Compensatory Mechanisms in C-V Dysfunction, Dysmetric Dyslexia and Dyspraxia

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OUR STUDY of dysmetric dyslexic and dyspraxic individuals revealed the presence of "silently" active compensatory and overcompensatory neurophysiological and neuropsychological mechanisms which attempt to neutralize and even deny the underlying cerebellar-vestibular disturbances and symptomatology. Namely, the prognosis for dysmetric dyslexic and dyspraxic children was found to be favorable, provided psychological and educational traumatization and scarring did not occur; their cerebellar-vestibular symptomatology appeared to diminish with age; their "soft signs" became "softer" and more difficult to diagnose, and their reading, writing, spelling and drawing orientation, coordination and balance so improved by latency and puberty that the authors wondered if so-called "late bloomers" and "developmental dyslexics" were not in fact dysmetric dyslexic and dyspraxic.

Blurring Speed Compensation

Analysis of our "blurring speed" data clearly indicated that the dysmetric dyslexic and dyspraxic blurring speeds significantly increased as a function of age and that the average Mode I* word-blurring speed approached normal by 10 to 12 years of age—

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despite the continued presence of abnormal clinical and ENG** findings.[1] It thus became apparent that the dysmetric dyslexic blurring speeds [dd(BS)] were not only a function of decreased cerebellar-vestibular tracking or scanning capacity, but a function of compensatory scanning capacity as well.

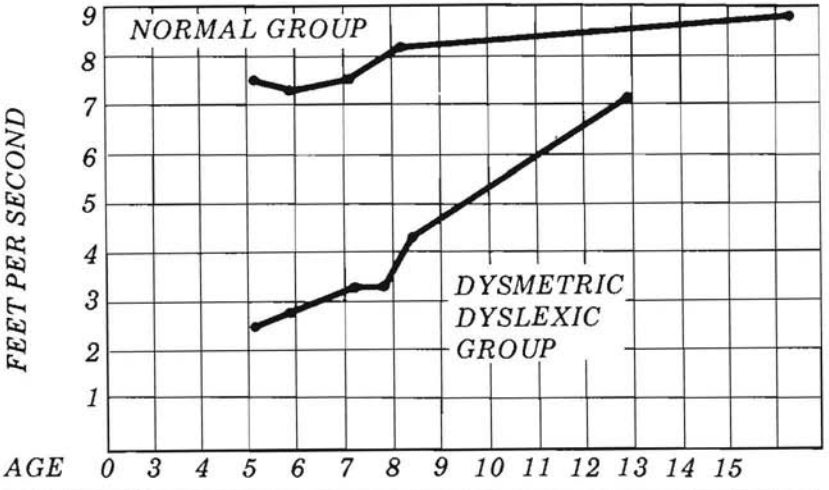
The blurring speed relationships can be simply expressed as follows:

$$BS = f(MITC) = f_1 + f_2 + f_3$$

where f = Maximum Induced Tracking Capacity; f_1 = inherent non-facilitated tracking capacity; f_2 = decreased tracking capacity or subclinical nystagmus; and f_3 = facilitated or compensatory tracking capacity. Figures 1, 2, and 3 indicate how compensatory scanning mechanisms (f_3) improve the dd(BS) as a f(age) and that the ddf_3 is significantly greater than (normal) nf_3.

Figure 1

Mode I Test; Clear Background



*Mode I: A foreground consisting of black lettered words and phrases is speeded up against a blank neutral background until blurring is reported by the observer and "blurring speed" is recorded. Mode II: The same foreground is speeded up against a fixed scenic background, and once again the "blurring speed" is recorded. Mode III: The foreground remains stationary while the scenic background is set in motion; and the presence or absence of foreground movement and/or blurring is recorded. The 3-D optical scanner is a projector system capable of beaming and recording the speed of independently moving foreground and background patterns. The task for the observer is analogous to attempting to read or fixate sign posts while riding on a train which is gradually accelerating.

**Electronystagmography (ENG) is an electrical technique for objectively detecting, recording and measuring nystagmus—a rapid involuntary oscillation of the eyeball.

Figure 2

Mode II; Scenic Background

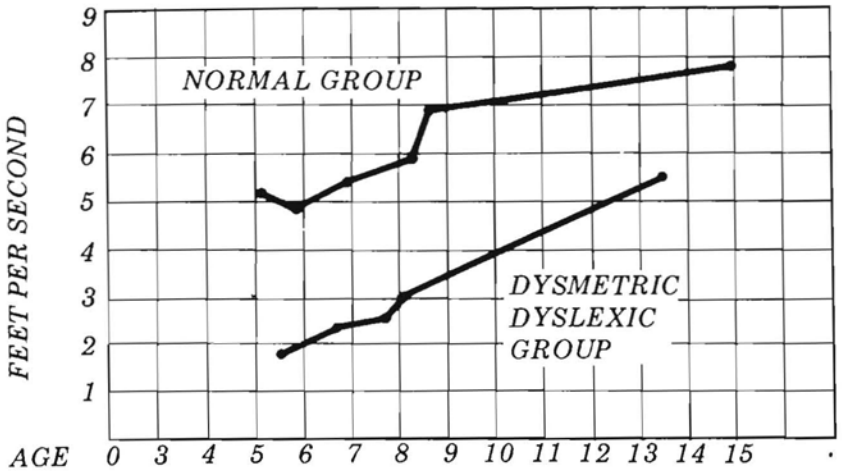
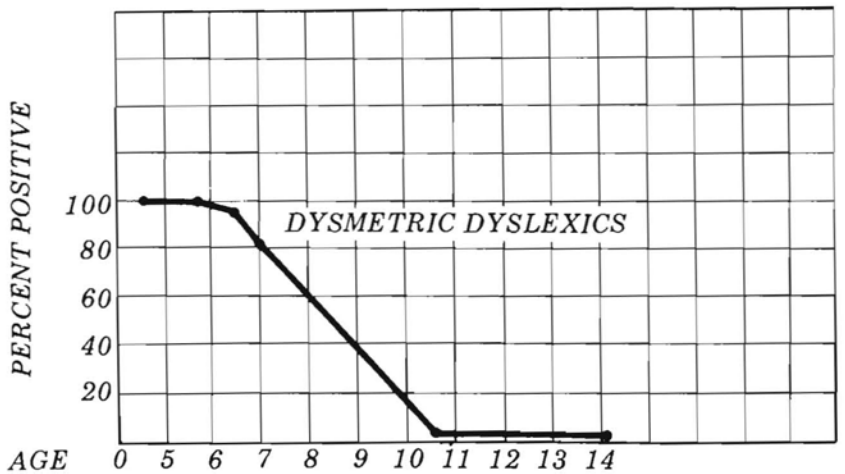


Figure 3

Mode III; Percent of Positive Responses vs. Age



It is certainly tempting to speculate that current adaptive biological need determines the ddf_3 and nf_3, and that these compensatory tracking mechanisms are "ontogenetic" recapitulations of a phylogenetic sequence of events in which man's pre-reading blurring speeds and cerebellar-vestibular circuits increased in relationship to continuously evolving and increasingly complex adaptive biological needs over an evolutionary time span.

We hypothesized that this increased compensatory scanning capacity (nf_3 and ddf_3) develops by means of a complex over-

determined resultant of "cerebellar learning" or facilitation and increasing cortical feedback control with continued myelinization and granulation of the cerebral cortex and its pathways.[2] Theoretically speaking, it is as if these developing cerebellar, cortical, and cortical-cerebellar feedback tracts and forces were better able to check, neutralize, and inhibit the subclinical nystagmus underlying dysmetric dyslexia and dyspraxia.

In view of the fact that the age range at which the average Mode I word-blurring speed approaches normal corresponds to the psychoanalytic latency period and the pedagogical "late blooming" period, one can anticipate that a clear understanding of the complex evolving neurophysiological and neuropsychological forces determinging blurring speed compensation would significantly contribute to an understanding of the interface between brain and mind.[3]

By virtue of the fact that blurring speeds were found to measure cerebellar-vestibular compensation, regression, and developmental lag, the use of this methodology took on broader and more dynamic dimensions. More specifically, by means of blurring speed measurements, we are able to develop maturation, compensatory, and regression curves as a function of time or age, medication, oculo-motor facilitation techniques, etc.

By means of our blurring speed measurements and expanding theoretical and clinical formulations, we have undertaken the study of dysmetric dyslexic and dyspraxic adult disorders and the factors leading to cerebellar-vestibular functional and blurring speed regression, factors such as multiple sclerosis, post-ECT states, concussion states, toxic states (alcohol and drugs), flu states, and the like. The investigation of the above and other newly discovered dysmetric dyslexic and dyspraxic disorders via ENG and blurring speeds has led to increased correlations between clinical cerebellar-vestibular findings, positive ENG results, and decreased blurring speeds. Furthermore, we have developed a broader conceptualization of dysmetric dyslexia and dyspraxia—the resulting symptoms depending upon age of onset, specific sites and type of lesion or dysfunction, and the directional vectors of the dynamic equilibrium existing between cerebellar-vestibular regressive and compensatory mechanisms and forces. It is anticipated that the continued study of adult dysmetric dyslexic and dyspraxic disorders will yield invaluable information as to the compensatory nature and function of the cerebellar-vestibular and related circuits.

The Concept of a "Subclinical Nystagmus"

Clinical and subclinical nystagmus of cerebellar-vestibular origin is a function of cerebellar-vestibular disorder and imbalance and serves no adapative tracking function. In acute and poorly compensated cases, the cerebellar-vestibular dysfunction and resulting nystagmus leads to an oscillating and unstable optical fixation (i.e., "blurred" vision, oscillopsia, etc.). With time and "compensation," this clinical nystagmus diminishes or disappears.

In dysmetric dyslexia, we demonstrated a cerebellar-vestibular dysfunction and decreased tracking capacity, and thus assumed the existence of a "subclinical nystagmus."

It is well known that nystagmus of vestibular origin is "suppressed" upon ocular fixation and "released from suppression" when the eyes are closed and no longer tracking. Why is this nystagmus "inhibited" during fixation and tracking on the one hand, and "released from inhibition" when not tracking or with eyes closed? *So that clear vision, tracking, and orientation can be maintained!*

We reason as follows: If vestibular nystagmus is suppressed during ocular fixation so that clear vision and tracking can be maintained, then closing one's eyes removes this adaptive tracking need as well as the need for "nystagmus suppression"; and, as a result of decreased inhibition, the vestibular nystagmus is "released" from suppression or inhibitory control.

As a result of active inhibition, clinical nystagmus becomes subclinical, and with decreased inhibition subclinical nystagmus becomes clinical. We have demonstrated a dynamic equilibrium existing between dsyfunctioning and compensatory mechanisms—and the clinical or subclinical resultant depending on quantitative vectors.

Of 72 dysmetric dyslexic children referred for "blind" ENG evaluations, 85 percent evidenced some vestibular abnormality; and only 20 percent were found to have a subclinical nystagmus (i.e., positional and/or spontaneous nystagmus).

Of 250 consecutive "dyslexic" children, 97 percent were found to have a cerebellar-vestibular dysfunction; and the incidence of clinical nystagmus was zero.

Thus, by means of the increased recording and measuring sensitivity of ENG as well as the methods used to diminish compensatory mechanisms, the incidence of detectable nystagmus of cerebellar-vestibular origin was raised from zero to 20 percent. One can either assume that the remaining 80 percent of dys-

metric dyslexic children have no subclinical nystagmus, or that all dysmetric dyslexic children have a subclinical nystagmus and that present-day ENG methodology can detect subclinical nystagmus directly in only 20 percent of the cases tested.

We have assumed the existence of a subclinical nystagmus in all cases with cerebellar-vestibular dysfunction and reasoned that this maladaptive cerebellar-vestibular nystagmus is actively inhibited by compensatory forces so as to allow dysmetric dyslexics to "see and track clearly" by maintaining adequate optical fixation and tracking capacity.

In normal individuals the "need to see and track" a moving visual sequence clearly triggers an adaptive optokinetic ocular tracking (nystagmus) response so as to keep the visual target in focus and thus preserve clear vision and tracking.

In the presence of a cerebellar-vestibular dysfunction and subclinical nystagmus, the inherent adaptive stimulus-tracking capacity is reduced and blurring of a moving stimulus sequence will occur at decreased blurring speeds.

In the absence of cerebellar-vestibular dysfunction and subclinical nystagmus, stimulus-blurring will occur at maximum blurring speeds because the adaptive tracking capacity is unimpaired.

As we said earlier, the blurring speed can be formulated as:

$$BS = f(MITC) = f_1 + f_2 + f_3$$

where f = Maximum Induced Tracking Capacity; f_1 = inherent nonfacilitated tracking capacity; f_2 = decreased tracking capacity or subclinical nystagmus; and f_3 = facilitated or compensatory tracking capacity. For all individuals, f_1 and f_3 reflect adaptive stimulus-dependent tracking capacities and are positive tracking forces; $f_2 = 0$ for normal individuals; <0 for dysmetric dyslexic individuals, is not stimulus-dependent, and thus has no adaptive tracking function and is a measure of cerebellar-vestibular dysfunction and decreased tracking capacity.

New blurring speed techniques which minimize f_3 in dysmetric dyslexic individuals will enable more exact determinations of f_2 and thus significantly enhance the diagnostic-screening value of the blurring speed methodology:

- If f_3 is minimized by special techniques, then $BS = f_1 + f_2$.
- For normal individuals $f_2 = 0$ and $n(BS) = f_1$.
- For dysmetric dyslexic individuals, $f_2 < 0$ and $dd(BS) = f_1 + f_2$.

If the nonfacilitated or noncompensated dd(BS) is sub-

tracted from the age and group appropriate n(BS) equivalent, a measure of f_2 is obtained:

$$n(BS) - dd(BS) = f_2$$

In addition, by obtaining blurring speeds with and without f_3, f_3 can be ascertained as well.

Our blurring speed methodology "triggers" or forces ocular tracking to its maximum neurophysiological capabilities, and the endpoint of this limit is the stimulus speed at which blurring occurs.

If blurring speed is now viewed as a function of the Maximum Induced Tracking Capacity (MITC), then:

$$BS = f(MITC) = f_1 + f_2 + f_3$$

If by special techniques f_3 is significantly reduced and approaches 0, then the above equation is further simplified and can be represented as follows:

$$BS = f(MITC) = f_1 + f_2$$

As stated earlier, by definition $f_2 = 0$ for normal individuals with intact cerebellar-vestibular function and thus:

$$n(BS) = f_1$$

If f_1 represents the normal nonfacilitated word-blurring speed value, it can be readily obtained from the average blurring speed values of young normal matched controls and thus represents a predetermined constant in this equation:

$$dd(BS) = (f_1) + f_2$$

If f_1 = constant, then any significant reduction in the normal blurring speed [n(BS) or f_1] is dependent on f_2 or the subclinical nystagmus:

$$dd(BS) = (f_1) + f_2$$

We have already demonstrated that nystagmus of cerebellar-vestibular origin is non-stimulus bound and nonadaptive for tracking purposes.

Our word-blurring speed data has clearly demonstrated a significant reduction in the average word-tracking capacity of dysmetric dyslexic children—even in the presence of ddf_3 or compensatory tracking values.

Our aforesaid formulations show that any significant reduction in the blurring speed value to be a direct function of the subclinical nystagmus, and the data statistically proves the existence of a subclinical nystagmus in dysmetric dyslexia and dyspraxia.

In summary, the subclinical nystagmus is a function of decreased cerebellar-vestibular tracking capacity, and has a negative (adaptive) tracking speed value in cerebellar-vestibular dysfunction; and our data has proven its presence in dysmetric dyslexia and dyspraxia. The directional component of the subclinical nystagmus when detected and recorded by means of an ENG may indicate the vector sum or direction or the cerebellar-vestibular imbalance—but this nystagmus is not visually oriented or stimulus-bound, and has no adaptive tracking significance (i.e., the blurring speeds in dysmetric dyslexia are equally decreased in opposite directions and appear independent of the direction of the subclinical nystagmus).

Oculo-Motor Tracking Compensation

The oculo-motor tracking patterns of normal and dysmetric dyslexic individuals of varying ages were recorded by means of electronystagmography during routine Modes I, II, and III blurring speed testing and analyzed as a function of age and compensation. See the Appendix of this article for case studies.

Modes I and II: Normal individuals have a pathognomic sudden and dramatic reduction or inhibition of their induced tracking rates at the blurring speed (Cases 1 and 2). Dysmetric dyslexic children at times show a similar reduction of their tracking rate at blurring as do normal individuals, and at other times they continue to track a visual sequence moving at two or more times their blurring speed. This "phantom scanning," as we initially labeled it, was puzzling indeed. The rate of this "phantom scanning" remained relatively constant even though the moving visual sequence was speeded up several fold and the "phantom scanning" immediately ceased when either the stimulus sequence came to rest or when the dysmetric dyslexic and dyspraxic individuals closed their eyes—thus disproving the existence of an "after nystagmus" (Cases 3 and 4). In addition, with increasing age and compensation, the dysmetric dyslexic and dyspraxic tracking curves became more eumetric, their tracking amplitudes increased and approached normal, and "phantom scanning" was minimized or eliminated (Case 5).

Mode III: Normal individuals do not experience foreground movement or blurring in Mode III, and their tracking recordings indicate a steady fixation pattern without any induced background nystagmus (Cases 1 and 2). Young dysmetric dyslexic individuals experience foreground movement and/or blurring, and their tracking recordings often

reveal a corresponding background-induced nystagmus. Foreground movement and/or blurring may occur without the ENG detection of an induced nystagmus and vice versa (Cases 3 and 4). With increasing age and compensation, foreground blurring first disappears together with the induced nystagmus, and then foreground movement follows suit (Case 5). The tracking and compensatory mechanisms were not only age dependent but were found to be stimulus-specific as well; and at times Mode III compensation in one direction would preceed Mode III compensation in both directions (Case 3).

Hypothetical Formulations

In order to explain the observed complex spread of facinating and intriguing blurring speed and tracking phenomena, we evolved the following hypotheses:

1. Visual tracking is modulated by separate but interacting macula-foreground and rod-peripheral circuits, and the cerebellum plays a vital role in their functional coordination and integration.

2. As a result of cerebellar-vestibular dysfunction, the tracking movements are dysmetric; and with cerebellar and cortical learning or facilitation, these tracking movements approach eumetria.

3. Dysmetric dyslexic and dyspraxic individuals with decreased blurring speeds, decreased tracking amplitudes, and functional narrowing of their visual fields adaptively decrease and sacrifice part of their visual tracking field and amplitude in order to maintain an optical fixation point and clear macula vision.

4. We have demonstrated the presence of a background-induced nystagmus in dysmetric dyslexic and dyspraxic individuals during Mode III testing, and have assumed this rod-background induced nystagmus to result from a failure in cerebellar background inhibition. We reasoned that, if the failure of cerebellar background inhibition result in foreground instability, then the silently active presence of cerebellar background inhibition must be crucial and essential to normal foreground perception.

5. We have demonstrated a clear-cut and pathognomonic reduction or inhibition of the tracking rate at blurring in normal individuals (Cases 1 and 2) and "phantom scanning"

in dysmetric dyslexic and dyspraxic individuals (Cases 4 and 5). We have assumed that in normal and fully-compensated dysmetric dyslexic and dyspraxic individuals, inhibition of the tracking rate occurs at the blurring speed and represents an adaptive attempt to retarget at a physiological tracking rate. "Phantom scanning," on the other hand results from:

- A neurophysiological cerebellar failure to properly inhibit the rate of the tracking nystagmus after it exceeded its adaptive tracking limit; and, thus, "phantom scanning" may be conceptualized as a special form of cerebellar past-pointing.
- A neuropsychological attempt to deny an organic defect; and, thus, it may be viewed as similar to "phantom seeing" in the blind and "phantom hearing" in the deaf.
- A neuropsycholgical and neurophysiological attempt to see during or despite blurring in a manner similar to the development of the "searching nystagmus" of the blind, and the "searching nystagmus" in miners and albinos. We have come to recognize that the cerebellum plays a vital role in maintaining orientation and clear vision, and is thus capable of initiating and modulating both adaptive and maladaptive attempts to preserve and regain these lost or impaired orienting and tracking functions.

6. The observation that some dysmetric dyslexic and dyspraxic children learn to read well despite significantly decreased blurring speeds suggests the adaptive development of a "functional enlargement" of the "macula optical fixation zone" as a result of "retinal-occipital" and cerebellar-vestibular facilitation.

It is therapeutically imperative to recognize the fact that the organism's ability to compensate and reverse dysfunction is often significantly dependent on age and critical developmental stage. If therapeutic intervention is delayed and attempted too late to trigger compensatory development, compensation may either be impossible or severely restricted. For example, if amblyopia secondary to strabismus is not diagnosed and treated early in children, the visual suppression of vision in one eye cannot be reversed or compensated, and remains irreversible. Similar crucial time factors must also play a role in the compensatory ability of dysmetric dyslexic and dyspraxic children.

Fallacious Assumptions in "Primary" Dyslexic Research

A reader might justifiably ask at this point: How can a "dyslexic" individual have normal or above-normal reading scores? Is there not then some contradiction in the use of the term *dyslexia*? We in turn, ask the reader several questions before replying. How is it possible for a polio victim to become a track star or athlete? How is it possible for an infectious disorder to be accompanied by below-normal temperatures? How is it possible for a diabetic to have below-normal blood sugars or hypoglycemia? Why should we expect any one nonspecific, variable, and overdetermined symptom of a disease to be constantly present in a fixed quantity 100 percent of the time? How is it possible for dyslexia researchers to have ignored the vast number of "compensated" and successful dyslexic individuals from their dyslexic studies and definitions?

One is obviously correct in being puzzled and asking questions. In fact, we have done the same. There are numerous contradictions in the conceptualization and use of the term *primary dyslexia*. Dyslexia has been unwittingly and interchangeably used to represent both a disorder of unknown origin (primary dyslexia) and one of its variable and nonspecific symptoms (i.e., decreased reading score). For example, when referring to a "severely dyslexic" child, one usually means a "dyslexic" child with a severe reading score symptom—and the underlying assumption or implication is that the primary dyslexic disorder is also severe. However, this assumption may also be fallacious. A child with a "mild" dyslexic disorder might be severely traumatized emotionally and/or educationally, and as a result may develop severe reading score difficulties as a direct result of the traumatization. We certainly have medical parallels: Is "severe" temperature elevation a measure of the severity of the underlying infectious disorder? Obviously not. "Mild" virus infections often are accompanied by "high" temperatures and "severe" or even terminal viral and or bacterial infections may be accompanied by "below" normal temperatures.

Temperature is a variable, fluctuating, nonspecific, and overdetermined symptom which is often indicative but never pathognomonic of infection; it can result entirely from noninfectious metabolic, toxic, central-nervous-system and environmental disturbances; and the latter disturbance may also occur together with infection—the resulting temperature disturbance representing a vector-resultant or summation of all contributing factors. It is the presence or quality of the temperature symptom which is diagnostically significant and not its quantity. (Namely, any

abnormality of temperature, high or low, is indicative of disturbance; the absence of temperature does not imply the absence of infection and, as stated earlier, the degree or quantity of temperature is no indication of the severity or etiology of its underlying disorder).

We have demonstrated the reasoning sketched above to be valid for reading score abnormalities in primary and/or dysmetric dyslexia and dyspraxia, as well as for any and all of the signs and symptoms found in dyslexia (i.e., spelling, writing, arithmatic, orientation, sequencing, and coordination disturbances). In retrospect, one can only wonder how and why this basic scientific reasoning had been so successfully avoided and denied in primary dyslexic research.

Selective Sampling

Primary, specific, and developmental dyslexia are usually or often conceptualized and defined by the degree of reading score impairment (i.e., that a child would be two or more years behind his or her peers under equal circumstances, etc.). (How does one *really* measure equal circumstances?)

We have demonstrated as fallacious the assumption that a disorder of unknown origin (primary dyslexia) must invariably by definition and conceptualization be accompanied by a symptom (decreased reading score) which can only manifest in "severe" form.

This assumption is not only fallacious but contrary to all that is known in clinical medicine and biology. Is it really true that all (primary) dyslexics have severe reading score difficulties? Or is it that only those dyslexics with severe reading score difficulties are recognized, referred, and thus clinically examined?

Our blurring speed data has clearly indicated that cerebellar-vestibular impaired children have (qualitative) ocular-fixation and scanning difficulties which may or may not lead to significant reading score difficulties—depending upon the degree of the disorder's severity, the emotional and educational climate, IQ, and the child's neurophysiological and neuropsychological compensatory abilities. Many of our young dysmetric dyslexic and dyspraxic children have responded favorably to maturation and or remedial and medical intervention, and have become academically successful and above-average readers. We have numerous dysmetric dyslexic and dyspraxic adults with cerebellar-vestibular signs and symptoms who have become academically successful—often on their own—and are now prominent leaders and teachers in the learning disabilities movement. Their reading scores are often above average—but they frequently have residual spelling,

writing, spatial, and coordination disturbances in varying degrees.

What should the diagnosis be for these individuals who have compensated and overcompensated for their reading score symptoms? If indeed "dyslexic" children must have severe reading score difficulties to be "truly" dyslexic, then the further assumption is unwittingly made, namely, that their reading score prognosis is hopeless. If this unwitting assumption is true, how can we explain the numerous people who have improved and even become famous on their own; and why are we attempting to screen, diagnose, remediate, and treat a group of "dyslexic" children who will not benefit in so far as their reading scores go?

What is the diagnosis of a primary dyslexic child who is two or more years behind his peers in reading score after his reading scores become normal or above normal; and what is the "diagnosis" of this same child who is waiting for his or her reading scores to become sufficiently below average and thus qualify as a "true" dyslexic?

Male/Female Dyslexic Incidence

In addition, we also strongly suspect that the reported male/female primary dyslexic incidence ratios of four to one, five to one, and even ten to one do not represent "true incidence" ratios but merely highly selected male/female samples of primary dyslexics with *severe reading score difficulties.* These reported ratios do not sample primary dyslexic males and females with mild, compensated, and even overcompensated reading score difficulties.

Inasmuch as primary dyslexic male/female children are almost exclusively referred (and defined) on the basis of *severe reading score difficulties*, it stands to reason that the resulting "apparent incidence" statistics will merely tell us the "referred incidence" or number of dyslexic male/female children with severe reading score difficulties and not the total or "true incidence" of primary dyslexia.

We have demonstrated in a previous paper that the dysmetric dyslexic and dyspraxic male/female referral ratio is a function of initial referral age, and that the male/female ratio increases from two to one for first graders to four to one for second through sixth graders.[4] We have assumed that the "true incidence of dysmetric dyslexia and dyspraxia is relatively constant between kindergarten and sixth grade, and that the "apparent" or "referral incidence" is due entirely to selective sampling and thus may indicate that dyslexic females compensate for their reading scores better than males.

A Theoretical Analysis of a "Scientific Neurosis"

One might justifiably ask at this time: If the authors are correct in demonstrating that many basic and unchallenged assumptions in primary dyslexic research are fallacious, contradictory, and unscientific, then how can this "scientific neurosis" or dilemma be scientifically explained?

We propose the following theoretical analysis:

1. A lesion affecting the angular gyrus of the dominant parietal hemisphere has been proven to be responsible for a severe reading disorder in adults in which the ability to interpret the meaning and significance of written symbols is lost (i.e., agnostic or cortical dyslexia). Inasmuch as the meaning or gnosis of written symbols is lost, cortical dyslexia invariably must result in severely decreased reading scores. One either understands the meaning of written symbols or one doesn't; and, as a result, the reading score symptom is present in severe form. The severity of this cortical disorder persists as the compensatory range is often minimal and the prognosis poor. Angular gyrus or agnostic dyslexia is an all-or-none "dyslexia" in the vast majority of cases; and, as a result, one runs into little "practical" or "scientific" difficulty by calling both the disorder and its leading symptom, severe reading score difficulty, by one and the same name.

2. The neurophysiological impairment in primary or idiopathic dyslexia was fallaciously assumed to be of cortical origin as well—and the reasoning and conclusions valid for cortical dyslexia were fallaciously applied with equal conviction to primary dyslexia.

3. A displacement of conviction was transferred unwittingly from the proven field of cortical or agnostic dyslexia in adults to the field of idiopathic or primary dyslexia in children.

4. Any challenge to the displaced cortical assumptions fallaciously applied to primary dyslexia was defended as if the challenge was made against the assumptions proven to be valid in cortical dyslexia.

5. A "scientific neurosis" or prejudice arose and blindly and vigorously attempted to resist any attempt at its own clarification and resolution.

In conclusion, assumptions became convictions, convictions developed into neurosis, and the neurosis resisted any attempt at its own resolution and dissolution.

As a result of this scientific neurosis, the crucial role of compensatory factors in primary dyslexia was ignored or denied, as were the compensated cases themselves. Compensation in the cortical or agnostic dyslexias is the exception to the rule. Compensation in primary or dysmetric dyslexia is the rule. If the *idiot-savant* is the exception to the cortical compensatory rule, then the "*non-idiot-savant*" is the compensatory rule for primary or dysmetric dyslexia. The neurotic defenses of denial and confabulation were of sufficient force to insure the survival of the fallacious assumptions in primary dyslexia.

Summary

An attempt has been made to summarize and simplify the complex, wholistic, and dynamic interaction of overlapping neurophysiological and neuropsychological compensatory processes in dysmetric dyslexia and dyspraxia. The elucidation of both the dysfunctioning and compensatory mechanisms underlying dysmetric dyslexia and dyspraxia led us to design neurophysiologically and neuropsychologically specific pedagogical, psychological, oculo-motor, and pharmacological procedures in an effort to treat and eventually prevent the disorder and its symptomatic expressions. The therapeutic procedures and results will be presented in follow-up papers.

APPENDIX

Joy L.—Case 1

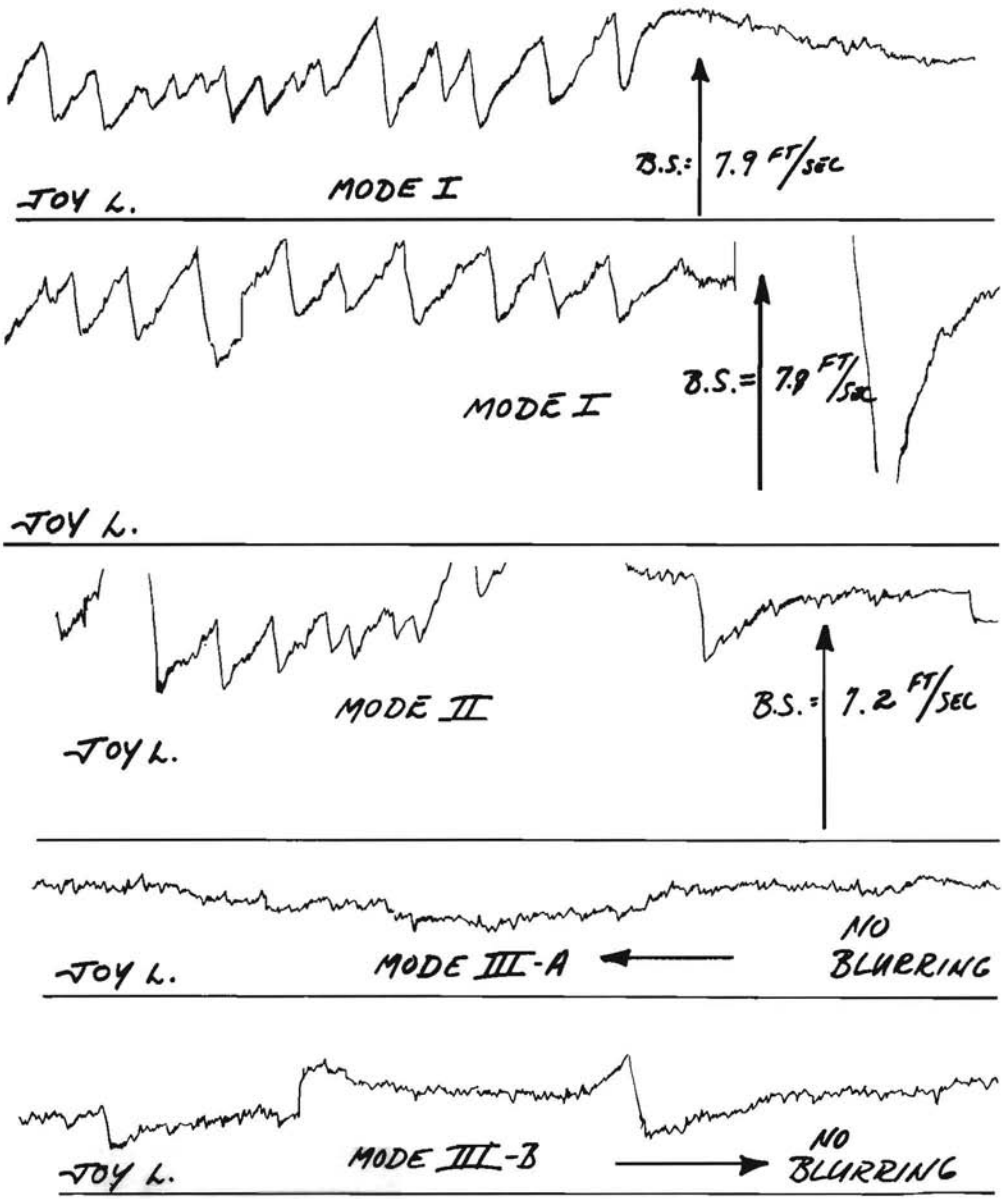
Joy L. is a normal five-year-old girl.

Mode I: Words were projected against a fixed blank background; and at the blurring threshold, the rate of the tracking nystagmus is reflexly reduced. (This is often accompanied by blinking as noted in the bottom vertical channel.)

Mode II: Words were projected against a fixed scenic background; and, once again, the rate of the tracking nystagmus is dramatically reduced at the blurring threshold and beyond. Blurring neurophysiologically took place shortly before it was reported.

Mode III: The child was instructed to fixate a stationary foreground, and a scenic background was set in motion. There was no induced nystagmus and the foreground remained clear. In III-A the scenic background moves from right to left, and in III-B the scenic background moves from left to right.

Figure 4
ENG Results for Case 1



Laura L.—Case 2

Laura L. is a normal seven-year-old child.

Mode I: Words were projected against a blank background. The arrow indicates that, when blurring takes place at 9 feet per second, there is a reflex reduction of the rate of nystagmus as an attempt to shut off and refocus the blurred visual input.

Mode II: Words were again projected against a fixed scenic background. The arrow indicates when blurring was reported. Note the reflex reduction of the rate of the induced optokinetic or tracking nystagmus at or shortly before the blurring speed.

Mode III: During foreground fixation and the background moving there was no induced nystagmus. In III-A the scenic background moves from right to left, and in III-B the scenic background moves from left to right.

Kathy K.—Case 3

Kathy K. is a six and a half year-old dysmetric dyslexic child.

Mode I: Moving words projected against a fixed blank background caused blurring at 3.4 feet per second with no significant decrease in the rate of the tracking nystagmus. The amplitude of the tracking nystagmus is less than half of the normal controls—indicating that the visual field is significantly restricted during tracking—so as to maintain fixation without blurring. In I-A the words move from right to left, and in I-B the words move from left to right.

Mode II: Moving words projected against a fixed scenic background caused blurring at 2.2 feet per second with initial reduction of the rate of the tracking nystagums. Once again, the amplitude of the tracking nystagmus is significantly reduced as compared to normal controls.

Mode III: Foreground blurring occurs when a stationary foreground is fixated and the scenic background is slowly set in motion. The induced nystagmus produces the typical zig-zag nystagmoid pattern so often noted. The moving background "catches the eye" and "pulls" it off the foreground fixation target. The patient tries again to fixate the stationary foreground but overshoots or undershoots it until another moving background object again "catches the eye" and pulls it off the fixation point. This labile fixation is a result of the patient's inability to suppress the background induced nystagmus—and blurring results. In III-A the scenic background moves from right to left, and in III-B the scenic background moves from left to right.

Figure 5
ENG Results for Case 2

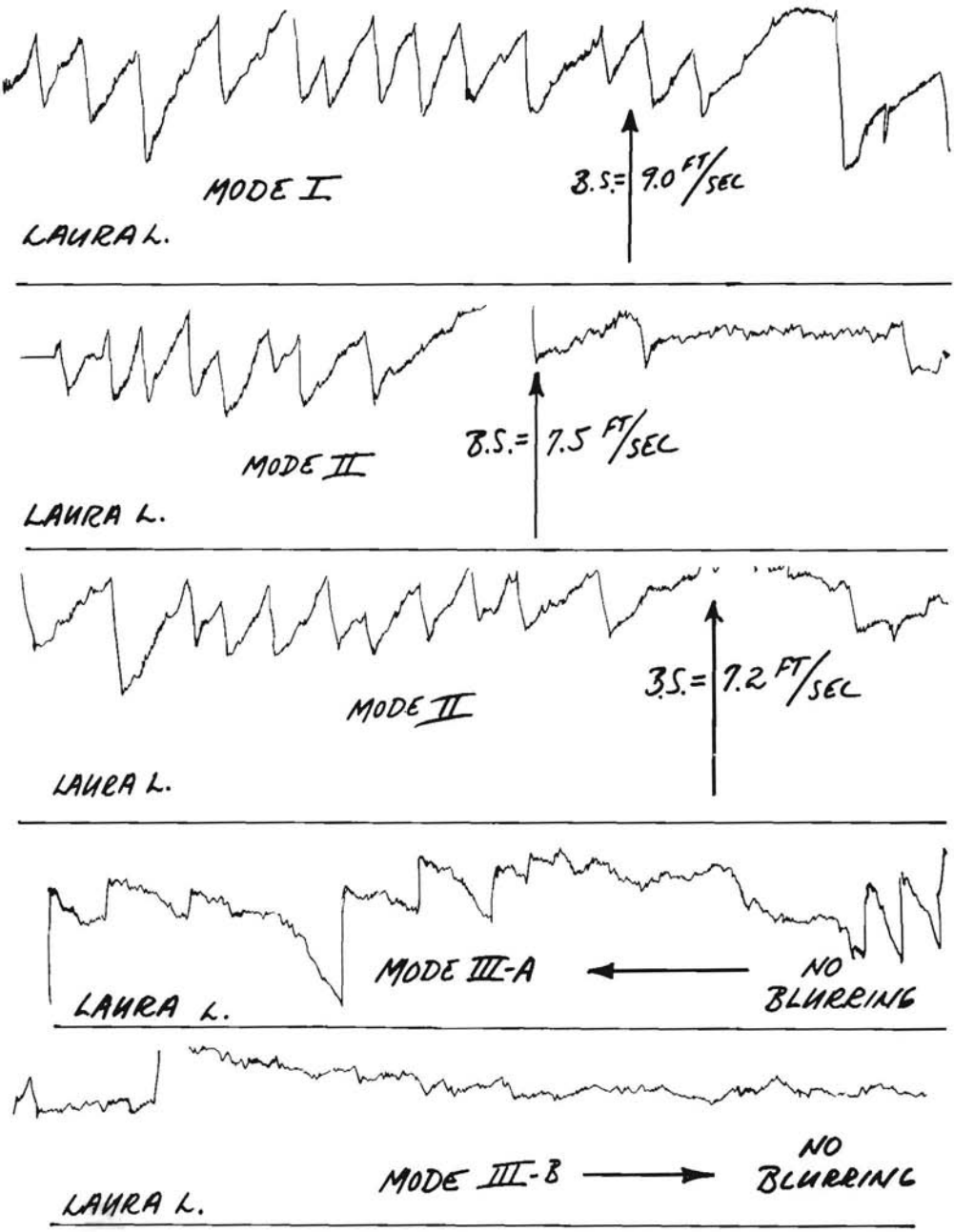
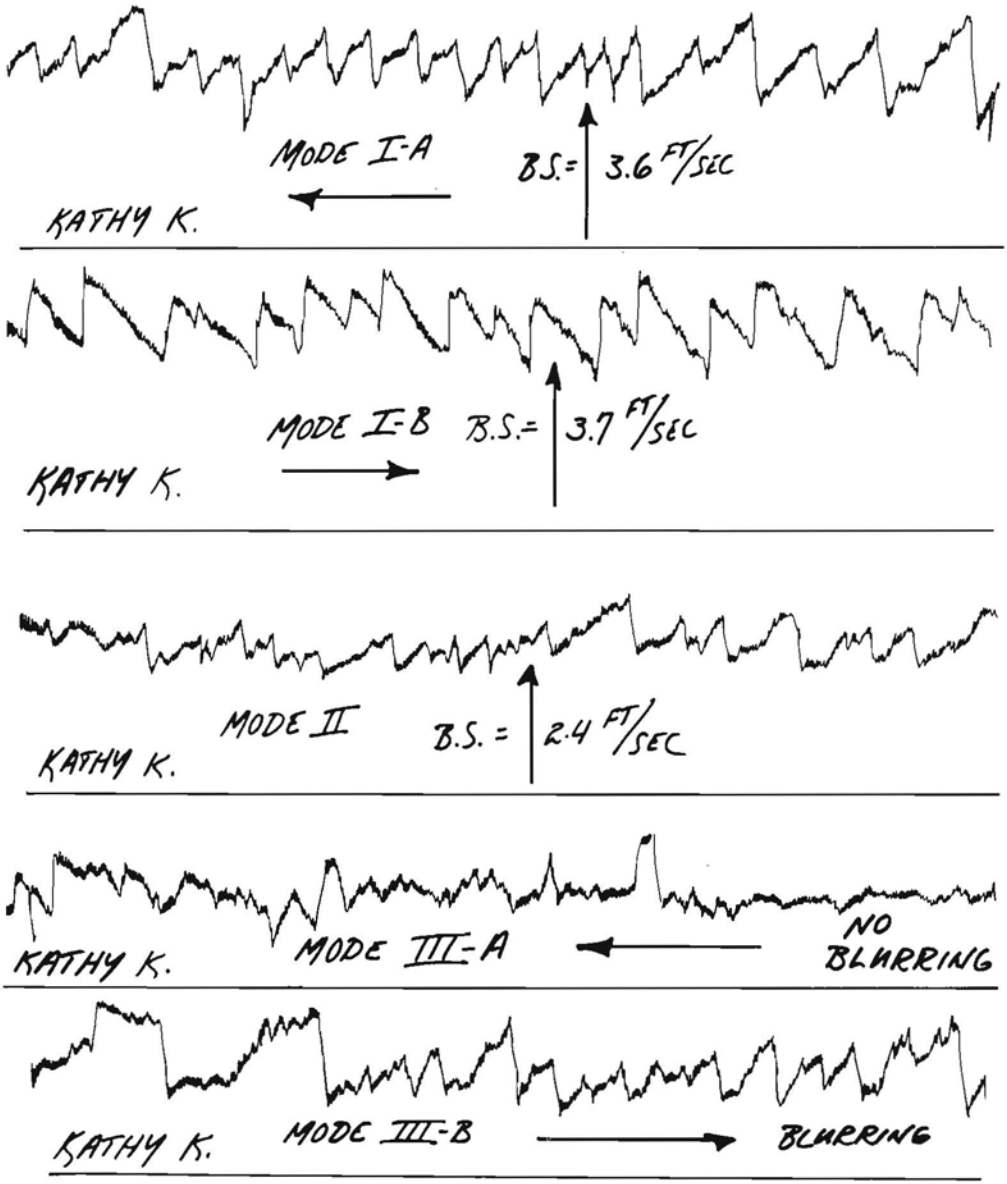


Figure 6
ENG Results for Case 3



Thomas L.—Case 4

Thomas L. is a nine and a half year-old dysmetric dyslexic boy.

Mode I: The moving words projected against a fixed scenic background resulted in blurring at 3.6 feet per second with no reduction of the rate of the tracking nystagmus after the blurring speed was exceeded ("phantom scanning"). The induced tracking nystagmus was dysmetric and of decreased amplitude, indicating a significant restriction of the tracking field.

Mode II: Moving words projected against a fixed scenic background resulted in blurring at 2.5 feet per second with no significant reduction of the rate of the tracking nystagmus after the blurring speed was exceeded.

Mode III: Foreground blurring occurred when the scenic background moved from right to left III-A and left to right III-B. The background-induced nystagmus produces the typical zig-zag nystagmoid pattern so often noted.

Douglas S.—Case 5

Douglas S. is a twelve-year-old blurring-speed compensated dysmetric dyslexic child.

Mode I: Words were projected against a fixed blank background and blurring was reported at 9 feet per second. The tracking amplitude is reduced but one can see a clear-cut reduction in the rate of the tracking nystagmus at the blurring threshold. The above was repeated. Here one can better see the dysmetric ocular pursuit in contrast to the clear-cut pursuit movements of even the younger controls. The reflex reduction of the rate of the tracking nystagmus at the blurring threshold of 9 feet per second is not appropriate.

Mode II: Moving words against a fixed scenic background caused blurring at 8 feet per second. The tracking amplitude is reduced.

Mode III: The patient fixated a stationary foreground, and the background was set in motion. Foreground blurring did not occur. The background nystagmus was suppressed, and as a result one did not get the zig-zag curve, and blurring did not occur.

NOTES

1. J. Frank and H. Levinson, "Dysmetric Dyslexia and Dyspraxia—Synopsis of a Continuing Research Project," *Academic Therapy* 11:2 (Winter 1975-76): 133-143; J. Frank and H. Levinson, "A New Method of Diagnosing Cerebellar-Vestibular Dysfunction and Predicting Dysmetric Dyslexia in Young Children" (submitted for publication, 1976).

Figure 7
ENG Results for Case 4

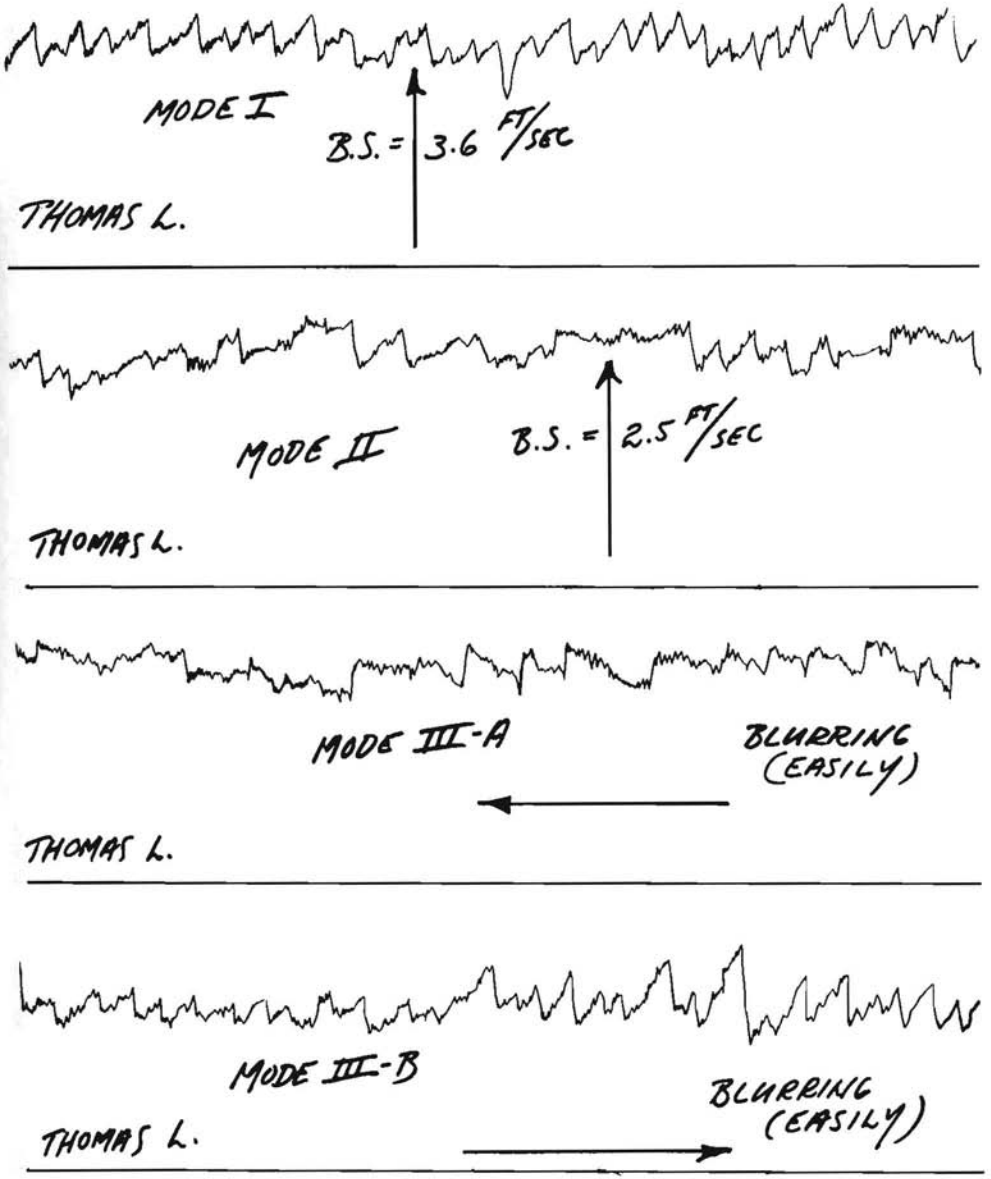
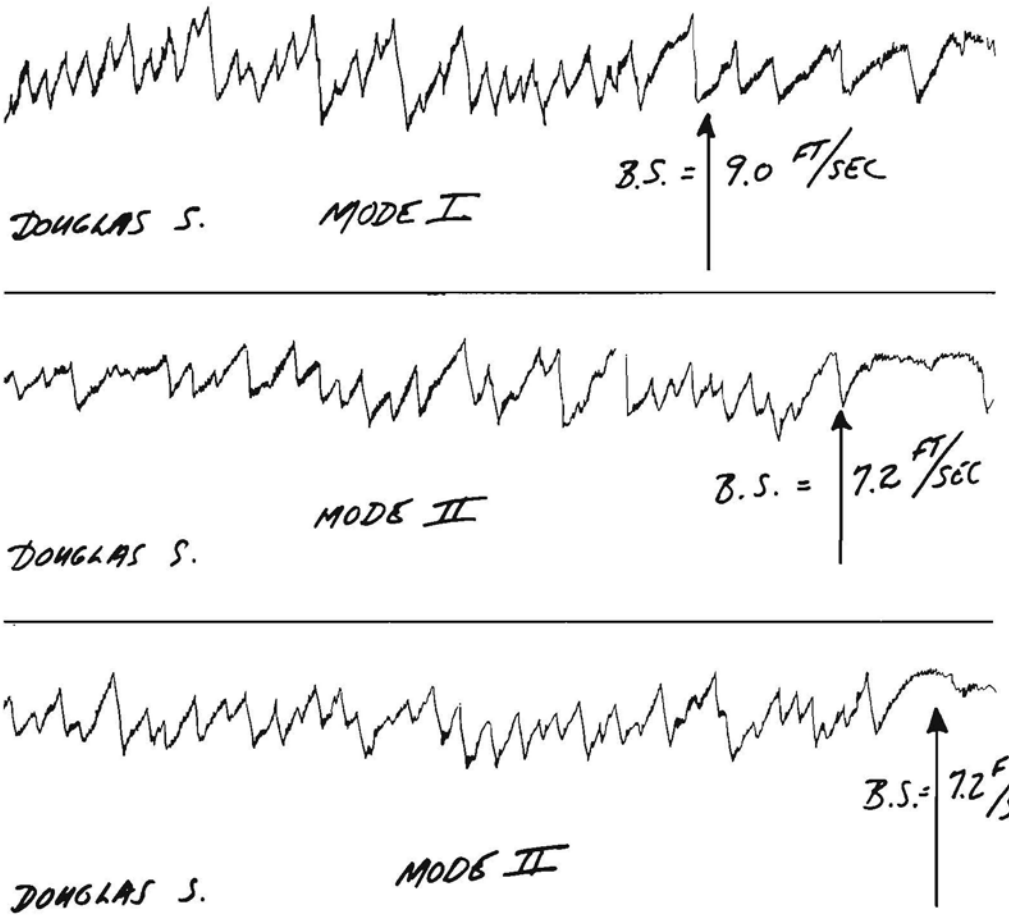


Figure 8
ENG Results for Case 5



2. J. C. Eccles, M. Ito, and J. Szentagothai, *The Cerebellum as a Neuronal Machine* (New York: Springer-Verlag, 1967): 314.
3. J. Frank and H. Levinson, *Dysmetric Dyslexia and Dyspraxia* (New York: W. W. Norton Publish Company, in press).
4. Frank and Levinson, "Dysmetric Dyslexia and Dyspraxia—Synopsis . . . ," *loc. cit.*

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