

DYSMETRIC DYSLEXIA AND DYSPRAXIA*
Hypothesis and Study

Jan Frank, M.D. and Harold Levinson, M.D.

Reprinted from

THE JOURNAL OF
THE AMERICAN ACADEMY OF
CHILD PSYCHIATRY

VOLUME TWELVE NO. 4, OCTOBER, 1973



Quadrangle The New York Times Book Co.
10 East 53rd Street, New York, N.Y. 10022

DYSMETRIC DYSLEXIA AND DYSPRAXIA*

Hypothesis and Study

Jan Frank, M.D. and Harold Levinson, M.D.

Of 115 consecutive dyslexic children selected and referred for psychiatric evaluation on the basis of their poor or refractory response to reading instruction, 112 (97%) children showed evidence of a cerebellar-vestibular dysfunction. This cerebellar-vestibular dysfunction manifested itself in the dyslexic children by: positive Rombergs, difficulty in tandem walking, articulatory speech disorders, dysdiadochokinesis, hypotonia, and various dysmetric or past-pointing disturbances during finger-to-nose, heel-to-toe, writing, drawing, as well as during ocular fixation and scanning testing (Dow and Moruzzi, 1958).

Goodenough figure drawings (1926; Bender, 1951) and Bender Gestalt designs (1938) revealed in all cases 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 angle formations, suggested that the

Dr. Frank is an Associate Professor of Psychiatry, Downstate Medical Center, State University of New York. Dr. Levinson is a Clinical Instructor of Psychiatry, Downstate Medical Center, State University of New York; School Psychiatrist, Bureau of Child Guidance, New York City Board of Education; Attending in Psychiatry, Long Island Jewish Hillside Medical Center.

Reprints may be requested from Dr. Frank, 45 East 82 Street, New York, N.Y. 10028.

**The term "dyspraxia" in "dysmetric dyslexia" is used as an analogy to the parietal dyspraxias and refers to the dysmetric, steering and spatial orientation disturbances noted in the writing and drawings of dysmetric dyslexic children.*

automatic copilot or the inner spatial steering and equilibrium mechanism of the vestibular apparatus and cerebellar-vestibular circuits was impaired.

SAMPLE

The 115 dyslexic children in this study were English-speaking, of average to above-average intelligence, ranging in age from 6½ to 14 and attending grades 1 through 6 in the New York City Public School system. They were selected as follows: 40 2nd- through 6th-graders in the Special Reading Service were referred for psychiatric evaluation on the basis of their poor or refractory response to 1 hour of remedial reading biweekly for at least 1 year; 15 2nd- through 6th-graders were referred for psychiatric evaluation on the basis of their being 2 or more years behind in their reading; 60 1st-graders in the Supportive Reading Program were selected and referred for psychiatric evaluation on the basis of their poor performance on an initial screening test and the supportive reading teacher's opinion of their slow progress. This refractory group of children represents a highly selected minority population among the total number of children in the Bureau of Special Reading Services and the Supportive Reading Program.¹ (Four mentally retarded cerebellar-vestibular positive children with diffuse brain dysfunction were excluded from this study.)

METHODS

The authors examined each of the 115 dyslexic children neurologically and psychiatrically. Seventeen children of this dyslexic group were randomly selected and referred for neurological ex-

¹ One of the authors, representing the Bureau of Child Guidance, together with Mr. Maurice Wollin, Community Superintendent, District 31, Borough of Richmond, organized and supervised the Supportive Reading Program for Staten Island. This program replaced and expanded the decentralized Bureau of Special Reading Services in 1971. The authors wish to thank the Bureau of Child Guidance, the Bureau of Special Reading Services, Mr. Maurice Wollin, and especially the supportive reading teachers, Cathleen Amaro, Ruth Baker, Louise Banks, Mary Brown, Francis DeMarco, Phyllis Eisner, Elaine Jensen, Marie Mainer, and Anne Padula, for their cooperation.

aminations and 30 for ear, nose, and throat, audiographic and electronystagmography examinations.²

RESULTS

All 17 cases referred for neurological examinations were found to have a cerebellar deficit. Twenty-six of the thirty electronystagmography examinations were abnormal or positive and revealed spontaneous and positional nystagmus, dysmetric ocular pursuit as well as asymmetric functioning of the vestibular apparatus ranging from hypo- to hyperfunction to directional preponderance of one labyrinth relative to the other in the presence of normal ear, nose, and throat, and audiographic findings. Two additional cases showed unilateral canal paresis when tested by cold caloric stimulation using the Kobrak technique (1919-20). Their audiograms were normal, but electronystagmography examinations were not performed. (One dyslexic child with left 8th cranial nerve deafness and left canal hyperactivity on the electronystagmogram was not counted in this study.)

HYPOTHESIS

The surprising finding of a cerebellar-vestibular dysfunction in 112 of 115 dyslexic children suggested the idea that the cerebellar-vestibular circuits may play an important etiological role in a form of dyslexia we call dysmetric dyslexia. We propose the following hypothesis:

1. The cerebellar-vestibular circuits provide a harmonious, well-integrated, and stable motor background for visual perception.
2. This motor background or motor Gestalt is nothing more than

² Dr. Sidney Carter, Professor of Neurology and Pediatrics, Columbia Presbyterian Hospital, examined 3 children neurologically, and Dr. Arnold Gold, Associate Professor of Neurology and Pediatrics, Columbia Presbyterian Hospital, examined 14. Dr. James Holman, Clinical Associate Professor of Otorhinolaryngology, Cornell University Medical College, and Dr. Noel Cohen, Clinical Associate Professor of Otolaryngology, New York University Medical College, were responsible for all but 2 (performed at Mt. Sinai Hospital) ear, nose, and throat, audiographic, and electronystagmography examinations. The cooperation of our colleagues was especially appreciated as they participated in this study without any identifying data.

the subliminal, automatic, integrated motor activity of the eye muscles, head, and neck so that ocular fixation and sequential scanning of letters and words can take place.

3. In the presence of a cerebellar-vestibular dysfunction and sub-clinical nystagmus, ocular fixation and sequential scanning of letters and words are disordered, and letter and word scrambling results.

4. This scrambling and resulting dysmetric visual perception lead to deficient comprehension or dyslexia.

DISCUSSION

The reading disturbance in dysmetric dyslexia can be compared to the normal reading difficulty one experiences when reading a sign-board from the window of a moving train. The ensuing fixation nystagmus interferes with intentional fixation on the one hand, and sequential scanning on the other, and results in letter and word scrambling. For example, the biggest or first letter of a word is often fixated first during the slow right-to-left phase of the nystagmus. The rapid left-to-right phase often skips over several letters or a whole word until another letter is automatically fixated and scrambling results.

If in dysmetric dyslexia one assumes the existence of a subclinical nystagmus, a resulting letter and word scrambling, and an accompanying secondary anxiety, then many of the otherwise confusing reading disturbances become understandable, i.e., visual attention difficulties, comprehension impairment in the absence of agnosia, and variable reversal tendencies.

The existence of a subclinical nystagmus in dysmetric dyslexia is: (1) strongly supported by the expected and usual occurrence of nystagmus in cerebellar-vestibular dysfunction; and (2) proven by the presence of spontaneous nystagmus during electronystagmography examinations when the eyes were closed and the position of the head changed—despite the absence of clinical nystagmus during repeated neurological examinations.

In addition, from the spatial orientation disturbances seen in dysmetric dyslexia we can explain the patient's confusion of letters and words which differ only or mainly in spatial placement, i.e., b=d=p=q, a=e, ε=3, c=u, m=w, saw=was, no=on. This visual

dysmetric perception is the ocular motor equivalent of onomatopoeic sounds and words so often noted in the early speech process of children.

Only 3 of the initial 40 dyslexic cases examined had evidence of a cortical dysfunction.³ It was our opinion, however, that the cortical dysfunction merely facilitated the emergence of the aforementioned reading disturbances, but was not primarily responsible for them. We consider cerebellar-vestibular-positive dyslexic children to be dysmetric dyslexic only in the presence of an intact cortex. We further believe that a primary cerebellar-vestibular dysfunction in children, together with its resulting dysmetric visual perception and accompanying anxiety, can lead to a maturational lag. The resulting symptoms can best be understood as follows (Goldstein, 1936):

Study of the symptoms in lesions of the cerebellum has shown that it is impossible to come to a real comprehension so long as we regard the symptoms only from the viewpoint of the lesion alone. Real comprehension can only come if we regard the single part as a part of the brain as a whole and the symptoms as performances of the brain deprived of one certain part. Thus, each theory of the function of one part is only possible in the frame of a theory of the brain and, further, of the organism as a whole. Only then, we are able to characterize the particular significance of one single part, that is, as its significance in the performances of the whole organism [p. 11].

³ Patricia J. is a 10-year-old 3rd-grader with a compensated expressive aphasia, word-finding difficulties, and a Wechsler Intelligence Scale for Children (WISC) of 76.

Maureen L. is an 8½-year-old 3rd-grader with a compensated expressive aphasia, word-finding difficulties, and a WISC of 86. She had a 1-minute seizure when 1 year old. Upon awakening, she screamed, her head and body were shaking, and her eyes rolled. Dr. A. Gold examined her in 1971 and noted: "At 2 she fell . . . and subsequently there were prominent periorbital hematomas which usually indicate an anterior fossa fracture. . . . Her EEG was generally abnormal with dysrhythmic features both in the resting state and during hyperventilation . . . generalized slow wave episodes at 3 CPS sometimes containing notched slow waves. With drowsiness the record became generally disorganized and slow with an increase in the episodic disturbances . . . more in the left temporal occipital region."

Patricia D. is an 8-year-old 3rd-grader with a WISC of 83, a history of 2 half-minute seizures between ages 2-4 and 1 at 5. "She'd fall with her mouth and eyes open and her eyes rolled" without tonic clonic movements or urinary or fecal incontinence. Numerous falling and tripping episodes resulted in 2 concussions during 1969-70. An EEG performed at Downstate Medical Center in 1971 was "mildly and diffusely slow."

Approximately 5 of the remaining 75 dyslexic children in this study had evidence of a cortical dysfunction. We are awaiting the results of more definitive neurological evaluations.

We are well aware, of course, that a correlation does not indicate a causation and that the cerebellar-vestibular disturbances found in these children may be indicative only of a general central nervous system difficulty or be an unrelated finding. To explore further the relation between cerebellar-vestibular dysfunction and dyslexia we are completing the neurological, ear, nose, and throat, audiographic, and electronystagmography examinations of 150 randomly selected dyslexic children in order: (1) to determine more accurately the incidence of cerebellar-vestibular dysfunction in dyslexia; (2) to study further the presence and role of subclinical nystagmus in dysmetric dyslexia; (3) to study the reading process during experimental alterations of the nystagmus with cerebellar-vestibular harmonizing agents such as cyclizine (Marezine); (4) to evaluate the use of cyclizine alone or in combination with low doses of methylphenidate (Ritalin) in the prevention and treatment of dysmetric dyslexia in cerebellar-vestibular-positive preschool and school children respectively. We suspect methylphenidate charges the neocortex via its stimulating effect on the mesencephalic reticular activating and alerting system of Magoun (1952) and, furthermore, that methylphenidate will therapeutically complement the harmonizing effect of cyclizine (Marezine) despite its pharmacological antagonism as a stimulant; (5) to compare cerebellar-vestibular-positive and cerebellar-vestibular-negative organically determined dyslexic cases in order to understand better the neurophysiological contributions of the cerebellar-vestibular circuits (and their related ascending and descending pathways) to the varied symptomatology seen in dyslexia, i.e., reversals, rotations, perceptual-motor deficits, etc.; (6) to study the possible role of otitis interna or (subclinical) labyrinthitis in the development of cerebellar-vestibular dysfunction and dysmetric dyslexia; and (7) to study hereditary dysmetric dyslexia. Cerebellar-vestibular dysfunction and dyslexia were noted in the fathers of 4 dysmetric dyslexic children. Thus, genetic loading may be etiologically significant in the development of cerebellar-vestibular dysfunction in some forms of dysmetric dyslexia.

In addition, we are investigating the reading processes, cerebellar-vestibular functioning, and electronystagmograms in the following control groups: (1) normal children without dyslexia; (2) reading disabilities that readily respond to remedial reading; (3) cases of

ocular or retinal nystagmus, i.e., congenital nystagmus, nystagmus associated with albinism, and miner's nystagmus; and (4) cases of cerebellar-nystagmus, i.e., multiple sclerosis and inferior cerebellar artery syndromes.

The correlation between refractory dyslexia and dysmetric dyslexia by no means represents an equation. Some of the dysmetric dyslexic children initially referred because of their poor response to remediation eventually scored close to or above average in their reading achievement—depending upon their endowment, compensatory ability, neurotic overlay, psychotherapeutic intervention, and the quality and intensity of their instruction. We believe the above correlation merely reflects the high incidence of dysmetric dyslexia among the organically determined dyslexias, which as a group are least likely to respond to ordinary reading remediation.

Should the incidence of cerebellar-vestibular-positive and electronystagmogram-positive dyslexic cases be as high as suspected relative to the total organic dyslexic population, then electronystagmography would prove highly useful as a rather simple procedure for early detection and prediction of organic (dysmetric) dyslexia in preschool and kindergarten children.

Furthermore, electronystagmography could serve as an invaluable aid in differentiating dysmetric dyslexia from the other organic forms of dyslexia and the nonorganic psychogenic and faulty teaching dyslexias as well (Blanchard, 1946; Pearson, 1952). The latter group, in our experience, accounts for the vast majority of reported reading disabilities. The difficulty in differentiating the organic from the nonorganic dyslexias can be accounted for in part by the fact that children with brain dysfunction are highly susceptible to neurotic and personality disorders. Thus, most, if not all, organic dyslexias have a neurotic superstructure and are in reality mixed organic-neurotic overdetermined symptom complexes or pathoneuroses (Rosen, 1955; Ferenczi, 1916-17).

The information obtained from the completed study should lead to an improved nosology for dyslexia based upon neuropsychological, neurophysiological, and pathogenetic principles and corresponding development of psychological, pedagogical, and medical treatment techniques, i.e., the use of cerebellar-vestibular harmonizing agents alone or in combination with low doses of methylphenidate for the prevention and treatment of dysmetric dyslexia.

INCIDENCE OF DYSMETRIC DYSLEXIA

The Supportive Reading Program of Staten Island, New York City Board of Education, screened over 850 of a total 1st-grade population of 3,273 children during September and October of 1971. The children were distributed throughout all 29 (Non-Title-I) elementary schools.

The supportive reading teachers selected all those children entering 1st grade who were thought by their kindergarten and 1st-grade teachers to be slow in acquiring reading skills and who might be helped by supportive reading techniques. Of these screened children 803 were selected for supportive reading instruction 2 to 3 times a week for 1 year. Of the 803 children in the reading program 75 were selected for neuropsychiatric evaluation on the basis of their poor performance on an initial screening test and the supportive reading teacher's opinion of their slow progress. Of the 75 children 51 have thus far been neurologically and psychiatrically evaluated and 49 (96%) had evidence of a cerebellar-vestibular dysfunction. Of the 49 cerebellar-vestibular-positive children 4 had evidence of a cortical dysfunction. If these 4 cases are removed from the group of 49, then 45 cerebellar-vestibular-positive children (88%) of the 51 1st-graders had no cortical dysfunction and were therefore considered dysmetric dyslexic. (Of these 51 children 19 were randomly selected for electronystagmography examinations, and 16 were positive.)⁴

Assuming 88 percent of all 75 children initially selected for neurological and psychiatric evaluation had only a cerebellar-vestibular dysfunction, 66 of a total 1st-grade population of 3,273 would be dysmetric dyslexic, or approximately 2 percent.

MALE/FEMALE RATIO IN DYSLEXIA

Cerebellar-Vestibular-Positive Dyslexia

The ratio of male/female cerebellar-vestibular-positive dyslexic children referred for neuropsychiatric evaluation increased with age and grade. This ratio was found to be 2.5/1 (42 males/16 females)

⁴ Only 1 of the 2 1st-grade children considered cerebellar-vestibular-negative had an electronystagmogram performed and it was negative. This child is a hyperactive distractible 7-year-old 1st-grader who showed neither letter reversals nor rotational or angle-formation difficulties on the Bender Gestalt drawings and responded favorably to methylphenidate.

for cerebellar-vestibular-positive dyslexic 1st-graders and 4/1 (43 males/11 females) for cerebellar-vestibular-positive dyslexic 2nd- to 6th-graders.

The Supportive Reading Program

The male/female ratio (436/264) in the Supportive Reading Program remained approximately 1.6/1 (385/244) even after the cerebellar-vestibular-positive dyslexic population was removed. As would be expected, the male/female ratio in the nonreferred 1st-grade population was 1/1 (1,135/1,155).

We propose the following explanation to account for the above male/female ratios:

1. The socially determined expectations and stress for male children to succeed are greater than for female children. This male/female differential stress factor is reflected in the 1.6/1 male/female ratio for the Supportive Reading Program population, the 2.5/1 male/female ratio for beginning first-grade cerebellar-vestibular-positive dyslexic children and the 4/1 male/female ratio for the 2nd-through 6th-grade cerebellar-vestibular-positive dyslexic children.

2. Cerebellar-vestibular-positive dyslexic males are more susceptible to this stress factor than nonorganic dyslexic males as reflected in the cerebellar-vestibular-positive dyslexic male/female ratios of 2.5/1 and 4/1, in contrast to the Supportive Reading Program male/female ratio of 1.6/1.

3. This socially determined male/female differential stress factor is operative not only from 1st grade onward but from earliest childhood and is reflected in the higher number of males already found in the beginning 1st-grade Supportive Reading Program (1.6/1) and cerebellar-vestibular-positive dyslexic population (2.5/1).

Although many alternative explanations are possible, our hypothesis can and will be tested when young preschool children are screened for cerebellar-vestibular dysfunction. A male/female cerebellar-vestibular-positive ratio approximating 1/1 would, in our opinion, prove this contention.

Left-Handedness in 1st Graders and Cerebellar-Vestibular-Positive Dyslexia

Of 3,000 1st-graders 360 (12%) were found to be left-handed. Of the 710 children in the Supportive Reading Program, 129 (18%) and of

2,290 in the nonreferred 1st-grade population 231 (10%) were left-handed.

Of 112 cerebellar-vestibular-positive dyslexic children 14 (11%) were left-handed. A left-handed genetic determinant was clearly evident in 10 of the 14 children: 7 had a left-handed parent; and 3 had either left-handed siblings or left-handed direct parental relatives.

Because of selective sampling, the above-reported differences in left-handedness are not considered statistically significant. We believe left-handedness to be a genetically determined variant in the general as well as in the dyslexic population; thus, it plays no special role in dyslexia.

SUMMARY

Of 115 consecutive dyslexic children selected and referred for psychiatric evaluation on the basis of their poor or refractory response to reading instruction, 112 (97%) of the children showed evidence of a cerebellar-vestibular dysfunction. Of this dyslexic group 17 children were randomly selected and referred for neurological examinations and 30 for ear, nose, and throat, audiographic, and electronystagmography studies. All 17 cases referred for neurological examinations were found to have a cerebellar deficit. Of the 30 electronystagmography examinations 26 were abnormal or positive and revealed spontaneous and positional nystagmus, dysmetric ocular pursuit, and asymmetric functioning of the vestibular apparatus in the presence of normal ear, nose, and throat, and audiographic findings. Two additional cases showed unilateral canal paresis when tested by cold caloric stimulation.

From these findings, we have evolved the following hypothesis which we plan to test in future studies:

1. The cerebellar-vestibular circuits provide a harmonious, well-integrated, and stable motor background for visual perception.
2. This motor background or motor Gestalt is nothing more than the subliminal, automatic, integrated motor activity of the eye muscles, head, and neck so that ocular fixation and sequential scanning of letters and words can take place.
3. In the presence of a cerebellar-vestibular dysfunction and sub-clinical nystagmus, ocular fixation and sequential scanning of letters and words are disordered and letter and word scrambling results.

4. This scrambling and resulting dysmetric visual perception lead to deficient comprehension or dyslexia.

5. A primary cerebellar-vestibular dysfunction in children, together with its resulting dysmetric visual perception and accompanying anxiety, can lead to a maturational lag.

6. The use of cerebellar-vestibular harmonizing agents such as cyclizine (Marezine) alone or in combination with reticular activating and alerting agents such as methylphenidate (Ritalin) is suggested for the prevention and treatment of dysmetric dyslexia in cerebellar-vestibular-positive preschool and school children respectively.

The existence of a subclinical nystagmus in dysmetric dyslexia has been clearly demonstrated by means of electronystagmography, and the incidence of dysmetric dyslexia in the 1st-grade population of Staten Island was found to be approximately 2 percent.

POSTSCRIPT

The authors have recently succeeded in proving the dysmetric dyslexic hypothesis by means of a new, rapid (2–5 minutes per child), highly accurate (98%), and specific dysmetric dyslexic mass screening procedure and instrument which can detect cerebellar-vestibular dysfunction and dysmetric dyslexia in prekindergarten (4–5-year-olds) and beginning school children without the need for the time-consuming, difficult, and cumbersome Barany caloric stimulation. The content, details, and statistical validation of the dysmetric dyslexic working hypothesis as well as the dysmetric dyslexic mass screening instrument will form the basis and subject matter of a second paper.

REFERENCES

- BARANY, R. (1901–1921), *Some New Methods for Functional Testing of the Vestibular Apparatus and Cerebellum*. Nobel Lectures, Physiology and Medicine 1901–1921. Amsterdam: Elsevier, 1967.
- BENDER, L. (1938), *A Visual Motor Gestalt Test and Its Clinical Use*. New York: American Orthopsychiatric Association Research Monograph No. 3.
- (1951), The Goodenough test (drawing-a-man) in chronic encephalitis in children. *Quart. J. Child Behav.*, 3:449–459.
- BLANCHARD, P. (1946), Psychoanalytic contributions to the problems of reading disabilities. *The Psychoanalytic Study of the Child*, 2:163–187. New York: International Universities Press.

- DOW, R. S. & MORUZZI, G. (1958), *The Physiology and Pathology of the Cerebellum*. Minneapolis: University of Minnesota Press.
- FERENCZI, S. (1916-17). Disease- or patho-neuroses. In: *Further Contributions to the Theory and Technique of Psychoanalysis*. New York: Basic Books, 1954, pp. 78-89.
- GOLDSTEIN, K. (1936), The function of the cerebellum from a clinical standpoint. *J. Nerv. Ment. Dis.*, 83:1-12.
- GOODENOUGH, F. L. (1926), *Draw-a-Man Test: The Measurement of Intelligence by Drawings*. Yonkers-on-Hudson: World Book.
- JONGKEES, L. & PHILIPSZOON, A. (1964), *Electronystagmography*. Stockholm: Oto-Laryngologica Supplementum 189.
- KOBRAK, F. (1919-20) Zur Frage einer exakten Messbarkeit der Sensibilität des Vestibularapparates. *Arch. Ohren. Nasen. u. Kehlkopf.*, 105:132-134.
- MAGOUN, H. W. (1952), The ascending reticular activating system. *A. Res. Nerv. & Ment. Dis. (Proc.)*, 1950, 30:480-492.
- PEARSON, G. H. J. (1952), A survey of learning difficulties in children. *The Psychoanalytic Study of the Child*, 7:322-386. New York: International Universities Press.
- ROSEN, V. H. (1955), Strophosymbolia: an intrasystemic disturbance of the synthetic function of the ego. *The Psychoanalytic Study of the Child*, 10:83-99. New York: International Universities Press.