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THE CEREBELLAR-VESTIBULAR BASIS OF LEARNING DISABILITIES IN CHILDREN, ADOLESCENTS AND ADULTS: HYPOTHESIS AND STUDY[1,2]

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Summary.—This paper provides a description of the cerebellar-vestibular-determined (CV) neurological and electronystagmographic (ENG) parameters characterizing 4,000 patients with learning disabilities. Of this sample, 1465 or 36.6% were children, 1156 or 28.9% adolescents, and 1379 or 34.5% adults. Using a set of diagnostic methods and criteria, the incidence of CV-dysfunction in this diverse sample was statistically equivalent to that reported by neurologists and neurotologists in a prior "blind" analysis of 115 dyslexic children. Over 94% of both the learning disabled and the dyslexic samples showed two or more abnormal neurological or ENG parameters indicating a CV-dysfunction whereas less than 1% evidenced hard neurological signs of a cerebral disorder. These and related data suggested that: (1) learning disabilities and dyslexia may be cerebellar-vestibular-based and reflect a single disorder and that (2) the varying academic, speech, concentration, activity, and related symptoms characterizing learning disabled persons seem to be shaped by a diverse group of cerebellar-vestibular-determining mechanisms rather than distinct neurophysiological disorders; also, (3) cerebellar-vestibular dysfunctioning and learning disabilities may secondarily trigger altered and/or compensatory cerebral processing and dominance mechanisms. (4) The cerebral cortex apparently plays a vital, compensatory role in shaping the final symptoms. A cerebellar-vestibular basis of learning disabilities is proposed. This conceptualization is consistent with, encompasses, and/or readily explains most of these clinical diagnostic, therapeutic, and research data as well as the many and varied hypotheses.

Background

Cerebral cortical explanations of dyslexia have dominated the neurophysiological literature ever since this disorder was initially described in 1896. Simultaneous with its recognition, there developed the natural assumption and conviction that dyslexia was based on the same angular gyrus defect shown to be responsible for acquired alexia in adults (Morgan, 1896; Kerr, 1897). Eventually significant symptomatic and prognostic differences were noted between the two disorders, and the expected neurological signs diagnostic of a cerebral deficit could not be found among dyslexics despite determined efforts. As a result, this notion of deficit was replaced by such concepts as cerebral dysfunction, cerebral immaturity, or developmental lag (Critchley, 1969), incomplete cerebral dominance (Orton, 1937),

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and even speculations relating alterations in cerebral cellular formations (Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985) and lateralization to adverse metabolic or hormonal effects (Geschwind & Behan, 1982; Geschwind, 1986). On the basis of computerized EEG studies or brain electrical activity mapping (BEAM), Duffy, Denckla, Bartels, and Sandini (1980) demonstrated differences in the cortical processing patterns of dyslexics and normal controls. However, this study did not show whether the measured processing differences were of a primary or secondary cortical nature.

A primary cerebellar-vestibular (CV) basis for dyslexia first became suspected in the late 1960s as a result of a chance clinical insight. The so-called "soft" balance, coordination, rhythmic, and direction-related neurological signs found characterizing dyslexia were recognized to be hard and fast diagnostic evidence of a localizing CV-determined disorder (Frank & Levinson, 1973). As a result, dyslexia was hypothesized to reflect a primary CV-impairment with resulting alterations (dysfunctional and compensatory) in interconnecting cerebral mechanisms. This conceptualization appeared readily to explain the seemingly paradoxical absence of cerebral cortical neurological signs and the presence of CV-localizing signs in dyslexic samples as well as the various suggestions that the cortex is significantly involved in dyslexia. In other words, the CV circuits were assumed to be analogous to the vertical and horizontal TV stabilizers which fine-tune the sensory input signals in a manner similar to their well-known neurophysiological role in fine-tuning the motor output. Accordingly, CV-dysfunctioning was reasoned to lead to the sensory-motor signal drifting, scrambling, and reversals or sensory-motor dysmetria and dyspraxia characterizing dyslexic symptomatology. Needless to say, a person with an intact cerebral cortex will secondarily experience difficulty in processing, storing, interpreting, transferring, and directing these CV-determined dysmetric and dyspraxic signals (Dow & Moruzzi, 1958) unless compensatory or altered processing takes place.

The hypothesis that dyslexia has a cerebellar-vestibular basis was initially tested and reported by Frank and Levinson (1973). Of 115 dyslexic children, 97% showed clear-cut neurological signs of a CV-dysfunction, and these findings were independently validated by well known neurologists in "blind" analyses. The reported CV signs included positive Romberg, difficulty in tandem walking, articulatory speech disorders, dysdiadochokinesis, hypotonia, and various dysmetric or dyspraxic past-pointing disturbances during finger-to-nose, heel-to-toe, writing, drawing, as well as during ocular fixation and tests for scanning (Dow & Moruzzi, 1958). Moreover, 90% of the dyslexic sample tested electronystagmographically evidenced vestibular abnormalities. These results were reported by neurotologists who participated in this "blind" analysis without any identifying data. In addition,

Goodenough figure drawings (1926; Bender, 1951) and Bender-Gestalt designs (1938) had indicated in all cases tested a disturbance in spatial orientation, i.e., rotations of the Bender-Gestalt cards, copying paper, drawn Bender-Gestalt figures, as well as rotations of the head and body. This, together with tilting of the Goodenough and Bender-Gestalt drawings from their intended horizontal and vertical axes and steering difficulties during formation of angles, suggested that the automatic co-pilot or the inner spatial steering and equilibrium mechanism of the vestibular apparatus and cerebellar-vestibular circuits might be impaired.

Since these earlier findings, several thousand dyslexics of varying ages were similarly studied and diagnosed (Levinson, 1980, 1984). In addition, an optokinetic method was devised to assess impaired ocular fixation and sequential scanning as well as a narrowed perceptual span or tunnel vision in dyslexics vs "normal" controls (Frank & Levinson, 1975-76; Levinson, 1980). Moreover, 75% of dyslexics treated with CV-stabilizing medications such as meclizine, cyclizine, etc., in clinical trials manifested rapid and often dramatic improvements in a wide variety of academic, concentration, behavioral, balance and coordination, speech, and mental-related symptoms (Frank & Levinson, 1976, 1977; Levinson, 1980, 1984, 1986). Although compensatory and overcompensatory mechanisms often resulted in average and above-average test scores in reading, writing, spelling, and mathematics (as well as improved balance and coordination functioning), there frequently remained some residual clinical evidence of the underlying dyslexic disorder and/or the associated CV-dysfunction (Frank & Levinson, 1976). This clearly suggested that a diagnosis of dyslexia cannot always be reliably made on the basis of either reading and educational test scores alone or on any one neurophysiological diagnostic modality. Dyslexia was eventually recognized as a syndrome characterized by a wide variety of symptoms affecting reading, writing, spelling, mathematics, memory, speech, grammar, direction, time, concentration, activity level, as well as balance and coordination-related functions. Each symptom, reading included, may vary in intensity from severely impaired to compensated and even overcompensated. Moreover, the CV-related mechanisms responsible for each of the various symptoms were carefully examined neuropsychologically (Levinson, 1980, 1984). These findings eventually led to the realization that dyslexia and learning disabilities reflect a single disorder and so must share a common group of CV-determining mechanisms and symptom-combinations. These insights appear consistent with the current definition of learning disabilities which includes dyslexia as a subcategory, according to Public Law 94-142, Education for All the Handicapped Children Act.

During the period in which the clinical data mentioned above were collected and published, a wide array of independently performed

neurophysiological and educational research has added significant depth and perspective to the role of the cerebellar-vestibular system in the development of academic or cognitive disabilities and even abilities. For example, de Quiros (1976), de Quiros and Schrager (1979) described CV-dysfunctioning mechanisms and neurological and electronystagmographic diagnostic parameters in learning disabled persons. Moreover, correlations between reading ability and postural control, known to be regulated by the CV system, have been observed in elementary school children tested in the USA, France, and Israel by Kohen-Raz and Hiriartborde (1979), clearly supporting a relationship between cerebellar-vestibular mechanisms and reading. In addition, Pavlides (1981) and Black, Collins, DeRouach, and Zubrick (1984) respectively recorded disturbances in saccades and smooth ocular pursuit of dyslexics, findings equivalent to the CV-determined dysfunction in ocular fixation and sequential scanning noted clinically and experimentally when an optokinetic blurring-speed method was used (Frank & Levinson, 1975-76; Levinson, 1980). The special learned or conditioned CV-nature and origin of the ocular fixation and sequential scanning required for normal reading was corroborated in a personal communication by Sir John Eccles (1987), Nobel Laureate in cerebellar neurophysiology. Eccles also has presumed these ocular fixation and scanning mechanisms to be impaired among dyslexics. Indeed, he distinguished the CV-nature of these fixation and scanning reading mechanisms from the cortical functions required for global perception, thereby clarifying the apparent clinical paradox whereby only letters, words, and numbers are reversed by dyslexics, and not objects. Even the tunnel vision reported by the author to characterize dyslexics was neurophysiologically supported by the research of Guedry, Lentz, and Jell (1979), Lovegrove, Heddle, and Slaghus (1980), and Dichgans (1977). These authors suggested that the vestibular system played an important role in the reciprocal coordination of peripheral and central vision. Needless to say, a CV-determined dyscoordination and decomposition (Dow & Moruzzi, 1958) of these visual mechanisms into their separate components could readily explain the development of a narrowed perceptual span or tunnel vision. Moreover, the author's clinical inference that the CV-system regulates the entire sensory input in a manner analogous to its well recognized role in coordinating the entire motor output is independently validated by the studies of Snider and Stowell (1944), Dow and Anderson (1942), and Adrian (1942).

Also, only CV-determined mechanisms appeared capable of readily explaining the acquired transient "space dyslexia" experienced by the astronauts who began mirror reading at zero gravity during the combined French-Russian space mission. These findings were reported both publicly at the Academia Rodinensis Conference of the Royal Society of Medicine in

London, England (1986) and in a personal communication (1987) with Francis Lestienne, Director of Research, National Center of Scientific Research, Paris, France. Equally consistent with, and explainable by, the proposed CV-basis hypothesized for dyslexia or learning disabilities are the findings of Zinkus, Gottlieb, and Shapiro (1978) and Silva, Kirkland, Simpson, Stewart, and Williams (1982). These studies indicated developmental speech, motor and psychoeducational sequelae of chronic otitis media. Similar conclusions were reported by the Committee on Early Childhood, Adoption and Dependent Care of the American Academy of Pediatrics (1984): "There is growing evidence demonstrating a correlation between middle-ear disease with hearing impairment and delays in the development of speech, language, and cognitive skills." These findings are compatible with a wide range of clinical evidence indicating that any injury to the CV-system (traumatic, toxic, infectious, allergic, metabolic, degenerative) may result in acquired dyslexic symptomatology or learning disabilities (Frank & Levinson, 1975-76; Levinson, 1980, 1984). Moreover, these data clearly support the author's observations and contention that the speech and language symptoms characterizing learning disabilities are of a primary CV origin and not of a primary cortical origin.

From the point of view of treatment and rehabilitation, Ayres (1972) has clearly shown a beneficial result of sensory integration or vestibular stimulation training of learning disabled persons. In a similar fashion, favorable responses in reading, writing, and concentration were reported for learning disabled individuals subjected to oculomotor fixation, scanning, and related perceptual-motor exercises which result in CV-facilitation (Halliwell & Solan, 1972; Pierce, 1977; Flax, Mozlin, & Solan, 1984). Recently Kohen-Raz (1986, pp. 182-186) reported a conspicuous reduction in academic failure for a large sample of culturally disadvantaged first graders who had been given physical education emphasizing exercises requiring CV-control. Even more specifically, Kaga, March, and Tanaka (1981) observed that vestibular-determined postural training in deaf toddlers suffering from gross motor and cognitive retardation normalized when their deficient or reduced labyrinthine function was stimulated. Finally, the beneficial effects of vestibular stimulation in triggering cognitive growth during infancy have been well documented (Korner & Thoman, 1970, 1972). These data clearly point to the crucial dual roles of the CV-system in modulating both the mechanisms, symptoms, and treatment modalities characterizing dyslexia or learning disabilities and the effect of stimulating cognitive or cortical development and compensation.

In retrospect, only the so-called prenatally determined cortical cellular anomalies in four dyslexics reported by Galaburda, *et al.* (1985) seemed significantly inconsistent with both the CV-hypothesis and the above-noted

data, especially the research correlating acquired forms of dyslexia or learning disabilities with middle-ear and cerebellar-vestibular origins. Moreover, the cerebral theory of dyslexia as explanation of these anomalies appeared as incapable of specifically explaining and encompassing most of these and related diagnostic signs, symptoms and symptom-determining mechanisms proposed for dyslexia as it was of accounting for the various CV-determined therapeutic responses. Interestingly, Eccles also believed these cortical cellular anomalies and the corresponding explanation were neuropathologically coincidental to dyslexia and has stated his views publicly [Academia Rodinensis Conference of the Royal Society of Medicine in London, England, 1986] and privately (personal communication, 1987).

Current Research Aims

In this article, a large and varied sample of learning disabled children, adolescents, and adults were assessed for positive neurological and ENG localizing evidence of CV-dysfunction. A significant association of a learning disability diagnosis with positive CV and negative cortical diagnostic parameters would significantly support the proposed CV-basis hypothesized for learning disabilities or dyslexia. Moreover, important diagnostic parameters were analyzed as functions of age, sex, handedness, and reading-score impairment to test a variety of assumptions in research on dyslexia.

As the symptoms and so-called "soft" neurological signs characterizing dyslexia or learning disabled subjects tend to improve with age, Critchley (1969) considered this disorder due to a primary functional CNS (cerebral) maturational lag. As a result, some assumed this disorder actually disappeared by adulthood. However, prior research indicated that the underlying CV-dysfunction remains essentially unchanged despite the development of compensating performance with increasing age (Levinson, 1980). This finding is consistent with the clinical observations indicating: (1) that a large number of adults do not significantly compensate with age and remain burdened and overwhelmed with the very same symptoms they had as children, and (2) that dyslexic symptoms may occasionally even reintensify with age (Levinson, 1980, 1984). To test these varying assumptions and clinical observations, the CV-determined neurological and ENG (and optokinetic) diagnostic parameters were obtained from children, adolescents, and adults with learning disabilities and the results were compared. A significant presence of abnormal CV-diagnostic parameters in learning disabled adults would tend to both support the author's contention and refute a primary developmental-lag theory of dyslexia or learning disabilities.

Inasmuch as referred learning disabled individuals often present a variety of leading symptoms or chief complaints such as concentration and distractibility symptoms (ADD), dysgraphia, dyscalculia, etc., and as such subjects and samples are known to be characterized by a wide array of

symptom-combinations identical to that described in highly selected, severely reading-score-impaired or dyslexic samples (Levinson, 1980, 1984), the extent of this symptomatic overlap was determined. High symptomatic overlap per subject in the present sample suggests that the various symptoms defining learning disabilities stem from a common denominator with varying determining mechanisms rather than from a separate series of unique and distinct neurophysiological disorders. This is an epidemiological approach limited to this large group from the practice of one physician. Persons are of unknown representativeness of the total population. No normal controls have been included at present. Follow-up analyses of this sample of referred patients have explored (1) the possible mechanisms which might underly the symptoms characterizing learning disabilities, (2) their abnormal optokinetic fixation and tracking mechanisms, (3) the response of learning disabled individuals and symptoms to a variety of CV-stabilizing medications, i.e., meclizine, cyclizine, etc., (4) the role of proposed CV-mechanisms in fears, phobias and related anxiety states.

METHOD

Subjects

On the basis of neuropsychological examinations, 4,000 consecutively studied individuals between the ages of 7 to 50 yr. completing both neurological and ENG testing qualified for a diagnosis of learning disabilities according to the definition utilized in Public Law 94-142 (The Education for All the Handicapped Children Act). Although this definition legally applies only to individuals $\leq$ 21 yr., all adults in this study, regardless of their ages, dated their learning-related symptoms back to childhood. All adults still manifested essentially the same qualitative and quantitative evidence required for a diagnosis of learning disability. Accordingly, the Civil Rights Mandate Section 504 Subpart E takes over from Public Law 94-142 and assures appropriate postsecondary education to learning disabled (and other handicapped) adults following graduation from high school.

All subjects in this sample were of normal or superior IQ and had experienced significant deficits in one or more of the following functional areas: reading, writing, spelling, mathematics, memory, speech, simple grammar, concentration, activity level, time and direction as well as associated motor difficulties involving balance, coordination, and rhythm. There was an absence of detectable primary emotional, social, educational, diffuse CNS, sensory, and medical determinants to their disorder. This sample's ages ranged from 7 to 50 yr., with a mean age of 19 $\pm$ 10.5 yr. Of the sample, 1,465 or 36.6% were children (7 to 12 yr.) with a mean age of 9.8 $\pm$ 1.6 yr., 1,156 or 28.9% adolescents (13 to 18 yr.) with a mean age of 15.3 $\pm$ 1.6 yr., 1,379 or 34.5% adults (19 to 50 yr.) with a mean age of 30.3 $\pm$

9.1 yr. (Sarnoff, 1980; Sharp, 1980). Also, the adult group was divided by age into three subsamples to facilitate intergroup comparisons and intragroup studies. The ages of these subsamples were 19 to 30 yr. with a mean age of 23.1 ± 3.1 yr., 31-40 yr. with a mean age of 34.5 ± 2.9 yr., and 41 to 50 yr. with a mean age of 43.9 ± 2.7 yr. The male/female ratio was 2.3/1.

Procedure

CV-diagnostic parameters and reading scores.—To evaluate the presence of CV-dysfunction, all 4,000 children, adolescents and adults in this study were given, and completed, neurological and ENG examinations. These findings are displayed in Tables 1 and 2 below. To test the validity of diagnosing dyslexia as a unique and separate disorder on the basis of individuals being ≥ 2 yr. behind their peers or potential in reading, important diagnostic variables were analyzed as functions of reading scores. These data appear in Tables 3 and 4 below. Those 1,399 individuals with independently determined reading scores were separated into three reading categories and the following percentages were obtained: 416 or 29.7% read at or above grade level, 312 or 22.3% read less than two years below grade level, and 671 or 48.0% read two or more years below grade level. The reading scores were based on a variety of tests, such as WRAT—R, Gray Oral Reading Test, Gates-MacGintie Reading Comprehension Test, Woodcock Reading Mastery Test, Peabody Individual Achievement Test, and Gilmore Oral Reading Tests. Where more than one test or subtest standard score was available, a mean was obtained for statistical analysis.

Independently obtained WRAT—R and/or Woodcock Mastery, and/or Peabody Individual achievement reading (and related) scores were available for 375 or 27.2% of adults 19 yr. and over. (All these tests are standardized for adults; and the first two tests are specifically stated to be reliable for persons aged up to 75 yr.) For continuity and statistical analysis, a 12th grade reading level was considered average for adults. Although the reading-score impairment required for a diagnosis of dyslexia is not defined as a function of age or compensation with age, it is readily apparent that a ≥ 2-yr. deficit for a child may be significantly different from that particular deficit for an adolescent or adult. Accordingly, the reading-scores were also obtained and analyzed separately for children, adolescents, and adults.

Reading symptoms.—As the ocular fixation and sequential scanning mechanisms required for reading activity are assumed to be CV-based (Eccles, personal correspondence), an attempt was made to estimate the percent of these learning disabled subjects who evidenced corresponding reading errors and symptoms. All 4,000 subjects (and/or parents) were asked if reading was characterized by continually losing one's place, needing a finger or marker, or having to slow down the tracking activity significantly

(slow reading) to fixate and refixate better, and if the following visually related symptoms were present: blurring, oscillopsia, scrambling, and reversals. Moreover, all were similarly questioned for past and/or present evidence of impaired reading memory or agnostic-like and conceptual reading difficulties.

Overlapping symptoms.—To test the validity of conceptualizing learning disabilities as a syndrome with a common neurophysiological denominator but involving varying mechanisms, the symptomatic overlap was examined in the 4,000 sample for frequently appearing symptoms of reading, writing, spelling, mathematics, memory, speech, direction, time, simple grammar, concentration, and activity level. In addition, the percent of these learning disabled groups demonstrating 1 to 11 of the above symptoms was tallied.

Handedness.—To test for possible relations of laterality and sex to dyslexia or learning disabilities, all subjects were examined for handedness. Mixed-handedness was said to be present when a given individual was able to perform one or more functions better or as well with the nondominant hand. For example, a subject who could naturally eat or write or bat or throw or catch, etc. as well or better with the nondominant as the dominant hand was considered mixed-handed. Accordingly, the remainder were either completely right-handed or were completely left-handed. The complete-right-handed/complete-left-handed/mixed-handed ratio was 8.8/1.4/1 or 78.2%, 12.9%, 8.9%. In further studies, handedness will be qualitatively and quantitatively rated according to the criteria utilized by Briggs and Nebes (1975) or Oldfield (1971) so accurate comparisons can be made with randomly selected samples and other learning disability studies (Geschwind, 1986).

Neurological examinations.—Standard neurological examinations were given to all 4,000 subjects. Inasmuch as CV-impaired individuals most frequently employ ocular fixation and concentration mechanisms to compensate for impaired sensory-motor functions, all subjects were tested in such a way as to minimize compensatory techniques and so maximize the emergence of abnormal CV signs. Accordingly, patients were examined for dysdiadochokinesis, finger-to-nose testing, and finger-to-thumb sequencing with eyes closed and upon distraction. In addition, eyes-closed Romberg testing was intensified when patients were instructed to balance themselves on one foot (Levinson, 1980).

Electronystagmographic measures.—All 4,000 subjects completed ENG testing. The ENG technique and evaluation is that used by Kenneth Brookler in private practice and while Chief of Otology and Neurotology at Lenox Hill Hospital in New York City and is consistent with that used by Noel Cohen, Chairman of Otolaryngology at NYU Medical Center. The examination consisted of positional testing for horizontal and vertical spontaneous and position-triggered nystagmus as well as monaural (alternate

binaural) and simultaneous bithermal caloric stimulation utilizing water at 30°C and 44°C.

Positional testing was performed on all 4,000 patients with eyes closed and used the supine 0° head up, head right, head left, right-lateral and left-lateral positions as well as the supine 30° position with head and neck straight ahead. (Head hanging and right and left Hallpike positions were not tested.) Nystagmus was considered present when three consecutive beats per 10 sec. period were recorded in any given position. Its presence is inconsistent with a normal vestibular system. The monaural or alternate bithermal and simultaneous bithermal caloric responses were measured for unilateral weakness and directional preponderance. Unilateral vestibular weakness or reduced vestibular response (RVR) was defined as a difference of 30% or more in slow-phase velocity on stimulation of the right versus left ear or as a "Type II" response (Brookler, 1971) on simultaneous caloric stimulation. Directional preponderance (DP) was defined as a difference of at least 30% in right- versus left-beating nystagmus, corresponding to a "Type III" response. "Type IV" responses (characterized by inconsistent vestibular responses to simultaneous binaural warm and cool water) were considered to be abnormal but of a nonlocalizing and nonspecific nature. The exact details of this ENG technique may be found elsewhere (Jongkees, Maas, & Philipszoon, 1962; Levinson, 1980). According to Brookler, the presence of any one neurological or ENG parameter is consistent with CV-dysfunction. Although these were the apparent criteria used in the "blind" assessments of dyslexic subjects by various clinicians in the 1973 study by Frank and Levinson, most subjects tested evidenced $\geq$ 2 abnormal parameters per diagnostic modality. To ensure reliability, present results were also obtained and reported on the basis of two or more abnormal parameters per diagnostic modality per subject.

Results

CV-diagnostic Data

When CV-dysfunction is determined on the basis of one or more abnormal parameters per diagnostic modality per subject, then in this sample of 4,000, 96.3% showed abnormal neurological dysfunction, 95.9% ENG dysfunction, and 99.5% neurological or ENG dysfunction. To ensure reliability, when CV-dysfunction is determined on the basis of two or more abnormal parameters per diagnostic modality per subject, then in this sample of 4,000, 81.6% showed abnormal neurological dysfunction, 69.7% ENG dysfunction, and 94.1% either neurological or ENG dysfunction. Despite variations, the incidence of abnormal diagnostic parameters was significantly high in the child, adolescent, and adult age groups studied, suggesting that the CV-disorder may be found across a wide age range. The specific neuro-

TABLE 1
NEUROLOGICAL PARAMETERS FOR THREE AGE GROUPS: CHILDREN, ADOLESCENTS, AND ADULTS

Signs	Total		Children		Adolescents	
	N	%	7 to 12 yr.		13 to 18 yr.	
			n	%	*n*	%
Sample size	4000		1465		1156	
1 or more signs	3852	96.3	1437	98.1	1092	94.4
2 or more signs	3265	81.6	1324	90.4	876	75.8
Ocular Dysmetria	3164	79.1	1201	82.0	858	74.2
Romberg-Monopedal	2366	59.2	917	62.6	601	52.0
Dysdiadochokinesis	987	24.7	549	37.5	225	19.5
Finger-nose	876	21.9	339	23.1	176	15.2
Finger-finger	2886	72.2	1272	86.8	756	65.4
Tandem Dysmetria	1380	34.5	702	47.9	285	24.7
Tremor	15	0.4	5	0.3	3	0.2
Hypotonia	0	0.0	0	0.0	0	0.0

	Adults							
	Total		19 to 30 yr.		31 to 40 yr.		41 to 50 yr.	
	N	%	*n*	%	*n*	%	*n*	%
Sample size	1379		784		331		264	
1 or more signs	1323	95.9	752	96.0	316	95.4	255	96.7
2 or more signs	1065	77.2	592	75.5	266	80.4	207	78.4
Ocular Dysmetria	1105	80.1	620	79.1	263	79.4	222	84.2
Romberg-Monopedal	848	61.4	448	57.1	227	68.6	173	65.5
Dysdiadochokinesis	213	15.4	124	15.8	50	15.1	39	14.8
Finger-nose	361	26.1	202	25.8	87	26.3	72	27.3
Finger-finger	858	62.2	488	62.2	211	63.7	159	60.2
Tandem Dysmetria	393	28.4	203	25.9	112	33.8	78	29.5
Tremor	7	0.5	5	0.6	2	0.6	0	0.0
Hypotonia	0	0.0	0	0.0	0	0.0	0	0.0

logical and ENG diagnostic parameters characterizing the entire sample are shown in Tables 1 and 2. Cerebral cortical signs were present in fewer than 1% of the sample.

Using a two-tailed t test, all diagnostic neurological and ENG parameters were analyzed for possible associations with sex, handedness, age groups, namely, children, adolescents and adults, and with reading scores. All neurological and ENG parameters except for reduced vestibular response (RVR) were statistically independent of sex, handedness, and reading scores (regardless of age group). Mixed-handedness, however, was significantly associated with the ENG parameter measuring reduced vestibular response (RVR) but not for completely left-handed and completely right-handed learning disabled subjects ($p < .01$). This finding suggests that asymmetric vestibular dysfunction may influence lateral or manual dominance and result in mixed-handedness. Tables 3 and 4 document the various neurological and

TABLE 2
ENG Parameters for Three Age Groups: Children, Adolescents, and Adults

Signs	Total		Children		Adolescents	
	N	%	7 to 12 yr.		13 to 18 yr.	
			n	%	n	%
Sample size	4000		1465		1156	
1 or more signs	3836	95.9	1416	96.6	1120	96.9
2 or more signs	2788	69.7	1080	73.7	813	70.3
Positional Dysfunction	3515	87.9	1291	88.1	1035	89.5
H. Nystagmus	1912	47.8	657	44.8	532	46.0
V. Nystagmus	3241	81.0	1203	82.1	975	84.3
Caloric Dysfunction	387	9.7	134	9.1	104	9.0
D.P.*	208	5.2	77	5.3	56	4.8
R.V.R.*	235	5.9	78	5.3	63	5.4
Simult. Cal. Dysfunction	3000	75.0	1170	79.9	869	75.2
Type 2*	1095	27.4	448	30.6	305	26.4
Type 3	412	10.3	183	12.5	104	9.0
Type 4	1493	37.3	537	36.7	459	39.7

	Adults							
	Total		19 to 30 yr.		31 to 40 yr.		41 to 50 yr.	
	N	%	n	%	n	%	n	%
Sample size	1379		784		331		264	
1 or more signs	1301	94.3	729	93.0	317	95.8	254	96.2
2 or more signs	895	64.9	503	64.2	218	66.0	174	65.9
Positional Dysfunction	1189	86.2	670	85.5	288	87.0	231	87.5
H. Nystagmus	723	52.4	379	48.3	189	57.1	155	58.9
V. Nystagmus	1063	77.0	598	76.3	255	77.0	210	79.4
Caloric Dysfunction	149	10.8	80	10.2	37	11.2	32	12.1
D.P.*	76	5.5	44	5.6	13	3.8	19	7.2
R.V.R.*	94	6.8	51	6.5	27	8.2	16	6.2
Simult. Cal. Dysfunction	961	69.6	535	68.2	235	71.0	191	72.2
Type 2*	341	24.7	199	25.4	85	25.6	57	21.5
Type 3	125	9.1	59	7.5	31	9.4	35	13.3
Type 4	497	36.0	278	35.5	119	36.0	100	37.8

*D.P.—Directional preponderance. R.V.R.—Reduced vestibular response. For definitions of Type 2, Type 3, and Type 4 caloric dysfunction, refer to p. 992.

ENG parameters as functions of reading score. These very same parameters are also shown as functions of sex and handedness in Tables 5 and 6.

As indicated in Table 1, the incidence of ≥ 2 CV neurological signs is significantly higher in children than in adolescents ($p < .001$) and adults ($p < .001$). Moreover, some of the neurological signs appear more often in adults than in adolescents ($p < .001$). In other words, the detectability of some CV neurological signs seems to decrease from childhood to adolescence and to increase from adolescence to adulthood. As noted in Table 2, although the ENG detected CV disorder remains constant with age, vertical

TABLE 3
NEUROLOGICAL PARAMETERS AS A FUNCTION OF READING GRADE

Signs	Total		Average or Above Grade		< 2 yr. Below Grade		≥ 2 yr. Below Grade	
	N	%	*n*	%	*n*	%	*n*	%
Sample Size	1399		416		312		671	
1 or more signs	1352	96.6	395	95.0	306	98.1	651	97.0
2 or more signs	1147	82.0	332	79.8	264	84.6	551	82.1
Ocular Dysmetria	1136	81.2	335	80.5	263	84.3	538	80.2
Romberg-Monpedal	817	58.4	237	57.0	192	61.5	388	57.8
Dysdiadochokinesis	370	26.4	107	25.7	92	29.5	171	25.5
Finger-nose	300	21.4	78	18.8	70	22.4	152	22.7
Finger-Finger	1000	71.5	275	66.1	234	75.0	491	73.2
Tandem Dysmetria	500	35.7	143	34.4	120	38.5	237	35.3
Tremor	1	0.1	0	0.0	1	0.3	0	0.0
Hypotonia	0	0.0	0	0.0	0	0.0	0	0.0

nystagmus occurs more frequently among children and adolescents than adults ($p < .05$), whereas horizontal nystagmus occurs more frequently

TABLE 4
ENG PARAMETERS AS A FUNCTION OF READING GRADE

Signs	Total		Average or Above Grade		< 2 yr. Below Grade		≥ 2 yr. Below Grade	
	N	%	*n*	%	*n*	%	*n*	%
Sample Size	1399		416		312		671	
1 or more signs	1346	96.2	405	97.4	299	95.8	642	95.7
2 or more signs	1000	71.5	298	71.6	225	72.1	477	71.1
Positional Dysfunction	1244	88.9	375	90.1	279	89.4	590	87.9
H. Nystagmus	702	50.2	220	52.9	146	46.8	336	50.1
V. Nystagmus	1153	82.4	348	83.7	266	85.3	539	80.3
Caloric Dysfunction	156	11.2	55	13.2	26	8.3	75	11.2
D.P.*	97	6.9	38	9.1	15	4.8	44	6.6
R.V.R.*	105	7.5	44	10.6	16	5.1	45	6.7
Simult. Cal. Dysfunction	1068	76.3	317	76.2	237	76.0	514	76.6
Type 2*	410	29.3	123	29.6	85	27.2	202	30.1
Type 3	167	11.9	59	14.2	39	12.5	69	10.3
Type 4	539	38.5	182	43.8	113	36.2	244	36.4

*D.P.—Directional preponderance. R.V.R.—Reduced vestibular response. For definitions of Type 2, Type 3, and Type 4 caloric dysfunction, refer to p. 993.

among adults than children and adolescents ($p < .05$). The import of these variations as well as the suggestion that these parameters vary with age as a result of the development of compensatory and/or decompensatory mechanisms remains to be explored.

TABLE 5
NEUROLOGICAL PARAMETERS AS A FUNCTION OF SEX AND HANDEDNESS

Signs	Total		Handedness							
			Total		Right		Left		Mixed	
	N	%	*n*	%	*n*	%	*n*	%	*n*	%
					Male					
Sample size	4000		2771		2156		343		272	
1 or more signs	3852	96.3	2680	96.8	2084	96.8	335	97.6	261	96.0
2 or more signs	3265	81.6	2262	81.7	1746	81.1	289	84.2	227	83.5
Ocular Dysmetria	3164	79.1	2191	79.1	1705	79.2	273	79.5	213	78.4
Romberg-Monopedal	2366	59.2	1596	57.6	1209	56.1	221	64.5	166	61.0
Dysdiadochokinesis	987	24.7	698	25.2	542	25.2	81	23.7	75	27.5
Finger-nose	876	21.9	589	21.3	437	20.3	75	22.0	77	28.2
Finger-finger	2886	72.2	2051	74.0	1593	73.9	260	75.7	198	72.8
Tandem Dysmetria	1380	34.5	918	33.1	704	32.7	119	34.6	95	34.9
Tremor	15	0.4	11	0.4	6	0.3	3	1.0	2	0.7
Hypotonia	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0
			Handedness							
			Total		Right		Left		Mixed	
			n	%	*n*	%	*n*	%	*n*	%
					Female					
Sample size			1229		973		173		83	
1 or more signs			1172	95.2	932	95.8	167	95.2	73	88.5
2 or more signs			1003	81.5	796	81.8	144	82.5	63	75.9
Ocular Dysmetria			974	79.0	773	79.4	140	79.8	61	73.0
Romberg-Monopedal			770	62.5	611	62.8	112	63.9	47	57.1
Dysdiadochokinesis			289	23.5	234	24.0	39	22.5	16	19.0
Finger-nose			287	23.3	228	23.4	40	22.7	20	23.9
Finger-finger			836	67.9	676	69.4	106	60.7	54	64.8
Tandem Dysmetria			462	37.5	363	37.4	67	38.4	32	38.1
Tremor			4	0.3	2	0.2	0	0.0	2	2.3
Hypotonia			0	0.0	0	0.0	0	0.0	0	0.0

Reading Symptoms

Of the 4,000 learning disabled, 3,821 or 95.5% gave a past or present history of reading symptoms. Analyzing the past and/or present reading symptoms characterizing the reading-impaired subsample of 3,821, 3,676 or 96.2% evidenced clear-cut ocular fixation and scanning errors, 3,366 or 88% reported instability of memory, 2,728 or 71.4% noted reversals, and only 84 or 2.2% expressed conceptual-like reading difficulties in association with the above reading errors. These data clearly indicate that ocular fixation, tracking and orientation errors (and underlying mechanisms) as well as related instability of memory consistent with CV-dysfunctioning characterize the reading disorders in this sample. Conceptual reading errors are seldom present and so may represent coexisting (cortical) variables.

TABLE 6
ENG PARAMETERS AS A FUNCTION OF SEX AND HANDEDNESS

Sex	Total		Handedness							
			Total		Right		Left		Mixed	
	N	%	*n*	%	*n*	%	*n*	%	*n*	%
					Male					
Sample size	4000		2771	69.3	2156	53.9	343	8.6	272	6.8
1 or more signs	3836	95.9	2664	96.1	2077	96.4	332	96.8	255	93.6
2 or more signs	2788	69.7	1929	69.6	1537	71.3	224	65.4	168	61.8
Positional Dysf.	3515	87.9	2435	87.9	1905	88.4	301	87.8	229	84.3
H. Nystagmus	1911	47.8	1293	46.7	995	46.1	161	46.8	138	50.8
V. Nystagmus	3241	81.0	2239	80.8	1752	81.3	282	82.1	206	75.6
Caloric Dysf.	387	9.7	258	9.3	209	9.7	24	6.9	26	9.7
D.P.*	208	5.2	133	4.8	111	5.1	11	3.2	11	4.0
R.V.R.*	234	5.9	155	5.6	118	5.4	16	4.6	22	8.3
Simult. Cal. Dysf.	3000	75.0	2085	75.3	1650	76.5	249	72.7	186	68.5
Type 2*	1094	27.4	734	26.5	598	27.7	78	22.6	59	21.7
Type 3	412	10.3	287	10.4	220	10.2	36	10.4	32	11.7
Type 4	1493	37.3	1063	38.4	831	38.6	137	39.9	95	35.0
			Handedness							
			Total		Right		Left		Mixed	
			n	%	*n*	%	*n*	%	*n*	%
					Female					
Sample size			1229	30.7	973	24.3	173	4.3	83	2.1
1 or more signs			1172	95.4	932	95.8	161	93.3	79	94.9
2 or more signs			859	69.9	693	71.2	104	59.9	62	74.3
Positional Dysf.			1080	87.8	861	88.5	143	82.5	76	91.5
H. Nystagmus			618	50.3	497	51.1	79	45.6	42	50.7
V. Nystagmus			1001	81.5	797	82.0	131	75.7	73	88.4
Caloric Dysf.			129	10.5	109	11.2	12	6.8	8	9.5
D.P.*			75	6.1	61	6.3	8	4.6	6	7.2
R.V.R.*			79	6.4	63	6.5	9	5.3	7	8.0
Simult. Cal. Dysf.			915	74.4	732	75.3	121	69.8	62	74.3
Type 2*			360	29.3	285	29.3	53	30.4	22	26.9
Type 3			125	10.1	101	10.3	15	8.4	9	11.2
Type 4			430	35.0	347	35.6	53	30.9	30	36.1

*D.P.—Directional preponderance. R.V.R.—Reduced vestibular response. For definitions of Type 2, Type 3, and Type 4 caloric dysfunction, refer to p. 992.

It is important to note here that many so-called "reading comprehension" tests and scores primarily and inadvertently measure memory functioning for content. For example, the vast majority of learning disabled subjects with reading impairments were shown to know the meaning and significance of letters, words, and phrases while reading. However, their ability to answer questions correctly on tests is almost invariably dependent on how well they fixate, track, orient or see and/or remember what was read as a function of time. These tracking and memory-related pseudocomprehension

TABLE 7
DISTRIBUTION OF SYMPTOMS IN LEARNING DISABILITIES (N = 4000)

Symptoms	Learning Disabled Sample (N = 4000)		ADD Subsample N = 3269 (82%)		NonADD Subsample N = 731 (18%)	
	n	%	n	%	n	%
Reading	3821	95.5	3212	98.3	609	83.3
Writing	3675	91.9	3060	93.1	615	84.2
Spelling	3107	77.7	2732	80.5	375	51.3
Mathematics	3341	83.5	2899	83.0	442	60.5
Memory	3501	87.5	3049	87.9	452	61.8
Time	2824	70.6	2526	71.9	298	40.8
Direction	2983	74.6	2608	72.7	375	51.3
Speech	3409	85.2	2890	84.7	519	71.1
Grammar	2591	64.8	2236	68.0	355	48.7
Activity Level	2412	60.3	2220	62.6	192	26.3
Concentration	3269	81.7	3269	100.0	0	0.0

errors must be distinguished from those errors resulting from subjects' inability to understand the meaning of seen letters, words, and sentences. Indeed, even some of these conceptual or agnostic-like reading errors appear due to CV-determined time delays in visual and/or auditory processing for read and heard content (Levinson, 1980, 1984).

Overlapping Symptoms

Protocols of all learning disabled patients were analyzed for the current presence and history of symptoms and mechanisms affecting reading, writing, spelling, mathematics, memory, time, direction, speech, simple

TABLE 8
NUMBER OF OVERLAPPING SYMPTOMS IN TOTAL LEARNING DISABLED SAMPLE AND ADD AND NONADD SUBSAMPLES

No. of Overlapping Symp.	Learning Disabled Sample (N = 4000)		ADD Subsample N = 3269 (82%)		NonADD Subsample N = 731 (18%)	
	%	Cum.	%	Cum.	%	Cum.
1	0.1	100.0	0.3	100.0	1.4	100.0
2	0.2	99.9	0.6	99.7	2.9	98.6
3	1.2	99.7	1.8	99.1	14.3	95.7
4	2.6	98.5	2.4	97.3	10.0	81.4
5	3.9	95.9	3.3	94.9	10.0	71.4
6	5.6	92.0	8.5	91.6	27.1	61.4
7	9.8	86.4	13.7	83.1	14.3	34.3
8	15.8	76.6	18.1	69.4	18.6	20.0
9	16.3	60.8	30.3	51.3	1.4	1.4
10	26.9	44.5	21.0	21.0	0.0	0.0
11	17.6	17.6				

Note.—The overlapping symptoms excluded concentration as a variable in ADD and NonADD subsamples. (%) is the cumulative percent.

grammar, activity levels, concentration, and distractability. The frequencies of incidence are tabulated in Table 7. As indicated, the incidence and distribution of each of the 11 symptoms is very high in the sample of 4,000. Table 8 documents the number and percent of the entire sample of 4,000 who show 1 to 11 of the above-mentioned symptoms. As noted, 95% of the subjects evidenced $\geq$ 5 symptoms, 61% $\geq$ 9 symptoms whereas 1% $\leq$ 3 symptoms, and 0.1% only 1 symptom. In other words, the entire learning disabled sample of 4,000 is characterized by subjects having a significant number of overlapping symptoms whereas subjects with monosymptomatic states (such as "pure" dyslexia, dysgraphia, dyscalculia, dysnomia and dysphasia, attention deficit disorder [ADD], hyperactivity, dyspraxia, etc.) are rare.

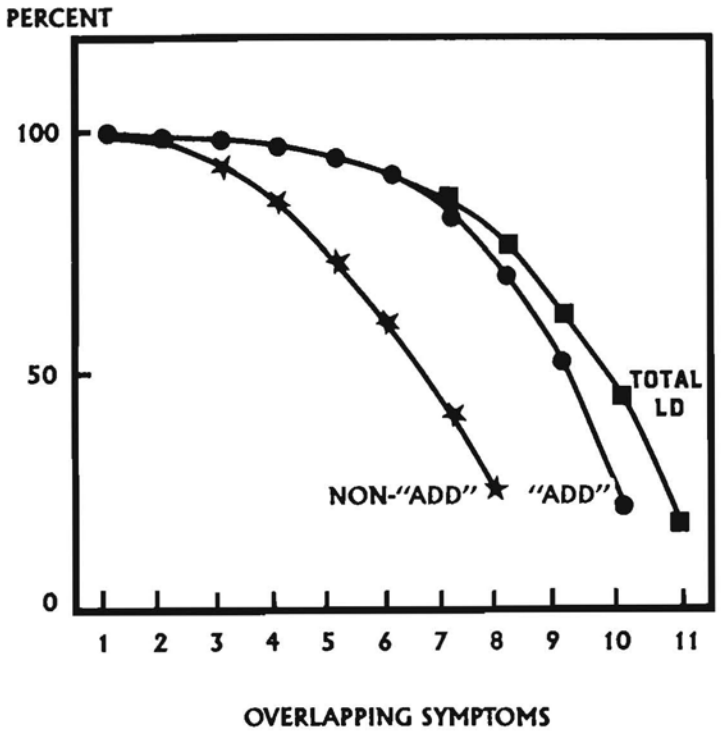

FIG. 1. Overlapping symptoms in total learning disabled (▪), ADD (Attention Deficit Disorder, (•), and nonADD subsamples (*)

Inasmuch as attention-deficit disorder (ADD) is currently viewed as a unique disorder, and as 3,269 or 81.7% of this sample evidenced concentration symptoms of an ADD quality and 731 or 8.3% did not, it appeared

reasonable to determine the symptomatic overlap for ADD and nonADD subsamples. As noted in Tables 7, 8, and Fig. 1, although distribution patterns of symptoms and their overlap for subjects in the ADD and nonADD subsamples were similar to each other and to the entire sample, the nonADD subsample had a lower percentage of symptoms and overlap. (The overlapping symptoms excluded concentration as a variable in ADD and nonADD subsamples.) This finding is consistent with clinical and experimental observations (Levinson, 1980) indicating that impaired concentration and distractability destabilize other sensorimotor functions and corresponding symptoms in learning disabilities and minimizes compensation. In other words, it appears very likely that ADD or attention-deficit disorder is as part of the learning disability syndrome as are the other 10 frequently occurring symptoms listed in Table 7.

Handedness Data

Inasmuch as Orton (1937) reported a higher incidence of dyslexia in left-handed and mixed-handed subjects and the reverse, and as Geschwind (1986) "clearly documented" the increased risk of the complete or strong left-hander (especially males) for learning disorder (or stuttering) to be 10 to 1 compared to a complete or strong right-hander, the frequencies of incidence of complete-right-handed and complete-left-handed subjects with learning disabilities were obtained. As detailed in Table 9, for the sample of 4,000 learning disabled, the incidence of males who are completely left-

TABLE 9
SEX AND HANDEDNESS VS READING GRADE IN LEARNING DISABILITIES

Group	Total		Learning Disabilities with Reading Grade							
	N	%	Subtotal		Average or Above		< 2 yr. Below		≥ 2 yr. Below	
			n	%	*n*	%	*n*	%	*n*	%
Sample Size	4000		1399		416		312		671	
Right handed	3129	78.2	1077	76.9	309	74.2	256	82.0	512	76.3
Left handed	516	12.9	164	11.7	55	13.2	29	9.3	80	11.9
Mixed handed	355	8.9	158	11.2	52	12.5	27	8.7	79	11.7
Male	2771	69.3	988	70.6	270	64.9	226	72.4	492	73.3
Right handed	2156	53.9	760	54.3	196	47.1	184	59.0	380	56.6
Left handed	343	8.6	111	7.9	34	8.2	19	6.1	58	8.6
Mixed handed	272	6.8	117	8.4	40	9.6	23	7.4	54	8.0
Female	1229	30.7	411	29.4	146	35.1	86	27.6	179	26.7
Right handed	973	24.3	317	22.7	113	27.2	72	23.1	132	19.7
Left handed	173	4.3	53	3.8	21	5.0	10	3.2	22	3.3
Mixed handed	83	2.1	41	2.9	12	2.9	4	1.3	25	3.7

Note.—In 4000 learning disabilities (total in the table), only 1399 (subtotal in table) had independently obtained reading grades.

handed is similar to that of females who are completely left-handed. The completely right-handed, completely left-handed, and mixed-handed percentages were 78.2%, 12.9%, 8.9%. The incidence of left-handedness and mixed-handedness in this sample was no higher than that characterizing a random sample (Briggs & Nebes, 1975). Moreover, handedness and reading scores were unrelated for the learning disabled subsample of 1,399 who had independently obtained reading scores. These data suggest that sex, handedness, and reading scores in dyslexia or learning disability are unrelated.

Discussion

In this study, 99.5% of 4,000 learning disabled subjects showed ≥ 1 neurological or ENG sign of CV-dysfunction. When using ≥ 2 abnormal diagnostic neurological or ENG parameters per subject as evidence of CV-dysfunction, 94.1% showed CV-impairment. These data were statistically equivalent to those obtained by Frank and Levinson (1973) in a more homogeneous sample of 115 dyslexic children for whom diagnostic findings were independently and "blindly" validated by neurologists and neurotologists without identifying data. Here, the CV-determined diagnostic parameters and learning disability symptoms were significantly present in all age groups. Moreover, the reading symptoms characterizing the vast majority of the 4,000 learning disabled were CV-related or consistent but not cortically determined. By contrast, the incidence of hard and fast cortical signs noted in both the present and 1973 studies fell below 1%. Also, the sample of 4,000 learning disabled was characterized by a significant symptomatic overlap whereas the presence of monosymptomatic subjects were rare. These and related data suggested (1) that learning disabilities and dyslexia are CV-based and represent one disorder, (2) that this CV-dysfunction continues with increasing age despite the development of coexisting compensatory and even decompensatory factors which result in symptomatic and neurophysiological improvements and/or regression. Also, (3) the symptomatic overlap within subjects and samples appears to reflect a common predisposing CV-basis with varying symptom-shaping and determining mechanisms rather than a diverse group of separate neurophysiological disorders. (4) A variety of so-called "pure" disorders and diagnostic terms (dyslexia, dygraphia, dyscalculia, dysphasia and dysnomia, dyspraxia, attention-deficit disorder [ADD], perceptual-motor disorder, etc.) merely reflect highly selected learning disabled symptoms and samples.

In cross-sectional analyses based on the present sample, reading scores (as well as all other symptoms) varied from severely impaired to overcompensated or above average, and all measured CV-diagnostic parameters were noted to be independent of groups created by reading scores. According to these analyses: (1) the reading score-dependent diagnosis of dyslexia appears to be arbitrary and incorrect, further suggesting that dyslexia and learning

disability represent just one disorder and (2) compensatory cortical, cerebellar (Eccles, 1986) and related CNS-function may significantly affect the final reading-score and symptomatic outcome in dyslexia or learning disabilities, regardless of the initial CV-predisposing and symptom-determining factors.

As some investigators who suggest that impaired cerebral dominance underlies dyslexia or learning disabilities have reported a significantly higher incidence of dyslexia in mixed-handed and/or left-handed individuals (Orton, 1937), and some have even reported a predisposition of left-handed males to learning disabilities (Geschwind & Behan, 1982; Geschwind, 1986), handedness was evaluated as a possible factor associated with various diagnostic neurological and ENG parameters and with reading scores. When using similar diagnostic criteria, the distribution of complete-left-handedness and mixed-handedness in this sample was no higher than that reported for a random sample by Briggs and Nebes (1975); and the incidence of males and females with complete-left-handedness was similar. Also, handedness was independent of reading scores, further refuting the assumed association among laterality, cerebral dominance, and dyslexia or learning disabilities. Consistent with these findings, Orton appeared to have significantly modified or reversed his original (1937) mixed-handed and left-handed-dyslexic associations in later years. For example, when discussing a paper by John G. Lynn, Orton (1942) stated:

> One probably makes a mistake in attempting to associate too closely conditions like reading disability with handedness pattern. The great majority of my patients with specific reading disability are right-handed. Many of them are also right-eyed and right-footed; in other words many of them have distinctly unilateral motor patterns, but this 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 have confusion in the motor patterns (p. 1064).

If a majority of Orton's dyslexic patients evidenced distinctly right-handed and unilateral motor patterns and if dyslexics with and without confused unilateral motor patterns have the same symptoms, then might we not assume that dyslexic symptoms and confused motor or left-handed patterns are probably unrelated? Also, if the speech-symptoms characterizing learning disabilities or dyslexia, stuttering included, are primarily CV-determined rather than of a dominant cortical origin (Levinson, 1980) and as dyslexia or learning disabilities are characterized by positive CV and negative cortical signs, then the theoretical basis for Orton's original assumptions relating dyslexia to impaired cerebral and lateral dominance may well have been in error. In support of this reasoning, the mixed-handed learning disabled persons in this study were significantly associated with the asymmetric vestibular functional parameter termed reduced vestibular response (RVR) compared to the complete-right-handed and complete-left-handed learning

disabled ($p < .01$). This finding suggests that asymmetric CV-dysfunction may result in destabilization and/or compensatory alterations in cerebrally modulated dominance and laterality functions which accounts for the association of mixed-handedness with reduced vestibular responses. If indeed this assumption is validated, then explanations which attribute or associate dyslexia or learning disabilities to impaired cerebral and lateral dominance may have, to some extent, reversed cause and effect. However, conclusions must be deferred until the handedness data obtained from the present clinical technique are compared with those of matched random controls and/or statistically correlated with corresponding data derived from the more objective handedness criteria of Briggs and Nebes (1975) or Oldfield (1971).

Inasmuch as the male:female learning disabled or dyslexic incidence has been reported as between 2:1 and 10:1, all diagnostic parameters here were analyzed as functions of sex. As noted, no statistical differences were apparent. These data are consistent with prior work (Frank & Levinson, 1973; Levinson, 1980) and suggest that although a sex-linked component may exist in some dyslexics, the male:female ratios are referral rather than incidence ratios and emotional, behavioral, social, and compensatory factors play a crucial role in shaping these ratios.

Although the statistical absence of both cerebral neurological signs and evidence of primarily impaired cerebral dominance mechanisms in learning disabilities suggest an absent primary cortical role, these data by no means imply an absence of cortical determinants. Indeed, the reverse is considered true. The CV system is in a continuous, interacting or feedback relation with the cerebral cortex (Snider & Stowell, 1944); and the latter is forced to cope, adapt and compensate for the sensory-motor dysmetria and dyspraxia resulting from CV dysfunction and learning disability (Frank & Levinson, 1976). Moreover, based on neural evidence and information processing theory, Leiner, Leiner, and Dow (1986) reasoned that "the phylogenetically newest structures of the cerebellum may contribute to mental skills in much the same way that phylogenetically older structures contribute to motor skills." Accordingly, a neocerebellar impairment may significantly interfere with cortically-dependent feedback circuits and result in both dyslexic symptoms and altered cortical processing patterns. One wonders: does the presence of alterations of cortical processing patterns in dyslexia (Duffy, *et al.*, 1980) permit the conclusion that dyslexia is of cerebral origin? Might not the reverse be true? Might not a CV-determined learning disability or dyslexia significantly alter functional or processing patterns of the cerebral cortex?

One may note in conclusion that the hypothesis suggesting a CV-basis of learning disabilities or dyslexia appeared to be clinically and statistically supported although no comparison with "normal" persons have been made

as yet. This hypothesis is consistent with most, if not all, research data, regardless of apparent contradictory quality. Also, this hypothesis appears capable of unifying and holistically explaining a heretofore varied and perplexing data base which often appeared characterized by seemingly paradoxical and conflicting findings. As a result of the presented research, many traditionally accepted assumptions and convictions regarding dyslexia or learning disabilities have been challenged. Moreover, the reported insights have led to new methods of diagnosis and treatment (Levinson, 1980, 1984) as well as a possible CV-basis for fears, phobias, and related anxiety states (Levinson, 1980, 1984, 1986).

A follow-up study will attempt to compare the presented data with "normal" controls matched for age, sex, and handedness. Needless to say, this controlled study is considered essential for both clarifying and validating the CV-basis of learning disabilities. It is anticipated that a significant incidence of so-called "normal" subjects will show CV-dysfunction without apparent or obvious symptoms. However, differences between the "normal" and learning disabled groups may highlight combinations and intensities of compensatory factors and diagnostic parameters crucial for symptom formation. On the basis of available data, clinicians interested in understanding the neurophysiological and psychological basis of learning disabilities or dyslexia should consider adding neurological, ENG, and optokinetic testing as well as qualitative analyses of the varied symptoms to their current assessments.

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