DYSMETRIC DYSLEXIA AND DYSPRAXIA

Synopsis of a Continuing Research Project

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

Reprinted from

ACADEMIC THERAPY PUBLICATIONS

VOLUME XI NUMBER 2, WINTER 1975-76

Dysmetric Dyslexia and Dyspraxia

Synopsis of a Continuing Research Project

Jan Frank
Harold N. Levinson

THIS ARTICLE reports on our collaboration in a joint dyslexic research project over a period of many years. During this time we have neuropsychiatrically examined over 1,000 children usually diagnosed as having specific, primary, or developmental dyslexia.

A retrospective analysis of our extensive examinations indicated that the signs and symptoms unique to the vast majority of our cases were of cerebellar-vestibular origin: 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. *Goodenough-Harris Drawing Test* and *Bender Gestalt* designs 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-Harris* and *Bender Gestalt* drawings from their intended horizontal and vertical axes and steering difficulties during angle formations, suggested that the "automatic copilot"—the inner spatial steering and equi-

Jan Frank, MD, 45 East 82nd Street, New York, New York 10028, is an associate professor of psychiatry, Downstate Medical Center, State University of New York. Harold N. Levinson, MD, 15 Lake Road, Lake Success, New York 11020, is a clinical instructor of psychiatry, Downstate Medical Center, State University of New York; he is also a school psychiatrist for the Bureau of Child Guidance, New York City Board of Education; and he is an attending physician in psychiatry at the Long Island Jewish Hillside Medical Center, and at Nassau Hospital.

librium mechanism of the vestibular apparatus and cerebellar-vestibular circuits—was impaired.

This observation led to a prospective study in which 97 percent of 250 consecutively referred "dyslexic" children were found to have definite evidence of cerebellar-vestibular dysfunction (i.e., dysmetric sensory-motor functioning).

In view of the dysmetria correlated with and underlying the dyslexic and dyspraxic signs and symptoms, we have coined the term "dysmetric dyslexia and dyspraxia" in order to define the disorder neurophysiologically. This new definition had two significant advantages:

- It was neurophysiologically specific and, thus, allowed other investigators defining "dyslexia" according to various criteria to reexamine their cases according to our cerebellar-vestibular criteria, and, thus, determine the incidence of cerebellar-vestibular dysfunction in their dyslexic populations.
- It avoided equating dysmetric dyslexia and dyspraxia with specific, primary, and developmental dyslexia. This equation would have led to a needless confusion as the above "dyslexic" definitions were nonspecific and left much to be desired.

In order to prove our dysmetric dyslexic hypothesis, namely that a cerebellar-vestibular dysfunction, a resulting subclinical nystagmus, and ensuing ocular fixation and sequential scanning dysfunction led to dyslexia, we designed a new instrument capable of measuring the ocular fixation and sequential scanning functions in children and adults. We clearly proved that dysmetric dyslexic children have an ocular fixation and sequential scanning defect, and that normal and nondyslexic children do not.

Fallacious Assumptions in Research

Prior to our investigations, "dyslexia" in children was believed by many investigators to result from a cerebral cortical dysfunction. Even the neurologists who examined our cases in a blind study believed dysmetric dyslexia to be of cortical origin—despite their own findings of positive cerebellar signs and negative cortical signs. They believed that their positive cerebellar findings in our dysmetric dyslexic children indicated diffuse brain and cortical dysfunction—even in the complete absence of diffuse brain and cortical findings in their own reports.

The cerebellar-vestibular circuits provide the spatial and

temporal harmony of the sensory input in a manner analagous to the function of the vertical and horizontal TV stabilizers. When the horizontal and/or vertical stabilizers go awry, the resulting visual scramble makes interpretation or perception difficult to impossible and "dysmetric dyslexia" results.

Dysmetric dyslexia must be sharply distinguished from conceptual or agnostic dyslexia or cortical origin in which, by analogy, the TV picture is clear but the observer cannot comprehend the meaning and symbolic significance of the visual communication or pattern.

Previous "dyslexic" investigators, in our opinion, must have reasoned as follows:

- Adults with lesions of the dominant angular gyrus develop "dyslexia" or an agnostic and conceptual reading impairment.
- Children of normal or superior IQ and absent cortical findings develop reading disturbances characterized by letter and word reversals as well as the other typical orientation and dysmetric dyspraxic symptoms defining primary, specific, developmental, and dysmetric dyslexia.

They concluded, therefore, that both groups of individuals with reading disorders have a cortical impairment.

We found such reasoning fallacious, since these same investigators did not recognize that:

- Adults with lesions of the dominant angular gyrus and conceptual or agnostic reading impairment lose the meaning of letters and words. If these adults are still capable of recognizing that letters and words are written symbols and guess at their meaning, their guessing is completely randomized (e.g., *b* equals *cat; was* equals *cat; dog* equals *garden; d* equals *z*). If they have right-left disturbances, it is because they have lost the symbolic meaning and significance of right and left. As a result, their responses to right-left questions are randomized. Because of their agnostic and conceptual impairments, their *Wechsler Intelligence Scale for Children* (WISC) Verbal IQ drops below normal; and the prognosis for their regaining and fully compensating lost functions is guarded or poor.
- Children with primary, specific, developmental, and dysmetric dyslexia have no conceptual or agnostic reading impairment. Their letter and word reversals reflect an orientation and sequential scanning and processing impairment (e.g., *b* equals *d* equals *p* equals *g; was* equals *saw; dog*

equals *god).* Their guessing is not randomized, but spatially determined. They know the symbolic significance of letters and words, but are confused by the letters' and words' spatial orientation. These children invariably have difficulty recalling the direction of right and left. They know the symbolic significance of right and left as directions, but are not sure where these directions are in space. When questioned as to right and left, they guess. Once they assign a right-left direction to space, all their ensuing replies are consistent with this guessed or assigned direction. In view of the fact that there is a scanning and orientation rather than a conceptual or agnostic (or cortical) impairment, their WISC Verbal IQ is normal or superior; and their prognosis for reading compensation is fair to good—despite their having to develop a reading and orientation function they never had. Indeed, many of these children often "overcompensate" and become teachers, physicians, artists, mathematicians, etc.

In view of the fact that dominant cortical lesions in adults lead to aphasic, agnostic, and conceptual dyslexia, it logically followed that cortical dyslexia correlates with decreased reading score (quantitative dyslexia) almost 100 percent of the time. The same assumption was made without thorough clinical inspection, for the disease or diseases called primary, specific, developmental, or dysmetric dyslexia (i.e., that the disease called "dyslexia" in children correlates highly with decreased reading score or quantitative dyslexia). In our study this assumption has also been proven fallacious.

Our initial investigations of primary, specific, and developmental dyslexic children revealed an underlying cerebellar-vestibular dysfunction, positive electronystagmograms, and deficient ocular-motor fixation and scanning ability.

Dyslexia: Disease vs Symptom

In screening large populations of children utilizing our "blurring speed" instrument and methodology [described later in this article], we discovered that the incidence of cerebellar-vestibular dysfunctioned children with decreased scanning ability is approximately 8 to 10 percent, and that only half of these children had significantly reduced reading scores. In other words, the *disease* called dyslexia must be clearly distinguished from the *symptom* called dyslexia or decreased reading score.

In dysmetric dyslexia and dyspraxia, the disease is a cerebellar-vestibular dysfunction and a resulting qualitative scanning defect which contributes to, but does not determine, decreased reading score or quantitative dyslexia. Cerebellar-vestibular dysfunction results in a qualitative dyslexia—and not necessarily a quantitative dyslexia.

Reading score is an overdetermined functional complex and distributes itself over a bell-shaped curve in dysmetric dyslexia, with somewhat more than half falling below the normal reading level. This finding correlates with the clinical observation that primary and dysmetric dyslexic children have a relatively good prognosis insofar as their reading score is concerned—provided that they are not psychologically traumatized.

Stated another way, children with cerebellar-vestibular dysfunction and qualitative dyslexia are capable of significantly compensating and overcompensating their quantitative dyslexia. They are often, however, predisposed to neurotic and character disturbances; and their dysmetria often manifests itself in poor handwriting, poor spelling, disinterest in reading, etc. We have demonstrated that dysmetric dyslexia starts out as a neurophysiological dysfunction which is significantly compensated for in time, and becomes a psychological dysfunction which intensifies with time.

The clinical picture is altogether different for the agnostic, conceptual, or cortical dyslexias. In this group, the disease invariably results in decreased reading score, and the compensatory ability is significantly restricted.

Male/Female Incidence of Dyslexia

The male/female incidence of primary dyslexia is reported to be two to one, four to one, or higher; and some authorities assumed sex-linked genetic factors were responsible. Prior to our research on dysmetric dyslexia and dyspraxia, no study on the male/female incidence ratios was reported on the basis of referral-age and specific dyslexic etiology.

Our initial study indicated that for dysmetric dyslexia, the male/female ratio increased from two to one to four to one as the age of initial referral increased. This finding strongly suggested nongenetic factors—especially because we had also demonstrated that the blurring speeds or scanning ability and reading scores of dysmetric dyslexics improve with age and usually spurt between ages 10 and 12. We have also demonstrated that the

male/female blurring speed ratio equals 1 for both normal and dysmetric dyslexic groups.

These findings strongly suggest:

- That the incidence of cerebellar-vestibular dysfuntion is the same for males and females.
- That psychological rather than neurophysiological factors determine the male/female referral ratio of two to one to four to one in dysmetric dyslexia.
- That the male/female referral ratio is different than the male/female incidence of dysmetric dyslexia.

Dysmetric dyslexics are usually referred for evaluation on the basis of "decreased reading score." Cerebellar-vestibular dysfunction contributes to quantitative dyslexia, but does not determine it—as reading score is an overdetermined functional complex even in dysmetric dyslexia. It thus appears that the male/female cerebellar-vestibular dysfunction ratio equals 1, and that boys with cerebellar-vestibular impairment are more affected by psychological stress factors (e.g., the need to succeed) than girls.

As a result of this male "psychological vulnerability," males with cerebellar-vestibular dysfunction and qualitative dyslexia cannot compensate in their reading scores as well as females—and as a result are referred for evaluation on the basis of "decreased reading score" in the ratio of two to one to four to one. This male/female psychological stress gradient and differential can also explain the higher incidence of reading score failures in male/female nonorganic "dyslexic" populations as well.

In summary, a deeper and more dynamic understanding of the neurophysiological and psychological determinants of dysmetric dyslexia reveals:

- There is no real difference in the male/female incidence of qualitative dysmetric dyslexia and dyspraxia.
- That males with qualitative dysmetric dyslexia do not compensate as well as females with respect to their reading scores.
- As a result of this differential male/female stress factor, males with dysmetric dyslexia are referred for evaluation on the basis of reading score in the ratio of two to one to four to one.
- The compensated quantitative dyslexics still have qualitative dysmetric dyslexia; and until now have escaped diagnosis, study, and suitable treatment. Our statistics suggest the incidence of this compensated group to be approximately four to five percent.

Blurring-Speed Methodology

We had assumed the ocular fixation and sequential scanning dysfunction in dysmetric dyslexics to be analagous to the tracking difficulty one might have while attempting to read a signboard from a rapidly moving train. The "need to see" the signboard will trigger the release of an optokinetic tracking response so that the optical fixation point is maintained and clear vision preserved. As the train accelerates, a speed is reached at which the physiological tracking capacity is exceeded, the optical fixation point is lost, and the visual sequence is scrambled or blurred. The speed of the train at which blurring or scrambling occurs is a measurable endpoint representing the maximum cerebellar-vestibular tracking capacity.

Utilizing this analogy, we designed a projector system capable of beaming and recording the speed of independently moving foreground and background visual patterns. Three measurements were recorded:

> *Mode I:* A foreground consisting of black lettered words and phrases was speeded up against a blank neutral background until blurring was reported by the observer and the "blurring speed" for Mode I was recorded.
>
> *Mode II:* The same foreground was speeded up against a fixed scenic background, and once again the "blurring speed" was recorded.
>
> *Mode III:* The observer was instructed to fixate the stationary foreground consisting of words while the scenic background was set in motion. The presence or absence of foreground movement and/or blurring was recorded.

In addition, ocular-motor tracking patterns were obtained during Modes I, II, and III, and proved diagnostically helpful in assessing "blurring" neurophysiologically.

The blurring speeds for young dysmetric dyslexic children were significantly lower than for normal children of similar age. Dysmetric dyslexic children reported foreground movement and/or blurring in Mode III. Normal children did not. The blurring speeds of dysmetric dyslexic children increased with age and tended to become normal at approximately 10 years of age—indicating the presence of neurophysiological compensatory processes. The word-blurring speeds for young, normal five-year-old children started out at a high, narrow range increased slightly over the next 50 years, and seemed relatively independent of the group tested.

A repetitive sequence of heavily saturated black elephants resulted in a wide-normal range, seemed group-dependent, and were more difficult to track despite its simpler visual gestalt.

These and other experiments tended to indicate that the tracking response is an Inbuilt Release Mechanism (IRM) which is stimulus-dependent and that different visual gestalt patterns are tracked by correspondingly different cerebellar-vestibular tracking circuits.

The word-gestalt tracking circuits, on the other hand, seemed genetically facilitated and ready to function at an early age.

Sir John Eccles hypothesizes learning to take place in the cerebellum as follows:

> The immense computational machinery of the cerebellum with a neuronal population that may exceed that of the rest of the nervous system gives rise to the concept that the cerebellar cortex is not simply a fixed computing device, but that it contains in its structure the neuronal connexions developed in relationship to learned skills. We have to envisage that the cerebellum plays a major role in the performance of all skilled actions and hence that it can learn from experience so that its performance to any given input is conditioned by this "remembered experience". As yet, of course, we have no knowledge of the structural and functional changes that form the basis of this learned response. However, one can speculate that the spine synapses on the dendrites in the molecular layer are especially concerned in this and that usage gives growth of the spines and particularly the formation of the secondary spines that Hamori and Szentagothai (1964) described on Purkinje dendrites. One can, therefore, imagine that in the learning of movements and skills there is the microgrowth of such structures giving increased synaptic function and that as a consequence the cerebellum is able to compute in an especially adapted way for each particular learned movement and thus can provide appropriate corrective information that keeps the movement on target.[1]

If we speculate that the neurophysiological and anatomical cerebellar changes in response to "current learning" recapitulates the neurophysiological and anatomical changes of "evolutionary learning" or "evolutionary genetic imprinting" just as ontogeny recapitulates phylogeny, then we can better explain the intimate relationships between stimulus and motor response, as well as the

reason and way the cerebellar cortex tripled in size in order to "cope" with or track an ever increasingly complex environment (e.g., reading and symbolic gestalts).

One might further speculate that genetically or congenitally determined cerebellar-vestibular lags and dysmetric dyslexia may represent an ontogenetic indication of an intermediate or pre-reading state in phylogeny.

Our studies to date indicate that incidence of genetically or congenitally determined dysmetric dyslexia to be approximately one to two percent of the population.

Etiological Factors in Dysmetric Dyslexia

Genetic:

- We previously speculated that one form of genetic dysmetric dyslexia might represent an ontogenetic recapitulation of a phylogenetic prereading state when the cerebellar cortex was one third its present mass. In other words, delayed maturation of the cerebellar-vestibular circuits as reflected in decreased word-blurring speeds might be genetically determined in some families and ontogenetically reflect the slow phylogenetic evolution of man's cerebellar function over several million years.
- In addition, genetically determined immunological patterns may predispose some families to viral, toxic, or allergic dysfunctions of the labyrinthine-vestibular-cerebellar circuitry.

Ear Infections:

The high incidence and specific localization of the cerebellar-vestibular dysfunction underlying dysmetric dyslexia strongly suggests that either "harmless" ear infections and/or the antibiotics used for treatment may result in damage to the labyrinthine-vestibular-cerebellar circuitry.

If there is any significance to the impression that the incidence of "dyslexia" appears to be increasing in Western civilization, then one might hypothesize that the widespread use of antibiotics in modern societies, together with a resulting change in the viral and immunological structure, is responsible. Our continuing research will attempt to elucidate the validity of our assumptions.

Emotional and Cultural Stimulus Deprivation:

The recognition that the cerebellar-vestibular tracking and orienting circuits are stimulus-dependent Inbuilt Release Mechan-

isms, which unfold in relation to vital emotional-stimulus "dialogue-imprints" and triggers, led the authors to classify dysmetric dyslexia and dyspraxia into Primary and Secondary types:

> *Primary Dysmetric Dyslexia and Dyspraxia* results from a primary or basic dysfunction of the cerebellar-vestibular tracking and orienting circuits (e.g., genetic, infectious, toxic, traumatic, etc.).
>
> *Secondary Dysmetric Dyslexia and Dyspraxia* results when normally inbuilt cerebellar-vestibular tracking and orienting circuits fail to develop or unfold secondary to severe emotional and cultural stimulus deprivation (e.g., "perceptual-motor" deficits in emotionally deprived children).

As one might anticipate, each type of dysmetric dyslexia and dyspraxia has its unique defining characteristics which will be elucidated in future papers. In addition, Primary and Secondary types may coexist with each other as well as in various combinations of organic and nonorganic dysfunctions—resulting in a biological spread of symptoms requiring careful etiological dissection and treatment.

Cerebellar-vestibular perceptual-motor performance can now be clinically assessed and conceptualized as a function of:

- age and developmental stage;
- cerebellar-vestibular circuit integrity or dysfunction;
- emotional-stimulus dialogue adequacy or deficiency;
- combinations of the above in relationship to the wholistic functioning of the central nervous system and organism as a whole.

Treatment

On the basis of Sir John Eccles' work on cerebellar learning and functioning, and our own discoveries of the compensatory mechanisms in dysmetric dyslexia, we have developed stimulus-dependent ocular-motor facilitation "exercises" at specific blurring speeds so as to induce cerebellar-vestibular or ocular-motor tracking compensation.

We have also developed a new reading technique which requires minimum scanning and thus avoids scrambling.

In addition, we have proven the usefulness of the "sea sickness" group of medications in the prevention and treatment of dysmetric dyslexia and dyspraxia.

Summary

We have attempted to define dysmetric dyslexia and dyspraxia as a medical disorder with specifics: neurophysiology and psychology, method of diagnosis and prediction, etiology, incidence, prognosis, and treatment. Needless to say, any summary of this kind leaves much to the imagination. However, this paper is intended to serve merely as an introduction to the papers and books we intend to publish in the near future, and was undertaken as a response and accomodation to the numerous requests of individuals sincerely interested in helping dyslexic children.

NOTE

1. J. C. Eccles, M. Ito, and J. Szentagothai, *The Cerebellum as a Neuronal Machine* (New York: Springer-Verlag, 1967): 314.

REFERENCES

Ayres, A. J. *Sensory Integration and Learning Disorders.* Los Angeles: Western Psychological Services, 1972.

Barany, R. *Some New Methods for Functional Testing of the Vestibular Apparatus and Cerebellum* [Nobel Lectures, Physiology and Medicine 1901-1921]. Amsterdam: Elsevier, 1967.

Dow, R. S. and Moruzzi, G. *The Physiology and Pathology of the Cerebellum.* Minneapolis: University of Minnesota Press, 1958.

Eccles, J. C. *The Inhibitory Pathways of the Central Nervous System* [The Sherrington Lectures IX]. Springfield, Illinois: Charles C Thomas, 1969.

Frank, J. and Levinson, H. "Dysmetric Dyslexia and Dyspraxia—Hypothesis and Study." *The Journal of the American Academy of Child Psychiatry* 12 (1973):690-701.

Jongkees, L. and Philipszoon, A. *Electronystagmography.* Stockholm: Oto-Laryngologica Supplementum 189, 1964.

Schiller, C. H. *Instinctive Behavior—The Development of a Modern Concept.* New York; International Universities Press, Inc., 1957.

Spitz, R. *The First Year of Life.* New York: International Universities Press, Inc., 1966.