Seasickness Mechanisms and Medications in Dysmetric Dyslexia and Dyspraxia

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Reprinted from
ACADEMIC THERAPY PUBLICATIONS

VOLUME XII NUMBER 2, WINTER 1976-77

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IN A SERIES OF PAPERS, we have conceptualized and defined dysmetric dyslexia and dyspraxia as a cerebellar-vestibular induced dysmetria or sensory-motor and spatio-temporal sequencing and processing disturbance in dynamic equilibrium with compensatory forces—resulting in a diverse spectrum of symptoms in varying states of compensation and overcompensation (i.e., reading, writing, spelling, arithmetic, drawing, speech, temporal orientation, and emotional difficulties).[1] The temporal and spatial sequencing of the sensory input and motor output is scrambled, "blurred," or uncoordinated, and results in sensory-motor "reversals" or dysmetria. As in any disease state, the resulting manifest symptoms are vector resultants of opposing forces and appear in various combinations, in varying degrees of intensity, and in varying degrees of compensation and overcompensation. We have, furthermore, characterized this dysmetric disorder in terms of methods of diagnosis and prediction, etiology, incidence, prognosis, and treatment.

The purpose of this article is to present the rationale used to treat dysmetric dyslexic and dyspraxic individuals (of all ages) with seasickness medications, as well as to summarize the

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insights gained by our investigative-therapeutic methodology. (See Table 1, following the text of this article.)

CEREBELLAR FUNCTION AND DYSFUNCTION

Research with dysmetric dyslexic and dyspraxic individuals utilizing our "blurring-recognition speed" methodology and "3D optical scanner" has led to a greater understanding of cerebellar function and dysfunction.

Blurring-Speed Methodology

As we noted in an earlier article, we had assumed the ocular fixation and sequential scanning dysfunction in dysmetric dyslexics to be analogous to the tracking difficulty one might have while attempting to read a signboard from a rapidly moving train.[2] 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 and implemented a 3D Optical Scanner capable of beaming and recording the speed of the moving gestalt sequence, ie sequential blurring speeds. Three measurements of cerebellar-vestibular function were recorded.*

The objectivity of the blurring speed methodology can be increased and simplified by measuring recognition speeds (i.e., the speed at which a blurred or non-recognizable sequence becomes recognizable or clear). This relationship can be expressed as follows:

$$\text{Sequential Blurring Speed} = \text{Sequential Recognition Speed} + \frac{x}{10} \text{ seconds}$$

Inasmuch as x divided by ten is approximately two tenths of a second, x divided by ten is diagnostically insignificant

*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 the "blurring speed" for Mode I 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 observer is instructed to fixate the stationary foreground consisting of words while the scenic background is set in motion. The presence or absence of foreground movement and/or blurring is recorded. Refer to Figures 4 through 8 in J. Frank and H. Levinson, "Compensatory Mechanisms in Cerebellar-Vestibular Dysfunction, Dysmetric Dyslexia, and Dyspraxia," *Academic Therapy* 12:1 (Fall 1976): 20-26.

and for all practical purposes may be considered as zero. Therefore, for testing purposes:

Sequential Blurring Speed = Sequential Recognition Speed

Thus, if a series of differing moving gestalts are initially set in motion above the blurring or recognition speed, and are slowed down until recognition results, one can avoid "confabulatory responses" and be able to test very young children without having to explain and demonstrate the meaning of blurring. In practice, we determine the maximum induced tracking speeds by alternatingly determining the blurring and recognition speeds until the difference between the two determinations is less than three tenths of a second.

In addition, oculo-motor tracking patterns were obtained during Modes I, II, and III by means of ENG* and proved diagnostically helpful in assessing blurring neurophysiologically and resulted in significant neurophysiological insights.

Modes I and II

Dysmetric dyslexic and dyspraxic individuals blurred a moving visual sequence at significant reduced input speeds during Mode I and II testing when compared to normal controls.

Blurring, scrambling, and non-recognition are "cortical endpoints" indicating maximum cerebellar fixation and tracking capacity. In other words, the cerebral cortex cannot properly interpret the meaning of a rapidly moving scrambled visual input, once the cerebellar-vestibular capacity to maintain an optical fixation point is exceeded. Metaphorically speaking, the cortex cannot interpret what it cannot "see," and is "blind" at rapid input and scrambling speeds.

In dysmetric dyslexia and dyspraxia, blurring and scrambling occurs at significantly reduced sensory input speeds.

We therefore hypothesize that the cerebellum plays a vital role in inhibiting, modulating, or slowing down the rate at which the sensory input is transmitted to the cerebral cortex for interpretation. In cerebellar-vestibular dysfunction the cerebellar inhibiting capacity is impaired; the sensory input speeds cannot be significantly reduced prior to cortical reception; and, as a result, the cortex receives the input at a speed or rate beyond its

* Electronystagmography (ENG) is a technique for objectively detecting, recording, and measuring nystagmus—a rapid involuntary oscillation of the eyeball. The electronystagmographic monitoring of eye movements is made possible by virtue of the positive corneal potential relative to the retina; and the resulting changes in electrical potential when the eyes move may be recorded by electrodes placed on the outer margins of the eyes.

interpretive threshold, and blurring is experienced and reported.

Further, inasmuch as the blurring, scrambling, and reversals observed in dysmetric dyslexia and dyspraxia reflect the cerebellar's failure to maintain the spatio-temporal order of the sensory input prior to cortical reception, we postulate that the cerebellum normally acts to maintain the spatio-temporal order or sequence of the sensory input, as well as regulating cortical reception speeds.

ENG recordings during Modes I and II testing reveal that normal individuals have a pathognomonic sudden and dramatic reduction or inhibition of their induced tracking rates at the blurring speed, whereas dysmetric dyslexic and dyspraxic individuals often continue tracking a visual sequence moving at two or three times their blurring speed—a phenomenon we call "phantom tracking."

These findings lead us, thus, to the hypothesis that, in normal and compensated dysmetric dyslexic and dyspraxic individuals, inhibition of the tracking rate at the blurring speed represents a cerebellar modulated attempt to retarget at a physiological tracking rate and phantom scanning represents an inhibitory failure of this adaptive retargeting cerebellar mechanism as well as a denial of the tracking or scanning defect.

Mode III

For cerebellar-vestibular normal individuals, the Mode III moving scenic background does not induce a tracking nystagmus; the ENG tracking patterns are devoid of any foreground ocular deflections; and the fixated foreground is invariably reported as clear and stationary.

The Mode III testing of dysmetric dyslexic and dyspraxic individuals with significantly reduced Mode I and II blurring speeds often results in a background-induced nystagmus—and foreground movement and/or blurring is reported. In other words, the moving scenic background induces or provokes an optokinetic nystagmus, and compensatory attempts to regain foreground fixation results in a zigzag foreground-background ENG ocular deflection pattern. This foreground-background nystagmus manifests itself symptomatically and clinically in reversals, scrambling, blurring, and so on.

A crucial insight into cerebellar functioning can be gained by simply restating the previous observations:

1. Dysmetric dyslexic and dyspraxic individuals with uncompensated cerebellar-vestibular dysfunction have dif-

ficulty inhibiting or suppressing the Mode III background-induced nystagmus, which results in foreground-background instability, reversals, scrambling, and blurring.

2. Cerebellar-vestibular normal and compensated dysmetric dyslexic and dyspraxic individuals inhibit or suppress the Mode III attempt to induce or provoke a background nystagmus and as a result are able to maintain foreground stability.

3. Therefore, the cerebellum plays a vital role in maintaining foreground-background separation by a process of active background inhibition—and thus to act as a dynamic sensory-motor and foreground-background filter.

In summary, we have developed a clinically based hypothesis of cerebellar function which postulates: For normal perception, gnosis, and conception to occur in the cerebral cortex, the cerebellum (through selective inhibition and facilitation) must separate foreground from background, slow down the rate, and maintain and coordinate the spatio-temporal order of the sensory input in a manner analogous to its role in regulating and coordinating the motor output.

Sir John Eccles states:

> There is general agreement among neuroscientists that every conscious experience—every perception, thought, and memory—has as its material counterpart some specific spatio-temporal activity in the vast neuronal network of the cerebral cortex and subcortical nuclei, that is woven of neuronal activities in space and time in the "enchanted-loom" so poetically described by Sherrington (1940).[3]

We postulate the cerebellum to play a silently active role in the function of this mysterious "enchanted-loom"—a role not inferior to that of the cerebral cortex.

In describing motor learning in the cerebellum, Eccles states:

> 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 a 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.[4]

If sensory learning is hypothesized to occur in the cerebellum in a manner analogous to motor learning, then we can explain the "pseudo-cerebral higher cerebellar functions" found impaired in cases exhibiting dysmetric dyslexia and dyspraxia (and previously attributed to a fallaciously assumed cerebral cortical dysfunction).

If indeed our cerebellar speculations are correct, then the application of the concepts mentioned previously to our dysmetric dyslexia and dyspraxia research might be a small step in a direction predicted by Eccles in the last sentence of *The Cerebellum as a Neuronal Machine*:

> We are confident that the enlightened discourse between such theorists (communication theorists and cyberneticists) on the one hand and neurobiologists on the other will lead to the development of revolutionary hypotheses of the way in which the cerebellum functions as a neuronal machine; and it can be predicted that these hypotheses will lead to revolutionary developments in experimental investigation.[5]

Neurophysiological Hypotheses

Prior to our dysmetric dyslexic and dyspraxic research, conscious and non-conscious (unconscious) mental and perceptual events could not be explained neurophysiologically. Our work, however, has led us to a new hypothesis of the cerebellar role in modulating conscious and non-conscious

events, and of the need for bilateral cerebral hemispheres (or "two brains").

Utilizing our 3D Optical Scanner and blurring-recognition speed methodology, we found blurring or non-recognition to be a *cerebral cortical indicator* of maximum cerebellar tracking capacity (i.e., the perceptual cortex cannot consciously interpret or perceive what it cannot "see," and the cerebral cortex is "blind," once the cerebellar tracking capacity is exceeded and the optical fixation point is lost).

"Reading score" compensated dysmetric dyslexic and dyspraxic individuals with significantly reduced blurring speeds frequently utilize a special form of "speed reading," and are able to absorb both fixated, conscious content as well as "blurred," "unseen," "background," or "non-conscious" content.

We were forced to assume that these dysmetric dyslexic and dyspraxic individuals were able to derive the meaning of a paragraph, chapter, or book by a process of peripheral or background non-conscious perception—for how else could one explain their overcompensated reading ability on the one hand, and their dysmetric dyslexia and dyspraxia and decreased blurring speeds on the other. As a gifted dysmetric dyslexic and dyspraxic writer put it: "I've always been amazed by how much I knew and how little I read. Reading was always so very difficult and frustrating for me. As a child I scored in the 98th percentile for reading comprehension." She was amazed to learn that her blurring speeds were one third of those of a normal five and a half year-old child, and that she saw "absolutely nothing" when words were moving across a screen at the average word-blurring speed of a child of that age.

If indeed "non-conscious perceptions" occur and can be recovered by questioning (by means of comprehension and reading tests), and if conscious cerebral perception depends upon the impulse speed the cortex receives, then we are in a position to speculate as to the central nervous system mechanisms determining conscious and non-conscious perception. Thus, the hypotheses:

1. Conscious and non-conscious perception depends on the transmission speeds impinging on or received by the cerebral cortex.
2. The cerebellum, through its processes of selective inhibition, disinhibition, and facilitation, modulates the input transmission speeds received by the cerebral cortex.
3. Through the processes of disinhibition and facilitation,

the cerebellum may either fail to slow down the sensory input, or even speed it up so that the sensory input speeds reaching the perceptual cortex exceed its "interpretive threshold," and "blurring" or "non-recognition" is perceived and reported.

4. The cerebellum, by regulating transmission speeds, dynamically influences conscious and non-conscious cerebral perception as well as foreground-background perception.

5. By virtue of controlling transmission speeds, the cerebellum is especially well suited to serve as a "dynamic sensory-motor filter"—capable of separating the sensory-motor input and output into foreground and background.

6. The cerebral hemispheres have developed as an extension to and in relationship to cerebellar function. Instead of viewing the cerebral hemispheres as dominant and non-dominant for gnostic perception, we consider both cerebral hemispheres as dominant—one cerebral hemisphere is dominant for foreground perception and the other is dominant for background perception. This hypothesis may serve to clarify the question: "Why two brains?" Holistically speaking, both cerebral hemispheres are in dynamic equilibrium with each other, the cerebellum, and the organism as a whole.

Should these hypotheses prove valid, we will have succeeded in bridging the theoretical gap artificially separating psychoanalysis and neurophysiology — hopefully providing a windfall harvest to both fields.

In attempting further to understand cerebellar morphology and function, Eccles traces the evolution of the cerebellum:

> With each further evolutionary development of the brain, this same cerebellar organization seemed to be a necessary adjunct, presumably because it possessed some unique mode of processing information. Hence, these newly evolved components of the brain colonized or developed areas of the cerebellum for this purpose; and most lately of all, the cerebral hemispheres have called forth the great development of the cerebellar lobes. With the evolutionary growth of the brain, the cerebellar hypertrophy has matched the hypertrophy of the cerebrum. This evolutionary story certainly gives rise to the concept that there is some highly significant and unique functional meaning in the neuronal organization of the cerebellum and in the processing of information that is accomplished thereby.[6]

Although Eccles states that "the cerebral hemispheres have called forth the great development of the cerebellar lobes," we postulate that, from a phylogenetic and functional point of view, the reverse was probably true; that is, the development of the cerebellar lobes stimulated the development of the cerebral hemispheres as a response to the organism's need to master increasingly complex environmental stimuli over an evolutionary time span. This cerebellar-cortical "developmental spurt" arose in relationship to man's attainment of an upright gait and need to master an exponentially expanded sensory-motor horizon.

The Cerebellar Role in Modulating Sensory Input

A search of the literature has revealed a series of animal experiments indicating the cerebellum receives and reacts to visual, tactile, and acoustical—as well as proprioceptive—stimuli.

> The unexpected discovery that the cerebellum, which Sherrington (1906) had called "the head ganglion of the proprioceptive system," is heavily impinged upon by exteroceptive impulses (touch, vision and hearing) was reported almost simultaneously by Snider and Stowell (1942, 1944), Dow and Anderson (1942), Adrian (1943) and Snider (1943).[7]

According to R. S. Snider and A. Stowell:

> It is an established and universally recognized fact that the cerebellum is a place of convergence of impulses from proprioceptors located throughout the body. The great emphasis placed on this fact, the tacit assumption that the spinocerebellar tracts convey only impulses of proprioceptive origin, and the nature of signs and symptoms resulting from cerebellar deficit doubtless have combined to discourage serious experimental examination of the possibility that impulses from other groups of receptors also pass to this organ. Yet a number of considerations warrant the hypothesis that at least some classes of exteroceptors possess a cerebellar representation. The present investigation was begun when the following facts were considered together: (i) tactile impulses are relayed by the nuclei gracilis and cuneatus to the thalamus and thence to the cerebral cortex, and (ii) these same nuclei, according to many workers, send fibers to the cerebellum by way of the external arcuate fibers. Since there is no good reason for supposing that all impulses carried from the nuclei of the posterior columns by the external arcuate system originate only

in proprioceptors, it seemed reasonable to attempt to determine whether impulses from the tactile end-organs pass to the cerebellum.

The success which has attended recent attempts to map areas of sensory projection in the brain by recording evoked potential changes led us to use this method. It soon was found that the application of an appropriate tactile stimulus to the region of the foot of a cat evokes discrete potential changes in definite cerebellar areas. The extension of this original observation enabled us to secure evidence of the existence of a topical projection of certain parts of the cutaneous tactile system to the cerebellum.

Studies on the auditory system were initiated when it was observed that discrete surface positive potentials of latencies shorter than those of the tactile responses were evoked during displacement of hairs around the external ear. Since the mechanical stimulator used to move the hairs made a low clicking sound and since the evoked potentials proved not to be tactile in origin, it became evident that we were dealing with a representation of the auditory system in the cerebellum. Subsequent experiments adequately established this fact. Naturally the question then arose as to whether any other major exteroceptive system sends impulses to the cerebellum. Subsequent experiments were carried out to examine this point, and it was soon found that impulses of retinal origin reach certain cerebellar cortical areas.

One of the most universally accepted teachings of clinical neurology is that the cerebellum is not concerned with any kind of sensation, for true sensory defects have not been found among the disorders which are produced by cerebellar lesions in man. Certainly there is no clinical evidence to indicate that any cerebellar deficit is accompanied by disturbances in touch, auditory or visual perception. Yet we cannot resist wondering whether loss of the cerebellar representations of these three exteroceptive systems does not produce objective and subjective effects which are so subtle that they have escaped present methods of study.[8]

Our clinical research findings have indicated that indeed there are significant disturbances in visual, tactile, auditory, and proprioceptive sensory functioning as a result of cerebellar-vestibular dysfunction and dysmetric dyslexia and dyspraxia; but that these dysfunctions were either denied or fallaciously

attributed to cerebral cortical dysfunction and such vague concepts as "minimal brain dysfunction" or "cerebral immaturity."

If indeed the cerebellum may be considered as the "head ganglion of the proprioceptive system" (Sherrington) and is neurophysiologically capable of harmonizing the total sensory input as it is known to modulate the total motor output, then our assumption that the cerebellum might be considered as the "head ganglion of the total sensory-motor system" seems scientifically justified.

B. Ghelarducci, M. Ito, and N. Yagi demonstrated vestibular signals entering the cerebellum (of a rabbit) as a mossy fiber input and visual signals as a climbing fiber input, and as output inhibits second-order neurons which mediate vestibulo-ocular reflexes. They state:

> It is conceivable that the flocculus used visual information to modify the vestibulo-ocular reflexes to obtain steady retinal image during head movement.[9]

The neurophysiological animal experimentation demonstrates the role of the cerebellum in receiving and modulating the sensory input, as well as regulating oculo-motor reflexes through centrifugal inhibition. The structure and function of the cerebellum is as suited to harmonize the sensory input as it is to regulate the motor output.

SEASICKNESS MECHANISMS AND SEASICKNESS MEDICATIONS

Our clinical research with dysmetric dyslexic and dyspraxic individuals has demonstrated that the cerebellum plays as vital a role in modulating the visual input (especially in relationship to upright gait) as it does in harmonizing the motor output; furthermore, that the cerebellum modulates the acoustic, tactile, and kinesthetic inputs.

We have developed a clinically based hypothesis of cerebellar function in which, through inhibitory-facilitory modulation and control, the cerebellum is theorized to play a vital role in regulating sensory (and motor) speed transmission prior to its reception by the cerebral cortex for interpretation, perception, or conceptualization. Specifically, we hypothesized that for normal cortical interpretation to take place, the cerebellum must slow down the sensory transmission speed so that it falls within the threshold speed range required for cortical interpretation.

When blurring, scrambling, or non-recognition is cortically perceived, the cerebellar's capacity to inhibit, slow down, and

maintain the spatio-temporal sequence and order of the sensory input has been exceeded; and the input sensory speed at which this scrambling or blurring endpoint occurs is a measure of cerebellar inhibitory and ordering capacity.

This inhibitory and sequential ordering capacity is significantly reduced in cerebellar-vestibular dysfunction and results in dysmetria. Blurring, scrambling, and non-recognition represent the inability of an intact cortex to properly understand, see, hear, or feel a sensory sequence which is transmitted to it at a rate and in a "scramble" beyond its threshold for decoding.

One might justifiably ask at this point: "What does all this have to do with motion sickness and the 'seasickness medications'?" We will attempt to answer this question by first asking two more:

1. Could it be that motion stimuli are also modulated by the cerebellum, as was determined or hypothesized for the visual, acoustic, and tactile stimuli?
2. If indeed motion stimuli are inhibited, slowed down, and regulated by the cerebellum as are the other sensory input stimuli (and motor outputs), might we not be justified in viewing motion sickness as an endpoint representing "cerebellar overloading" or "cerebellar inhibitory capacity" for modulating motion input?

Thus, by analogy, blurring and seasickness may be considered cortical and autonomic nervous system indicators, or signals, that the cerebellar's sensory inhibitory and sequential ordering capacity has been exceeded.

If so, then seasickness or motion sickness might be viewed as an Inbuilt Release Mechanism (IRM) which is triggered when the motion input exceeds an adaptive rate and thus acts as a warning signal to the organism that something is wrong. One may further speculate that this unpleasant seasickness response may serve to force the central nervous system to change its physiologically dangerous state by:

1. flight, and/or
2. expenditure of the "energy overloading" through energy-discharging reflexes: wretching, vomiting, etc.

By analogy, motion sickness and anxiety may now be viewed as homeostatic IRMs which are genetically and environmentally imprinted with "survival" triggering information so that the individual might avoid injury and dangerous situations. Indeed, the seasickness IRMs and the anxiety IRMs are adaptively inter-

related; and one often triggers the other.

This cerebellar-vestibular overloading and "adaptive" theory of motion sickness could then explain the many puzzling and as yet unanswered questions regarding motion sickness:

1. If motion sickness requires vestibular stimulation, how can motion sickness occur without *motion*, and thus without any apparent stimulation of the motion receptors of the vestibular apparatus?

2. How can motion sickness be conditioned and thus be triggered by smell or some other conditioned stimulus? Once again, motion sickness can be triggered by a "learned" or "conditioned" non-motion and non-vestibular stimulus. The vestibular apparatus and motion receptor is not crucial in modulating this complex learned or conditioned motion sickness response.

3. How and where does "habituation" to rotation and specific motion take place? Apparently not in the vestibular apparatus.

4. How can psychogenic factors associated with fear and anxiety lead to the symptoms of motion sickness without motion or primary vestibular stimulation?

5. Why do the amphetamines and other stimulants act as other antimotion sickness or antiemetic drugs—and yet the stimulants are believed to have no direct effect on the vestibular system?

In view of the above unanswered questions, C. D. Wood and A. Graybiel are justified in stating: "The central nervous system mechanisms involved with motion sickness are incompletely understood, hence the actions of the drugs in preventing motion sickness are also not understood."[10] And H. A. Bickerton is equally justified in stating, "The precise pharmacologic activity and site of action of the antimotion sickness drugs remain uncertain."[11] In introducing the topic he states: "Motion sickness has been a recognized clinical entity for over 2,000 years; it is mentioned by Hippocrates. Glaser has referred to it as a 'unique affliction which, in common with childbirth, can cause complete temporary incapacitation without any pathological basis and entirely by reflex mechanisms, though unlike childbirth, it serves no obvious purpose at all'."[12]

If, however, we take into account the neglected role and function of the cerebellum in "motion sickness" as we did in dysmetric dyslexia, then we have a unique theory which can attempt to answer the questions raised previously regarding

motion sickness. Briefly, if we acknowledge that the cerebellum plays a major role in modulating, harmonizing, controlling, or communicating with both "outer world" and "inner world" (autonomic nervous system) sensory-motor systems through its proven function of centrifugal (outgoing) peripheral selective inhibition and facilitation and its demonstrated ability to order and coordinate almost all centripetal or incoming sensory-motor signals, then we are in a position to hypothesize about "motion sickness" as we had previously hypothesized about dysmetric dyslexia and dyspraxia.

Cerebellar Inhibition and Motion Sickness

Ocular fixation is known to suppress or inhibit nystagmus of vestibular origin. Ocular fixation is known and was found to suppress, diminish and even prevent motion sickness (i.e., dysmetric dyslexic and dyspraxic individuals prone to motion sickness prefer or are compelled to drive a moving vehicle rather than be passengers so as to avoid triggering the motion sickness response). On the basis of the above observations, we assumed that ocular fixation and concentration trigger inhibitory mechanisms which diminish vestibular reactivity, vestibular nystagmus and motion sickness.

Independent neurophysiological animal experiments indirectly support the contention and thus the role of the cerebellum in motion sickness:

> The cat flocculus receives excitatory input from the labyrinth by the mossy fiber (MF) route from the optic nerve by the climbing fiber (CF) route, as in the rabbit . . . and MF and CF inputs from neck afferents. . . . Excitation of labyrinthine and visual origin converge on the same Purkinje cells. . . . which also can be excited by neck afferents (Wilson, Maeda and Franck, unpublished observations). In addition to these excitatory actions, stimulation of the labyrinth or visual system causes inhibition at the granule or Purkinje cell level. . . . In this paper we will show that simulation of neck afferents also produces intracerebellar inhibition, *and that the inputs from the three modalities inhibit each other.* [Italics have been added for emphasis.]
>
> Not only excitatory but also inhibitory action originating in labyrinth, visual system and neck afferents converge onto the same areas of the cat flocfulus and onto the same population of Purkinje cells. It is well known that floccular Purkinje cells modulate

> vestibulo-ocular reflexes (e.g., ref. 5) and the presence of excitatory and inhibitory neck inputs to these neurons suggests that information from neck receptors plays a role in this modulation. Whether some of the Purkinje cells excited or inhibited by neck afferents exert an influence, at the level of the vestibular nuclei, on cervico-ocular reflexes . . . or on vestibular reflexes to neck motoneurons remains to be determined.[13]

The Motion Sickness Drugs

We should consider the following hypothesis: When overloaded, the cerebellum is no longer efficiently able to slow down, maintain the sequence, and coordinate sensory-motor impulses or stimuli. Under the stress of and as a reaction to the specific overloading of motion stimuli, the autonomic nervous system is triggered on the one hand and selectively released from cerebellar modulating control on the other, and reacts with nausea and vomiting. This "motion sickness reaction" may be viewed similarly to the warning and discharge function of anxiety—in the service of adaptation and homeostasis.

Just as cerebellar dexterity and coordination varies from individual to individual, so does the cerebellar's ability to modulate selective sensory (and motor) stimuli vary from individual to individual—thus accounting for the significant variation in susceptibility to motion sickness.

The cerebellum is capable of "habituation," conditioning, or "learning"; and this function can explain the decreased motion sickness response to repeated motion if we assume that Eccles' formulation of motor "learning in the cerebellum" (cited earlier) applies equally well for "sensory learning." This assumption enables us to understand simply how visual, auditory, or conditioned smell stimuli can trigger the cerebellum to react with motion sickness in the absence of motion input and in the absence of vestibular stimulation.

We may then conclude that any disease state of the cerebellum, toxic or otherwise, might selectively trigger the specific "learned" or "imprinted" motion sickness mechanisms within its neuronal sphere.

The cerebellum plays an essential role in many adaptive and homeostatic mechanisms. Thus, fear and anxiety may similarly trigger this cerebellar controlled motion sickness response.

The amphetamines or stimulants are known to stimulate the cerebral cortex. In our initial paper, we postulated that the

stimulants activate the reticular activating system which in turn activates or alerts the cerebral cortex.[14] It is now possible to hypothesize that, through reticular-cerebellar and cortical-cerebellar feedback loops, the amphetamimes or stimulants lead to "cerebellar activation" and thus strengthen the ability of the cerebral cortex to modulate, coordinate, and integrate sensory and motor sequence stimuli in a harmonious fashion.

The various other "antimotion" or "antinauseant" medications must directly or indirectly accomplish a similar type of increased cerebellar control, regardless of their varying sites of action of pharmacologic activity.

Increased cerebellar control leads to its increased capacity to modulate, coordinate, order, slow down, and harmonize its sensory and motor input and output.

If our hypotheses are correct, then the "antinauseants" or "antimotion" medication groups are "cerebellar (or cerebellar-vestibular) harmonizing agents," and thus may prove effective for dysmetric dyslexia and dyspraxia.

We experimentally administered "cerebellar-vestibular harmonizing agents" to dysmetric dyslexic and dyspraxic individuals and found that these agents improved their cerebellar and cerebellar-vestibular functioning.

This experiment validated our hypotheses that:

1. Motion sickness is a cerebellar or cerebellar-vestibular dysfunction;
2. Antimotion sickness medications and many of the antinauseants and antiemetics are cerebellar or cerebellar-vestibular harmonizing agents;
3. Cerebellar or cerebellar-vestibular harmonizing agents are effective in dysmetric dyslexia and dyspraxia;
4. Cerebellar or cerebellar-vestibular harmonizing agents are or can be effective in diminishing cerebellar symptoms of various types;
5. The therapeutic use of these cerebellar-vestibular harmonizing agents can be useful in investigating the function and dysfunction of the cerebellar and cerebellar-vestibular circuits in dysmetric dyslexic and dyspraxic individuals.

Positive Response Pattern

In a follow-up article we will present the clinical data from which the following Positive Dysmetric Dyslexia and Dyspraxia Composite Response Pattern summary was abstracted. As the

reader will note, the results of our double-edged investigative-therapeutic pharmacological study have independently corroborated our conceptualization of cerebellar function and dysfunction—as derived from our clinical dysmetric dyslexia and dyspraxia observations and blurring-speed data.

Summary

On the basis of our clinical observations of dysmetric dyslexia and dyspraxia, blurring-speed data, and corresponding ENGs, we have developed a new conceptualization of cerebellar function and dysfunction, motion sickness medications, and the use of motion sickness medications in dysmetric dyslexia and dyspraxia. The Positive Dysmetric Dyslexia and Dyspraxia Response Pattern to the Motion Sickness Medication is presented—and provides the basis for a new treatment of dysmetric dyslexic and dyspraxic individuals, as well as a new tool with which to investigate cerebellar function and dysfunction pharmacologically.

We have attempted to condense a massive amount of clinical and theoretical dysmetric dyslexic and dyspraxic data so as to keep the reader abreast of our continuing, ever-expanding dymetric dyslexia and dyspraxia research efforts. Follow-up articles and books will, we hope, round out and fill in the sketch or summary, and provide the reader with a coordinated, dynamic, and holistic portrait of a research effort which expands exponentially at every twist and turn.

Table 1

Positive Dysmetric Dyslexic and Dyspraxic Response Patterns to Seasickness Medications

Reading Activity

Increased spontaneous reading activity
Diminished dysmetric tracking and finger pointing
Improved fixation ability
Improved foreground-background differentiation (i.e., decreased blurring and increase in degree of letter blackness)
Decreased or eliminated reading reversals
Increased reading speed and accuracy
Increased interest in reading

Writing Activity

Increased spontaneous writing activity
Smoother rhythm and increased legibility
Improved spacing between letters and words
Increased horizontality in writing
Increased use of cursive writing (printing usually easier)
Decreased writing reversals
Increased use of grammatical details (i.e., periods, commas, etc.)
Increased writing speed
Increased word content
Decreased number of spelling errors

Spelling

Increased spelling-recall and decreased letter reversals (i.e., insertion and omissions

Arithmetic

Increased mechanical alignment
Increased memory for calculations

Directionality, Spatial Organization and Planning

Increased right-left differentiation
Decreased rotations
Increased detail in drawing
Improvement in *Goodenough* figure drawings
Improved spacing in writing
Improved relationships to spatial coordination tasks (i.e., ball playing, catching, throwing, batting, etc.)
Increased ability to tie shoelaces, etc.

Balance and Coordination

Increased ability to ride a bike, dribble basketball, etc.
Decreased clumsiness (i.e., tripping, falling, and various past-pointing and pre-pointing activities)
Increased feeling of internal steadiness

Foreground-Background Activity (Sensory)

Increased foreground clarity
Improved background suppression of irrelevant and distracting events (i.e., visual, acoustic, etc.)
Decreased acoustical blurring and scrambling

Speech

Increased spontaneity of speech
Decreased slurring, where present
Increased rate and improved rhythm of speech
Increased verbal content
Decreased stuttering, stammering and hesitations

Sequence Activity and Memory

Increase in sequence memory (i.e., days of the week, months of the year, spelling, multiplication, etc.)

Time Sense

Increased sense of time and time sequences

Concentration and State of Consciousness

Improved and increased clarity of consciousness—and associated improvement in memory

Mood

Improved and increased stability of mood

Self-Image

Decreased feelings of inferiority and stupidity
Decreased defensive attitude
Increased self-assertiveness
Increased positive attitude

Body Image

Improved—as reflected in *Goodenough* figure drawings and generalized sensory-motor activity
Improved visual, acoustic, tactile, temperature, olfactory and proprioceptive modulation

Frustration Tolerance

Increased frustration tolerance
Increased concentration and attention span

Anxiety Tolerance

Increased anxiety tolerance

Socialization

Increased and improved socialization—especially with peers

Acceptance of Symptoms

Decreased denial
Increased ability to tackle, understand, and accept symptoms
Increased ability to ask questions spontaneously

Dysmetric Dyslexic and Dyspraxic Phobias, Inhibitions, Counterphobias, Characterological Development

Improved

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2. Frank and Levinson, "Dysmetric Dyslexia and Dyspraxia—Synopsis . . . ," *loc. cit.*: 139.
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8. R. S. Snider and A. Stowell, "Receiving Areas of the Tactile, Auditory, and Visual Systems in the Cerebellum," *Journal of Neurophysiology* 11 (1944): 331-357.
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14. Frank and Levinson, "Dysmetric Dyslexia and Dyspraxia—Hypothesis . . . ," *loc. cit.*

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