DYSLEXIA: A BIOLOGICIAL PERSPECTIVE

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Abstract

The purpose of this paper is to provide the behavioral optometrist with an improved understanding of the biological precursors of dyslexia and their effect on therapeutic clinical decisions. The distinctions between dyslexia and reading disability and their prevalences are addressed. In order to understand the pathogenesis of dyslexia, it is necessary to review the normal ontogenesis of cerebral asymmetry and its influence on language development. Special attention is given to the left Sylvian Fissure and the perisylvian region that have been identified as the principal language area in the cerebral cortex. The specific biological abnormalities that lead to dyslexia are explained in terms of the aberrant patterns of asymmetry, symmetry, and cerebral dominance that are characteristic of dyslexia. It remains unclear, however, why this unexpected educational anomaly emerges in some individuals and why others are affected with the more prevalent variety of reading disability. Some of the potential precursors of dyslexia such as autoimmune deficiency, pre- and perinatal disorders, and genetic models are discussed.

Key Words

dyslexia, reading disability, visual handicaps, cerebral asymmetry, planum temporale, autoimmune disorders

Dyslexia is generally understood to be a significant and an unexpected difficulty in learning to read and spell.[1] For the purposes of this paper, dyslexia is limited to individuals who are affected by a special form of inherent biological insult that is constitutional in origin and, ideally, results in a unitary condition.[2] According to Denckla,[3,4] dyslexia is dominated by disorders of verbal learning and language. Spoken language, however, does not present a persistent problem in elementary school when reading acquisition is the problem. Galaburda,[5] whose seminal research is examined below, also has characterized this condition as an auditory-linguistic deficiency. Although dyslexics usually are not learning-disabled and have average or better than average intelligence, their reading performance may be a standard deviation below prediction.

In summary, dyslexia is described as a severe, albeit specific, disability in reading and is characterized by normal intellectual capacity and disorders in language processing. The acquisition of reading skills is slow and laborious, and the results of most remedial educational programs have not been effective for individuals with this condition.

Behavioral optometrists should not confuse dyslexia with the more commonly encountered reading disability (RD). Dyslexics comprise 3% or 4% of the elementary school children,[6] while RD affects 15% to 20% and is included under the umbrella of learning disabilities (LD).[7] RD has many antecedents. It is often accompanied, in various degrees, by other academic disorders, auditory, visual, developmental, perceptual, and visual-motor disabilities, and secondary emotional problems. Because many optometrists are involved in treating children who have been identified as either dyslexic or reading-disabled, this distinction is important in completely defining and understanding the child's academic problem and in determining an optimal treatment plan and prognosis. An example is the dyslexic child who has been receiving individual reading instruction but continues to make poor progress in reading, although vision therapy appears to be ameliorating a concomitant visual dysfunction. The optometrist should become quite concerned about the lack of transfer. At the very least, consultation with the school psychologist is warranted.

Educators and clinicians have found that dyslexics fail to learn to read in spite of at least average intelligence,[a] abundant opportunities to learn, normal auditory and visual acuity, the absence of frank brain damage, no primary emotional disturbance, and normal culturation.[8] Although visual handicaps usually are excluded as primary determinants, visual problems sometimes are contributory since they could impede the dyslexic individual's ability to respond to educational intervention. However, although dyslexia may occur concomitantly with visual and other handicapping conditions, the visual dysfunction most often does not account for the depth of the reading problem that exists. In this paper, I shall discuss this

relatively small special group, the 3% or 4% of the total school population identified as dyslexic.

The precise details of the biological substrates that lead to the pathogenesis of dyslexia are not perfectly understood. However, what is known at present about these substrates is useful in understanding why this "unexpected" educational anomaly emerges in some individuals and why the usual pedagogical interventions often fail. Moreover, research in this area lends support to the notion that dyslexia is not solely the product of developmental delay, but rather is the result of specific biological aberrations. In order to understand aberrant development, however, a knowledge of normal development is necessary.

Normal Development of Cerebral Asymmetry

Normally, the acquisition and control of language are lateralized in the left hemisphere. Incomplete cerebral dominance, ill-defined laterality, deficits in lateral awareness, and directionality are known to be common in dyslexics. Consequently, post-mortem histological brain studies and animal research that investigated the anatomical correlates to cerebral dominance were a logical first step. Anatomical evidence of cerebral asymmetry is a prominent characteristic that could have an impact on reading.[9]

In the normal human brain an easily detectable asymmetry, the *planum temporale*, located in the Sylvian Fissure, is observable in the fetus and the neonate. The planum temporale constitutes a substantial portion of the temporal-parietal speech and language region. The contiguity of the planum temporale to Heschl's gyrus, the primary auditory area, and Wernicke's area that mediates the understanding of language is illustrated in Figures 1a and 1b. Although the left hemisphere develops more slowly than the right, the left planum temporale is normally larger and has a greater number of neurons than the homologous area in the right hemisphere. Connected to Wernicke's area by the *arcuate fasciculus* is the frontal speech (Broca's) area. Its function is to actuate the spoken word and to monitor speech.

Ordinarily, neural development is orderly and sequential. After the development of the neural tube and the period of cell proliferation, cells migrate to their permanent locations from the 8th through the 36th week. Cellular differentiation and maturation continue to take place and connections are established well into the postnatal period. The earlier developing right hemisphere usually produces neurons in the prenatal period in excess of the number of available synaptic sites for the neurons to target.[6] When an intrahemispheric or contralateral neuronal connection is not completed, a process of cell death takes place. This asymmetrical reduction of the excess neurons in the right hemisphere during corticogenesis contributes to the normal development of left hemisphere dominance. However, since the right hemisphere matures prior to the left, certain adverse conditions could provide an opportunity for it to affect the normal development of the left hemisphere. Any developmental deviation that alters (i.e., increases) the availability of alternative postsynaptic sites for neurons of the right hemisphere, including transcallosal, can result in the reshaping of neuronal circuits and the abnormal development of the right hemisphere.

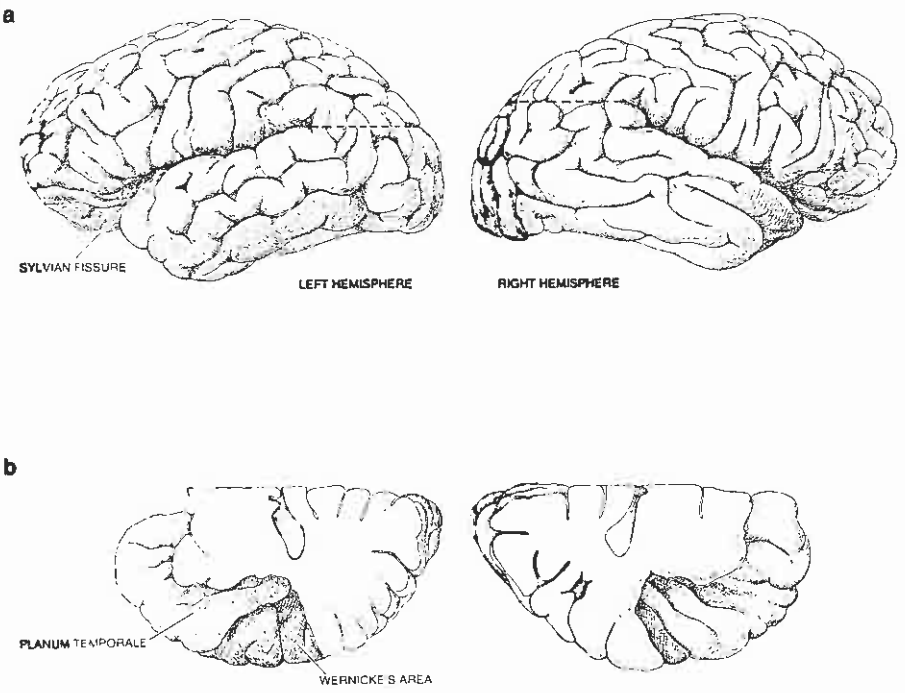

Figure 1a and Figure 1b. Anatomical asymmetry of the human cortex. The Sylvian Fissure, which defines the upper margin of the temporal lobe, rises more steeply on the right side of the brain (Figure 1a). A more striking asymmetry is found on the planum temporale, which forms the upper surface of the temporal lobe, and can be seen only when the Sylvian Fissure is opened. The enlarged region is part of Wernicke's area, the auditory association cortex (Figure 1b).

From: Geschwind N. Specialization of the human brain. Sci Am, 1979; 241 (3): 180-199. (Reprinted with permission.)

Pathogenesis of Childhood Dyslexia

Investigations of the pathogenesis of childhood dyslexia suggest that the size of the left planum temporale is the same as in the non-dyslexic brain, but the size of the right can vary significantly.[5] In the dyslexic brain, the size of the right planum may be equal to the left, making the two symmetrical, and occasionally the right is slightly larger. In the normal brain, asymmetry results from the dominant and nondominant homologous areas having different numbers of neurons. Therefore, we may reason that not only does the absence of asymmetry in dyslexic brains result in a similar number of neurons in the left and right planum, but also a greater total number of neurons than in the nondyslexic brain. What are the underlying mechanisms of these developmental aberrations?

To answer this question, we must first understand which aberrant developmental events may be capable of decreasing cell death. These events cause abnormal changes in cortical structure and the usual

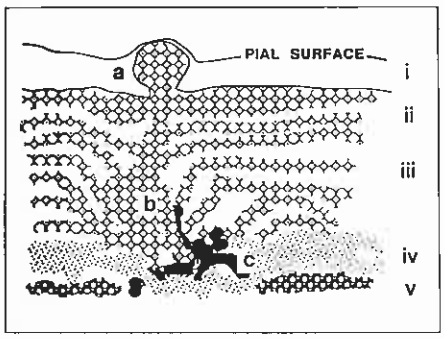

Figure 2. Semistylized, composite diagram of types of cerebrocortical dysgenesis seen in the brain of dyslexics. An ectopic collection of neurons is seen in layer i-a; an associated disorganization of the cortical lamination (dysplasia) is seen predominantly in the upper layers b; abnormal microarchitecture of cortical vessels often accompanies the dysplasia (c). (Reprinted with permission from Galaburda AM. Pathogenesis of childhood dyslexia. In: Language, communications, and the brain. New York: Raven Press, 1988: 127-137.)

Figure 3. Asymmetrical brain areas exhibit side differences in the number of neurons and have a relatively smaller region on one side. Brains with symmetrical cortical areas contain a relatively greater total number of neurons in those areas than brains with asymmetrical cortical areas. (Reprinted with permission from Galaburda AM. Pathogenesis of childhood dyslexia. In: Language, communications, and the brain. New York: Raven Press, 1988: 127-137.)

asymmetry of the brain and, consequently, in cerebral dominance. Research has indicated that these developmental anomalies are the consequences of disordered neural migration primarily to the region of the left perisylvian cortex during the second half of gestation.[10] This microdysgenesis that results in ectopias (abnormally placed neurons) and dysplasia (distorted cortical architecture) rarely is seen in normal brains, but frequently in dyslexics (see Figure 2). They are relatively minor migration defects that, heretofore, have been considered insignificant by neuropathologists since they were not thought to be associated with neurological impairment.

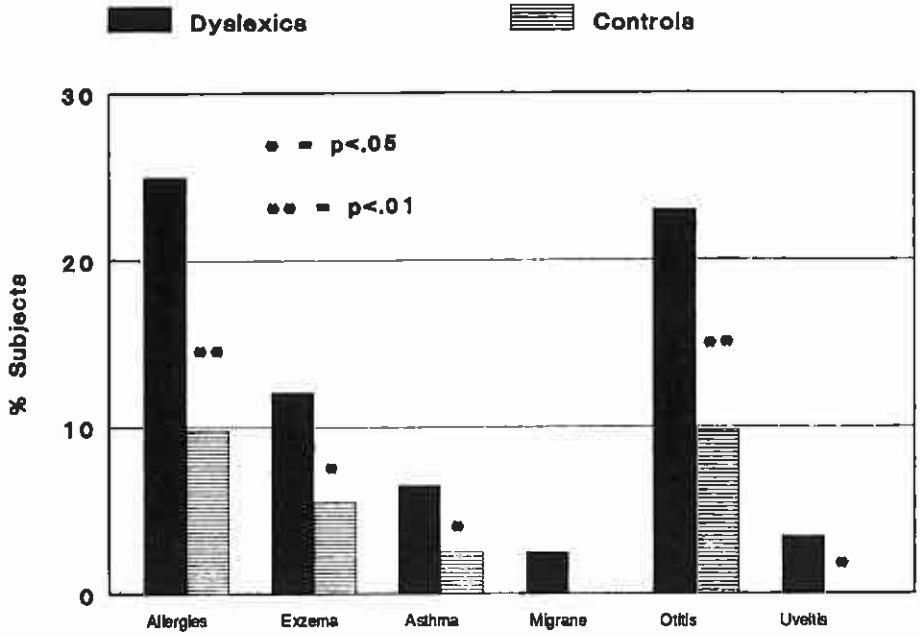

Figure 4. Percent of subjects in the dyslexic and control groups with various immune diseases. Reprinted with permission of the publishers from Dyslexia and Development by Albert M. Galabruda, Cambridge, Mass.: Harvard University Press, Copyright 1993 by the President and Fellows of Harvard College.

Due to the microdysgenesis in the individuals destined to become dyslexic and the delay in left cortical development, many more neurons of the earlier developing right hemisphere cross the callosum and randomly connect with the left hemisphere targets. Therefore, the number of available targets for subsequent left intrahemispheric connections is reduced. Further, the pattern of these random connections is not discreet and produces more "noise" in the system. Each of these factors affects the quality of the functional properties of the left hemisphere.

Consequently, in the intact right hemisphere the competition to establish intrahemispheric connections is reduced. There is diminished cell death, and the size of the right planum temporale is increased. *Symmetry replaces asymmetry* (see Figure 3). The potential for such an individual to become gifted in skills normally attributed to the right hemisphere such as mathematics, music and painting should not be overlooked. Geschwind and Galaburda speak of this as the "Pathology of Superiority."[5 (page 65)] In summary, Galaburda postulates that the focal ectopias and dysplasias reflect insult to the brain in the course of corticogenesis. This leads to rearrangement of cortical architecture of neurons and connections and to the abnormal patterns of asymmetry, symmetry, and cerebral dominance that are characteristic of dyslexia.

Biological Substrates of Dyslexia

We have discussed the distinction between dyslexia and reading disability, as it is understood today, in very general terms. More specifically, we have examined the pathogenesis of dyslexia in terms of Galaburda and Geschwind's hypothesis that the brains of dyslexic individuals reveal the presence of focal areas of cortical malformation and symmetry in a basic language area of the cerebral cortex.[11] It is now reasonable to consider some of the underlying biological substrates that may be precursors of these anomalies.

During the past several years, evidence of an elevated frequency of autoimmune disorders, especially childhood allergies and migraine, has been reported among individuals who have been identified as dyslexic.[12,13] Although the grounds are not clear, extensive research tends to support the theory that a pathogenic linkage exists between cerebrocortical dysgenesis and autoimmune disorders, albeit not necessarily cause and effect (see Figure 4).[5,14,15]

Exogenous causes are also considered to be risk factors in any central nervous system abnormality. Pre-, peri-, and postnatal stress and diseases have been implicated in the etiology of reading disabilities.[11,16] Episodes of staining and hemorrhaging, especially during the first trimester of pregnancy, and extended delivery times that contribute to fetal hy-

poxia have been reported frequently.[16] Prematurity and low birth weight are also noted two to three times more often in disabled than in normal readers.[16] Postnatal episodes, such as convulsions, concussions, dehydration, extended high temperatures, need for oxygen, and chronic ear infections, are not unusual.[16]

Although some investigators are prone to accept a genetic analysis of dyslexia, the current data are not viewed as convincing, largely due to the lack of a good phenotype definition. The explanation of dyslexia that I have presented encompasses a number of entities, each with its own genetic model. They include microdysgenesis, autoimmune deficiency and allergies, cerebral dominance, left-handedness, and certain artistic talents. The variations of expressivity within a family not only make it very difficult to determine the phenotype, but make any effort to elucidate the underlying genotype futile.[17] The geneticist reminds us that what is inherited is not a particular phenotypic trait or character but rather a genotypic potential for an organism's developmental response to the environment. Given a certain genotype and a certain sequence of environmental situations, development follows a certain path. Therefore, a given genotype might well develop phenotypically along different paths under different environmental influences.[18] The observed phenotype variance has both a genetic and an environmental component. Understanding the genetic component contributes to the appreciation of the environmental component and its potential for manipulation (i.e., treatment).

Ultimately, an increased awareness of the biological precursors of dyslexia should enable the behavioral optometrist to intervene earlier with more effective preventive and corrective procedures. Knowledge of the historical factors that place a child "at risk" for dyslexia facilitates the recognition of delays in those developmental conditions that are harbingers of subsequent reading and learning disorders.

Conclusion

Since many optometrists are engaged in treating children who have been identified as reading- and learning-disabled, it is important that we become aware of the biological antecedents of dyslexia. An appreciation of these antecedents can often explain why the successful treatment of functionally-based visual disorders can have different effects on reading ability among optometric patients. Understanding the biological perspectives of dyslexia, hemispheric asymmetry, and brain specialization enables us to appreciate more fully why the diagnosis, management, and remediation of dyslexia are so complex and why the resolution of this problem has eluded researchers and clinicians for more than a century. Clinically, the responsibility of the optometrist is not limited to the treatment of associated visual disorders, but s/he should be prepared to document the presence of a concomitant language disorder and make the appropriate referrals if indicated. We should continue to support a multidisciplinary approach so that each dyslexic child will benefit from the combined professional skills that are available.

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Endnotes

a. Operationally, there is a difference between a child who tests well on IQ and poorly on reading, and the one who tests poorly on both. When one considers the impact of language, reading, and environmental factors on IQ, we are not on firm ground if we explain defective reading solely by low IQ scores.

b. The spatial relations, emotions, and attention areas located in the right hemisphere phylogenetically preceded the development of the language and analytical areas in the left hemisphere. Ontogenetic development often recapitulates this phylogenetic order.

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