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ABSTRACT

Studies on autopsy of the brains of dyslexics and those measuring brain functioning during cognition during the past decade and a half have shed some light on the possible causes of developmental or specific dyslexia. This article briefly reviews some of the most pertinent research and offers some guidelines for the prognosis and treatment of dyslexic individuals and literary options for reading teachers and specialists to use in working with dyslexia.

INTRODUCTION

Authorities have offered a variety of opinions concerning causes of developmental dyslexia as a neurologically based disorder ranging from brain damage to brain maturational lags and everything in between. An early authority, Hinshelwood (1917), a British physician, became interested in dyslexia when he treated a male patient who suddenly lost the ability to read. Hinshelwood learned later that his patient, a school teacher, had a lesion in his left temporal lobe. He then reasoned that children who had difficulty in learning to read might be similarly afflicted, an opinion he propagated in his book, *Congenital Word Blindness* (1917). Those who support brain damage hypotheses include the following causes of brain defects in maternally induced drug or alcohol abuse and anoxia, forced labor, premature birth and high forceps use, during the birth process.

Neurologically based arguments for dyslexia are relative to the acquisition of cerebral dominance. Orton's (1937) well-known hypothesis of "strephosymbolia," or twisted symbols, is related to a condition in which engrams in the two hemispheres vie for recognition thereby causing a reading disability, a position that has been discredited, although dominance enthusiasts still persist.

Rabinovitch (1962, 1968) has maintained that there is some unknown developmental neurological deficit in cases of dyslexia and along with Bender (1957) and DeHirsch and Jansky (1968) has maintained that the case of developmental dyslexia is a delay or slow development of reading related areas in the brain. These hypotheses imply that the ability to read will improve when the afflicted child reaches an optimum age.

Interest in neurologically based causes of reading disability has persisted to the present time. Fortunately, the development of technology has afforded medical researchers the opportunity to study the brain of poor readers in ways never before possible. Current research, then, has provided new insights into the relationship between developmental dyslexia and neuroanotomical finding. The purpose of this paper is to examine some recent medical neuroanotomical findings relative to developmental dyslexia and to present some implications they may have for reading teachers and reading specialists.

More specifically, there are two questions that the paper addresses. First, is there a causal relationship between neuroanotomical disorders and developmental dyslexia? Second, what can the reading teacher or specialist do?

In order to answer these questions we decided to focus mainly on three researchers whose work seem to be particularly relevant. We are aware that there are numerous articles on neuroanotomical findings reflecting keen interest in the topic, but much of the research we have focused on is by Albert Galaburda, M.D., whose work has captured the attention of professionals in several fields (Galaburda, 1989; Sherman, Galaburda and Geschwind, 1985; Galaburda, 1983). We also have focused on the research of Livingstone (Livingstone, Rosen, Drislane, and Galaburda, 1991) and Flowers (1993) because their research has an obvious connection to Galaburda's findings.

In "Ordinary and Extraordinary Brain Development," Galaburda (1989) reported on his findings of 21 human brains, eight of which were from individuals who were alleged to be dyslexics. Significant about Galaburda's research findings is that the eight dyslexic brains shared the same condition in every case, a remarkable incidence of consistency if only for a relatively small number of cases. No definition of dyslexia was offered in the article, although he referred to a definition he used in an earlier article.

> *Although the wording may vary somewhat, most workers in the field will accept dyslexia as a condition manifested by difficulty with learning to read and write efficiently despite the presence of normal intelligence, adequate educational opportunities, and normal psychiatric makeup. Most definitions exclude individuals with major sensory deficits. Between five percent and fifteen percent of the school-age population in the United States fit this definition (Galaburda, 1985, p. 22)*

One can infer that dyslexia has a constitutional basis, a position held widely by the medical community.

Galaburda offered an explanation as to why the brains of dyslexics must be different from the brains of non-dyslexics in an earlier article (1983, p. 46) by describing the process of cell migration during the early development of humans. He pointed out that the development of the cortex or outer layer of the brain takes place in stages. Early in brain development, certain nerve cells develop in clusters, or germinal zones. These immature cells, or progenitor neuronal cells, later migrate to their final positions in the cortex or subcortex. Once mature, these cells assume very specific functions vital to normal brain functioning. At the time certain neuronal cells mature, other neuronal cells in the cortex die off in large numbers while the mature cells survive. The net result of the opposing processes of selective cellular drop-out and cellular maturation cause asymmetry in the sizes of the brain cortexes of non-dyslexics. Upon microscopic analysis of dyslexia brains, Galaburda found that the neurons that would normally drop out remain viable. He also noted that large groups, or nests, of neocortical cells were dumped in superficial layers or clumps on the cerebral cortex, instead of migrating in the specific pattern found in non-dyslexic brains. A resultant effect of this aborted migration was the failure of the neuronal cells to exert an inhibitory influence on other nerve cells. If migration had occurred, the traveling neurons would have settled in close proximity to other types of neurons, thereby causing an inhibitory influence on them. He concluded, then, that possibly for this reason the dyslexic brains had more nerve cells than the asymmetric but normal brains of the non-dyslexics.

Galaburda has had reason to rethink the effect of symmetry on dyslexia because of additional research conducted by Steinmetz and Galaburda (1991). The researchers reported that study of eight normal reading left-handed individuals showed symmetry in the brains of these individuals similar to the symmetry found in the brains of dyslexics studied earlier (Galaburda, 1989). Steinmetz and Galaburda concluded on the basis of the new information that planum symmetry alone could not account for the presence of dyslexia. Additionally, Galaburda's (1989) findings also centered on the corpus collosum, the band of fibers connecting the two hemispheres. The corpus collosum in dyslexic brains was much larger than the corpus collosum in non-dyslexic brains. One possible explanation for this phenomenon is that in dyslexics the brain tries to adjust for a deficiency in one hemisphere by making extra connections in the other resulting in a larger cell mass in the corpus collosum.

Although, by his own admission, causal conclusions are premature, it appears to be obvious from Galaburda's research that the anomalies found in dyslexic brains are themselves caused by a variety of factors. Sherman, Galaburda, and Geschwind, (1985) reported a possible cause of abnormal development in dyslexic brains. They

noted that immune deficient mice exhibited an abnormal development found in humans with developmental dyslexia. The mice brains were scarred from a condition called lupus, an autoimmune disease that causes inflammation of the blood vessels resulting in swollen vessels and occlusion of normal blood flow. The link between the condition of the mice and human beings is that women with active lupus have given birth to dyslexic children. One child who died with juvenile lupus and dyslexia had the same type of scarring found in immune deficient mice. Furthermore, the same type of scarring was found in the brains of three dyslexic women. The link between these clinical findings is that immune deficiency states may result in autoimmune diseases such as lupus, which are more commonly found in women. Immune regulation, lacking in lupus patients, involves complex processes occurring at the molecular genetic level.

When developmental dyslexia is analyzed from a molecular genetic viewpoint, many possibilities for causes of developmental dyslexia come to mind. Any number of random mutations during or prior to embryonic development could result in the type of arrested neocortical brain development seen in dyslexia. Similarly, mutations of this sort could be transmitted as an inherited trait. Finally, various pathophysiologic processes occurring during the final stages of brain development, ranging from lupus to temporary brain anoxia, could alter or destroy the molecular-genetic blueprint or machinery necessary to complete neocortical development.

The findings of brain abnormalities and deleterious effects of autoimmune diseases on neocortical development do not necessarily explain their connection to developmental dyslexia. Recently, however, the research of Livingstone, et al. (1991) revealed a possible link between the two entities. Livingstone's work reported in the Proceedings of the National Academy of Sciences (1991) involved the sense of vision whose role in dyslexia has been discounted by vision specialists having found no differences between the eyes of dyslexics and normal readers. Livingstone reported that it is likely that dyslexics process visual information more slowly than normal readers. She added that dyslexics also have trouble distinguishing between the order of two rapidly flashing visual stimuli but perform normally on tests with prolonged presentation of stimuli. Autopsies on five dyslexic brains and five non-dyslexic brains concluded that one of the two major visual pathways, the magnocellular system, malfunctioned in dyslexic brains. This system is composed of large cells which carry out fast visual processes and was more disorganized and consisted of smaller cell bodies than the magnocellular system in non-dyslexic brains. The magnocellular systems are used for perceiving motion, depth perception, low contrast, and locating objects in space. The parvocellular system composed of small cells that carry out slower visual processes, were similar in both types of brains. The parvocellular system specializes in color perception, details of forms, perceiving stationary images, and recognizing high contrasts.

The connection between Livingstone's, et al., (1991) work and Galaburda's (1989) may reside in the findings of research scientists who found that animals form antibodies that destroy a protein peculiar to the magnocellular system thereby causing the magno system to respond sluggishly. Galaburda interprets this finding as evidence that dyslexia might be an autoimmune disease acquired congenitally.

Flowers (1993), interested in constitutional causes for dyslexia, reported on the structure and physiological aspects of dyslexia relating her findings to core left-hemisphere language functions. She found that by measuring brain function during cognition, there was a left hemisphere deficit similar to Galaburda's conclusions concerning left-hemisphere deficits in his samples. Unlike Livingstone, et al., (1991), who ascribed the disability to a possible mal-timing in the visual system, Flowers identified patterns for the individuals she studied presenting evidence of disability associated with phonological awareness. Livingstone, et al., (1991) stated that although many authors have argued that dyslexia is a linguistic rather than a visual problem, linguistic defects may be related to perceptual difficulties. However, Flowers' (1993) work may indicate that dyslexic children may also have problems with transitions inherent in auditory phonemic discriminations as well.

CONCLUSIONS OF NEUROANOTOMICAL RESEARCH

The findings from the studies we have reported raise several questions. First, do the data show a causal relationship between neuroanotomical disorders and developmental dyslexia? Second, what can the classroom teacher/reading specialist do?

Is there a causal relationship? At the present time we don't know. Even though the evidence of symmetry and disorganization between dyslexic and non-dyslexic brains that have been studied is impressive, we don't know if the pathology reported is the cause of dyslexia or if both the brain abnormalities and the dyslexia are the result of some underlying cause. Furthermore, one might argue that there are too few cases from which to generalize. However, Duane (1989) stated that in the psychological literature, large numbers of cases to interpret phenomena are important; but in neurology, "... a single well-studied brain can provide insights into broad mechanisms of function and dysfunction" (p. 219). Galaburda (1989) analyzed eight dyslexic brains, six males and two females, with all males showing numerous malformations of the cerebral cortex in language relevant regions and to some extent bilaterally. The degree of consistency among Galaburda's findings across the dyslexic brains is compelling, but a direct causal relationship has not been established in spite of these compelling findings. Questions about the causal effect between the possible deficiencies in the visual system of dyslexic children reported in the vision studies by Livingstone, et al. (1991) and the phonological system reported by Flowers (1993) also exist.

WHAT CAN THE READING TEACHER DO?

Although the evidence, including the personal experience of the authors, strongly indicates that the developmental dyslexics have undue difficulty attaining the kind of automatic word recognition that would make them facile readers, the reading teacher or remedial specialist does have some options.

> *1. The use of multisensory approach is indicated. First, dyslexic individuals usually have difficulty learning under any circumstances; however, the conventional visual-auditory approaches that work for most children do not alone produce the required results — that is, helping the child make the automatic responses to letters and words. It may be that the conditions described by Galaburda (1989), who reported structural and developmental anomalies; Livingstone, et al., (1991) who reported visual processing anomalies; and Flowers (1993) who reported phonological anomalies, are causes of the inability of the dyslexic individual to profit solely from visual-auditory programs. Unfortunately, these programs are the substance of basal programs or literature based programs which supplement with phonics-structural analysis approaches.*

One technique that has produced some results is the use of the VAKT technique. VAKT stands for visual, auditory, kinesthetic, tactile. The technique was first popularized by Grace Fernald (1943) in her text *Remedial Techniques in Basic School Subjects*. She explained that the task of learning to decode includes the presentation of word forms is made in a variety of ways and is basic to conventional approaches that combine visual discrimination exercises with their auditory counterpart. Auditory methods include learning the sound of the alphabet (although not every proponent agrees on this requirement); repeating phonograms, associating the phonograms with orthographic or visual symbols and so on. It doesn't matter if synthetic or part-to whole phonics is used, the approach is essentially auditory supplemented by visual associations. To the two conventional senses, Fernald adds the kinesthetic and tactile. Fernald (1943, pp. 26-27) points out that if one uses material that the child understands conceptually, the word forms for these concepts can be taught by some kinesthetic technique. Kinesthesia involves eye movements, lip-throat movements, and hand kinesthetic movements. The latter specifically are involved in the tactile use of the fingers in tracing the letters and words. The teacher, at first, guides the child's hand, helps him to draw the letters while looking at the whole word, saying it as he traces it and then repeating the process while writing the word from

memory. In this manner, including all four methods, the child can and does learn to read usually to a more efficient degree than when conventional visual-phonics programs alone have failed to produce positive results.

VAKT is usually used at first in conjunction with an experience approach which draws upon the child's background of experience and the child then learns to recognize the word forms that represent the concepts he already knows. There is no vocabulary control nor lock-step learning involved. In cases of partial disability, VAKT has been especially useful. With cases of developmental or specific dyslexia, VAKT has produced learning to read, even if to a more limited extent than with cases of partial disability, but superior to the results affected by conventional phonics-structural analysis approaches.

2. Another method useful in teaching reading-disabled students is espoused by the Orton Institute. The Institute recommends the Gillingham-Stillman approach (1963) which uses VAKT but presents the technique in a much more formal and incremental way than VAKT alone. Although the approach places a heavy emphasis on VAKT, conventional phonics and structural analysis learning is an important part of the program.

The validity of the two methods described above lies in the fact that reading specialists have used the approaches with positive results with poor readers for over fifty years when conventional methods have failed. Harris and Sipay (1990) stated that some research concerning the effects of the Fernald or VAKT method has produced positive results in individuals who have repeatedly failed to learn to read (pp. 500-502). They added, however, that research on Orton-Gillingham approach is extremely limited (p. 504). For our experience, we found VAKT alone yields positive results without the highly formal instruction and lengthy training required by Gillingham-Stillman. It may be more feasible to use the VAKT although proponents of Gillingham-Stillman strongly advocate their approach.

3. If Livingstone's (1991) work has any meaning for reading instruction, it may be that her research on the visual system has some implications for Irlen's (1991) work. Irlen has had some success with cases of reading disability in using colored filters in conjunction with reading instruction.

4. Another new , albeit controversial remedial approach, is embodied in the work of Tallal. Nash (1996) reported that Tallal has presented data that have linked dyslexia with deficits in the rate with which dyslexic children process rapidly presented sensory arid motor information. Tallal suspects that some types of dyslexia

may stem from their inability to process auditory information rapidly enough. Tallal has developed computer-based programs that use animated video games. The basis of the therapy program is a speech processing program that permits the researchers to slow down the speed of auditory clues so that the dyslexic children can process them thereby enabling the children to learn the requisite decoding skills.

It is interesting to note that Livingstone's (1991) work focuses on the visual system while Flower's (1993) work focuses on auditory functioning, because the two remedial approaches reported above by Irlen and Tallal involve visual and auditory training respectively. There may be some credence to the belief that there are different kinds of dyslexias.

Additionally, teachers can tape lessons from textbooks that dyslexic students are required to read thereby enabling them to learn the material despite the fact that they cannot decode the text. Teachers can also pair a dyslexic child with an effective reader who can read to his/her classmate and discuss the requisite material. If a school can subscribe to the services of the Books for Blind Association, any text can be taped for the student and the teacher can select taped texts from among thousands of titles that are presently available. The point is, then, that while the reading-decoding avenue to learning is in the process of improving, children can learn the required content subject information. Certainly, teachers can use oral tests (recorded tests) and untimed tests that are better suited to the reading disabled child.

Perhaps the day will come when science will do more than tell us what is wrong and effect changes in basic human structures like the brain that will prevent conditions like dyslexia. Until that day arrives, we may be the only hope for the dyslexic child — a responsibility we do not take lightly.

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