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Neuropsychology and Specific Learning Disabilities: Lessons from the Past As a Guide to Present Controversies and Future Clinical Practice

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Learning disabilities and neuropsychology have always been intertwined, even before Ralph Reitan put neuropsychology on the map in the 1950s or Sam Kirk coined the term *learning disabilities* in 1963. The history of specific learning disabilities (SLDs) is steeped in the tradition of brain damage and brain dysfunction, whether one traces the roots of SLD to the perceptual processing disorder approach of Kurt Goldstein and Alfred Strauss or to the developmental language disorder conceptualization of Samuel Orton and James Hinshelwood (Shepherd, 2001). And if the past endorses the strong relationship between SLDs and neuropsychology, that endorsement is no less powerful than the impact of present research or future applications of technology on the essential role of neuropsychology on the assessment of SLD.

The history of SLD is not a linear or chronological one but rather an uneasy amalgam of two traditions that are conceptually distinct and seemingly resistant to integration. The Goldstein-Strauss-Werner history—based initially on Kurt Goldstein’s (1942) studies of the perceptual, cognitive, attentional, and mood disorders of soldiers who sustained head injuries—emphasizes disorders of perception, especially visual perception. Indeed, it is the deficit in perceptual processing that is considered the specific learning *disability* (there is no room in this model for specific learning *disabilities*). However, a different history of SLD that predates Goldstein first began

appearing in Europe in the 1890s with accounts of an adult patient who lost the ability to read following a stroke, though he could speak and write fluently, remember details, and understand easily (Dejerine, 1892); and accounts of a 14-year-old nonreader, Percy F.: “I might add that the boy is bright and of average intelligence in conversation. . . . The schoolmaster who has taught him for some years says that he would be the smartest lad in school if the instruction were entirely oral” (Morgan, 1896, p. 1,378). This tradition, popularized by Orton and Hinshelwood, produced an impressive literature following Dr. Pringle Morgan’s 1896 account of Percy, which depicted clear-cut cases of individuals with learning disabilities specific to reading and writing (e.g., Kerr, 1897; Morgan, 1914) and later on specific to arithmetic (Schmitt, 1921). Hinshelwood (1917) believed the problem to be a congenital lesion in the left angular gyrus, which impaired the ability to store and remember visual memory for letters and words; Orton (1937) hypothesized a functional brain disorder associated with the inability of one hemisphere to become dominant over the other for handling language, but he nonetheless “accepted the notion of the origin of dyslexia in the angular gyrus region” (Spreeen, 2001, p. 285). Both agreed that SLD was a function of a developmental disorder of written language.

Occasionally, neuropsychologists who write about the history of SLD blend the Goldstein-Strauss-Werner tradition with the Hinshelwood-Orton approach: “Orton’s theory remained a theory until, in 1947, Strauss and Lehtinen called attention to the frequent appearance of neurological signs in learning-disabled children” (Spreeen, 2001, p. 286). But usually the two traditions are treated separately.

Indeed, the two historical roots of SLD could not be more different in conception, origination, or research methodology. Yet they converge in their basic premise that neurology and neuropsychology are the keys for understanding learning problems and ultimately treating them. Even the founding fathers of the developmental language disorder approach, while relying on a field of neuroscience that was in its infancy, did not agree on the neurological causation of the problem. Yet the fact remains that, regardless of the orientation of the early SLD pioneers, and regardless of whether one’s intuitive understanding of SLD is more aligned with a specific perceptual disorder or an array of specific disorders in language development, all paths to the present field of SLD come through the fields of neurology and neuropsychology.

THE GOLDSTEIN-STRAUSS-WERNER VISUAL PERCEPTUAL APPROACH TO SLD

The Goldstein-Strauss-Werner theory posited that a disorder of visual perception, along with the concomitant attentional problems, impairs learning on tasks that depend on perception and attention. Fix the perceptual disorder

of these brain-damaged individuals (in a learning environment that reduces distraction and inattention), and you have fixed the learning problem (even Mental Retardation). Goldstein's student, Alfred Strauss, extended his mentor's work to mentally retarded adolescents and observed the same kinds of perceptual, mood, and learning disorders in this low-IQ population that Goldstein had found with head-injured soldiers (Strauss & Werner, 1943). These researchers attributed the disorders to brain damage and concluded that (a) there was a difference between Mental Retardation caused by brain injury and Mental Retardation that was familial, (b) brain injury produced specific perceptual and behavioral deficits, and (c) special education aimed at treating the observed perceptual and behavioral problems would be effective with Mental Retardation due to brain injury but not due to inheritance from parents. Strauss worked with an educator, Laura Lehtinen, to implement the perceptual training (Strauss & Lehtinen, 1947), emphasizing the point that remediation of learning and behavior problems worked hand-in-hand with identification of learning and behavior problems from the inception of the perceptual disorder movement.

The next logical step to extend the theory was to study children, not just adolescents and adults, and to investigate children with normal or near-normal intelligence. These studies included children with known brain damage, such as cerebral palsy (Cruickshank, Bice, & Wallen, 1957), and, intriguingly, samples of children who evidenced learning and behavior problems *but did not show clinical signs of brain damage* (Strauss & Kephart, 1955)—that moved the field forward in a dramatic way. Goldstein, Strauss, Werner, Cruickshank, and Kephart were the pioneers who established the concept of a learning and behavior disability caused by *minimal brain dysfunction* (i.e., not detectable through standard clinical procedures, but brain injury nonetheless) that was distinct from Mental Retardation.

Lehtinen's early work suggested that remediation of the perceptual disorders was feasible, and a plethora of visual-perceptual-motor training programs began to predominate in the 1960s, with names like Frostig, Ayres, Getman, Kephart, and Barsch associated with different methodologies on the same theme. However, subsequent systematic reviews of 81 research studies, encompassing more than 500 different statistical comparisons, concluded that "none of the treatments was particularly effective in stimulating cognitive, linguistic, academic, or school readiness abilities and that there was a serious question as to whether the training activities even have value for enhancing visual perception and/or motor skills in children indicated" (Hammill & Bartel, 1978, p. 371). Yet, this lack of research support did not stop the visual training in the schools and it did not slow down the movement that endorsed learning disabilities (usually known then as *minimal brain dysfunction* or *perceptual disorder*) as problems with perception (usually visual but sometimes auditory). In fact, several influential special educators who have studied SLD history (e.g., Kavale & Forness, 1995; Torgesen, 1998)

believe that the Goldstein-Strauss-Werner “view influenced the definition of ‘specific learning disability’ in federal laws and also influenced U.S. public school practices” (Shepherd, 2001, p. 5).

THE ORTON-HINSHELWOOD DEVELOPMENTAL WRITTEN LANGUAGE APPROACH TO SLD

Like the Goldstein-Strauss-Werner approach, the Orton-Hinshelwood view of SLD had its roots in the learning problems and behaviors of adults with brain damage. Rather than focusing on war-injured soldiers, the developmental language pioneers were impressed by late-nineteenth century accounts of adults in Great Britain, France, and Germany who suffered known brain damage to specific regions of the brain and lost the ability to read—despite retaining writing and spelling skills (Shepherd, 2001). In the early part of the twentieth century, the accounts began to include children who were seemingly normal with no overt signs of brain damage, but (like the brain-damaged adults) had a specific disability in reading, writing, or arithmetic despite normal abilities in other areas of cognition and achievement. Though Hinshelwood (1895), an ophthalmologist, initially focused on acquired word blindness based on an adult patient who could not read subsequent to injury to the angular gyrus, he was impressed by Morgan’s (1896) first reporting of congenital word blindness in children. He became intrigued by subsequent accounts published by physicians (including himself) of 14 cases in Europe and North America of children and adolescents with reading disorders that were apparently congenital and not due to any known brain injury (Spreen, 2001).

This accumulation of clinical cases impelled Hinshelwood (1917) to publish a widely read monograph, *Congenital Word Blindness*, that included detailed descriptions of these children, such as a boy of 12 who was brought by his mother to have his eyesight checked: “He could barely read by sight more than two or three words, but came to a standstill every second or third word. . . . [But he] read all combinations of figures with the greatest of fluency up to millions” (p. 21). And just as Sally Shaywitz (2003) insists that the diagnosis of dyslexia is no less accurate or science-based than nearly any other medical diagnosis, Hinshelwood (1917) said virtually the same thing about a century earlier—that it’s fairly easy to diagnose congenital word blindness because the condition is as clear-cut and distinct as any other medical pathology.

Orton (1937) coined the term *strephosymbolia* (twisted symbols) to describe what later came to be known as *dyslexia*. He provided excellent clinical descriptions of children with reading disorders who, he observed, had special difficulty with letter and word reversals—the kinds of transpositions that suggested to Orton that these children read from right to left. He was

a firm believer in thorough assessment, including the recording of extensive family and school histories and the administration of IQ and achievement tests. He was especially interested in children's performances on different areas of academic achievement to confirm his belief that children with a reading disability would score lower on reading and spelling tests than on arithmetic tests; and that children with writing disabilities would score lower on tests of spelling than on arithmetic tests. Orton did not feature disorders of mood or attention as aspects of the learning disability (as did the perceptual theorists), but he noted that many of his patients with reading disorders also had speech and motor disorders; they were predominantly male; they tended to have life-long difficulties with academic skills; and he often treated several members of the same family.

Hinshelwood (1917) limited the diagnosis of congenital word blindness to those who demonstrated the gravity of the defect and evidenced a purity of symptoms, but he excluded children who were just a bit slow in acquiring reading skills. Orton's definition was not as stringent: "Our experience in studying and retraining several hundred such cases has convinced us that they form a *graded series including all degrees of severity of the handicap*" (Spreen, 2001, p. 285).

Hinshelwood advocated assessment methods that were remarkably similar to Orton's and they both strongly favored remediation that was targeted directly at the academic problem. For example, they both emphasized a phonics approach to teaching reading, differing only in Hinshelwood's preference for teaching sound to letter correspondence versus Orton's method of teaching letter to sound correspondence (Shepherd, 2001). As with the perceptual disorder theorists, neurology was believed to be at the root of the learning problem (brain damage to Hinshelwood and failure to establish dominance to Orton). However, the brain damage or dysfunction was tied directly to the specific language disorders that the children displayed—not to a single process such as visual perception. And contrary to the Goldstein-Strauss-Werner theorists, remediation was aimed at improving the specific area of learning deficit (such as spelling or reading), not at strengthening a supposed underlying process. Both theories of the historical roots of SLD emphasized developmental disorders, but these brain-related disorders were either perceptual in nature (Goldstein-Strauss-Werner) or associated with written language (Hinshelwood-Orton)—the distinction between minimal brain dysfunction and developmental dyslexia, respectively.

SAM KIRK'S INTEGRATION OF THE TWO MODELS

Kirk (1963) coined the term *learning disabilities* when he delivered a speech to a large group of parents whose children were having school difficulties and to a smaller group of professionals with a keen interest in the topic. All were

seeking a label for these children that Kirk referred to as having developmental deficits of one kind or another (which encompasses developmental disorders of both perception and written language). Kirk's label had a decided educational flavor, focusing on the nature of the problem rather than the hypothesized cause, and it was the precursor for the federal definitions and laws of the late 1960s and 1970s that proclaimed *specific learning disabilities* as a disorder that entitled special education services to anyone with an SLD diagnosis. When reading the text of Kirk's (1963) speech, it is clear that his notion of learning disabilities was more aligned with Hinshelwood-Orton than Goldstein-Strauss-Werner as he referred to "a group of children who have disorders in development in language, speech, reading and associated communication skills needed for social interaction" (p. 3). However, like the perceptual theorists, Kirk stressed that the disorder involved a processing disorder. But, unlike those theorists, he believed the processing disorders to be *psycholinguistic* in nature, not visual perceptual. He believed that these psycholinguistic disorders led directly to disorders in reading, language, and so forth—an approach that is consistent with the Hinshelwood-Orton belief that brain damage or brain organization is related specifically to written language disabilities. Kirk, however, was more consistent with the perceptual theorists regarding his model of remediation: He believed that a child's weak psycholinguistic processes (as measured by his Illinois Test of Psycholinguistic Abilities, described in his 1963 speech, but not published until 1968) needed direct remediation in order to treat a child's learning disability. Unfortunately, subsequent research on the effectiveness of psycholinguistic training yielded the same dismal conclusions that were reached for perceptual training (Newcomer & Hammill, 1976).

THE FEDERAL DEFINITION OF SLD

The definition of SLDs that was inaugurated in the Children with Specific Learning Disabilities Act of 1969 was retained in the Right to Education for All Handicapped Children's Act of 1975 and has remained intact for IDEA 1997 and IDEA 2004. The first part of this definition is as follows:

The term "specific learning disability" means a disorder in one or more of the basic psychological processes involved in understanding or in using language, spoken or written, which disorder may manifest itself in imperfect ability to listen, think, speak, read, write, spell, or do mathematical calculations.

This definition is clearly a derivative of Kirk's approach to the disorder, but when it is related to the two separate historical roots of SLD it is unquestionably the voice of Goldstein-Strauss-Werner, not Orton or Hinshelwood. Processing disorders had no role in the notion of developmental disorders of written language.

The second part of the definition is an amalgam of the two historical traditions:

DISORDERS INCLUDED—Such term includes such conditions as perceptual disabilities, brain injury, minimal brain dysfunction, dyslexia, and developmental aphasia. (*Federal Register*, 2006)

The terms *perceptual disabilities* and *minimal brain dysfunction* are associated with Goldstein and Strauss, whereas the terms *dyslexia* and *developmental aphasia* are Hinshelwood-Orton concepts. The definition is literally built by committee, which undoubtedly accounts for much of the controversy that has hounded the field of SLD from its inception and that has grown exponentially over the past decade.

Part of this controversy concerns the need to identify a processing disorder as part of the diagnostic process (Hale et al., 2004), a mandate of the IDEA 2004 SLD definition that tends to be ignored or trivialized by those who favor a Response-to-Intervention (RTI) only approach for diagnosing SLD (e.g., Gresham, 2002). Interestingly, processing disorders are part of the SLD definition from the Goldstein-Strauss-Werner perspective, but, as noted, such disorders play no part in the Hinshelwood-Orton definition. Therefore, from a historical perspective, the necessity of identifying a processing disorder receives only mixed support.

However, with history as a guide to practice, the need for neuropsychological assessment as part of the diagnostic process receives broad-based support. Regardless of the tradition with which one identifies most closely, history is unanimous in associating brain damage, brain dysfunction, or brain organization with SLDs. Whether or not the problem is a disordered process or a kind of brain dysfunction specifically associated with reading, writing, or arithmetic, neuropsychological assessment is necessary to better understand the individual's learning disability and to treat it. Kirk did not specifically endorse the neurological basis of SLD, but he did endorse the need for assessment: "The concept of learning disability as used in education does not deny or reject a neurological deficit. . . . The major emphasis is on the use of psychological tests and/or observation for the purpose of organizing a remedial program. Such a program is . . . very dependent upon the determination of psychological abilities and disabilities" (Kirk & Kirk, 1971, pp. 12–13). Indeed, all historical approaches to SLD emphasize the spared or intact abilities that stand in stark contrast to the deficient abilities, as well as the necessity of developing a remedial program based to some extent on test results. Although the programs designed to remediate perceptual and psycholinguistic processes have not proven effective either to improve the disordered process or "cure" the learning disability, there is a growing body of neuropsychological literature that supports neuropsychological assessment both to map the areas of the brain that are associated with specific

aspects of the reading process and to inform intervention (Shaywitz, 2003; Spreen, 2001).

NEUROPSYCHOLOGICAL RESEARCH AND SLD

Neurology and neuropsychology have been intimately associated with SLD for more than a century. The wealth of neuropsychological research that has blossomed steadily since the early case studies of head-injured soldiers with mood and perceptual disorders (Goldstein), and of adults and adolescents with specific reading disabilities (Hinshelwood), makes it imperative that neuropsychological assessment retain that intimate link to SLD diagnosis and treatment. However, from a historical perspective, it is also true that much of the association between brain damage or dysfunction and SLD has been by presumption and implication. The clinical cases of adults with reading disorders following known brain injury provided hard data of a link between brain damage and SLD. But the dozens of clinical reports of children or adolescents with so-called congenital disabilities were based on the assumption of central nervous system dysfunction (i.e., soft data, not hard science; Benton, 1982). As recently as a decade ago, the National Joint Committee on Learning Disabilities (1998) emphasized that SLD was intrinsic to the individual and *presumed* to be caused by CNS dysfunction. Given the behavioral orientation of the RTI movement during the first decade of the twenty-first century, it is important to ask whether the scientific data now support a hard link between neuropsychology and SLD or the relationship remains presumptive.

Initial evidence of a neurological link focused on soft signs (such as poor motor coordination, left-right confusion), which were more prevalent in SLD than normal populations (e.g., Hertzog, 1983). Though these soft signs were often criticized because they were developmental in nature and disappeared over time, data suggested otherwise: Spreen (1988), in the Victoria study of 203 children diagnosed with SLDs, showed that soft signs observed at ages 8 to 12 years persisted or even increased through age 25. Despite Spreen's findings of the stability of soft signs, this line of research proved a virtual dead end because soft signs "rarely point to specific locations in the cortex" (Spreen, 2001, p. 286). More compelling data came from autopsy studies of a total of six individuals with dyslexia (Drake, 1968; Galaburda & Kemper, 1978; Humphreys, Kaufmann, & Galaburda, 1990), which consistently showed "dyslexic brains" to differ from normal brains: "The autopsy studies showed microdysgenesis with ectopias and dysplasias bilaterally along the Sylvian fissure frontally and along the planum temporale, in the left more than in the right hemisphere" (Spreen, 2001, p. 287). However, computerized tomography (CT) studies have only occasionally supported the asymmetries reported in the autopsy studies, and have sometimes contradicted

the findings, for example, when age and brain size are controlled (Schultz et al., 1994); reviews of the CT literature do not consistently support asymmetry of the plana in individuals with dyslexia (e.g., Morgan & Hynd, 1998). Another line of research suggested corpus callosum abnormalities in adults with dyslexia (e.g., Duara et al., 1991), but a review of pertinent studies indicated that the results have not been replicated across samples of individuals with dyslexia (Beaton, 1997).

Different avenues of research have explored *functional* abnormalities in children with SLDs, using positron emission tomography (PET), single-photon emission tomography (SPECT), functional magnetic resonance imaging (fMRI), and electrophysiological (EEG) techniques, an advance over the strictly *structural* abnormality approach of the CT scan studies (Bigler, Lajiness-O'Neill, & Howes, 1998; Spreen, 2001). The metabolic imaging studies have provided intriguing results based on changes in blood flow and blood oxygenation while individuals with and without dyslexia are performing specific reading tasks such as phonological processing, lexical-semantic processing, orthographic-visual processing, auditory processing, and so forth (e.g., Shaywitz et al., 1998; Rumsey et al., 1997). These studies have helped localize the brain areas involved in each aspect of reading and have identified differences in processing between normal individuals and those diagnosed with dyslexia, as well as gender differences in phonological processing. Again, different teams of researchers differ in their conclusions, in part because of different methodologies, different tasks, comorbidity, small sample sizes, and inadequate descriptions of the populations of individuals with dyslexia (Spreen, 2001). Probably the best conclusions of the burgeoning literature that has attempted to demonstrate a clear association between neuropsychology and SLDs are (a) "the neurological basis is no longer 'presumed,' although it is not always confirmed, and less specific than we would like it to be" (Spreen, 2001, p. 301); and (b) "the technological advances in imaging the brain along with examining electrophysiological and metabolic correlates of function have been impressive. Although these techniques . . . are very sensitive in detecting certain abnormal neurologic conditions, these techniques have not yielded much clinical or diagnostic utility in the assessment of the individual with a learning disorder" (Bigler et al., 1998, p. 79).

But, despite these cautions, there is much reason for optimism based on the accumulating evidence from an impressive array of studies using EEG and metabolic imaging techniques, such as the innovative studies conducted by Shaywitz and her colleagues. In their studies of normal readers they discovered clear-cut differences in how men and women read: "men activated the left inferior frontal gyrus, while women activated the right as well as the left" (Shaywitz, 2003, p.77). Of the three neural pathways for reading, their research and the imaging studies of other researchers indicated

that beginning readers rely primarily on two relatively slow, analytic routes (parieto-temporal and frontal), whereas experienced, skilled readers depend on an express pathway—occipito-temporal. And when comparing good readers to dyslexic readers, Shaywitz (2003) notes: “As they read, good readers activate the back of the brain and also, to some extent, the front of the brain. In contrast, dyslexic readers show a fault in the system: underactivation of neural pathways in the back of the brain” (p. 81). Indeed, there has been widespread support from a variety of fMRI and PET scan studies to indicate that individuals with dyslexia demonstrate reduced left temporo-parietal responses relative to controls during reading tasks such as phonological processing—a finding that was first observed in adults (e.g., Rumsey, 1992; Shaywitz et al., 1998) and then verified with children (e.g., Shaywitz et al., 2002). Recent studies that utilize diffusion tensor imaging (DTI), a form of MRI, provide evidence that the integrity of the *white matter structure* of the neural pathways in the left temporo-parietal region differs for good versus poor readers, as indices based on the white-matter structure correlated significantly with reading ability (Beaulieu et al., 2005; Deutsch et al., 2005).

Also of great interest is the dramatic series of studies now underway by Sally Shaywitz, Jack Fletcher, and others—the application of brain imaging studies to directly evaluate how the neural systems used for reading respond to specific interventions (Shaywitz et al., 2004). For example, Shaywitz and her colleagues used fMRI to study poor readers’ responses to the implementation of a 1-year experimental intervention program: “The final set of images obtained one year after the intervention had ended was startling. Not only were the right-side auxiliary pathways much less prominent but, more important, there was further development of the primary neural systems on the left side of the brain. . . . [T]hese activation patterns were comparable to those obtained from children who had always been good readers” (Shaywitz, 2003, pp. 85–86). These exciting, positive, educationally relevant results stand in stark contrast to the hundreds of failed intervention studies in the 1960s and 1970s that featured the training of perceptual and psycholinguistic processes (Hammill & Bartel, 1978; Newcomer & Hammill, 1976).

Specific details about Shaywitz’s and other researchers’ neuroimaging studies, as they apply both to diagnosis and remediation of SLD, appear throughout this volume (see, especially, the chapters by Erin Bigler, Gayle Deutsch, Jack Fletcher, Elaine Fletcher-Janzen, Jane Joseph, Byron Rourke, and Sally Shaywitz). The exciting results and implications of these studies far outweigh the inconsistencies and occasional contradictions in the CT, EEG, and imaging literature. The history of SLD is steeped in neuropsychology and neuroscience, including a diverse set of studies with the Halstead-Reitan Neuropsychological Battery by Rourke and others that consistently identified neuropsychological deficits in individuals with SLD while also distinguishing those with SLD from patients with brain damage and from normal controls (Reitan & Wolfson, 2001). The present is enriched by novel and insightful

applications of neuroimaging technology to SLD diagnosis and treatment, and the future of SLD assessment should continue to embrace the essential link between brain functioning and the identification of *learning abilities* as well as learning disabilities.

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