndromes. Developmental Medicine and Child Neurology, 1975, 17, 150-163.
- McFie, J. The effects of hemispherectomy on intellectual functioning in cases of linguistic hemiplegia. Journal of Neurology, Neurosurgery and Psychiatry, 1961, 24, 240-249.
- McKeever, W. F., & Huling, M. D. Lateral dominance in tachistoscopic word recognitions of children at two levels of ability. Quarterly Journal of Experimental Psychology, 1970, 22, 600-604.
- McKeever, W. F., & VanDeventer, A. D. Dyslexic adolescents: Evidence of impaired visual and auditory language processing associated with normal lateralization and visual responsivity. Cortex, 1975, 11, 361-378.
- McLeod, J. A comparison of WISC subtest scores of pre-adolescent successful and unsuccessful readers. Australian Journal of Psychology, 1965, 17, 220-228.

- Miller, L. K., & Butler, D. The effect of set size on hemifield asymmetries in letter recognition. Brain and Language, 1980, 9, 307-314.
- Money, J. On learning and not learning to read. In J. Money (Ed.), The disabled reader: Education of the dyslexic child. Baltimore: John Hopkins University Press, 1966.
- Monroe, M. Children who cannot read. Chicago: University of Chicago Press, 1932.
- Morgan, W. P. A case of congenital word blindness. British Medical Journal, 1896, 2, 1378.
- Moscovitch, M., & Catlin, J. Interhemispheric transmission of information: Measurement in normal man. Psychonomic Science, 1970, 18, 211-213.
- Moscovitch, M., & Klein, D. Material-specific perceptual interference for visual words and faces: Implications for models of capacity limitations, attention, and laterality. Journal of Experimental Psychology: Human Perception and Performance, 1980, 6, 590-604.
- Myers, R. E. Function of corpus callosum in interocular transfer. Brain, 1956, 79, 358-363.
- Myers, R. E., & Sperry, R. W. Interhemispheric communication through the corpus callosum. Archives of Neurology and Psychiatry, 1958, 80, 298-303.
- Myklebust, H. R. Learning disabilities: Definition and overview. In H. R. Myklebust (Ed.), Progress in learning disabilities (Vol. 1). New York: Grune & Stratton, 1968.
- Myklebust, H. R. Toward a science of dyslexiology. In H. R. Myklebust (Ed.), Progress in learning disabilities (Vol. 4). New York: Grune & Stratton, 1978.
- Naidoo, S. Specific dyslexia. New York: John Wiley & Sons, 1972.
- Nakamura, R. K., & Gazzaniga, M. S. Processing difficulties following commissurotomy in the monkey. Experimental Neurology, 1977, 56, 323-333.
- Nauta, W. J. H., & Bucher, V. M. Efferent connections of the striate cortex in the albino rat. Journal of Comparative Neurology, 1954, 100, 257-259.
- Nebes, R. D., & Sperry, R. W. Hemispheric disconnection syndrome with cerebral birth injury in the dominant arm area. Neuropsychologia, 1971, 9, 247-259.

- Nielson, J. M. Agnosia, apraxia, aphasia: Their value in cerebral localization. New York: Hoeber, 1946.
- Obrador, S. Nervous integration after hemispherectomy in man. In G. Schaltenbrand & C. W. Woolsey (Eds.), Cerebral localization and organization. Madison: University of Wisconsin Press, 1964.
- O'Leary, D. S. A developmental study of interhemispheric transfer in children aged five to ten. Child Development, 1980, 51, 743-750.
- Ornstein, R., Herron, J., Johnstone, J., & Swencionis, C. Differential right hemisphere involvement in two reading tasks. Psychophysiology, 1979, 16, 398-401.
- Orton, S. T. "Word-blindness" in school children. Archives of Neurology and Psychiatry, 1925, 14, 581-615.
- Orton, S. T. An impediment to learning to read--A neurological explanation of the reading disability. School and Society, 1928a, 28, 286-299.
- Orton, S. T. A physiological theory of reading disability and stuttering in children. New England Journal of Medicine, 1928b, 199, 1046-1052.
- Orton, S. T. Specific reading disability--Strephosymbolia. The Journal of the American Medical Association, 1928c, 90, 1095-1097.
- Orton, S. T. Some studies in the language function. Research Publications of the Association for Research in Nervous and Mental Disease, 1932, 13, 614-633.
- Orton, S. T. Reading, writing and speech problems in children. New York: Norton, 1937.
- Orton, S. T. Some studies in the reading disability. (Unpublished address presented at the Medical Convocation at the University of Pennsylvania, June 18, 1945.) "Word-blindness" in school children and other papers on strephosymbolia: (specific language disability dyslexia) 1925-1946. Pomfret, Connecticut: The Orton Society, Inc., 1966.
- Papanicolaou, A. C. Cerebral excitation profiles in language processing: The photic probe paradigm. Brain and Language, 1980, 9, 269-280.
- Parsons, O. A., Majumder, R. K., & Chandler, P. J. Impaired flicker detection in visual fields subserved by non-damaged hemispheres. Cortex, 1967, 3, 307-316.
- Parsons, O. A., Majumder, R. K., & Chandler, P. J. Brain damage, light-to-cycle ratio and flicker detection. Cortex, 1968, 4, 269-279.

- Petrauskas, R. J., & Rourke, B. P. Identification of subtypes of retarded readers: A neuropsychological, multivariate approach. Journal of Clinical Neuropsychology, 1979, 1, 17-37.
- Pirozzolo, F. J. The neuropsychology of developmental reading disorders. New York: Praeger Publishers, 1979.
- Pirozzolo, F. J., & Rayner, K. Cerebral organization and reading disability. Neuropsychologia, 1979, 17, 485-491.
- Rabinovitch, R. D., Drew, A. L., De Jong, R. N., Ingram, W., & Withey, L. A research approach to reading retardation. Research Publications of the Association for Research in Nervous and Mental Disease, 1954, 34, 363-396.
- Rakic, P., & Yakovlev, P. I. Development of the corpus callosum and cavum septi in man. Journal of Comparative Neurology, 1968, 132, 45-72.
- Rebert, C. S., & Mahoney, R. A. Functional cerebral asymmetry and performance III. Reaction time as a function of task, hand, and EEG asymmetry. Psychophysiology, 1978, 15, 9-16.
- Reitan, R. M. Relationships between neurological and psychological variables and their implications for reading instruction. In H. A. Robinson (Ed.), Meeting individual differences in reading. Chicago: University of Chicago Press, 1964.
- Rourke, B. P. Brain-behavior relationships in children with learning disabilities: A research program. American Psychologist, September 1975, pp. 911-920.
- Rourke, B. P. Reading retardation in children: Developmental lag or deficit. In R. M. Knights & D. J. Bakker (Eds.), The neuropsychology of learning disorders: Theoretical approaches. Baltimore: University Park Press, 1976.
- Rudel, R. G., & Denckla, M. B. Relationship of IQ and reading score to visual, spatial, and temporal matching tasks. Journal of Learning Disabilities, 1976, 9, 169-178.
- Rudel, R. G., Denckla, M. B., & Spalten, E. Paired associate learning of morse code and braile letter names by dyslexic and normal children. Cortex, 1976, 12, 61-70.
- Russell, W. R., & Espir, M. L. R. Traumatic aphasia: Its syndromes, psychopathology and treatment. London: Oxford University Press, 1961.
- Rutter, M. Prevalence and types of dyslexia. In A. L. Benton & D. Pearl (Eds.), Dyslexia: An appraisal of current knowledge. New York: Oxford University Press, 1978.

- Rutter, M., Tizard, J., & Whitmore, K. Education, health and behavior. London: Longman, 1970.
- Rutter, M., Tizard, J., Yule, W., Graham, P., & Whitmore, K. Isle of Wright studies, 1964-1974. Psychological Medicine, 1976, 6, 313-332.
- Rutter, M., & Yule, W. The concept of specific reading retardation. Journal of Child Psychology and Psychiatry and Allied Disciplines, 1975, 16, 181-197.
- Sadick, T. L., & Ginsburg, B. E. The development of the lateral functions and reading ability. Cortex, 1978, 14, 3-11.
- Salamy, A. Commissural transmission: Maturational changes in humans. Science, 1978, 200, 1409-1411.
- Satz, P., & Friel, J. Some predictive antecedents of specific reading disability: A preliminary two-year follow-up. Journal of Learning Disabilities, 1974, 7, 437-444.
- Satz, P., Friel, J., & Goebel, R. A. Some predictive antecedents of specific reading disability: A three-year follow-up. Bulletin of the Orton Society, 1975, 25, 91-110.
- Satz, P., Friel, J., & Rudegeair, F. Differential changes in the acquisition of developmental skills in children who later became dyslexic: Three-year follow-up. In D. Stein & N. Butters (Eds.), Plasticity and recovery of function in the central nervous system. New York: Academic Press, 1974.
- Satz, P., Rardin, D., & Ross, J. An evaluation of a theory of specific developmental dyslexia. Child Development, 1971, 42, 2009-2021.
- Satz, P., & Sparrow, S. S. Specific developmental dyslexia: A theoretical formulation. In D. J. Bakker & P. Satz (Eds.), Specific reading disability: Advances in theory and method. Rotterdam: Rotterdam University Press, 1970.
- Satz, P., & Van Nostrand, G. K. Developmental dyslexia: An evaluation of a theory. In P. Satz & J. J. Ross (Eds.), The disabled learner: Early detection and intervention. Rotterdam: Rotterdam University Press, 1973.
- Schaltenbrand, G., Spuler, H., & Wahren, W. Electroanatomy of the corpus callosum radiation according to the facts of stereotactic stimulation in man. Zeitschrift für Neurologie, 1970, 198, 79-92.
- Schaltenbrand, G., Spuler, H., & Wahren, W. The anatomy of the corpus callosum determined electrically during stereotactic stimulation in man. Confinia Neurologica, 1972, 34, 169-172.

- Schilder, P. Congenital alexia and its relation to optic perception. The Journal of Genetic Psychology, 1944, 65, 67-88.
- Searleman, A. A review of right hemisphere linguistic capabilities. Psychological Bulletin, 1977, 84, 503-528.
- Selenes, O. A. The corpus callosum: Some anatomical and functional considerations with special reference to language. Brain and Language, 1974, 1, 111-139.
- Senf, G. M., & Freundl, P. C. Memory and attention factors in specific learning disabilities. Journal of Learning Disabilities, 1971, 4, 36-48.
- Shankweiler, D. P. A study of developmental dyslexia. Neuropsychologia, 1963, 1, 267-286.
- Shatz, C. Abnormal interhemispheric connections in the visual system of Boston Siamese cats: A physiological study. Journal of Comparative Neurology, 1977, 171, 229-246.
- Sheer, D. E. Focused arousal and 40-Hz EEG. In R. M. Knights & D. J. Bakker (Eds.), The neuropsychology of learning disorders: Theoretical approaches. Baltimore: University Park Press, 1976.
- Silverberg, R., Bentin, S., Gaziel, T., Obler, L. K., & Albert, M. L. Shift of visual field preference for english words in native Hebrew speakers. Brain and Language, 1979, 8, 184-190.
- Sklar, B., Hanley, J., & Simmons, W. W. An EEG experiment aimed toward identifying dyslexic children. Nature, 1972, 240, 414-416.
- Sklar, B., Hanley, J., & Simmons, W. W. A computer analysis of EEG spectral signatures from normal and dyslexic children. Institute of Electronics and Electrical Engineering Transactions of Biomedical Engineering, 1973, 20, 20-26.
- Smith, A. Speech and other functions after left (dominant) hemispherectomy. Journal of Neurology, Neurosurgery and Psychiatry, 1966, 29, 467.
- Smith, A., & Burkland, C. W. Dominant hemispherectomy: Preliminary report on neuropsychological sequelae. Science, 1966, 153, 1280-1282.
- Smith, A., & Sugar, O. Development of above normal language and intelligence 21 years after left hemispherectomy. Neurology, 1975, 25, 813-818.
- Smith, F. Understanding reading: A psycholinguistic analysis of reading and learning to read. New York: Holt, Rinehart and Winston, 1971.

- Spatz, W. B., Tigges, J., & Tigges, M. Subcortical projections, cortical associations and some intrinsic interlaminar connections in the striate cortex in the squirrel monkey (Saimiri). Journal of Comparative Neurology, 1970, 140, 155-174.
- Sperry, R. W. Split-brain approach to learning problems. In G. C. Quarten, T. Melnechuk & F. O. Schmitt (Eds.), The neurosciences: A study program. New York: Rockefeller University Press, 1967.
- Sperry, R. W. Mental unity following surgical disconnection of the cerebral hemispheres. Harvey Lecture, 1968a, 62, 293-323.
- Sperry, R. W. Hemisphere deconnection and unity in conscious awareness. American Psychologist, 1968b, 23, 723-733.
- Sperry, R. Perception in the absence of the neocortical commissures. Research Publications of the Association for Research in Nervous and Mental Disease, 1970, 47, 123-138.
- Sperry, R. W. Lateral specialization in the surgically separated hemispheres. In F. O. Schmitt & F. G. Worden (Eds.), The neurosciences: Third study program. Cambridge, Massachusetts: MIT Press, 1974.
- Sperry, R. W. Changing concepts of consciousness and free will. Perspectives in Biology and Medicine, 1976, Autumn, pp. 9-19.
- Spreen, O. Postscript: Review and outlook. In D. J. Bakker & P. Satz (Eds.), Specific reading disability: Advances in theory and method. Rotterdam: Rotterdam University Press, 1970.
- Spreen, O. Neuropsychology of learning disorders: Post-conference review. In R. M. Knights & D. J. Bakker (Eds.), The neuropsychology of learning disorders: Theoretical approaches. Baltimore: University Park Press, 1976.
- Springer, S. P., & Gazzaniga, M. S. Dichotic testing of partial and complete commissurotomed patients. Neuropsychologia, 1975, 13, 345-346.
- Sroufe, L. A., Sonies, B. C., West, W. D., & Wright, F. S. Anticipatory heart rate deceleration and reaction time in children with and without referral for learning disability. Child Development, 1973, 44, 267-275.
- Stanley, G. Two-part stimulus integration and specific reading disability. Perceptual and Motor Skills, 1975, 41, 873-874.
- Stanley, G. The processing of digits by children with specific reading disability (dyslexia). British Journal of Educational Psychology, 1976, 46, 81-84.

- Stanley, G., & Hall, R. Short-term visual information processing in dyslexics. Child Development, 1973, 44, 841-844.
- Sterritt, G. M., & Rudnick, M. Auditory and rhythm perception in relation to reading ability in fourth grade boys. Perceptual and Motor Skills, 1966, 22, 859-864.
- Subirana, A. Handedness and cerebral dominance. In P. J. Vinken & G. W. Bruyn (Eds.), Handbook of clinical neurology (Vol. 4). Amsterdam: North-Holland Publishing Co., 1969.
- Sykes, D., Douglas, V. I., & Morgenstern, G. Sustained attention in hyperactive children. Journal of Child Psychology and Psychiatry, 1973, 14, 213-220.
- Symmes, J. S. Deficit models, spatial visualization, and reading disability. Bulletin of the Orton Society, 1972, 22, 54-68.
- Symmes, J. S., & Rapoport, J. L. Unexpected reading failure. American Journal of Orthopsychiatry, 1972, 42, 82-91.
- Tallal, P. Auditory temporal perception, phonetics, and reading disabilities in children. Brain and Language, 1980, 9, 182-198.
- Tallal, P., & Piercy, M. Developmental aphasia: Impaired rate of non-verbal processing as a function of sensory modality. Neuropsychologia, 1973, 11, 389-398.
- Tallal, P., & Piercy, M. Developmental aphasia: Rate of auditory processing and selective impairment of consonant perception. Neuropsychologia, 1974, 12, 83-93.
- Taubman, R. E. Studies in judged number: I. The judgement of auditory number. The Journal of General Psychology, 1950a, 43, 167-194.
- Taubman, R. E. Studies in judged number: II. The judgement of visual number. The Journal of General Psychology, 1950b, 43, 195-219.
- Teng, E. L., & Sperry, R. W. Interhemispheric interaction during simultaneous bilateral presentation of letters or digits in commissurotomy patients. Neuropsychologia, 1973, 11, 131-140.
- Thompson, J. The total number of functional nerve cells in the cerebral cortex of man and of the percentage of the total volume of the cortex composed of nerve cell bodies calculated from Karl Hammarberg's data; together with a comparison of the number of giant cells with the number of pyramidal fibers. Journal of Comparative Neurology, 1899, 20, 113.
- Tomasch, J. Size, distribution, and number of fibres in the human corpus callosum. Anatomical Record, 1954, 119, 119-135.

- Tomlinson-Keasy, C., & Kelly, R. R. Is hemisphere specialization important to scholastic achievement? Cortex, 1979, 15, 97-107.
- Toyama, K., Matsunami, K., & Ohno, T. Antidromic identification of association, commissural and corticofugal efferent cells in cat visual cortex. Brain Research, 1969, 14, 513-517.
- Trevarthen, C. B. Experimental evidence for a brain-stem contribution to visual perception in man. Brain, Behavior and Evolution, 1970, 3, 338-352.
- Trevarthen, C. B. Manipulative strategies of baboons, and the origins of cerebral asymmetry. In M. Kinsbourne (Ed.), Hemispheric asymmetry of function, London: Tavistock, 1974.
- Trevarthen, C. B., & Sperry, R. W. Perceptual unity of the ambient visual field in human commissurotomy patients. Brain, 1973, 96, 547-570.
- Trites, R. L., & Fiedorowicz, C. Follow-up study of children with specific (or primary) reading disability. In R. M. Knights & D. J. Bakker (Eds.), The neuropsychology of learning disorders: Theoretical approaches. Baltimore: University Park Press, 1976.
- Vellutino, F.R. Alternative conceptualizations of dyslexia: Evidence in support of a verbal-deficit hypothesis. Harvard Educational Review, 1977, 47, 334-354.
- Vellutino, F. R. Dyslexia: Theory and Research. Cambridge, Massachusetts: MIT Press, 1979.
- Vellutino, F. R., Steger, J. A., & Kandel, G. Reading disability: An investigation of the perceptual deficit hypothesis. Cortex, 1972, 8, 106-118.
- Vernon, M. D. Backwardness in reading. Cambridge: Cambridge University Press, 1957.
- Vesbayeva, C., Whitteridge, D., & Wilson, M. E. Callosal connexions of the cortex representing the area centralis. Journal of Physiology, 1967, 191, 79P-80P.
- Vincent, F. M., Sadowsky, C. H., Saunders, R. L., & Reeves, A. G. Alexia without agraphia, hemianopia, or color-naming defect: A disconnection syndrome. Neurology, 1977, 27, 689-691.
- Walter, D. O., Rhodes, J. M., Brown, D., & Adey, W. R. Comprehensive spectral analysis of human generators in posterior cerebral regions. Electroencephalography and Clinical Neurophysiology, 1966, 20, 224-237.

- Warm, J. S., Schumsky, D. A., & Hawley, D. K. Ear asymmetry and the temporal uncertainty of signals in sustained attention. Bulletin of the Psychonomic Society, 1976, 7, 413-416.
- Wender, P. H. Minimal brain dysfunction in children. New York: Wiley-Interscience, 1971.
- White, M. J. Laterality differences in perception: A review. Psychological Bulletin, 1969, 72, 387-405.
- Whitteridge, D. Area 18 and the vertical meridian of the visual field. In E. G. Ettlinger (Ed.), Functions of the corpus callosum. Boston: Little Brown & Co., 1965.
- Wilson, D. H., Culver, C., Waddington, M., & Gazzaniga, M. Disconnection of the cerebral hemispheres. Neurology, 1975, 25, 1149-1153.
- Wilson, M. E. Cortico-cortical connexions of the cat visual areas. Journal of Anatomy, 1968, 102, 375-386.
- Witelson, S. F. Sex and the single hemisphere: Specialization of the right hemisphere for spatial processing. Science, 1976, 193, 425-427.
- Woodrow, H. The effect of pattern upon simultaneous letter-span. American Journal of Psychology, 1938, 51, 83-96.
- Yakovlev, P. I., & Lecours, A. The myelogenetic cycles of regional maturation of the brain. In A. Minkowski (Ed.), Regional development of the brain in early life. Philadelphia: F. A. Davis Company, 1967.
- Yeni-Komshian, G. H., Isenberg, D., & Goldberg, H. Cerebral dominance and reading disability: Left visual field deficit in poor readers. Neuropsychologia, 1975, 13, 83-94.
- Zaidel, E. Auditory vocabulary of the right hemisphere following brain bisection or hemidecortication. Cortex, 1976, 12, 191-211.
- Zaidel, E. Auditory language comprehension in the right hemisphere following cerebral commissurotomy and hemispherectomy: A comparison with child language and aphasia. In A. Carmazza & E. B. Zurif (Eds.), Language acquisition and language breakdown: Parallels and divergencies. Baltimore: John Hopkins University Press, 1978.
- Zaidel, E. The split and half brains as models of congenital language disability. In C. L. Ludlow & M. E. Doran-Quine (Eds.), The neurological bases of language disorders in children: Methods and directions for research (NINCDS Monograph No. 22, U. S. Department of Health, Education and Welfare). Washington, D. C.: U. S. Government Printing Office, 1979.

- Zangwill, O. L. Cerebral dominance and its relation to psychological function. Edinburgh, Scotland: Oliver & Boyd, 1960.
- Zangwill, O. L. The brain and disorders of communication. The current status of cerebral dominance. Research Publications of the Association for Research in Nervous and Mental Disease, 1964, 42, 103-118.
- Zangwill, O. L. Consciousness and the cerebral hemispheres. In S. J. Dimond & J. G. Beaumont (Eds.), Hemisphere function in the human brain. New York: Halsted Press, 1974.
- Zeki, S. M. The secondary visual areas of the monkey. Brain Research, 1969, 13, 197-226.
- Zeki, S. M. Interhemispheric connections of prestriate cortex in monkey. Brain Research, 1970, 19, 63-75.
- Zurif, E. F., & Carson, G. Dyslexia in relation to cerebral dominance and temporal analysis. Neuropsychologia, 1970, 8, 351-361.