

NEUROLOGICAL-BEHAVIORAL MODEL OF DYSLEXIA

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ABSTRACT

This article addresses the distinction between non-specific and specific reading disabilities and dismisses the notion that they are of a homogeneous nature. Based on neurological evidence, a neurological-behavioral model is proposed which allows for identification and operational definition of distinct types of dyslexia. These types of dyslexia may be directly determined by characteristic coding dysfunctions of the individual. Disability-specific instructional strategies can be implemented once the specific coding dysfunction is known. This model also offers a solution to previous reporting in which researchers unknowingly mixed the types of dyslexia within their samples to contaminate research results.

KEY WORDS

dyslexia, reading disability, learning disability, neurology, behavior, dyseidnesia, dysphonesia, decoding, encoding, vision therapy

This article is dedicated to optometrists and other professionals who help dyslexic individuals. It offers a neurological-behavioral model for the differential diagnosis of dyslexia and co-management of its various types. Important questions emphasized in this article include: Do different types of dyslexia require different interventions? Are research results on dyslexia contaminated by mixing the types of dyslexia in sampling? What differential diagnostic tools can be administered? Do promising instructional strategies exist in educational therapy for individuals who have dyslexia?

We will briefly review the following: previous work on dyslexia, reading disability versus dyslexia, neurology of the different types of dyslexia, neurological-behavioral models, and practical considerations.

PREVIOUS WORK ON DYSLEXIA

The term dyslexia, which is used to describe individuals with a specific reading disability, originated in the late nineteenth century. According to Hinshelwood,¹ initial reports were made in 1877 by Kussmaul who described an acquired inability to read in adults with normal vision; he called this condition "word blindness." The term dyslexia was first used by Berlin in 1887, but it was Hinshelwood himself, in 1896, who differentiated the syndrome of complete "word blindness" (alexia) from the partial written language deficit of dyslexia.

Orton² proposed a theory to explain this condition and elaborated on his hypothesis in 1937. He contended that there was an inappropriate development of dominance between the right and left hemispheres of the brain. This was hypothesized to explain the apparent common difficulty of "dyslexics" in correctly orienting letters and numbers, e.g., "d" instead of "b." According to Orton, the lack of development of cerebral dominance leads to a "directional confusion" causing great difficulty for the individual with the symbols of written language. He used the term "strephosymbolia," meaning twisted symbol, to describe dyslexia. Orton was the first advocate for individuals with dyslexia, contending that their difficulty with the "symbolic" aspects of written language did not imply low intelligence. However, Orton's theory of cerebral dominance causing dyslexia has been largely refuted on the basis of more recent knowledge of functional neurology.^{3,4}

In recent years a great deal of work has been done in the area of determining the neurological deficits and differential brain function in dyslexia.⁵⁻¹² As a result of these efforts a new understanding of the nature of the individual with dyslexia, and how to help him or her, is available.

GENERAL READING DISABILITY VERSUS DYSLEXIA

Before discussing the recognized types of dyslexia a distinction between dyslexia and general reading disability

must be made. Critchley¹³ attempted to clarify the confusion regarding the difference between a dyslexic individual and a non-dyslexic poor reader. He was a proponent of the exclusionary diagnosis of dyslexia. Critchley stated that dyslexia was "... a disorder manifested by difficulty in learning to read despite conventional instruction, adequate intelligence and sociocultural opportunity. It is dependent upon fundamental cognitive abilities which frequently are constitutional in origin."

Of further importance to our discussion is the point made by Kolb and Winshaw,¹⁴ and Adelman and Taylor¹⁵ regarding the confusion surrounding the use of the term dyslexia. They stated that the basis for this confusion is that most people were using the term dyslexia as a label for everyone with a reading problem rather than restricting the term to those who had a specific neurological dysfunction.

These two contentions, the exclusionary diagnosis definition and the CNS dysfunction specification, give rise to two broad categories of reading disability. They are: 1. general problems causing non-specific reading disability (non-dyslexia) and 2. coding problems causing specific reading disability (dyslexia). (Refer to Table 1.) Specific reading disability (dyslexia) is a deficit in an individual's ability to interpret the symbols of written language due to minimal brain dysfunction and/or differential brain function.¹⁶

Table 1
READING DISABILITY
(broadly classified)

Non-Specific (general) Reading Disability

(Caused by one, or a combination of a number of factors as listed below)

- Low intelligence
- Educational deprivation
- Sociocultural deprivation
- Primary emotional problems
- Sensory impairment (visual, auditory, etc.)
- Poor motivation
- Attentional problems
- Etc.

Specific Reading Disability (dyslexia)

Dyslexia is a coding problem;¹⁶⁻¹⁸ the dyslexic has difficulty breaking the code of the written language. Coding ability involves decoding (determining the sound of a word from the printed symbols) and encoding (determining the letters which form the written word from a dictated word).

The lack of understanding that dyslexia is a coding problem has contributed to the confusion regarding the use of the term. For example, the relationship between comprehension and dyslexia is often poorly understood. However, Share and Silva¹⁹ found test-specific factors unique to comprehension versus word recognition (i.e., decoding) and that dyslexia is not significantly correlated with IQ. Most authorities agree that comprehension would not generally be a problem for individuals with dyslexia if they were helped to decode the words they did not know. (Dyslexics can usually comprehend very well when they listen to what is read to them.) On the other hand, there are individuals who have no decoding problem and yet have very poor comprehension; strictly speaking, they should not be considered dyslexic.

NEUROLOGY OF DYSLEXIC TYPES

Dyslexia is probably not a single, homogeneous entity.^{16-18,20-22} Two distinct cognitive processes for decoding (recognizing) and encoding (spelling) words have been confirmed on neurophysiological and anatomical bases.^{5-11,23,24} These two processes are: 1. phonological (phonetic coding) and 2. lexical (eidetic or whole-word coding). For example, the phonetic process is used to decode an unfamiliar, regular word such as "abandon." The individual first sounds out each syllable of the word (uh-ban-duhn), then blends the sounds of the syllables to form the sound for the word. Once the individual "hears" the word and knows it, the word can be spoken with syllable stressing according to the dialect of the individual (e.g., uh-BAN-duhn). The eidetic process, on the other hand, is used to decode familiar words which are included in an individual's sight-word lexicon. Many English words must be processed eidetically to a large degree if they are spelled irregularly and do not closely follow the rules of phonics. Ex-

amples are does (duz), should (shud), and foreign (foren). Reading such a word depends greatly on eidetic decoding since an attempt to decode the word phonetically would not result in the appropriate sound for the word.

These two written language processes of coding (i.e., phonetic and eidetic) serve as the fundamental basis for discussing research on the neurology of several types of dyslexia. As evidence of these processes is presented, the various types of dyslexia will be elucidated.

As stated by Benton and Pearl²⁵ "... the opinion of most professional workers is that endogenous factors reflected in anomalies of central nervous system function constitute the primary basis for specific reading retardation. It is for this reason that identification of the neurological correlates of the disability is generally regarded as the major research task today." This task has been further delineated by the suggestions of Pirozzolo and Witrock²⁷ and amplified by Hynd and Semrud-Clikeman.¹² They indicate that the interface between the cognitive and neurological sciences will lead to a fuller understanding of disabled readers. Therefore we present the research regarding brain function involving the cognitive-linguistic processes of phonetic and eidetic coding. This provides an updated neurological model of the types of dyslexia and information regarding diagnosis, counseling, and educational therapy. Before discussing the specific neurology of dyslexia, an overview of how the brain functions for the specialized task of reading is presented.

We do not suggest that there is a single, discrete area in the brain which functions as a "reading center." However, we also do not believe that the entire brain must always be totally involved in reading. Luria²⁶ sums up the function of the brain by stating that there are discrete brain areas of cortical activity which act in coordination to provide a functional system for a particular cognitive activity.

NEUROLOGICAL PERSPECTIVES ON READING

The following is an updated neuroanatomical model of the functional system involved in dyslexia. In keeping with Luria's contention, is there an area of the brain responsible for the cognitive

process of eidetic (whole-word) coding? Boder and Jarrico²⁷ hypothesized that a compromised right hemisphere, which mediates many visual-spatial abilities, was responsible for eidetic (whole-word) coding problems. Boder¹⁷ called this dyseidetic dyslexia. Geschwind⁵ was not in agreement with this hypothesis. He felt that the functional brain area responsible for whole-word coding was in the left hemisphere. Specifically, he proposed that the area involved was the left angular gyrus, located in the posterior parietal lobe. Further support for the left angular gyrus being the brain area involved in dyseidetic (for right-handed individuals) was provided by Roeltgen and Heilman.¹⁰ They described four cases showing the characteristics of dyseidetic dyslexia (which they labeled "lexical agraphia"). These patients all had lesions in which the left posterior parietal lobe was involved. Additionally, Flynn and Deering¹¹ performed electroencephalographic (EEG) studies on non-acquired dyseidetic dyslexics and determined that they showed an increase in theta from resting baseline. The significant difference in brain activity, increased theta waves, occurred in the area of the left angular gyrus. Naour²⁸ has indicated that increased theta is a signal of cerebral inefficiency.

The next question is whether there is an area of the brain responsible for phonetic coding. Boder¹⁷ introduced a type of specific reading disability involving phonetic coding problems and called it dysphonetic dyslexia. Hynd and Hynd⁹ proposed that the angular gyrus was responsible for phonetic coding (or "grapheme-phoneme correspondence," as they stated it). Hynd's theory, however, has not been supported by other researchers^{8,10} who studied patients with severely impaired phonetic ability (disability sounding out nonsense words phonetically, or poor phonetic spelling of dictated words). Based on computerized tomographic (CT) scans, it was determined that a portion of the supramarginal gyrus (SMG) of the left hemisphere was the most likely anatomical substrate responsible for the phonetic coding difficulties (dysphonia). Furthermore, it was noted that the angular gyrus had been spared in these patients who, although they manifested what was described as dysphonia (the authors used the term "phonological dyslexia"), did not exhibit

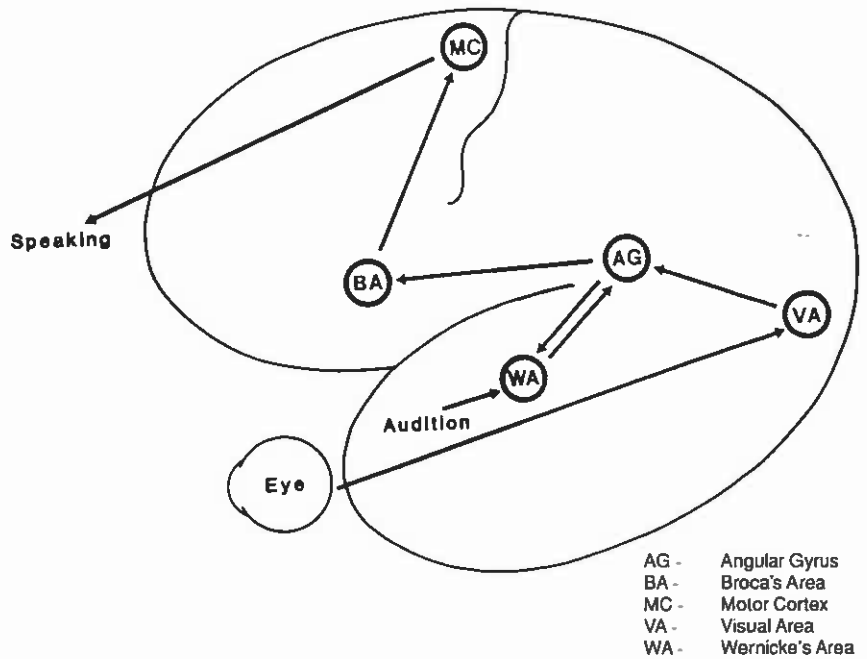


Figure 1. The Oral Decoding Process

the whole-word coding difficulties of dyseidetic dyslexia. Griffin and Walton¹⁶ speculated that the site of cortical dysfunction causing dysphonia was in Wernicke's area. The SMG is included in Wernicke's area.²⁹

The above substantiation of neuroanatomical loci, responsible for the eidetic and phonetic coding functions of written language, provides a rationale for presenting a functional neuroanatomic model based on EEG studies.⁷ It was determined that significant differences in the brain activity occurred in dyslexics versus non-dyslexics during reading tasks. During resting periods there were no differences in the EEG recordings. The significant differences which were recorded during the act of reading occurred in Broca's area in the left temporal lobe and in the angular gyrus.

Furthermore, support for the neuroanatomic model is provided by evidence indicating that written language deficits of the dyslexias are based on specific, minimal, developmental, brain anomalies. Cytoarchitectonic studies have demonstrated that developmental brain anomalies do exist in dyslexics.^{6,24}

Therefore the following neuroanatomic model of the types of dyslexia is presented on the basis of: 1. the evidence for separate neuroanatomic loci for coding functions, 2. EEG studies showing characteristic differences between individuals with and without dys-

lexia, and 3. evidence that dyslexia is based on specific developmental brain anomalies.

NEUROLOGICAL-BEHAVIORAL MODELS

From our interpretation of the literature, we propose the following neuroanatomic model for the decoding of written words. The neuroanatomic loci involved in the decoding of words are shown in Figure 1. A written word is presented to the individual. The visual image is received by the eyes and neurological impulses are sent to the visual area (VA) in the occipital lobes via the visual pathway. The impulses then travel to the left angular gyrus (AG). If the word presented is one with which the person is familiar (it is part of his sight-word recognition), then a sight-sound match is made quickly. If the word is one for which the subject has not developed sight recognition, then the impulses travel to a point in Wernicke's area (WA), perhaps the SMG. Here the word can be syllabicated, sounded out, and blended (i.e., phonetically decoded). This additional interaction between the angular gyrus and Wernicke's area, besides being relatively slow, may not always be effective in decoding irregular words, e.g., "foreign" and "should." If the person is able to decode the word phonetically, impulses are sent via the arcuate fasciculus to Broca's area

(BA) where, among other functions such as grammar sequencing and motor programming, speech formation is initiated. The signal is then sent to the motor cortex (MC) and relayed to the muscles around the mouth and throat, which are involved in articulation, so the word can be spoken.

This over-simplification serves as a neurologic model for the process of decoding and allows one to understand the reading and spelling problems associated with the three generally recognized types of dyslexia.

These types are dyseidnesia, dysphonia and the combined form, dysphonidnesia.^{16,17,21,27,30}

The whole-word coding problems of the individual with dyseidnesia are primarily due to a deficit involving the angular gyrus. Common spelling errors include phonetic equivalents for irregularly spelled words, e.g., "shud" for should. Reading is laborious and comprehension is usually reduced due to decoding slowness and the inability to decode irregularly spelled words when phonetic decoding is relied upon.

The dysphonetic type of dyslexia (dysphonia) causes an individual to have characteristic spelling errors and reduced ability to decode unfamiliar words during reading tasks. The attempted spelling is not a good phonetic equivalent of the word, e.g., "anaple" for abandon. Another error often made in these cases is semantic substitution. Since the individual has problems in attacking unknown words phonetically, it may be read, for example, "home" instead of house. Reading may be fairly good until the dysphonetic comes to an unfamiliar word. Such individuals have great difficulty applying the rules of phonics in decoding, although they may be able to store words very well for sight recognition.

The individual with dysphonidnesia (combined type of dyslexia) is severely limited in his or her ability to interpret the symbols of written language. Decoding and encoding errors are characteristic of both dyseidnesia and dysphonia.

A commonly held viewpoint is that dyslexia is synonymous with letter reversals, e.g., problems writing "b's" and "d's". A separate type of dyslexia, dysnemkinesia, addresses this issue.¹⁶ However, this type probably does not involve a significant neurological deficit for

coding and its prevalence is relatively low compared with dyseidnesia and/or dysphonia. It is more likely due to a developmental delay in the acquisition of laterality and directionality awareness in young children. Dysnemkinesia may, however, occasionally accompany the coding dysfunctions of dyseidnesia and dysphonia. Dysnemkinesia is usually amenable to therapy.³¹

PRACTICAL CONSIDERATIONS

There are practical considerations for helping the person with dyslexia based on this model. These include early diagnosis, multidisciplinary care, counseling, and appropriate educational intervention and remediation. As a result of this neurological-behavioral approach to reading disabilities, methods of differentially diagnosing the types and severities of dyslexia have been developed.^{16,27} In addition, a quick and easy-to-administer screening method has been developed.³² By employing these detection and/or diagnostic approaches, children with specific reading disabilities may be identified and provided with immediate educational intervention. When children with dyslexia and their parents and teachers are all informed about the specific deficiencies that exist, the individual is less likely to develop secondary emotional problems.³³

When appropriate methods of diagnosis are employed and children with specific types of dyslexia are identified, counseling and proper educational therapy may be instituted. This allows the youngsters to develop optimally in reading, writing and spelling. They may never reach the levels of most non-dyslexic readers for written language tasks.³⁴⁻³⁶ However, they can be helped by using their strength areas to "work around" their specific disabilities. The ability to implement such therapy is based on proper diagnosis using the methods suggested above. Specific educational therapeutic approaches for the various types of dyslexia have been described in detail,³¹ although additional research is needed in this area.

Of interest is the question of dyslexia and genetics. Reports have indicated that a genetic component seemed to be involved in dyslexia.³⁷⁻³⁹ In recent years evidence has demonstrated that the dyseidetic type of dyslexia follows an autosomal dominant mode of transmis-

sion.⁴⁰ By using the classification system of dyslexia, based on the model presented here, the genetic characteristics of at least one type of dyslexia appear to have been elucidated.

When all types of dyslexia are lumped together and statistical analysis is applied, results are often unclear. Flynn and Deering¹¹ found this to be the case in their EEG brain-function study which compared dyslexic types to non-dyslexics. When the EEG patterns of specific dyslexic types were compared to those of normal readers, significant and substantive differences were found. However, when all individuals with dyslexia were lumped together and compared to the non-dyslexics, the findings were inconclusive. This illustrates an important consideration regarding the characteristics of dyslexia. Dyslexic types should be categorized in future studies. In this way we may continue to learn more about the specifics of dyseidnesia and dysphonia and prevent the occurrence of spurious results from research studies which consider dyslexia to be a homogeneous disorder.

CONCLUSIONS

An updated neurological-behavioral model of dyslexia and its specific types allows for understanding of the problems facing the individual with dyslexia. By employing the principles implicit in this model it is possible to diagnose specific types of dyslexia, counsel affected individuals, recommend appropriate educational therapy, and clearly differentiate dyslexia from other problems which may cause reading disability. When an individual is experiencing a learning or reading problem optometrists are frequently among the first professionals consulted in order to determine if faulty visual functioning is a contributing factor. Consequently, the optometrist often becomes a key person in the co-management of patients with learning disability in general, and dyslexia specifically. When dyslexia and vision problems are detected and diagnosed, co-management with educational therapists and other professions of the multidisciplinary team is necessary.⁴¹

We believe the clinical application of this neurological-behavioral model can help optometrists explain the problem to their dyslexic patients. Consequently,

secondary emotional problems caused by past reading failure can be avoided. Furthermore, by knowing the type and severity of the patient's dyslexia, together with information from the patients and teachers, more effective optometric vision therapy can be provided. For example, a dysphonetic individual relies heavily on eidetic decoding in reading. It, therefore, behooves the optometrist to treat any vision and visual-perceptual-motor problems the patient may have, not only to have average visual skills, but to achieve excellence. On the other hand, a dyseidetic individual relies heavily on phonetics and can benefit from optometric vision therapy to improve such functions as visual sequencing and visual-auditory integration. Consequently, in these ways the vision therapy or enhancement is differentially applied to maximize the patient's reading ability.

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