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THE DIAGNOSTIC VALUE OF CEREBELLAR-VESTIBULAR TESTS IN DETECTING LEARNING DISABILITIES, DYSLEXIA, AND ATTENTION DEFICIT DISORDER[1]

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Summary.—Neurological and optokinetic measures of cerebellar-vestibular (CV) dysfunctioning were shown to be of significant diagnostic value in differentiating between learning disabled subjects and controls matched for chronological age, sex, handedness, IQ, and background ($ns = 35$). Although traditionally used electronystagmographic positional and caloric parameters were not similarly discriminating, quantitative measures of vertical nystagmus in various eyes-closed positions appeared to have diagnostic potential and were related significantly to such CV-determined neurological signs as positive monopedal Romberg. As a substantial majority of learning disabled (82.9%) evidenced ADD-like symptoms and since learning disabled subsamples with and without Attention Deficit Disorder (ADD) shared similar coexisting symptoms and CV signs, it appeared probable that learning disabilities and ADD were reflections of the same underlying CV determinants.

According to evidence corroborated by Frank and Levinson (1973, 1975-76, 1976) and Levinson (1980, 1988, 1989a, 1990), clinical neurological, electronystagmographic (ENG), and oculomotor signs indicative of cerebellar-vestibular (CV) dysfunction characterized populations defined variously as dyslexic or learning disabled, including a majority of subjects with attention deficit disorder (ADD). Moreover, a wide spectrum of antimotion-sickness antihistamines and stimulants applied in clinical practice were shown to alleviate significantly the CV, learning disability-related, and ADD signs and symptoms characterizing these patients (Frank & Levinson, 1976, 1976-77, 1977; Levinson, 1980, 1984, 1986, 1990).

Although no known study has attempted to replicate exactly these diagnostic and therapeutic findings, an important series of other investigations supported directly and indirectly the hypothesis of the CV origins of learning disorders (Cheek, 1969; Ayres, 1972; DeQuiros, 1976, 1979; Kohen-Raz, 1986) and were recently referenced in detail (Levinson, 1988, 1989a). On the other hand, the results of two ENG studies appeared to challenge this hypothesis (Stockwell, Sherard, & Schuler, 1976; Polatajko, 1985). Besides a wide variety of shortcomings such as Stockwell, *et al.*'s small control group of five subjects and Polatajko's conviction of a cerebrovestibular dysfunction in the absence of any cerebral signs and her reliance entirely on only traditional

[1]Special thanks are extended to Professor Kohen-Raz, Dr. Jose Alvir, and my staff at the Medical Dyslexic Treatment Center, especially Jin Yao, for dedicated help in preparing this paper for publication. Address requests for reprints to Harold N. Levinson, M.D., Medical Dyslexic Treatment Center, 600 Northern Boulevard, Suite 118, Great Neck, New York 11021.

ENG rotation procedures as the core instrument of differential diagnosis, neither study included meticulous screening of the "normal" controls for a possible incidence of mild or compensated and even overcompensated academic and CV problems. Despite Polatajko's failure to use any other tests diagnostic of CV dysfunctioning and sidestepping the sensitivity and reliability issues of ENG to detect CV disorders, she bluntly states that she "found *no* support for the notion that LD children suffer from vestibular dysfunction. . . . This conclusion is in direct opposition to the theories of Ayres, Frank and Levinson, and DeQuiros" (p. 290). In marked contrast, Stockwell, *et al.* state with due reservation: "The present study does not rule out the possibility that vestibular-cerebellar dysfunction is an etiology of dyslexia or that some test might be devised to detect such a disorder. But it does indicate that this form of dyslexia is either rare or that it is not revealed by standard clinical ENG procedures" (p. 242). In other words, Polatajko equated the absence of discriminating ENG signs in learning disabilities with the absence of CV dysfunction whereas Stockwell, *et al.* recognized the possible limitations of this diagnostic modality.

Current Aims

The present study was undertaken in the light of this apparent controversy and on the basis of doubts raised by other authors as to the sensitivity and reliability of the standard ENG procedures to measure vestibular changes (Martin & Oosterveld, 1970). Accordingly, the primary aims of this study were to evaluate CV-based diagnostic modalities and to substantiate further the CV origins of learning disabilities (including ADD) by comparing neurological, ENG, optokinetic and perceptual span parameters for a sample of learning disabled adolescents and their carefully matched controls. Moreover, for the purposes of assessing the frequency distribution and overlap of the various general behavioral symptoms characterizing learning disabled youth with and without ADD and even controlled samples, a detailed case history questionnaire for learning disabilities was administered.[2] Without the use of such an historical screening questionnaire, control samples may mistakenly be considered "normal" on the basis of only limited quantitative factors, i.e., whether or not scores on reading and related academic tests are within normal limits, ignoring the incidence of symptom-compensated dyslexics or learning disabled.

Method

Subjects

Thirty-five learning disabled adolescents were selected from a pool of

[2]This historical or symptomatic questionnaire is on file with Microfiche Publications in Document NAPS-04640. Remit $12.25 for photocopy or $4.00 for fiche to Microfiche Publications, POB 3513, Grand Central Station, New York 10017.

4000 outpatients of the Medical Dyslexic Treatment Center described in a prior paper (Levinson, 1988). All of them had been previously tested with the instruments and methods to be described below. The selection was performed by a computer program which matched the outpatient control sample by age, sex, handedness, socioeconomic background, and IQ. These controls were volunteers from neighborhood schools adjacent to the Medical Dyslexic Treatment Center. They were all high school students except for three who were just beginning college. All had above-average IQs, based on clinical testing and WISC—R or WAIS—R scores, and were of a middle-class background. As a result of the selective matching, both samples' mean ages were 16.9 ± 1.1 yr. (range 14.4—18.6). The male/female sex ratios were 1.2/1 and their completely right-handed to completely left-handed/mixed-handed ratios and percentages were 13.5/3/1 and 77.1%/17.1%/5.7%, respectively. Mixed-handedness was considered to be present when an individual naturally performed any function queried or volunteered (writing, throwing, eating, etc.) as well or better with the nondominant hand. (Numbers of questions varied.) The remainder were either completely right-handed or completely left-handed.

On the basis of prior neuropsychological testing, all 35 subjects had been diagnosed as learning disabled according to the definition utilized in Public Law 94-142 (The Education for All Handicapped Children Act) and a later amendment (United States Congress, 1975; Federal Register, 1977). All had experienced significant deficits in one or more of the general functional areas or symptom-categories of reading, writing, spelling, mathematics, memory, speech, simple grammar, concentration, activity level, as well as associated difficulties in direction, time, balance, coordination, and rhythm. They showed no overt or detectable evidence of primary emotional, social, educational, diffuse CNS, sensory, and medical determinants in their learning disorder. All had either normal or corrected 20/20 visual acuity as tested independently and by means of a Snellen chart.

Procedure

Both learning disabled and control groups were evaluated and examined by the following methods: (1) an Historical or Symptom Questionnaire for Diagnosing Dyslexia or Learning Disabilities,[2] (2) neurological examination, (3) ENG, and (4) newly revised optokinetic fixation, tracking and perceptual span measures. All subjects completed all testing modalities.

An historical or symptom questionnaire for learning disabilities.[2]—This questionnaire registers the general symptoms (past and present) and underlying mechanisms found to characterize dyslexia or learning disabilities. In this study, only the 11 most frequently noted general symptom-categories were evaluated, i.e., difficulties in reading, writing, spelling, mathematics, memory, time, direction, speech, simple grammar, activity level, and concentration.

Neurological examination.—Standard neurological examinations were given to all 35 learning disabled and 35 control 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 so that compensatory techniques were minimized and the emergence of abnormal CV signs would be maximized. Patients were examined for dysdiadochokinesis, finger-to-nose testing, and finger-to-thumb sequencing with eyes closed and upon distraction. In addition, the eyes-closed Romberg was given in the bipedal position and intensified when patients were instructed to balance themselves on one foot, i.e., monopedal position (Levinson, 1980).

Electronystagmographic examination.—Since the ENG methodology has been recently discussed in detail and referenced (Levinson, 1988), only the examinations used in the context of this study are briefly explained.

(a) *Standard Positional ENG* tests were performed with eyes closed and in the positions of supine 0° head up, head right, head left, right-lateral and left-lateral as well as the supine 30° position with head and neck straight ahead. Nystagmus was considered abnormally present when three consecutive beats per 10-sec. period were recorded in any given position.

(b) *Caloric ENG* tests were carried out by measuring the monaural or alternate bithermal and simultaneous bithermal caloric responses for unilateral weakness and directional preponderance. *Abnormal* unilateral vestibular weakness or *abnormal* 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 on simultaneous caloric stimulation. *Abnormal* directional preponderance (DP) was defined as a difference of at least 30% in right-beating nystagmus 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. To enhance reliability, present results were reported on the basis of two or more abnormal parameters per diagnostic modality per subject.

(c) An original modification of *Positional ENG* was performed by assessing horizontal and vertical nystagmus during the bipedal *Romberg position* with eyes open and closed (Levinson, 1980). The amount of nystagmus was measured for all relevant ENG parameters and was summarized and statistically expressed as means and standard deviations.

Tests of optokinetic fixation, scanning, and perceptual span.—A revised optokinetic tracking method was used to measure the functions of ocular fixation, sequential scanning, and perceptual span. A detailed description of this method and instrument has been reported and illustrated previously (Levinson, 1980, 1989a, 1989b).

A series of seven black elephants projected against a blank, white surround characterized the Mode I gestalt. Subjects were asked to concentrate and fixate on the center elephant of the stationary seven-elephant sequence and to report the total number of elephants they could clearly recognize in detail without moving their eyes.

The Mode II gestalt consisted of a visual display of seven black elephants set against a colored, floral background. The elephant foreground was slowly accelerated against the stationary floral background until the onset of blurring of the elephant-sequence was reported by the examinee in the absence of compensatory concentration and voluntary tracking techniques. The speed of the elephant-sequence triggering blurring (i.e., "blurring-speed") was assumed to be a measure of the maximum reflex oculomotor tracking capacity. To measure the perceptual span in this mode, observers were asked to report the number of elephants they could clearly recognize just before the blurring-speed was reached.

The Mode III gestalt consisted of a moving optokinetic foreground (black stripes resembling a picket fence) projected against a stationary background consisting of a visual span of seven black elephants set against a blank, white surround (Mode I gestalt). Observers were asked to concentrate on the moving foreground and to report whether the elephant-sequence was experienced as blurred and to describe whatever they saw. Experiencing background-blurring was assumed to be an indicator of impaired capacity for foreground/background fixation and refixation. Movement illusions consisted in experiencing foreground/background movement reversals, i.e., subjects experienced themselves or the stationary elephants in motion. Both background-blurring and movement illusions were considered probable indicators of perceptual instability.

Results

The learning disabled group and the controls were compared on 11 major symptom-categories as well as neurological, ENG, and optokinetic tracking and perceptual span parameters using two-tailed t tests and χ^2 tests. The χ^2 tests employed the Yates correction. Similar analyses were done comparing ADD versus nonADD and monopedal Romberg-positive versus Romberg-negative samples within the learning disabled group.

Comparison of Historical or Symptomatic Parameters

The 11 general symptom-categories previously found to characterize learning disabilities were significantly ($p<.001$) more frequent among the learning disabled patients than control subjects. These data are shown in Table 1. As noted in Fig. 1, 14 or 40% of the control group qualitatively evidenced some past or present learning-disability-related symptoms, however mild or compensated. For the majority of these 14 subjects, the overlap of

TABLE 1
DISTRIBUTION OF SYMPTOMS IN LEARNING DISABLED AND CONTROL ADOLESCENTS ($ns = 35$)

Symptoms-Categories (General)	Learning Disabled		Control Group		χ_1^2	p
	n	%	n	%		
Reading	32	91.4	4	11.4	41.7	<.001
Writing	32	91.4	11	31.4	24.1	<.001
Spelling	27	77.1	3	8.6	30.9	<.001
Mathematics	28	80.0	5	14.3	27.8	<.001
Memory	30	85.7	4	11.4	35.7	<.001
Time	24	68.6	1	2.9	30.1	<.001
Direction	25	71.4	4	11.4	23.6	<.001
Speech	29	82.9	5	14.3	30.3	<.001
Grammar	16	45.7	1	2.9	15.2	<.001
Activity Level	20	57.1	3	8.6	16.6	<.001
Concentration	29	82.9	4	11.4	33.0	<.001

symptoms appeared significantly less than that characterizing the learning disabled sample of 35. The learning disabled group had a mean number of 8.3 ± 2.0 overlapping symptoms as compared to 1.3 ± 2.2 in the control group ($t_{68} = 14.2$, $p < .001$). These results are consistent with the expectations that subjects with severe symptoms will most likely be referred and that

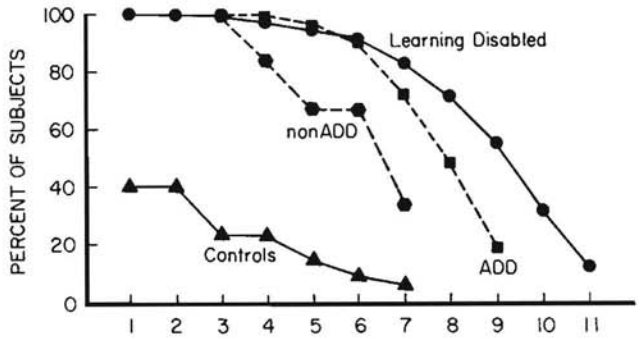

FIG. 1. The comparison of 11 overlapping symptom-categories in Learning Disabled vs Control groups (—) and comparison of 9 (nonconcentration and nonactivity) symptom-categories in ADD vs nonADD subsamples of Learning Disabled groups (---)

milder and perhaps hidden or compensated forms of this disorder may exist in matched random and even so-called "normal" control groups. Since none of the control sample showed more than seven overlapping symptoms but 71.4% of the learning disabled group did so, the number of overlapping symptoms may prove useful for intergroup separations in subsequent studies.

To explore a possible relation between ADD and learning disabilities, the learning disabled sample was divided into those with and without

ADD-like concentration and activity symptoms (Table 2). The nine general nonADD symptom-categories as well as the amount of overlapping of symptoms were estimated for these subgroups. The ADD group had a mean number of 7.2 ± 1.4 overlapping symptoms as compared to 5.5 ± 1.9 in the nonADD group ($t_{33} = 2.7$, $p = .01$). As noted in Fig. 1, while the ADD group had more symptoms than the nonADD group, the quality and over-all patterns of the nonconcentration and nonactivity symptoms in both ADD and nonADD learning disabled subsamples are basically similar. It appears reasonable to suspect that ADD and learning disabilities were reflections of the same disorder. Since the frequency and overlap of all related symptoms was significantly higher in the ADD than in the nonADD subsamples (Fig. 1), it appeared likely that impaired concentration destabilized the underlying mechanisms responsible for learning disability-related symptom formation and/or compensation. This reasoning is consistent with a wide range of neurophysiological data and clearly accounts for the higher frequency of symptoms and overlapping in the ADD subsample (Levinson, 1980, 1988, 1989a, 1990).

TABLE 2

DISTRIBUTION OF SYMPTOMS IN ADD* AND NONADD SUBGROUPS (nS = 35)

Symptom-Categories (General)	ADD Subgroup		NonADD Subgroup		χ_1^2	p
	n	%	n	%		
Subgroup Size	29	82.9	6	17.1		
Reading	27	93.1	5	83.3	0.0	ns
Writing	27	93.1	5	83.3	0.0	ns
Spelling	23	79.3	4	66.7	0.0	ns
Mathematics	24	82.8	4	66.7	0.1	ns
Memory	26	89.7	4	66.7	0.7	ns
Time	21	72.4	3	50.0	0.4	ns
Direction	23	79.3	2	33.3	3.1	.08
Speech	26	89.7	3	50.0	3.1	.08
Grammar	13	44.8	3	50.0	0.0	ns
Activity Level	20	69.0	0	0.0		
Concentration	29	100.0	0	0.0		

*The ADD subsample contains all learning disabled subjects with concentration and/or activity symptoms.

Comparison of Neurological Parameters

As noted in Table 3 and Fig. 2, CV neurological signs differentiated significantly between the learning disabled sample and the controls. None of the learning disabled or control samples had any cerebral signs. Specifically, in these comparisons a majority of the CV signs (Romberg-monopedal, tandem dysmetria, and finger-to-finger sequencing) were significantly higher for

TABLE 3
NEUROLOGICAL PARAMETERS FOR LEARNING DISABLED VS CONTROLS ($ns = 35$)

Neurological Parameters	Learning Disabled		Control Group		χ_1^2	p
	n	%	n	%		
CV Signs						
Ocular Dysmetria	27	77.1	26	74.3	0.0	ns
Romberg-monopedal	18	51.4	6	17.1	7.7	.006
Dysdiadochokinesis	7	20.0	3	8.6	1.1	ns
Finger-nose Dysmetria	7	20.0	1	2.9	3.5	.06
Finger-finger Sequencing	20	57.1	3	8.6	16.6	<.001
Tandem Dysmetria	13	37.1	1	2.9	10.8	<.001
Cerebral Signs	0	0.0	0	0.0	0.0	ns

the learning disabled group while finger-nose dysmetria showed a trend in the same direction. The comparisons on the three parameters which were statistically significantly different remained significant when a more stringent statistical criterion of $p = .008$ was used to adjust for the six multiple comparisons being made. Although the fact that 20% of learning disabled evidenced dysdiadochokinesis compared to 8.6% for the controls appeared clinically significant, the number of patients was too small for meaningful

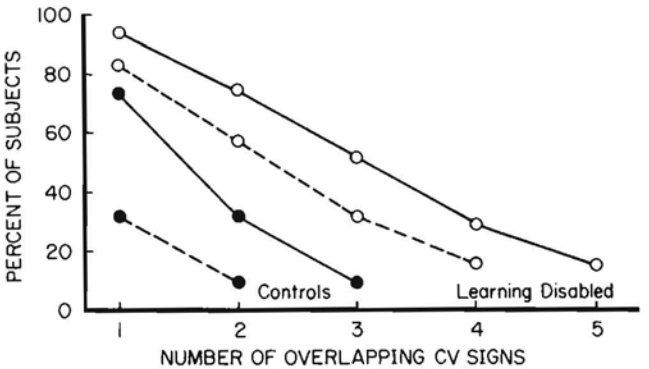

FIG. 2. The two comparisons indicate overlapping CV signs, including ocular dysmetria, in the Learning Disabled and Control groups (—) and overlapping CV signs, excluding ocular dysmetria (---).

statistical interpretation. These data further supported the hypothesis that learning disabilities or dyslexia was of a primary CV origin. As clinically tested, ocular dysmetria was only slightly higher in the learning disabled (77.1%) than in the control group (74.3%). Accordingly, one might conclude that either this parameter is improperly assessed or that a majority of learning disabled and control subjects evidence ocular dysmetria. Thus Fig. 2 compares learning disabled and control subjects on overlapping CV signs

with and without including ocular dysmetria. Also the presence of CV signs in the control group justifies this type of neurological examination in learning disability studies using so-called "normal" samples.

Comparison of ENG Parameters

No significant differences in frequency were noted for the traditional positional and caloric ENG parameters which characterized the learning disabled and control groups (Table 4). These results are consistent with those of Stockwell and associates and Polatajko; they suggest that the traditionally used ENG method and parameters described here are not helpful in distinguishing learning disabled from control adolescent patients.

TABLE 4
ENG PARAMETERS: LEARNING DISABLED VS CONTROLS ($ns = 35$)

ENG Parameters*	Learning Disabled		Control Group		χ_1^2	p
	n	%	n	%		
Positional Dysfunction	32	91.4	32	91.4	0.2	ns
Standard						
Horizontal Nystagmus	20	57.1	18	51.4	0.1	ns
Vertical Nystagmus	33	94.3	32	91.4	0.0	ns
Caloric Dysfunction	4	11.4	2	5.7	0.2	ns
Abnormal Directional Preponderance	3	8.6	2	5.7	0.0	ns
Abnormal Reduced Vestibular Response	3	8.6	0	0.0	1.4	ns
Simultaneous Caloric Dysfunction	24	68.6	22	62.9	0.1	ns
Type 2	9	25.7	5	14.3	0.8	ns
Type 3	4	9.7	1	2.9	0.9	ns
Type 4	13	37.1	15	42.9	0.1	ns
ENG Abnormalities						
1 or more signs	35	100.0	34	97.1	0.0	ns
2 or more signs	25	71.4	24	68.6	0.0	ns
Romberg Position (bipedal)						
Eyes open						
Horizontal Nystagmus	0	0.0	0	0.0	0.0	ns
Vertical Nystagmus	1	2.9	0	0.0	0.0	ns
Eyes closed						
Horizontal Nystagmus	5	14.3	4	11.4	0.0	ns
Vertical Nystagmus	25	71.4	24	68.6	0.0	ns

*Abnormal results are reported on the basis of traditionally used ENG criteria and definitions.

However, since a significant percent of dyslexics or learning disabled reported balance-related symptoms and showed positive monopedal Romberg signs and some evidenced rapid eye movements, especially of a vertical nature during eyes-closed testing, it appeared reasonable to suspect the underlying presence of, and to test for, vertical nystagmus and especially the degree in standard positions and a new ENG bipedal Romberg position

(Levinson, 1980, 1988, 1989d). Respective multivariate tests for over-all group effects on the set of 14 standard ENG parameters (multivariate $F_{14,55} = 1.46$, $p = .16$) and Romberg positional parameters with computable data (multivariate $F_{3,66} = 2.02$, $p = .12$) indicated no significant over-all differences between the learning disabled and control groups. (See Table 5 below.)

TABLE 5
DEGREE OF POSITIONAL AND DIRECTIONAL NYSTAGMUS (DEGREE/SEC.): LEARNING DISABLED VS CONTROL GROUPS ($ns = 35$)

Positional Parameters	Learning Disabled		Control Group		t_{68}	p
	M (degree/sec.)	SD	M (degree/sec.)	SD		
Supine 0° head up						
Horizontal Nystagmus	0.4	1.0	0.2	0.6	1.2	ns
Vertical Nystagmus	7.3	7.6	3.4	4.8	2.5	.01
Supine 30° head/neck/body, straight ahead						
Horizontal Nystagmus	0.2	1.0	0.2	0.6	0.2	ns
Vertical Nystagmus	6.7	8.4	5.0	6.7	0.9	ns
Right neck torsion						
Horizontal Nystagmus	0.5	1.3	0.2	0.7	1.3	ns
Vertical Nystagmus	3.2	4.0	2.1	3.0	1.2	ns
Left neck torsion						
Horizontal Nystagmus	0.3	1.1	0.2	0.1	0.8	ns
Vertical Nystagmus	3.1	6.0	1.7	2.9	1.3	ns
Right lateral						
Horizontal Nystagmus	0.5	1.3	0.2	0.1	1.3	ns
Vertical Nystagmus	3.5	8.8	2.7	4.7	0.6	ns
Left lateral						
Horizontal Nystagmus	0.4	1.2	0.3	0.7	0.2	ns
Vertical Nystagmus	2.1	3.1	1.5	2.4	0.9	ns
Caloric Reactivity						
Directional Preponderance	10.1	14.9	11.1	10.3	0.3	ns
Reduced Vestibular Response	13.6	15.9	8.1	7.4	1.9	.07
Romberg Position (bipedal)						
Eyes open						
Horizontal Nystagmus	0.0	0.0	0.0	0.0		
Vertical Nystagmus	0.1	0.6	0.0	0.0	1.0	ns
Eyes closed						
Horizontal Nystagmus	0.7	1.3	0.4	1.0	0.7	ns
Vertical Nystagmus	7.1	7.5	4.1	4.0	2.3	.04

However in the univariate analysis noted in Table 5, the degree (rather than the abnormal presence) of vertical nystagmus in the eyes-closed bipedal Romberg ($p = .04$) and in the supine 0° ($p = .01$) position and the degree of reduced vestibular response ($p = .07$) were significantly higher or tended to be higher in learning disabled than in the control group. Moreover, three

subjects of the 70 adolescents evidencing abnormal reduced vestibular responses (RVR) as traditionally diagnosed were in the learning disabled group (Table 4).

To assess a possible relationship between the positive monopedal Romberg sign and the degrees of reduced vestibular response, directional preponderance and vertical nystagmus, measures of the latter parameters were compared in learning disabled subsamples with and without clinically positive monopedal Romberg neurological signs. As seen in Table 6, the above-mentioned ENG parameters reflected greater impairments in the 18 learning disabled patients who had positive monopedal Rombergs (multivariate $F_{9,25} = 2.53$, $p = .032$) compared to the Romberg-negative group. How-

TABLE 6
ROMBERG-MONOPEDAL, VERTICAL NYSTAGMUS, AND VESTIBULAR REACTIVITY IN THE LEARNING DISABLED SUBJECTS (nS = 35)

Measures	+ Romberg, $n = 18$		− Romberg, $n = 17$		t_{33}	p
	M	SD	M	SD		
Degree of Vertical Nystagmus (degree/sec.)						
Supine 0° head up	9.0	10.0	5.5	3.2	1.4	ns
Supine 30° head/neck/body, straight ahead	9.5	9.0	3.9	1.8	2.2	.04
Right neck torsion	4.1	4.6	2.5	3.3	1.4	ns
Left neck torsion	5.3	7.8	0.8	0.7	2.4	.03
Right lateral	5.1	6.1	1.8	1.3	1.9	.06
Left lateral	3.1	3.7	0.8	1.5	2.1	.05
Romberg Position (bipedal) , eyes closed	10.9	7.8	3.3	3.3	3.6	.001
Degree of Vestibular Reactivity						
Reduced Vestibular Response	15.6	15.3	11.6	9.3	0.7	ns
Directional Preponderance	8.7	5.5	11.5	6.4	0.5	ns

ever, only the degrees of vertical nystagmus in the eyes-closed ENG Romberg position ($p = .001$), in the supine 30° position ($p = .04$), left-neck torsion position ($p = .03$) and left-lateral position ($p = .05$) were significantly higher in the learning disabled group who manifested abnormal monopedal Romberg neurological signs. There was also a trend ($p = .06$) for vertical nystagmus in the right-lateral position to be higher in the Romberg-positive group. Were these findings to be substantiated in larger samples, then the need for modifying the standard ENG procedures should be seriously considered. Moreover, a heretofore hidden relationship between CV-based neurological and ENG parameters appears to have been highlighted for more thorough exploration.

Comparison of Optokinetic and Related Parameters

As noted in Table 7, all six optokinetic-related parameters used significantly differentiated the learning disabled and control groups. Univariate

analysis with t tests indicated that the mean scores measuring functions related to oculomotor tracking capacity (blurring-speed) and perceptual spans during Modes I, II and III testing were significantly impaired ($p < .001$) for the learning disabled vs control samples. Similarly, the frequency of fixation and perceptual instability (background-blurring [$p = .01$] and movement illusions [$p = .02$]) were significantly higher in learning disabled than in control groups. Background-blurring and movement illusions were statistically significantly different for the two groups even when a more stringent criterion of $p = .025$ is used to adjust for the comparison of two correlated parameters.

TABLE 7
OPTOKINETIC TRACKING PARAMETERS: LEARNING DISABLED VS CONTROLS (nS = 35)

Optokinentic Parameters	Learning Disabled		Control Group		t_{68}	p
	M	*SD*	*M*	*SD*		
Maximum Tracking Capacity or Blurring Speed (ft./sec.)	1.7	0.8	3.1	1.8	4.6	<.001
Perceptual Span or Number of Elephants Seen						
Mode I Testing	2.0	1.5	3.8	1.8	4.6	<.001
Mode II Testing	2.2	1.4	4.3	1.7	5.7	<.001
Mode III Testing	1.8	1.2	4.0	1.7	6.4	<.001
Perceptual Instability	*n*	%	*n*	%	χ_1^2	*p*
Impaired Fixation, Refixation or Background-blurring	19	54.0	8	23.0	6.0	.01
Movement Illusions	12	34.0	3	9.0	5.4	.02

All the above-mentioned oculomotor findings appear consistent with the clinically-derived origin of these parameters, i.e., their relationship to the frequency with which dyslexics or learning disabled with reading symptoms report fixation and tracking-related reading errors as well as such corresponding symptoms as blurring, oscillopsia, and tunnel vision or "single targeting" when evaluated using the historical symptomatic questionnaire.[2] Moreover, these data are consistent with the findings of other researchers indicating that the CV circuits modulate optokinetically induced ocular fixation and tracking capacities (Baloh, Honrubia, & Sills, 1977; Ito, 1984; Eccles, personal correspondence, 1987).

DISCUSSION

The results of this analysis appear to have validated the diagnostic value and significance of the CV-based neurological and optokinetic parameters in dyslexia or learning disabilities. Although the traditional ENG method did not distinguish learning disabled from the control adolescents, modifications of this technique and scoring showed diagnostic potential. Moreover, present

and prior analyses of the high prevalence of ADD in learning disabled as well as the higher frequency of related symptoms and overlap in ADD-subgroups of learning disabled suggested (1) that learning disabilities or dyslexia and ADD might be reflections of the same underlying CV disorder (Levinson, 1988, 1990[3]) and (2) that concentration and activity symptoms may significantly destabilize the determining and/or compensatory mechanisms in learning disabled and so maximize the appearance of symptom formation. Conversely, these latter findings are consistent with overwhelming clinical and experimental data indicating that enhanced concentration plays a vital role in compensating for CV-dysfunctioning as well as for the related neurological, ENG and oculomotor signs and symptoms including those characterizing dyslexia, vertigo, and motion sickness.

In a compendium on screening in child health care, North (1974) states: "Nothing has been learned in the last 70 years to refute Osler's maxim that 90% of diagnosis (and insight) is based on history" (p. 635). Since the historical screening questionnaire used here was shown significantly reliable in assessing and differentiating the symptom-categories and overlap characterizing the various samples and subgroups in this study and formed the basis for noting 40% of the controls had experienced mild or compensated past and/or present forms of learning-disability-related symptoms, it appears reasonable to suggest that subsequent research use a similar screening procedure to examine corresponding groups, especially those considered normal.

As previously noted, changes in the ENG procedure and scoring led to new measures with diagnostic potential and significant insights concerning a possible relation between the CV neurological signs in learning disabled (i.e., positive monopedal Romberg) and the amount of vertical nystagmus, especially in the bipedal ENG Romberg position. In retrospect, it appeared reasonable to wonder whether mechanisms of CV-determined imbalance in the learning disabled group were not responsible for triggering abnormal monopedal Romberg neurological signs and related vestibular asymmetry as evidenced by the greater reduced vestibular response and correspondingly lower directional preponderance as well as the greater vertical nystagmus, especially in the bipedal Romberg ENG position. In prior studies (Levinson, 1988, 1989c), abnormal reduced vestibular response (RVR) was statistically associated with mixed-handedness and fears of height. Such data suggested that a significant amount of vestibular asymmetry might secondarily destabilize the coordination and related somatopsychic mechanisms responsible for complete right-handedness and left-handedness as well as cerebral dominance

[3]These findings are consistent with Wender (1971, 1987), Safer and Allen (1976), and Lambert and Sandoval (1980), indicating that up to 50% of those diagnosed ADD have dyslexia or learning disabilities as well as balance and coordination signs.

and lead to a higher incidence of compensatory mixed-lateral and mixed-central dominance while triggering imbalance mechanisms expressed as fears of losing one's footing, falling, or fears of height. Perhaps the use of these new quantitative and qualitative ENG variables may highlight the CV mechanisms and/or the extent required for symptom formation in learning disabilities and salvage the diagnostic basis of the ENG methodology.

Moreover, the CV neurological and related data reported by this author here and in prior works dating back to 1973 were remarkably consistent with the "mysteriously neglected" findings of Orton as reviewed by Geschwind (1982):

> He pointed out the *frequency of clumsiness in dyslexics.* Although others have commented on this, it still remains a mysterious and not adequately studied problem. It is all the more mysterious in view of the fact that many of these clumsy children go on to successes in areas in which high degrees of manual dexterity are absolutely necessary! (p. 17).

In retrospect, the frequency of clumsiness in dyslexia as described by Orton and Geschwind suggests also a cerebellar-vestibular (CV) rather than a cerebral origin and supports the neurophsyiological data and explanation presented in this paper. Moreover, CV-determined balance, coordination and rhythmic or clumsiness symptoms, including speech delays as well as stuttering and articulation errors (i.e., "soft signs"), are readily compensated by practice or repetition, maturation, and CV-related therapies (Levinson, 1984, 1988, 1989a). Indeed, subjects with poor gross motor coordination may demonstrate normal or even exceptional fine motor coordination skills, and the reverse, depending on the specific patterns of impaired vs compensatory CV functioning. [Even the higher male/female ratios noted in this disorder appear determined more by secondarily related emotional and referral determinants than genetic or constitutional factors (Levinson, 1988)]. These many considerations together with findings indicating that handedness and dyslexia or learning disabilities are significantly unrelated (Levinson, 1980, 1988; Orton, 1942) tend to refute both the primary cerebral dominance theories of dyslexia and their underlying assumptions (Orton, 1937; Geschwind, 1982, 1986; Geschwind & Behan, 1982).

Is it really true that the role of clumsiness and its compensation in dyslexia has been inadequately studied as stated by Geschwind or has this role and its CV determinants been significantly denied (Levinson, 1980)? Might Orton's association of dyslexia with clumsiness, speech disorders including stuttering, and mixed-laterality as well as his resulting cerebral dominance theory have overlooked primary CV determinants and related neurophysiological signs and considerations such as those reported here?

Obviously, follow-up and independent studies of substantially larger samples using additional objective methods are needed to cross-validate these findings.

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