Understanding and Evaluating Learning and Developmental Disorders in Children

– Sam Goldstein –
About the Presenter

Sam Goldstein

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Chapter 6

Learning Disabilities

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Learning disabilities, including reading disabilities, are the most prevalent group of handicapping neurobehavioral disorders served in the public schools (U.S. Department of Education, 1994). Researchers find that it is difficult to estimate the prevalence of learning disabilities in adults (Corley & Taymans, 2002). A genetic component has been found to these disabilities (Astrom, Wadsworth, & DeFries, 2007; Kovas, Haworth, Dale, & Plomin, 2007; Kovas, Haworth, Petrill, & Plomin, 2007; Kovas, Y., & Plomin, R. (2007). Therefore, a chapter addressing the neurobehavioral and genetic aspects of learning disabilities is an appropriate topic for inclusion in this text. Unlike most other genetic disorders, learning disabilities are not a single, relatively well-defined entity or syndrome. Rather, learning disabilities encompass an extremely heterogeneous group of problems with diverse characteristics which can result from a variety of biological influences, including genetic factors, environmental insults to the brain, and possibly, as recent research on brain development suggests, from extreme lack of early environmental stimulation. As a result, the multifaceted field of learning disabilities is complex and often contentious with many competing theories, definitions, diagnostic procedures, and suggested avenues of intervention.

Within the framework of this chapter it is not possible to adequately describe or attempt to integrate the many competing viewpoints and claims surrounding the construct of learning disabilities. This task has admirably been undertaken by other writers in the field who have approached learning disabilities from a broad, historical perspective as well as from the viewpoint of best current practices (Lerner, 1993; Mercer, 1991; Torgesen, 1991; Swanson, Harris & Graham, 2003). This chapter will approach learning disabilities from biomedical, neuropsychological, and information-processing perspectives.

The Concept of Learning Disabilities

Learning disabilities as a category of human exceptionality evolved from observations of physicians and educators as they studied and attempted to assist brain-injured children. Alfred Strauss and Laura Lehtinen published their classic work *Psychopathology and Education of the Brain-Injured Child*, in 1947. In 1966, Clements, as head of a task force sponsored by the U.S. Department of Health, Education and Welfare, strongly supported use of the term *minimal brain dysfunction* which became popularized as MBD (Mercer, 1991).

The terms minimal brain injury or minimal brain dysfunction were used to describe children of normal intelligence who appeared similar to some individuals with known brain injury in that they exhibited a combination of hard or soft signs of neurological deficiency concomitantly with educational and sometimes behavioral disorders. Minimal brain dysfunction was believed to be responsible for observed deficits in processes such as auditory and visual perception, symbol learning, short- and long-term memory, concept formation and reasoning, fine and gross motor functions, and integrative functions, resulting in disorders of receptive and expressive language, reading, writing, mathematics, physical skill development and interpersonal adjustment. In addition, behavioral traits such as distractibility, impulsivity, perseveration, and disinhibition were often found in children with minimal brain dysfunction syndrome (Cruickshank, Bentzen, Ratzeburg & Tannhauser, 1961; Gardner, 1973; Johnson & Myklebust, 1967; Fletcher, Shaywitz & Shaywitz, 1999). Thus, from the first, the field of learning disabilities centered around a medical model with the term minimal brain dysfunction being applied to an extremely heterogeneous group of individuals.

Johnson and Myklebust (1967) discussed the limitations of extant terminologies. They suggested that *minimal* was inappropriate to describe individuals whose resulting disabilities had much greater than minimal impact on their learning functions and that the words *brain injury* or *brain dysfunction* were viewed as too stigmatizing by many individuals with learning disorders and their parents.

In 1963 at a national organizing conference of concerned parents and professionals held in Chicago, Samuel Kirk proposed use of the term *learning disabilities* (LD) (Lerner, 1993). This term was quickly accepted by parents and continued to gain ascendance when federal and state governments adopted it at the time special education services were expanded to include students of average or better intelligence with otherwise unexplained academic learning disorders.
Kirk viewed learning disabilities from a psycholinguistic perspective which proposed that underlying specific deficiencies in central nervous system functioning result in deficits in psychoneurological learning processes which, in turn, explain observed learning disabilities. Based on the psycholinguistic-process model of Charles Osgood, Kirk described learning disabilities according to learning channels (auditory/verbal or visual/motor), learning levels (rote or conceptual), and specific processes (perception, reception, memory, integration, expression, etc.) (Kirk & Kirk, 1971). Naglieri and Das (2002), based on Luria’s model of intellectual processes described four critical processes essential for effective learning. Luria’s PASS model involves planning, simultaneous processing, attention and successive processing. Weaknesses in various combinations of these processes have been associated with specific learning disabilities (Naglieri & Das, 2002).

While the view of learning disabilities as neurologically-based process deficits remained widespread, during the 1970’s a behavioral approach to the topic was promulgated. Process deficits were roundly criticized as hypothetical constructs which could not validly or reliably be diagnosed and which had little or no demonstrable relationship to effective interventions (Hammill and Larsen, 1974, 1996; Larsen, Parker & Hammill, 1982). Proponents of this view advocated criterion-referenced or curriculum-based assessment of a multitude of specific skills and interventions based on a detailed analysis of the component parts of each skill to be taught/learned along with ecological analysis and modification of the learning environment. Well designed and group-validated approaches to curriculum instruction were held to be appropriate and effective for all students, including slow learners, without reference to supposed internal processing deficits or disabilities. This approach, and now referred to as response to intervention (RTI) has become increasingly popular and advocated for within special education programs in public schools.

While debate raged, a third approach to understanding and assisting those with learning disabilities added a new dimension. Based on research centered at the University of Virginia (Hallahan, 1980) and the University of Kansas (Schumaker, Deshler, Alley & Warner, 1983), cognitive learning models were applied to the understanding and treatment of learning disabilities. From a cognitive framework, learners are viewed as directing their own learning by focusing on topics and skills which are personally meaningful and by developing active strategies for information acquisition. One outgrowth of cognitive theory has been the holistic or constructivist approach to teaching and learning, including whole language methods of reading instruction. While the tenets of cognitive theory have been applied to the learning disabled population in a number of ways, a major emphasis has been helping students to develop more reflective, accurate, and efficient approaches to learning tasks (i.e., learning how to learn). Students are taught to consciously employ self-monitoring strategies and effective learning/study strategies. This model, which emphasizes an approach of focus on how students learn versus what a student learns, may have influenced the scientific discipline away from more deficit-based conceptualizations when explaining learning disabilities (Wong, 1987).

**Prevalence of Learning Disabilities**

Determining prevalence rates, or the frequency of occurrence, of learning disabilities in the population might at first glance appear to be a relatively straightforward process. However, since prevalence rates for any disease or disability are dependent on having a clear-cut definition of the disorder under consideration and since there is no consensually accepted or experimentally validated definition of learning disabilities, the process of determining the prevalence of learning disabilities is a quagmire. At the present time, incidence figures for this nondefinitive disorder or group of disorders cannot be determined precisely and are essentially broad estimates. However, “the DSM-IV-Text Revision does acknowledge that the prevalence of different LD types is difficult to establish because many studies focus on the prevalence of Learning Disorders in general without careful separation into specific Disorders of Reading, Mathematics, or Written Expression” (p. 55; Corley & Taymans, 2002; American Psychological Association, 1994).

Important considerations regarding the determination of LD prevalence were presented by MacMillan (1993) and Lyon (1996b). In a discussion of operationalizing disability definitions MacMillan described **prevalence rate** as referring to the total percentage of the population that is affected by a disorder while **detection rate** refers to the number of known or identified cases. For learning disabilities, prevalence and detection rates may, indeed likely do, differ. Depending on the stringency of identification criteria, prevalence estimates for learning disabilities have varied from as low as 1% to as high as 30% of the school-age population (Lerner, 1993). Mercer (1991) suggested that those with severe specific learning disabilities might comprise approximately 1.5% of students, while the inclusion of students with mild learning disabilities could raise that figure to about 4% or 5%. Other studies focusing on a specific classification of learning disabilities identifies from 5-8% of school age children exhibit arithmetical disabilities (Geary, 2003) and 5-17.5% with dyslexia (Shaywitz, 1998). The prevalence of children that have a classification of LD extends to 4.6 million children.
with half of students in special education services (Pastor, & Reuben, 2008). National estimates indicate that 6% to 9.7% of children suffer from learning disabilities (Pastor, & Reuben, 2008).

There are many factors associated with what some view as the burgeoning or even epidemic identification rate for learning disability. These factors can be divided into four groups: those related to definition/classification, available diagnostic instrumentation, systems operation, and sociopolitical realities. The four factors will be discussed in detail because they are central to some of the most important and persistently ineluctable issues in the field of learning disabilities.

As was mentioned, the primary factor underlying widely varying prevalence estimates for LD is lack of a clear-cut definition and lack of classification procedures derived from coherent theory. Lyon (1996b) stated: "Valid prevalence estimates depend upon a set of criteria for identification that are clear, observable, measurable, and agreed upon" (p. 58). While research-based theory building in terms of definition and classification is proceeding apace in the areas of phonologically based reading disorders and nonverbal learning disabilities (Torgeson, 1993), for the broad field of learning disabilities this remains a distant goal. At present, the concept of learning disabilities is multifaceted with diversity engendered by a wide array of associated medical and pedagogical disciplines including neurology, psychology, neuropsychology, speech and language pathology, optometry, occupational and physical therapy, and education as manifested through university research, regular education, special education, and private clinical assessment/tutoring. Each profession brings its own set of theoretical considerations, methodologies, and predilections. In addition, there is much variability within professional orientations. For example, among public school special education programs, the criteria for designating students learning disabled vary widely. Mercer, Forgnone, and Wolking (1976) and Mercer, King, Sears, and Mercer (1990) documented the range of definitions of learning disability and the lack of agreement in the diagnostic criteria adopted by state boards of education across the United States. The concept of an IQ/achievement discrepancy, which is presently the central feature of LD diagnosis in public school, is operationalized differently by states with arbitrary cut-off points for both intellectual level and achievement level. One result of highly flexible decision-making is that the percentage of children labeled learning disabled differs from state to state, varying from 2.85% to 9.43%; a threefold difference (Reschly, & John, 2004).

Another factor affecting the detection rate for learning disabilities is the availability of an appropriate range of reliable and valid assessment measures. Although there is currently much criticism directed toward the concept of IQ/achievement discrepancy as the basis for determining presence or absence of learning disability (Lyon, 1996b; MacMillan, 1993; Mather & Roberts, 1994; Stanovich, 1993; Toth & Siegel, 1994; Zigmond, 1993), intelligence and achievement will likely always be essential constructs for understanding individuals with learning disabilities and, therefore, must be measured as accurately as possible. In addition, to the extent that learning disabilities are viewed as reflecting specific process deficits or information-processing deficiencies, there must be a variety of well standardized instruments for quantifying processing strengths and weaknesses. For a given individual, it may be important to assess any of the following: phonemic awareness, phonological segmentation, grammatical and semantic comprehension, rapid automatic naming (quick label retrieval), digit/sentence repetition, oral expression, tactile perception, directional perception, spatial organization, social perception, verbal and nonverbal concept formation, concrete and abstract problem-solving, processing speed, and motor coordination in its many forms. While there are measures available to assess these constructs, their specificity, adequacy, quality of standardization, and breadth of dissemination are highly variable. The development of psychometrically sound diagnostic instruments remains a primary goal which will reduce misidentification and eventually have a positive impact on our understanding of the prevalence of learning disability as well as specific subtypes of learning disability.

A third important though somewhat overlooked factor affecting LD prevalence rates has to do with what MacMillan (1993) has termed system identification variables. This factor overlaps to some degree with the fourth factor, the effects of sociopolitical realities. Broadly, system identification encompasses the ecological processes within families, schools, and clinics which increase or decrease the likelihood that a given individual will be referred for evaluation and classified learning disabled. Family system variables affecting whether parents seek LD assessment for a child, or whether an individual seeks assessment for himself, could include socioeconomic status (SES), family values regarding educational attainment for males and for females, the presence or absence of comorbid conditions which create additional functional difficulties, the presence or absence of medical insurance coverage, etc. Variables within educational systems which influence the prevalence of learning disabilities include the educational philosophy of the school system, financial incentives for identification, the nature and quality of basic education provided for all students, class size, training of regular class teachers to accept and deal with diversity, availability of other types of supportive services for students and
teachers, training/competence of special education diagnosticians, etc. LD prevalence rates are also affected by sociopolitical factors such as increased attention to learning disabilities as a result of public awareness and political advocacy; ambiguity in the definitions and overlap of disability categories; and the social desirability of a learning disability classification rather than a classification of intellectual disability or mental retardation. (Keogh 1993; Macmillan, 1993).

MacMillan (1993 citing Zigler and Hodapp, 1986), suggested that for learning disabilities"...the ratio of detected to undetected cases may (and probably does) vary by age, IQ level, racial group, gender, and socioeconomic status" (p. 143).

Within school populations, data has been gathered on the incidence of learning disabilities by age, race, and gender. A report from the U.S. Office of Education (USOE, 2007) presented data showing that 1.22% of the student population in kindergarten were receiving special education services for LDs, whereas 6.49% of fifth grade class were enrolled in special education services due to LDs. Lerner (1993, citing USDE, 1991) reported the number of LD children served in special education at each age level increases rapidly from six years to nine years, peaks and levels off for children ages ten to twelve, and then gradually declines to age eighteen. Thus, it appears that the majority of LD children are first identified during their primary and intermediate years of elementary school and that far fewer students are first identified during secondary school. Lerner suggested that the decrease in the number of LD students served during their teen years may partially be accounted for by the number of teenagers with LD who drop out of school.

The U.S. Office of Civil Rights has been concerned about the number of students from racial and ethnic minority groups that are identified as disabled and enrolled in special education. A survey by that Office (OSEP, 1994, cited by Reschly, 1996) reported that a total of 8% of African-American students, 6.5% of Caucasian students, and 5.6% of Hispanic students were enrolled in special education under the categories of mild mental retardation, severe emotional disability, and learning disability. While African-American students may be over represented and Hispanic students under represented overall, according to figures from this survey the percentage of students from each group categorized learning disabled was relatively constant at 5.0% of African-Americans, 5.0% of Caucasians and 4.7% of Hispanics.

The prevalence of learning disabilities by gender has long been a topic of discussion and concern. Lerner (1993, citing U.S. General Accounting Office, 1981) reported that of the special education students classified learning disabled, approximately 72% are boys and 28% are girls. Research studies of individuals with reading disability have typically estimated a gender ratio of males to females in the range of 2:1 to 5:1 (Huston, 1992). A variety of explanations have been suggested to account for the preponderance of males with reading disability; for example, genetic factors, factors associated with differences in prenatal brain development, sex-linked differences in hemispheric specialization, postnatal maturational differences, and system identification variables (Kelley, 1993; Pennington, 1991; Thomson, 1990). Genetic factors as explanations to account for sex differences have been equivocal with some studies finding no differential genetic etiology between males and females in the development of reading difficulties (Hawke, Wadsworth, & Defries, 2006). While there may be some variation in male/female prevalence or severity of reading disability that can be attributed to biological differences, findings from three of the universities in the LD Research Network indicate that nearly an equal number of males and females manifest dyslexia (Lyon, 1996b; Kolata, 1990; Shaywitz, Shaywitz, Fletcher & Escobar, 1990). Moreover, recent studies have found that there are confounds in the research examining sex differences in the prevalence of dyslexia making it appear as if more males suffer from the disorder (Berninger, Nielsen, Abbott, Wijsman, & Raskind, 2008).

When prevalence estimates are determined through research-based epidemiological studies in which every child in a given cohort of children is assessed for reading disability, the ratio of males to females is close to 1:1; when estimates are determined through incidence figures for school-identified or clinic-identified populations based on teacher or parent referral, the ratio of males to females is much higher. System identification variables resulting in gender-based ascertainment bias appear to account for the majority of this difference. As is true for boys in general, boys with reading difficulty display more behavioral problems, including regulation of activity level, than do girls. Since their functional difficulties are more readily perceived as problematic, more boys than girls are referred for assessment and consequently more are classified to receive special education services (Chaise, et al., 1999).

Studies of adopted children have shown that learning disabilities are diagnosed four to five times more frequently in this group than in an equivalent group of non-adoptees (Kenny, 1967; Silver, 1970, 1989). Because information about the biological parents of adoptees is often limited or confidential, it is difficult to determine the reasons for the high rate of learning disability among adopted children.
Etiology and Genetics of Learning Disabilities

From the time of the earliest medical reports which described cases of dyslexia, learning disabilities have been viewed as stemming from central nervous system dysfunction, more precisely, from dysfunction of specific portions of the cerebral cortex (Doris, 1986; Huston, 1992). This long-standing presumption is being reinforced and validated by modern cognitive neuroscience. Language specific processing of the brain in areas surrounded by the Sylvian Fissure have been associated with a variety of language functions. The temporoparietal cortex receives projections containing but not limited to visual and auditory information. The posterior superior temporal gyrus or Wernickers area is associated with a variety of language functions, particularly involving comprehension. However, it is likely over-simplistic to describe temporoparietal areas of those responsible for the reception of language whereas frontal regions responsible for expressive language. It is more likely that a distributed network is responsible for full coherence of the language system (Joseph, Nobel & Eden, 2001).

PET and fMRI have been used extensively to extend an understanding of how specific components of learning map onto the brain (Ghilardi et al., 2000; Grahn, Parkinson, & Owen, 2009; Hubert at al., 2007; Rumsey, Howorwitz, Donahue, Maisog & Andreason, 1997; Thiel, 2003). As these techniques have become more refined and technologically advanced, an understanding of structural differences implicated in learning disabilities has progressed. Despite their limitations, these techniques have revealed much about structures of the brain associated with visual word form (Fritch, Friston, Liddle and Frack, 1991), orthography (Flowers, Wood & Nailer, 1991), phonology (Rumsey, et al., 1997) and semantics (Pugh, Shaywitz, Shaywitz, Constable, et al., 1996). However, a great deal of variability has been found then and between studies such that multiple sites within similar regions of the brain have been implicated in these processes (Poeppel, 1996).

Very few functional neuroimaging studies have been conducted with children. In part due to the fact that PET requires the application of radioactive material. At least one study using fMRI has mapped language dominance in children with partial epilepsy, finding results similar to those observed in adults (Hertz-Pannier, Gaillard, Mott, Cuenod, Bookheimer, et al., 1997). Readers interested in an extended discussion of learning and brain imaging are referred to Berninger (2004). Learning disabilities have traditionally been viewed as neurological deficits intrinsic to genetic and other biological factors within the individual and not of environmental origin. However, current research is documenting the intimate connection between environment and neuroanatomical development (Dawson & Fischer, 1994; Hutlenlocher, 1991). The pervasive effects of early environmental programming on the formation and pruning of neural networks and the theoretical relationship of this process to the occurrence of neurologically-based specific learning disabilities is an area that is only beginning to be considered.

The prenatal, perinatal, and postnatal environmental factors associated with brain development and brain injury are best viewed as potential causes of learning disability due to uncertainties and inconsistencies in the relationships between age at onset, the severity of circumstance or condition, the degree of transient or permanent brain dysfunction, and the broad range of possible effects on learning. For example, clinical studies have documented cases in which major structural deficits, even loss of an entire brain hemisphere, result in few observable signs of learning disability while many individuals with severe learning disabilities have no obvious structural deficits (Bigler, 1992; Satz, 1990). In addition, confounding variables such as socioeconomic status, parenting style, and early interventions mediate the degree to which a neurological abnormality will result in impaired learning. In many or most cases of learning disability etiology is, presumably, not a factor. However in some cases an environmental cause is directly known or fairly certain while in other cases, the environmental contribution to etiology is cloudy, involving a subtle interplay of potential factors which may be undocumented or unknown. In the last forty years, experimental research has provided strong support for a genetic factor in some forms of learning disability. The familial occurrence of reading, spelling, and writing disabilities has been investigated using a variety of methodologies such as study of family history and pedigree analysis, determination of concordance rates among identical and fraternal twins, comparison of linear regression in reading scores between identical and fraternal twins, and chromosomal analysis of family members.

The earliest widely cited family pedigree study of reading disorder, conducted by Hallgren in 1950 (cited in Pennington, 1991), consisted of a statistical analysis of dyslexia in 112 families. Among first-degree relatives (parents and siblings of an identified child), the risk for co-occurrence of this disorder was 41%, which is much higher than the usual prevalence estimates for the general population of 5% to 10%. Huston (1992), reporting on Hallgren's study, indicated that of the 112 families, 90 families, one parent was dyslexic; in 3 families both parents were dyslexic; and in 19...
families neither parent had dyslexia. While Hallgren's study has been criticized for methodological flaws, later studies carried out with greater technical precision, such as that of Finucci, Guthrie, Childs, Abbey & Childs (1976, cited in DeFries, 1991), have found similar familial rates in the range of 35% to 45%. Finucci (1978) also published a critical review of the early investigations of dyslexia and genetics. Even more recent studies continue to demonstrate considerable evidence supporting that dyslexia and even dysgraphia have a developmental, genetic influence (Raskind, 2001).

The Colorado Family Reading Study, begun in 1973, compared reading abilities of 125 reading-disabled children (probands) and their family members to 125 matched control children who were not reading disabled and their family members. The total number of subjects in this study was 1,044, making it an extensive family study. Results clearly demonstrated that reading disorders are familial in nature. Scores for siblings of proband subjects were significantly lower than scores for siblings of control subjects on measures of both reading and symbol processing speed. A similar pattern of significant results was observed for the parents of probands and controls. An interesting finding was that, on average, brothers of probands were significantly more reading impaired than sisters of probands. Similarly, fathers of probands were, on average, less skilled readers than mothers of probands; however, the score difference between fathers and mothers was less than the score difference between male and female siblings (DeFries, 1991). Although reading disabilities have now conclusively been shown to be familial in nature, familial occurrence suggests but does not demonstrate genetic heritability. Empirical investigations to ascertain the genetic inheritance of learning disabilities, specifically reading disability, have included concordance studies of twins, multiple regression studies of twins, segregation analysis studies, and chromosomal linkage studies. Comparison of pairs of identical and fraternal twins has been used to investigate the genetic component of reading disability in the same way that other twin studies have researched the heritability of intelligence and a variety of other personal characteristics. Many twin studies have employed a comparison of concordance rates to test for genetic etiology. A pair of twins is concordant for reading disability if both twins are reading disabled; if just one twin is reading disabled, the pair is discordant. Identical twins share an identical genetic makeup while fraternal twins share about 50% of heritable variation (LaBuda & DeFries, 1990). To the extent that reading disability is genetically determined, the concordance rate for pairs of identical twins should be considerably higher than for pairs of fraternal twins when at least one member of each identical and fraternal pair has been identified as reading disabled.

Two of the earlier reports of concordance rates for reading disability in twins were those of Hermann (1959) and Zerbin-Rudin (1967). Both of these researchers pooled the findings of smaller previous studies, possibly with some overlap in their reporting of cases. The concordance rates reported by both authors were nearly identical. Based on their combined data, as reported by Huston (1992), there was, on average, 100% concordance for twenty-nine identical twin pairs and about 34% concordance for sixty-seven fraternal twin pairs.

Due to technical differences in the method for determining concordance rates, different authors sometimes report different concordance figures for the same study, i.e., some authors report pairwise concordance rates and others report probandwise concordance rates. The first method counts each concordant twin pair one time. The latter method considers each member of a concordant pair as a separate research subject and, therefore, counts each concordant pair twice. Using probandwise concordance increases the percentage of concordance for both identical and fraternal twin pairs (LaBuda & DeFries, 1990). For example, in the Zerbin-Rudin study, a pairwise concordance rate for fraternal twin pairs was 34% (12 of 34 cases) as reported by Huston (1992); however, the probandwise concordance rate for those same twin pairs was 52% (24 [12 + 12] of 46 [34 + 12]) cases as reported by DeFries (1991).

Bakwin (1973, cited by LaBuda and DeFries, 1990) studied 31 pairs of identical and 31 pairs of fraternal twins, finding 84% pairwise concordance for identical twin males and 83% for identical twin females. Interestingly, the pairwise concordance rate for male fraternal twins was 42% while the rate for female fraternal twins was just 8%. Bakwin also investigated the environmental factors of birthweight and birth order as predictors of reading disability but found no significant differences between normally-reading twins and reading-disabled twins on these variables.

Stevenson, Graham, Fredman and McLoughlin (1987, cited by Thomson 1990), conducted a large-scale study of the reading and spelling abilities of 285 13-year-old twins divided into several subgroups according to type and severity of skill deficiencies. In contrast to other concordance studies of twins, these authors reported, overall, relatively similar pairwise concordance rates for identical and fraternal twin pairs, 32% and 21%, respectively. Their findings suggest a fairly low level of heritability for reading disorder. However, with IQ controlled, Stevenson, et al. found a strong genetic influence on spelling ability.
The most technologically sound large-scale twin study, the Colorado Twin Study, was begun in 1982 as part of the Colorado Reading Project. With IQ controlled (Verbal or Performance IQ = 90 or above) and other types of selection criteria in place, the Colorado Study examined reading disability in 101 pairs of identical twins and 114 pairs of fraternal twins. The pairwise concordance rate of 52% for identical twins was lower than for most earlier studies while the rate for fraternal twins was fairly typical at 33% (LaBuda & DeFries, 1990). Although there is some variation in the concordance figures generated by different studies, taken as a whole, they do provide strong evidence for a genetic factor in the etiology of reading disability.

In the search for the genetic mechanisms underlying reading disability, two primary strategies have been employed, chromosomal linkage studies and segregation analysis. Working from phenotype (clinical manifestation of disability) to genotype (underlying genetic substrate of disability), segregation analysis involves testing all members of affected families for the presence of a learning disorder and then fitting the data to potential models of genetic transmission, e.g., autosomal dominant, autosomal recessive, codominant, or polygenic models. Pennington, Gilger, Pauls, Smith, Smith and DeFries (1991) after performing segregation analysis on four subject samples, found support for a major gene model in which dyslexia in some families is transmitted by one or more dominant or partially dominant genes. They also found support for genetic heterogeneity, i.e., multiple genetic mechanisms in the transmission of dyslexia. Further research with more sophisticated segregation analysis also has pointed toward a major dominant gene effect which is frequently occurring (57% of the population) and which, when present, increases an individual's liability for reading problems (Gilger, Vorecki, DeFries & Pennington, 1994). However, this putative gene is of low penetrance such that only 3% of individuals having one or two copies of the defective allele demonstrated reading deficits greater than 1.96 s.d. below the population mean. Non-affected individuals (43% of the population) with two normal alleles and no copies of the defective allele had an extremely low probability (p = .0027) of being classified reading disabled. Genetic links to reading disabilities have been complicated by studies that have found that although alleles in specific genes have been linked to reading disabilities, the genes themselves do not contribute (Smith et al., 2001).

Working from genotype to phenotype, linkage studies have been conducted to identify the specific chromosomes and the genetic loci on those chromosomes that are associated with dyslexia. Through cytogenic studies of families in which there are a number of persons identified as dyslexic, the search for a gene or genes that may cause dyslexia can be narrowed. Smith, Pennington, Kimberling and Ing (1990) and DeFries and Gillis (1993) have summarized the complex principles of linkage analysis which involve investigating both the link between marker genes and the disability gene on a chromosome and the link between that chromosome and the phenotypic occurrence of reading disability.

The pioneering linkage study of Smith, Kimberling, Pennington and Lubs (1983) found evidence in some families for a link between reading disability and a marker on chromosome 15p. A later study with a larger number of subjects provided additional support for this finding (Smith et al., 1990) and further suggested that the apparent linkage was present in approximately 15-20% of families with multiple cases of reading disability.

A second possible genetic locus for reading disability in families not linked to chromosome 15 was suggested by the observation of the co-occurrence of dyslexia and disorders of the immune system which are coded to the human leukocyte antigen (HLA) region of chromosome 6 (Geschwind & Behar, 1982; Pennington, Smith, Kimberling, Greene & Haith, 1987; Smith, Kimberling & Pennington, 1991). Subsequent research to test this hypothesis (Cardon et al., 1994) studied linkage in two independent samples, 126 sibling pairs and 50 fraternal twin pairs, in which at least one member of each pair was reading disabled. Analyses of the reading performance of subject pairs genotyped for DNA markers localized the reading disability trait to a small region within chromosome 6 between markers D6S109 and D6S1260 (Turic et al., 2003). Other evidence points to the KIAA0319 gene on chromosome 6 as influencing reading ability and susceptibility to dyslexia (Paracchini et al., 2008). Additionally, chromosome 15 and the region including D15S146 and D15S994 on chromosome 15 have been linked to reading disabilities (Morris et al., 2000; Schumacher et al., 2008).

A high incidence of reading disability is found in individuals with abnormalities in sex chromosome karyotypes, the most common of which is the 47 XXX karyotype in males (Klinefelter's Syndrome), occurring in approximately 1/1,000 births (Berkow & Fletcher, 1992; Pennington, Bender, Puck, Salbenblatt & Robinson, 1982). Although not a frequent occurrence in the learning disabled population, the strong association between some sex chromosome anomalies and reading disorders provides additional evidence for the genetic heterogeneity of reading disability.

There is evidence that reading disability per se is not inherited but that genetic variations influence specific subskills connected to the reading process. Olson, Wise, Conners, Rack and Fulker (1989) found significant heritability for a phonological coding task but not for an orthographic coding task. Pauls (1996) reported genetic linkage studies of dyslexic subjects and their family members assigned to one of four research groups according to the primary deficient
process evident in their reading difficulty: phonological segmentation, non-word reading, rapid namings and single word identification. Similar to previous findings, phonological segmentation showed linkage to the HLA region of chromosome 6. There was no evidence for a connection between word identification and chromosome 6; however, there was some evidence that the word identification phenotype is tied to a variation in the same portion of chromosome 15 that was first implicated by Smith, et al. (1983).

In summary, family studies, concordance studies of twins, and multiple regression studies of twins have shown that reading disabilities run in families, that they are heritable, and that the heritable component is approximately 50%. Presently, segregation analyses point to genetic transmission via a partially dominant or dominant major gene effect. Genetic linkage studies have provided strong evidence that in some families and subject populations studied, reading disability is linked to chromosome 6p or chromosome 15p. Both segregation analyses and linkage analyses have led to the conclusion that phenotypic reading disability is genotypically heterogeneous, i.e., increased susceptibility to reading disability can be produced by multiple genetic profiles. Furthermore, there is preliminary evidence which suggests that within a single individual the component processes of reading may be influenced by separate genes at different loci.

**Subtyping Learning Disabilities**

Although public agencies have primarily chosen to define learning disability based upon a discrepancy between achievement and IQ based estimates of potential achievement, this statistical definition does little to facilitate an understanding of the underlying processes that contribute to successful and in this case, unsuccessful achievement. Although it has been suggested that learning disability is a broad, non-specific symptom for which cause must be identified, it has yet to be demonstrated that different causes lead to different types of learning disability or for that matter require different treatments.

The work of Boder (1973) and Bakker (1979), though thirty years old, exemplify efforts to classify and identify learning disability on the basis of educational criteria. Boder described three subtypes of children with learning disability: (1) a dysphonetic group lacking word analysis skills and having difficulty with phonetics; (2) a dyseidetic group experiencing impairment in visual memory and discrimination; and (3) a mixed dysphonetic, dyseidetic group. The dysphonetic group included two thirds of those identified as learning disabled with the dyseidetic group constituting approximately 10%. Bakker's work described L- and P-type Dyslexias. Children with L-type Dyslexia read quickly but made errors of omission, additions and word mutilation. The P-type group tended to work slowly and make time consuming errors involving fragmentations and repetitions. Among the interesting and promising attempts to define learning disability are those studies involving multivariate analysis. Efforts to subgroup learning disability using such analyses find that differences between good and poor readers may reflect impairment in minor skills such as oral word rhyming, vocabulary, discrimination of reversed figures, speed of perception for visual forms and sequential processing (Doehring, 1968). In 1979, Petrauskas and Rourke utilized a factor analytic method to describe the difficulties of a group of deficient readers. They found these readers falling statistically into four subtypes: (1) primarily verbal problems; (2) primarily visual problems; (3) difficulty with conceptual flexibility and linguistic skills; and (4) no identified specific weakness. The first of these two groups corresponds with Boder's analysis. The third may reflect children with weaker intellectual skills while the fourth may in fact reflect the long-standing, clinical perception that there are a group of children who experience achievement problems possibly secondary to non-neurologic factors (e.g., emotional disorder).

Mattis, French and Rapin (1975) identified three distinct syndromes of learning disability based upon a factor analysis. These included: (1) children struggling to read as the result of language problems; (2) children with articulation and graphomotor problems affecting academic achievement; and (3) children with visual spatial perceptual disorder. The third group displayed better verbal than non-verbal intellectual abilities. Almost 80% of the impaired children fell in the first two groups. Deneckla (1972, 1977) reported similar statistics noting that approximately 16% of learning disabled children experienced some type of visual-spatial or perceptual motor problem.

Thus, there is a consensus among factor analytic studies attributing a large group of children with problems related to verbal weaknesses and a smaller but significant group related to perceptual weaknesses. Joscho and Rourke (1985), based upon an analysis of the Wechsler Intelligence Scale for Children, found a clear distinction between children with learning problems stemming from verbal weaknesses and those whose problems stem from non-verbal weaknesses.

Satz and Morris (1981) found five distinct groups of reading disabled children, again along this verbal-nonverbal continuum. These included: (1) those with language impairment; (2) those with specific language problems related to naming; (3) those with mixed global language and perceptual problems; (4) those with perceptual-motor impairment only; and (5) an expected group similar to that reported by Petraskas and Rourke (1979) in which no significant impairments
were identified. Some researchers have hypothesized that this group of children simply has not experienced adequate
to develop essential achievement skills while others, as noted, suggest an emotional basis for this group of
profile of five learning disabled subtypes, including individuals with normal test scores, auditory processing problems,
difficulty with receptive and expressive language, spatial weaknesses and a global pattern of low test scores.
Rourke (1989) concluded that cluster-analytic studies have identified some association between learning delay
and a wide variety of perceptual, linguistic, sequential and cognitive skills. This finding is reinforced by the work of
others over nearly a forty-year period (Benton, 1975). According to Swartz (1974) a pattern consisting of depressed
scores on four Wechsler subtests, the ACID pattern (an acronym for Arithmetic, Coding, Information and Digit Span
subtest) characterize the weaknesses of most learning disabled children. Although this view is held by many others and
has been most recently advanced by Kaufman (1997), not all learning disabled children display this pattern. Children who
do, however, are thought to have a particularly poor prognosis for academic performance in reading, spelling and
arithmetic (Ackerman, Dykman & Peters, 1977). Some researchers have suggested that in a population of learning
disabled children demonstrating this pattern, one subgroup experiences particularly poor auditory-verbal memory and
sequencing while a second group experiences poor visual-spatial abilities. This distinction is similar to that described by
Joschko and Rourke in 1985. However, these authors reported a further distinction in the ACID pattern by age between a
younger group five to eight years old and an older group, nine to fifteen years old. On the basis of an extensive
neuropsychological battery, these authors found a distinct pattern of differences resulting in four subtypes. Joschko and
Rourke (1985) noted that "although the ACID subtypes generated in this research do not differ significantly in terms of
level of academic performance, the plots of the factor score profiles for each of the reliable subtests indicate that they have
qualitatively different ability profiles which may have practical applications" (pg. 77). However, even these authors noted
that effective remediation has not been clearly tied to this manner of ability profiling.

The inclusion of learning disability among the disorders evaluated and diagnosed by the medical and mental
health community has been considered an adjunct to formal psychiatric, psychological or neuropsychological evaluation.
However, as it has been recognized that learning disabled children appear more likely than others to develop psychiatric
problems, efforts have been made to refine the clinical diagnosis of learning impairments. The Diagnostic and Statistical
Manual of Mental Disorders - 4th Edition TR (DSM IV-TR) lists four academic skill disorders (American Psychiatric
Association, 2000). These are: Reading Disorder, Mathematics Disorder, Disorder of Written Expression and Learning
Disorder, Not Otherwise Specified. All four are qualified as reflecting the collection of standardized test data, indicating
performance substantially below what would be expected, based upon the individual's age, intelligence and educational
experience. According to these definitive criteria, the problem must interfere with the child's academic performance or
activities of daily living. The "Not Otherwise Specified" category reflects learning disability as an isolated weakness, for
example, difficulty with spelling independent of other written language problems. The DSM-IV-TR also contains a
Developmental Coordination Disorder diagnosis reflecting weak large or fine motor skills that may interfere with
academic achievement or daily living but are not due to a specific medical condition. Readers interested in an extensive
discussion of subtypes of learning disorders in childhood are referred to Silver and Hagan (1990) or Swanson, Harris and

A Neuropsychological Model to Assess Learning Disability

The consensus in current factor-analytic research is that there are two broad groups of skills necessary for
efficient learning:
1. Auditory-verbal processes. Weaknesses in these areas result in reading disorders and other language-
based learning problems.
2. Visual, perceptual and motor processes. Weaknesses in these areas may result in reading problems but
more likely affect handwriting, mathematics and certain social skills. Tables 6.1 and 6.2 present a model
for conceptualizing these skills and examples of them in a two-by-two grid.
The model conceptualizes learning skills on rote/automatic and conceptual levels, linguistically and visually.
As it has also been demonstrated that there is a significant but small group of children experiencing achievement
problems in the absence of either of these sets of skill weaknesses, professionals are also urged to consider the impact of
foundational skills such as an environment conducive to learning, problems with attention and impulse control, self-
esteeam as a learner and other emotional (e.g., depression/anxiety) and behavioral (oppositional defiance, conduct disorder)
problems as contributing to delayed achievement (Goldstein & Mather, 1998).
As can be seen in this model, language-based learning disorders are directly related to impaired language skills, especially those related to phonological processes (Pennington, 1991; Bishop & Adams, 1990; Scarborough, 1990, 1998). A solid body of emerging research suggests that the capacity to process information sequentially may lie at the root of impairments in phonological processing (Naglieri & Das, 2002). Further, for many children, poor comprehension results from poor rote language skills such as inability to distinguish similar sounds which then leads to poor auditory discrimination and weak phonetics. Problems with verbal short-term memory are also common among reading impaired individuals. Memory requires phonological skill. Poor readers may experience problems recalling letters, digits, words or phrases in exact sequence. While the majority of children with language based learning disabilities struggle to master basic foundational academic skills, others are capable of learning to read but when the curriculum begins to accelerate in third or fourth grade and they must read to learn, they struggle as the result of weak conceptual, linguistic skills. It is also not surprising that related language based skills such as spelling and writing are impaired in reading disabled children. For many, spelling is even more impaired than reading (Snowling & Hulme, 1991).

Weaknesses in visual-motor skills tend to cause problems with arithmetic and handwriting, often independent of associated reading disability. Included in problems for this group of children are difficulties involving social awareness and judgment. These problems do not appear to be primarily language-based and have been referred to collectively in the neuropsychology literature as nonverbal learning disabilities (NLD) (Pennington, 1991; Rourke, 1989). Children with this pattern have been reported to experience problems with spatial organization, attention to visual detail, procedural skills and mathematics; problems shifting psychological set from one operation to another, graphomotor weaknesses, poor factual memory, and poor judgment and reasoning (Rourke, 1985). Neuropsychologists can reliably conclude that children with nonverbal learning disability experience greatest deficits in visual, perceptual and organizational skills, psychomotor coordination and complex tactile perceptual abilities (Harnadek & Rourke, 1994). Finally, it is also suspected that individuals with nonverbal learning disability experience greater internalizing problems related to depression and anxiety than those with language based learning disability. It is unclear whether this pattern contributes to or is a consequence of the disability.

In the PASS model, Naglieri and Das (2002) note characteristic weaknesses in planning and attention processes for youth receiving diagnoses of ADHD, isolated weaknesses in planning for youth with mathematics learning disability and isolated weaknesses in successive processes for youth with phonics based reading disability. Readers interested in the PASS model are referred to Naglieri (1999).

**Evaluating Learning Disability in the Context of a Comprehensive Neuropsychological Evaluation**

A number of volumes provide thorough, indepth models for assessment of learning disabilities utilizing a myriad of tests and batteries. Interested readers are referred to Reynolds and Fletcher-Janzen (2006), Goldstein (1997), Mather and Goldstein (2008). Due to space limitations, this section will briefly review assessment measures. The basic task facing the professional is to answer questions concerning underlying neuropsychological skills essential to learning. Assets and liabilities must be identified. Screening of basic academic skills must also be completed. In many situations, the neuropsychologist can rely upon data collected at school to provide these basic achievement measures. The most widely used of these instruments, the Woodcock-Johnson III Tests of Achievement (Woodcock, McGrew & Mather, 2001) is the most comprehensive. It offers by far the most thorough, well-developed assessment of academic skills. The factor analytic model fits well with the concepts presented in this chapter concerning the underlying neuropsychological deficits contributing to learning disability. Subtest analysis often reveals patterns consistent with verbal, visual, rote or conceptual weaknesses. Although achievement/intelligence discrepancies are most widely used to identify learning disabilities, the issue of high I.Q. individuals with average achievement identified as learning disabled continues to be controversial. An age/achievement discrepancy nonetheless is a good target for the neuropsychologist, with a standard deviation and a half below the age mean used as a cut off.

In the absence of a comprehensive battery such as the Woodcock, it is recommended that neuropsychologists address collection of basic achievement data as follows:

1. **Reading.** A measure should be used to obtain single word reading reflecting phonetics skill and sight word achievement. An estimate of the ability to read within context and comprehend what is read should also be obtained. Achievement tests such as the Woodcock-Johnson III Tests of Achievement (Woodcock, McGrew & Mather, 2001), the Gilmore (Gilmore & Gilmore, 1968), the Gray Oral Reading
2. **Spelling.** Estimates of sight word memory for spelling and phonetic ability can be analyzed qualitatively utilizing the Wide Range Achievement Test-3 (Wilkinson, 1993).

3. **Mathematics.** The Wide Range Achievement Test-3 (Wilkinson, 1993) or the Key Math Diagnostic Inventory - Revised (Connoly, Nachtman & Pritchett, 1976) can be utilized to generate observations of conceptual versus rote sequential mathematics skills.

4. **Written Language.** Written language skills of thematic maturity, vocabulary, capacity to organize ideas, grammar, punctuation, and general execution can be observed utilizing the Story Writing subtest from the Test of Written Language - 3 (Hammill & Larsen, 1996).

### Learning Disabilities and Comorbid Disorders

Caron and Rutter (1991) reviewed the concept of psychiatric comorbidity and summarized different scenarios for the existence of either true or artificial comorbidity. As discussed by Lyytinen (1995) the term comorbidity, broadly applied, may refer to (1) the co-occurrence of two or more presumably separate neurocognitive disorders; (2) the co-occurrence of two or more independent conditions that are mediated by a common genetic or environmental etiology; (3) conditions, possibly related, which share the same or overlapping risk factors; (4) conditions which co-occur because of the secondary effects of one disorder on the other; (5) comorbid characteristics which combine to define a meaningful syndrome; and (6) conditions which result from interactive combinations of the five foregoing comorbid states. The frequently observed comorbid conditions associated with learning disabilities illustrate all six types of comorbidity although due to present empirical limitations, in a given instance the specific forms of comorbidity may be questionable or undetermined. Observed comorbidities could be a function of the current definitions of two or more of the disorders under consideration or of the diagnostic procedures used for the classification rather than representing true comorbidity. In addition, it may be unclear whether a given characteristic (e.g., psychosocial deficit) is an integral part of a learning disability pattern or whether that characteristic constitutes an associated comorbid condition (Lyytinen, 1995).

Attention Deficit Hyperactivity Disorder (ADHD) is likely the DSM-IV diagnostic category with the highest rate of comorbidity with LD. Among children diagnosed learning disabled, the reported rate of ADHD has generally ranged from 15% (Shaywitz, Fletcher & Shaywitz, 1992, cited in Lyon, 1996b) to as high as 85% (Safer & Allen, 1976), with most studies such as that of Shaywitz and Shaywitz (1988) reporting a comorbidity figure between 30% to 40%. There have been conflicting views about whether LD and ADHD are independent disorders or whether they are both manifestations of the same underlying brain dysfunction. While James and Selz (1997) suggest that due to methodological flaws in accumulated research, this question has not been fully resolved, many experts have concluded that although they frequently co-occur, there is strong evidence for the separateness of LD and ADHD (Barkley, 1990; Goldstein, 1997; Pennington, Grossier & Welsh, 1993; Shaywitz, Fletcher & Shaywitz, 1995; Silver, 1990). After reviewing several possible mechanisms for the comorbidity of ADHD with dyslexia, Pennington (1991) suggested that in most cases dyslexia leads to ADHD as a secondary symptom but that in a small percentage of cases there may be genetic correlation between the two disorders. In taking a new look at LD/ADHD comorbidity in twin data, Light, Pennington, Gilger, and DeFries (cited in Lyytinen, 1995), concluded that shared genetic influences account for a substantial portion of the covariance between dyslexia and ADHD.

It is widely believed that both LD and ADHD are comorbid to a greater than average degree with other categories of psychiatric disorder such as conduct disorder, anxiety, and depression. To date, few methodologically sound studies with well defined LD subject populations have evaluated this premise. Porter and Rourke (1985), employing the parent interview Personality Inventory for Children (Wirt, Lachar, Klinedinst & Seat, 1977) identified four distinct personality patterns or subtypes which characterized the majority of the LD children in their study. Of those classified, 44% displayed balanced, well-adjusted socioemotional functioning; 26% exhibited marked internalizing psychological disturbances (depression, anxiety, low social skills); 13% displayed roughly normal personality functioning but with a high degree of somatic concerns; and 17% exhibited behavioral disturbance reflected in hyperkinetic, aggressive, and antisocial behaviors. Three studies which used the MMPI to assess emotional functioning in learning disabled adults (Balow and Blomquist, 1965; Gregg, Hoy, King, Moreland & Jagota, 1992; Spreen, 1998, cited by Hooper and Olley, 1996) were consistent in reporting more maladjustment and serious psychopathology such as depression, anxiety, social withdrawal, phobias, acting-out tendencies, disorganized thoughts, etc. in LD adults than in their normal counterparts. In contrast, Lamm and Epstein (1992) found few differences in degree of psychopathology between individuals with
learning disabilities and control subjects when assessed via a structured waiting scale. As reviewed by James and Selz (1997), the emotional consequences of learning disability may vary by subtype but conflicting results have been reported. The most widely referenced psychopathology by subtype is the association of significant internalizing problems including social isolation, anxiety, depression, and suicide with non-verbal, or as is often termed, right-hemisphere-based learning disability (Bigler, 1989; Hooper & Olley, 1996; James & Selz, 1997; Rourke, 1989; Voeller, 1986).

“Psychosocial adjustment difficulties frequently are assumed to be the major social-emotional manifestation of learning disabilities” (Hooper & Olley, 1992, p. 170). Two of the earlier and persistent voices underscoring the need for a sociological perspective toward learning disabilities were those of Kronick (1974; 1976) and Bryan (1974; 1978; 1991). Kronick explored ways in which difficulties with attention, concentration, perception, inference, labeling of emotions, and communication of feelings interfere with identity formation, disrupt family relationships, and produce interactional dysfunction.

Bryan (1991) provided a comprehensive review of research on the attitudes of learning disabled children in adolescence toward themselves, their social competence, their communicative competence, and teachers’ judgments of their school behavior. In all areas, with the exception of knowledge of social norms, LD students were found to be less socially competent than their normally-achieving classmates. Voeller (1986) described children with non-verbal learning disabilities (NLD) who often fail to perceive social cues and thus have difficulty correctly interpreting their social environment. NLD children tended to push and crowd their peers, get into arguments or fights with peers, and have difficulty maintaining friendships. They were frequently considered strange or weird by their classmates.

Adolescents with learning disabilities are statistically at increased risk for juvenile delinquency and substance abuse, but no causal link has been established between learning disabilities and either of these conditions (Morrison & Cosden, 1997). The reported prevalence of learning disability among juvenile offenders generally ranges from 35% to 65% but due to the methodological limitations of research, these figures cannot be accepted at face value. No specific incidence figures for substance abuse among learning disabled adolescents were located. The risk for both juvenile delinquency and substance abuse is increased when LD is accompanied by hyperactivity and/or conduct disorder (Morrison & Cosden, 1997).

A number of hypotheses have been proposed to account for the link between LD and JD or substance abuse. The most frequently offered explanations suggest the following: low self-esteem and stresses associated with school failure lead to delinquency or substance abuse; LD youth are more susceptible to delinquent acts as a result of impulsivity, limited understanding of cause and effect, or poor social judgment; and young LD offenders lack the strategic planning skills to avoid being caught or to conceal their behavior when being questioned by legal authorities. A study by Waldie and Spreen (1993) provided some support for the susceptibility hypothesis that for those who are LD, comorbid JD is linked to impulsivity and poor judgment. For reviews of the literature on LD, JD, and substance abuse, refer to Murray (1976), Lane (1980), Skaret and Wilgosh (1989), and Morrison and Cosden (1997).

After reviewing relevant literature, Polloway, Smith, and Patton (1984) concluded that the social skill deficits observed in children with learning disabilities persists into adulthood. In a group of ninety-three adults diagnosed learning disabled at the Learning Disabilities Clinic at Northwestern University, 25% expressed concerns about social difficulties (Blalock & Johnson, 1987). Their social problems included difficulties in making and keeping friends as well as problems related to their specific disabilities, for example, following and participating in conversational exchanges, locating addresses, dancing, playing cards or word games, writing personal notes and letters, etc. In general, the adults with NLD experience the greatest social problems and social isolation but were not always aware of the social handicaps.

The frequently cited comorbidity of psychopathology and psychosocial difficulties with learning disabilities may result from several different direct or indirect mechanisms or a dynamic combination of mechanisms. As described by Hooper and Olley (1996) these mechanisms could include:

1. Behavioral disruption that arises directly from abnormal brain activity; 2. heightened exposure to failure, frustration, and social stigma due associated disabilities; 3. the possible effects of brain damage or anomalous neurodevelopment on subsequent temperament and personality development; 4. adverse family reactions ranging from overprotection to scapegoating; 5. the individual’s own reaction to being handicapped and its effect on his or her actual capacity to cope and compete; and 6. possible adverse effects from treatments themselves (e.g., lack of or poor treatment for specific learning problems) that may restrict normal activities and socialization (p. 164).
Although individuals with learning disabilities are increased risk for psychopathology and psychosocial deficits, there is wide variation in their patterns of socioemotional adjustment and groups do not dictate individual outcomes. The majority of children and adults with learning disabilities do not exhibit significant emotional disorders and function well in society. Morrison and Cosden (1997) view learning disabilities as a risk factor that in and of itself does not predict positive or negative outcomes. They propose that other internal and environmental risk/protective factors interact with the presence of a learning disability to mediate nonacademic outcomes such as emotional adjustment, adolescent problems (e.g., dropping out of school, juvenile delinquency, substance abuse) and adult adaptation. While the present discussion of socioemotional development and adjustment in persons with learning disabilities has been limited in scope, Mercer (1991) and the Learning Disabilities Quarterly: Special Issue, Fall, 1994, provide a more extensive review of these issues. Two additional types of comorbidity which occur with learning disabilities will be mentioned. First, one diagnostic category or subtype of learning disability may be viewed as comorbid with another. Since reading is an essential educational tool and since 80% of identified LD children have difficulty acquiring reading skills (Lerner, 1993) reading disability or dyslexia is often considered a child’s primary deficit with co-occurring deficits in other skill areas such as mathematics or graphomotor production considered secondary or comorbid deficits. It is possible for two distinct patterns or subtypes of learning disability to co-occur (e.g., deficits in phonemic awareness and processing with deficits in visual-spatial perception and spatial organization, affecting one or more academic skill areas. However, two co-occurring academic deficits (e.g., reading disability and math disability) may actually be manifestations of a single pattern of neurocognitive deficit with a shared information-processing bottleneck that impedes acquisition of skills in both academic areas (Lyytinen, 1995). Lyytinen cites two companion articles (Ackerman & Dykman, 1995; Räsänen & Ahonen, 1995) which examine the co-occurrence of reading and mathematical disorders. Light and DeFries (1995) assessed the genetic and environmental etiologies of comorbid reading and math deficits in identical and fraternal twin pairs. Their data indicated that approximately 26% of observed reading deficit was due to genetic factors which also influenced math performance.

A final category of comorbidity involves learning disability patterns which are observed in association with specific disorders or genetic syndromes, example, Klinefelter’s Syndrome, Turner’s Syndrome, Fragile X Syndrome, Tourette Disorder (Syndrome), neurofibromatosis, etc. Learning disabilities are a frequent comitant of syndromes associates with sex chromosome abberations. In Klinefelter’s Syndrome (XXY in males) affected individuals are of normal intelligence but frequently demonstrates specific deficits in verbal I.Q., auditory processing, efficient use of language, and reading (Berkow & Fletcher, 1992). Turner’s Syndrome (complete or partial absence of one X chromosome usually observed in females) impacts cognitive and learning processes to a variable degree. A typical phenotypic presentation involves characteristics associated with nonverbal learning disabilities such as visuo-spatial deficits, weakness in numerical and mathematical understanding, and social learning deficits (Mazzocco, 1996). Additional information about the learning ability/disability patterns which accompany specific genetic disorders is presented in other chapters of this text.

Comorbidity in neurocognitive disorders and learning disabilities is a complex topic which is currently receiving considerable emphasis in the research literature (e.g., Developmental Neuropsychology: Special Issue, 11(3), 1995). Future investigations of comorbidity will assist practitioners to view individuals with learning disabilities from a more holistic perspective, encouraging greater intervention efforts aimed at modifying the non-academic risk or protective factors which impede or enhance successful long-term outcomes. Further, explorations of comorbidity among learning disability categories or subotypes will contribute to the all important process of defining more distinct and empirically defensible LD subtypes. Subtypes of learning disabilities comorbid with other conditions have been identified in the contemporary empirical literature (Hendriksen et al., 2007). Recent studies have examined comorbidity of learning disabilities with attention deficit hyperactivity disorder and autism (Barnard, Muldoon, Hasan, O’Brien, & Stewart, 2008; Karande et al., 2007).

Interventions for Learning Disabilities

Since views differ regarding the nature and etiology of learning disabilities, views also differ about what constitutes appropriate and effective interventions for individuals with learning disabilities. Lyon and Moats (1988) discussed critical issues in the instruction of learning disabled students. Numerous authors, representing different theoretical orientations and instructional paradigms, have presented intervention methodologies developed or adapted for disabled learners. These include: psycholingusitc process or specific abilities approach (Johnson & Myklebust, 1967; Kirk & Kirk, 1971); behavioral approaches, including direct instruction and data-based instruction (Lindsley, 1971;
Lovitt, 1984; Marston & Tindal, 1995; White, 1986); cognitive approaches, including constructivism and instruction in learning strategies (Deshler & Lenz, 1989; "Implications," 1994; Swanson, 1993; Wong, 1991); and neuropsychological approaches (Rourke, Fisk & Strang, 1986; Hooper, Willis, and Stone, 1996). Mercer (1991) and Lerner (1993) have provided a lucid discussion of these instructional approaches and their application to individuals with learning disabilities. Mercer and Mercer (1993), Mather (1991), Mather and Jaffe (1992), and Lerner (1993) outline a broad array of specific teaching strategies and techniques which have been utilized successfully with atypical learners including those with learning disabilities.

Recent research is leading to better development of causal theories of learning disability and to promising avenues of intervention for the LD subtypes or specific information-processing weaknesses explicated by those theories. As Torgesen (1993) reports: "The two most completely developed current causal theories of learning disabilities are the nonverbal learning disabilities syndrome... and the theory of reading disabilities involving limitations in phonological processing" (p. 158).

A great deal of attention and research has been directed toward understanding phonological processing skills and their relation to the development of reading skills (Lyon, 1996b; Pennington, 1991; Shaywitz, 1996; Stanovich, 1993; Stanovich & Siegel, 1994; Torgesen, Wagner & Rashotte, 1994; Wagner & Torgesen, 1987). A number of well designed, longitudinal studies have documented the efficacy of instruction in phonological awareness and/or phonemic analysis and synthesis for the initial development of reading skills and for improving reading in reading deficient children (Ball & Blachman, 1988; Blachman, Ball, Black & Tangel, 1994 cited in Lyon, 1996b; Hatcher, Hulme & Ellis, 1994; Lundberg, Frost & Petersen, 1988).

At the conclusion of their research report, Hatcher, et al. (1994) suggest that children differ in their ability to acquire phonological competence and pose the question of how to best facilitate acquisition of underlying phonological skills. In this critical area of instruction, research-based practices are emerging. In a recent contribution, Torgesen, Wagner, and Rashotte (1997) discussed approaches to the prevention and remediation of phonologically-based reading disabilities. Research with the Auditory Discrimination In Depth Program (Lindemood & Lindemood, 1969) has shown that intensive instruction led to significant gains in reading and spelling skills for 281 subjects, ages five to fifty-five years old (Truch, 1994). Employing a new and promising approach, Merzenich, Jenkins, Johnston, Schreiner, Miller & Tallal (1996) acoustically modified speech to train sound discrimination abilities in children with language-based learning impairments. Subjects engaged in highly motivating discrimination tasks with speech stimuli altered by a computer algorithm which stretched the duration or increased the volume of sound elements critical to the discrimination process. After a few weeks' instruction, children in the study markedly improved their ability to discriminate phonemes and recognize both brief and fast sequences of speech stimuli. They also showed significant improvement in language comprehension abilities. While acoustically modified speech is a logically-conceived and an exciting intervention concept, experts in the field of dyslexia and learning disabilities, as reported by Travis (1996), suggested caution in regard to its potential benefits. Recent research has raised further doubts about the efficacy of this intervention (Watson, Kidd, Horner, Connell, Lowther, et al., 2003).

Just as well designed research can validate intervention practices and techniques for learning disabilities, it can also identify methods which are contraindicated for many LD students. In education as a whole, and in special education, there continues a great debate about the relative merits of code-oriented versus whole-language approaches to reading instruction (Foorman, 1995). Based on available research, most professionals in the field of learning disabilities have concluded that when used as the primary mode of instruction, the whole-language method is less effective than structured, explicit instruction in phonics for children with reading disabilities (Iverson & Tunmer, 1993; LDA Newsbriefs, 1995; Liberman & Liberman, 1992; Pressley & Rankin, 1994; Shapiro, 1992; Stanovich, 1994; Torgesen, et. al., 1994).

Summary

A neuropsychological perspective of learning disabilities provides an understanding of the underlying forces that impact rate and level of achievement across academic domains. Neuropsychological assessment has increasingly been utilized in academic settings. A neuropsychological perspective provides an understanding of the reasons why some children struggle academically. Pediatric professionals must be well-versed in academic assessment and school issues. This chapter provided an overview of the current literature concerning the history, etiology, definition, evaluation, treatment and efforts to accelerate achievement for youth with learning disabilities. There is an increasing appreciation within educational systems for the role underlying neuropsychological processes play in rates of achievement. An
increasing body of research is demonstrating that not only do brain structures and function impact learning but in a bi-directional manner achievement over time changes the brain.

References available upon request

Table 6.1. Categories of Academic Skills

<table>
<thead>
<tr>
<th>Auditory-Verbal</th>
<th>Visual Motor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conceptual</td>
<td>Visual non-verbal conceptual</td>
</tr>
<tr>
<td>Rote/Automatic</td>
<td></td>
</tr>
<tr>
<td>Auditory-motor</td>
<td>Letter perception</td>
</tr>
<tr>
<td>Auditory perceptual</td>
<td>Spatial organization and non-verbal integration</td>
</tr>
<tr>
<td>Rote auditory-sequential memory</td>
<td>Rote visual-sequential memory and retrieval</td>
</tr>
<tr>
<td>Rote and association memory and retrieval</td>
<td>Motor sequencing and fine motor control</td>
</tr>
</tbody>
</table>

Note: Adapted from table prepared by Sally I. Ingalls. Copyright 1991 by Neurology, Learning and Behavior Center, Salt Lake City, UT. Adapted with permission.
Table 6.2 - Levels of Processing Related to LD and Disability Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Auditory-Verbal</th>
<th>Visual-Motor</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Conceptual</strong></td>
<td>Language semantics; word meaning, definition, vocabulary</td>
<td>Social insight and reasoning; understand strategies of games, jokes, motives of others, social conventions, tact</td>
</tr>
<tr>
<td></td>
<td>Listening comprehension; understanding and memory of overall ideas</td>
<td>Mathematical concepts; use of 0 in +, -, x; place value; money equivalencies; missing elements, etc.</td>
</tr>
<tr>
<td></td>
<td>Specificity and variety of verbal concepts for oral or written expression</td>
<td>Inferential reading comprehension; draw conclusions</td>
</tr>
<tr>
<td></td>
<td>Verbal reasoning and logic</td>
<td>Understand relationship of historical events across time; understand scientific concepts</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Structure ideas hierarchically; outlining skills</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Generalization abilities</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Integrate material into a well-organized report</td>
</tr>
<tr>
<td><strong>Rote/Automatic</strong></td>
<td>Early speech; naming objects</td>
<td>Assemble puzzles and build with construction toys</td>
</tr>
<tr>
<td></td>
<td>Auditory processing; clear enunciation of speech; pronouncing sounds or syllables in correct order</td>
<td>Social perception and awareness of environment</td>
</tr>
<tr>
<td></td>
<td>Name colors</td>
<td>Time sense; doesn't ask, &quot;Is this the last recess?&quot;</td>
</tr>
<tr>
<td></td>
<td>Recall birthdate, phone number, address, etc.</td>
<td>Remember and execute correct sequence for tying shoes</td>
</tr>
<tr>
<td></td>
<td>Say alphabet and other lists (days, months) in order</td>
<td>Easily negotiate stairs; climb on play equipment; learn athletic skills; ride bike</td>
</tr>
<tr>
<td></td>
<td>Easily select and sequence words with proper grammatical structure for oral or written expression</td>
<td>Execute daily living skills such as pouring without spilling, spreading a sandwich, dressing self correctly</td>
</tr>
<tr>
<td></td>
<td>Auditory &quot;dyslexia&quot;: discriminate sounds, esp. vowels, auditorily; blend sounds to words; distinguish words that sound alike, e.g., mine/mind</td>
<td>Use the correct sequence of strokes to form manuscript or cursive letters</td>
</tr>
<tr>
<td></td>
<td>Labeling and retrieval reading disorder: auditory and visual perception okay but continually mislabels letters, sounds, common syllables, sight words (b/d, her/her)</td>
<td>Eye-hand coordination for drawing, assembling art projects, and handwriting</td>
</tr>
<tr>
<td></td>
<td>Poor phonic spelling</td>
<td>Directional stability for top/bottom and left/right tracking</td>
</tr>
<tr>
<td></td>
<td>Poor listening and reading comprehension due to poor short-term memory, especially for rote facts</td>
<td>Copy from board accurately</td>
</tr>
<tr>
<td></td>
<td>Labeling and retrieval math disorder: trouble counting sequentially; mislabels numbers (e.g., 16/60); poor memory for facts about numbers and sequences of steps for computation (e.g., long division)</td>
<td>Visual &quot;dyslexia&quot; confused when viewing visual symbols; poor visual discrimination; reversals/inversions/ transpositions due to poor directionality; may not recognize the shape or form of a word that has been seen many times before, i.e., &quot;word-blind&quot;</td>
</tr>
<tr>
<td></td>
<td>Recall names, dates, and historical facts</td>
<td>Spelling: poor visual memory for the nonphonetic elements of words</td>
</tr>
<tr>
<td></td>
<td>Learn and retain new scientific terminology</td>
<td></td>
</tr>
</tbody>
</table>

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