SPECIFIC LEARNING DISABILITIES: A NEUROPSYCHOLOGICAL PERSPECTIVE

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A dispersion in cognitive abilities is expected in normal populations. Specific learning disabilities would represent an extreme polarity in a continuum of normal cognitive dispersion. Three propositions relative to learning disabilities are advanced in the present paper. First, specific learning disabilities are expected to be found for diverse cognitive functions, even though some of these specific learning disorders have yet to be described in scientific literature. Second, it is noted that specific “factors” can be affected in cases of learning disabilities. Lastly, a parallel between focal neuropsychological syndromes and specific learning disabilities is proposed. Developmental learning difficulties would represent dysfunctional or dysmaturational defects; whereas neuropsychological syndromes represent a consequence of acquired structural brain pathology.

Keywords: Learning disabilities; dyslexia; individual differences; neuropsychological testing; brain dysmaturating

Specific learning disabilities, despite extensive research, continue to be a polemic area of study, generating controversy and confusion among the scientific community. Disagreement among researchers is evident even in the naming of learning disabilities; these disorders are variously referred to as “specific learning disabilities,” “developmental disabilities,” “learning disorders,” “developmental disorders,” or even “minimal brain dysfunctions” (Kinsbourne, 1985; Lubar & Deering, 1981; Morrison & Siegel, 1991).

In order to approach these learning defects, it is important to depart from the premise that cognitive abilities do not constitute a homogeneous phenomenon within a particular individual. Rather, a dispersion of cognitive abilities is expected.

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A degree of intrasubject dispersion in cognitive abilities is not the exception, but rather the rule (Anastasi, 1982; Cronbach, 1990). When clinical assessment reveals a discrepancy between normal levels of intellectual functioning and significant subaverage performance in a given cognitive domain, a specific learning disability should be assumed. Specific learning disabilities, therefore, refer to selective deficits for some particular type of learning.

In neuropsychology, a relatively modest interest in the analysis of individual differences has existed (e.g., Vernon, 1994). In a certain way, it is usually assumed that some basic cognitive abilities should be observed in any individual with a normal brain: Any normal person (i.e., any individual without evident brain damage) should recognize faces, discriminate language sounds, follow a route, possess a basic repertoire of learned movements, be able to copy a complex figure, etc. As a matter of fact, most neuropsychological tests infer a virtually perfect performance in normal subjects and, from a psychometric perspective, they have a quite low ceiling.

However, in clinical application, variability in the performance of any neuropsychological test is to be expected (Lezak, 1995). If, for instance, the Rey-Osterrieth Complex Figure (Osterrieth, 1944), or the Boston Naming Test (Goodglass, Kaplan & Weintraub, 1983) were administered to a normal population sample, by statistical definition some 2.5% of the subjects would obtain a score two standard deviations below the mean. Moreover, less than 0.5% (one out of 200) of the subjects would score three standard deviations below the mean. These subjects, from the vantage point of neuropsychological measurement, would present an abnormal performance with a probability level below .01. “Abnormal” in neuropsychology is usually regarded as “brain damage or dysfunction.” These subjects, inherently, do not present with structural brain damage. If these two tests (the Rey-Osterrieth Complex Figure and the Boston Naming Test) were both given, and assuming that the instruments represent noncorrelated tests, the number of “abnormal” subjects would increase twofold (one out of 100). If five noncorrelated tests were administered (assuming five independent factors) the number of “abnormal” subjects would be spuriously increased (about one out of 40). Conversely, from a statistical perspective, (assuming a normal distribution) an equivalent number of subjects with superior abilities two–three standard deviations above the mean) would be expected to occur in these same cognitive domains. Given, however, that neuropsychological tests have very low ceilings, they are not appropriate instruments to detect superior levels of cognitive ability.

In brief, there are important individual differences in the performance of neuropsychological tests developed to investigate brain integrity. It is expected that some percentage of the general population will obtain scores that, from a statistical perspective, should be considered “pathological” (Nadler, Mittenberg,
DePiano & Schneider, 1994). These individual differences should be taken into consideration in any attempt to understand brain organization of cognition in general and learning disabilities in particular.

**DYSLEXIA**

Dyslexia represents the prototype of a specific learning disability. Dyslexia has been defined by the World Federation of Neurology as a disorder manifested by difficulty in learning to read despite conventional instruction, adequate intelligence, and sociocultural opportunity. It is dependent upon fundamental cognitive disabilities which are frequently of constitutional origin (cf. Critchley, 1985). This is basically the same definition used for every type of specific learning disability. There are several points in the definition that merit particular consideration: (1) the difficulty is specific, and general intelligence is normal; otherwise, it would represent a nonspecific or global intellectual impairment (mental retardation); (2) it represents a cognitive defect, not a behavioral disorder; and (3) its origin can be “constitutional,” that is, it can appear in family groups; or simply, some genetic factors can be involved (Lubs et al., 1994; Olson, Wise, Conners & Rack, 1990; Vogler, DeFries & Decker, 1985).

Dyslexia can be associated with some “soft neurological signs,” such as synkinesias, difficulties to recognize fingers, right-left confusions, defects in visual scanning, etc. (Critchley, 1985; Pirozzolo, 1979). Furthermore, children with dyslexia exhibit difficulties with sequential series learning (letters, numbers, months, etc.), difficulties learning to read analog clocks, impairments in the use of spatial relations (e.g., up-down), and what has been named as the “developmental Gerstmann syndrome” (dysgraphia, dyscalculia, right-left recognition defects, and finger recognition deficits) (Benson & Geschwind, 1970; Biscaldi, Fischer & Aiple, 1994; Brachacki, Fawcett & Nicolson, 1994; Eden, Stein, Wood & Wood, 1994; Evans, Drasom & Richards, 1994; Lubar & Deering, 1981; Critchley, 1985; Singh, Broota & Gupta, 1993; Toth & Siegel, 1994) (Table I). In brief, dyslexia is much more than failure in learning to read.

<table>
<thead>
<tr>
<th>TABLE I</th>
<th>Some defects frequently associated with dyslexia.</th>
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<tr>
<td>Synkinesias</td>
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<td>Difficulties to recognize fingers</td>
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<td>Right-left confusions</td>
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<td>Disorders in eye-movements</td>
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<tr>
<td>Defective serialisation</td>
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<tr>
<td>Difficulties in learning to read the clock</td>
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<tr>
<td>Impairments in the use of spatial relations (e.g., up-down)</td>
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<tr>
<td>“Developmental Gerstmann syndrome”</td>
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</table>
It is usually accepted that dyslexia does not represent a homogeneous disorder, and several subtypes of dyslexia can be distinguished (Bakker, 1979; Boder, 1973; Bravo-Valdivieso, 1980; Masutto, Bravar & Fabbro, 1994; Mattis, French & Rapin, 1975; Rourke, Bakker, Fisk & Strang, 1983; Rispens, Van der Stege & Bode, 1994) (Table II). Furthermore, dyslexia can depend of the idiosyncrasies of a linguistic writing system (Bravo-Valdivieso, 1988; Wimmer, 1993). Hence, dyslexia in Spanish is not completely equivalent to dyslexia in English or Chinese. Cognitive abilities required to read in Spanish, English, and Chinese do not seem to have perfect correspondence.

Despite the different nosologic classifications that have been proposed, it seems evident that at least two distinct types of dyslexia can be identified: (1) dyslexia resulting from difficulties in auditory processing and in matching graphic symbols with language sounds; and (2) dyslexia resulting from deficits in the visuo-perceptual processing, in the orderly ability to scan the written text, and to obtain a simultaneous recognition of those graphemes included in a word. Additionally, reading difficulties could also eventually be associated with some retardation in language acquisition, and difficulties in writing may be correlated with deficits in graphomotor abilities.

<table>
<thead>
<tr>
<th>TABLE II</th>
<th>Some classifications of dyslexia.</th>
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<tbody>
<tr>
<td>1.</td>
<td>P-type dyslexia</td>
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<td>2.</td>
<td>L-type dyslexia</td>
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<td>(Bakker, 1979)</td>
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<tr>
<td>1.</td>
<td>Dysphonetic dyslexia</td>
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<td>2.</td>
<td>Dyseidetic dyslexia</td>
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<td>(Boder, 1973)</td>
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<tr>
<td>1.</td>
<td>Developmental phonological dyslexia</td>
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<td>2.</td>
<td>Developmental surface dyslexia</td>
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<tr>
<td>(Ellis, 1993)</td>
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<tr>
<td>1.</td>
<td>Audiophonic dyslexia</td>
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<td>2.</td>
<td>Visuospatial dyslexia</td>
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<tr>
<td>(Johnson &amp; Myklebust, 1971)</td>
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<tr>
<td>1.</td>
<td>Perceptual deficits</td>
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<tr>
<td>2.</td>
<td>Defects in articulatory and graphomotor abilities</td>
</tr>
<tr>
<td>3.</td>
<td>Dyslexia associated with language problems</td>
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<tr>
<td>4.</td>
<td>Difficulties in temporal sequencing</td>
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<tr>
<td>(Mattis et al., 1975)</td>
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<tr>
<td>1.</td>
<td>Auditory-linguistic subtype</td>
</tr>
<tr>
<td>2.</td>
<td>Visual-spatial subtype</td>
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<tr>
<td>(Pirozzolo, 1979)</td>
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<tr>
<td>1.</td>
<td>Central auditory processing dyslexia</td>
</tr>
<tr>
<td>2.</td>
<td>Visual-perceptual dyslexia</td>
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<td>(Quiros, 1964)</td>
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</table>
Consequently, it seems reasonable to conclude that: (1) there are at least two different cognitive defects (linguistic and visuospatial) that can eventually result in an inability or delay in normal reading skill acquisition; and (2) dyslexia is not a specific impairment in the acquisition of reading ability. Learning-to-read difficulty is just a particular manifestation of some, more fundamental cognitive deficits which can eventually result in difficulties with learning to read in a normal way.

Reading represents a supramodal skill, there are cognitive abilities that serve support functions and abilities that are fundamental in reading acquisition. It does not seem reasonable to infer that a specific cognitive ability governs the ability to read. Reading represents a complex functional system, requiring, and based on different cognitive abilities ("factors"). Therefore, it does not seem reasonable to attribute reading to a specific ability, just as it is unreasonable to postulate that a specific cognitive factor governs one's ability to use computers. As a matter of fact, different authors have supported this interpretation of dyslexia: Dyslexia is just a single manifestation of one or several more fundamental cognitive defects (see, Kinsbourne, 1985; Pirozzolo, 1979; Stanovich, 1994).

It is interesting that a specific intellectual profile has been reported in cases of dyslexic children (Naidoo, 1972). This profile becomes most evident when using standard intellectual measures such as the Wechsler Intelligence Scale. According to this profile, known as the “ACID pattern,” it is expected that Arithmetic, Coding, Information and Digit Span subtests are significantly depressed in dyslexic individuals, whereas all other subtest scores are normal or even superior (Rugel, 1974; Kolb & Whishaw, 1990). However, this particular profile refers to dyslexia in general, and does not distinguish between dyslexia subtypes. If dyslexia subtypes were separated, there is greater likelihood that intellectual profiles would become even more specific.

NONVERBAL LEARNING DISABILITIES

A “nonclassical” learning disability could be taken as a second example. Johnson and Myklebust (1971) identified a group of children who performed poorly in arithmetic and also were impaired in interpreting social signals, pretending and making abstractions. They labeled these disorders as “nonverbal learning disabilities,” and emphasized the impaired social perception in these children. Later studies supported this proposal (e.g., Brumbach, Staton & Wilson, 1983; Denckla, 1983; Loveland, Fletcher & Baile, 1990; Rourke, 1989; Rourke & Finlayson, 1978; Voeller, 1986, 1991; Tanel et al., 1987). Weintraub and Mesulam (1983) analyzed this nonverbal learning syndrome that begins early in life in a 14-patient sample and is characterized by emotional and interpersonal difficulties, shyness, visuospatial disturbances, and inadequate paralinguistic communicative abilities (e.g., inappropriate prosody).
Examination revealed neurologic and neuropsychological signs consistent with right-hemisphere dysfunction. In many of these individuals, a family history of similar symptoms was disclosed. The authors proposed that, just as developmental involvement of the left hemisphere may produce dyslexia, dysfunction of the right hemisphere may lead to chronic emotional difficulties, disturbances in interpersonal skills, and poor visuospatial abilities. Tranel, Hall, Olson, and Tranel (1987) pointed to a similar spectrum of behaviors in these individuals (chronic social isolation, shyness, withdrawal, visual spatial deficits, poor eye contact, impaired emotional prosody and gestures); these authors also attributed the syndrome to right hemisphere dysfunction.

This proposal is particularly important for any theoretical interpretation of learning disabilities: learning disabilities represent a diverse collection of syndromes and can go far beyond the classic disturbances conventionally taught in academic institutions.

**DSM-IV APPROACH TO LEARNING DISABILITIES**

It is important to take into account how learning disabilities are considered in the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (DSM-IV) (Table III). DSM-IV (American Psychiatric Association, 1994) includes a section designated as “Learning Disorders” (formerly named “Disorders in Academic

| TABLE III  Classification of developmental disorders  (“Disorders Usually First Diagnosed in Infancy, Childhood, or Adolescence”; American Psychiatric Association, 1994). Only Learning Disorders, Motor Skill Disorders, and Communication Disorders, could be considered as specific learning disabilities.  

| Mental retardation  
| Learning Disorders  
| Reading disorder  
| Mathematic disorder  
| Disorder of written expression  
| Motor skills disorders  
| Developmental coordination disorder  
| Communication disorders  
| Expressive language disorder  
| Mixed receptive-expressive language disorder  
| Phonological disorder  
| Stuttering  
| Pervasive developmental disorder  
| Attention deficit and disruptive disorder  
| Feeding and eating disorders of infancy or early childhood  
| Tics disorders  
| Elimination disorders  
| Other disorders of infancy, childhood, or adolescence |
Abilities”) in the chapter devoted to the “Disorders Usually First Diagnosed in Infancy, Childhood, or Adolescence.” DSM-IV presents that a diagnosis of a learning disorder should be made, “when the individual’s achievement on individually administered, standardized tests in reading, mathematics, or written expression is substantially below the expected for age, schooling, and level of intelligence” (p. 46). Substantially below is further defined as, “a discrepancy of more than two standard deviations between achievement and IQ.” Three subtypes of learning disorders are distinguished in the DSM-IV: Reading Disorder, Mathematics Disorder, and Disorder of Written Expression. The chapter of “Disorders Usually First Diagnosed in Infancy, Childhood, or Adolescence” additionally recognizes Motor Skills Disorder (Developmental Coordination Disorder), and a group of four Communication Disorders (Expressive Language Disorder, Mixed Receptive-Expressive Language Disorder, Phonological Disorder, and Stuttering).

Several observations could be mentioned:

(1) The criterion used in the DSM-IV for classifying learning disabilities represents a purely school-performance based criterion. Thus, if reading difficulties exist, the disability as a consequence a “Reading Disorder” (dyslexia) is diagnosed; if difficulties are observed in writing, then a “Disorder of Written Expression” exists; and finally, if defects are observed in calculation abilities, the diagnosis of “Mathematics Disorder” is made.

(2) Because of a reliance on purely academic criteria, this classification by necessity is contingent upon which learning modalities are considered as most functional in our current educational system. For instance, constructional difficulties, musical disabilities, and defects in social skill acquisition are not considered learning disorders.

(3) Finally, it is evident that academic abilities are not a good referent point for neuropsychology. To read or to perform arithmetical operations represents complex functional systems. In neuropsychology it is well known that different types of reading and calculation disorders can be observed in case of brain pathology; that is, there exist different subtypes of alexias and acalculias (e.g., Ardila & Rosselli, 1990; Benson & Ardila, 1996; Ellis, 1993). The human brain is specialized for a particular type and level of information processing, potentially useful for reading or performing calculations (Benson, 1994). But the brain is not per se specialized for reading or for performing calculations (Ardila, 1993).

MORE LEARNING DISABILITIES

During recent years the idea that, not only most “classical” learning disabilities (i.e., dyslexia, dysphasia, and dyscalculia) can be distinguished, but also many other types of learning difficulties, has tended to prevail (Rourke, 1988). The
existence of a variety of specific learning difficulties have been proposed in perceptual recognition (e.g., prosopodysgnosia, or simply, developmental prosopagnosia; McConachie, 1976), in memory abilities (specific developmental memory difficulties or developmental dysmnesias; De Renzi & Lucchelli, 1990), etc. Moreover, if learning disabilities were interpreted as a result of certain brain dysmaturation or dysfunction, it could be assumed that, theoretically, as many developmental learning disabilities as basic cognitive abilities must exist. For instance, although it has yet to be described in the scientific literature, a dysmusia or developmental amusia must theoretically exist. Furthermore, it should be possible to find learning difficulties equivalent to every known neuropsychological syndrome. Or, more exactly, a parallel between learning disabilities and neuropsychological syndromes should exist. Thus, developmental learning difficulties represent dysfunctional or dysmaturation defects; whereas neuropsychological syndromes represent a consequence of a structural brain pathology (Table IV).

TOWARD AN EXPLANATION OF LEARNING DISABILITIES

Obtaining an acceptable explanation of learning disabilities has not been an easy task. Efforts were initially focused on the analysis of dyslexia (Fletcher et al., 1994). Abnormalities in language lateralization and defects in handedness were

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Learning disability</th>
<th>Factor</th>
<th>Author</th>
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<tbody>
<tr>
<td>Anarithmetia</td>
<td>Discalculia</td>
<td>Numerical</td>
<td>Several</td>
</tr>
<tr>
<td>Prosopagnosia</td>
<td>Developmental</td>
<td>Visuospatial</td>
<td>McConachie, 1976</td>
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<tr>
<td></td>
<td>prosopagnosia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spatial agnosia</td>
<td>Spatial-type</td>
<td>Visuospatial</td>
<td>Johnson &amp; Myklebust, 1971</td>
</tr>
<tr>
<td></td>
<td>learning disabilities</td>
<td></td>
<td>Not described yet</td>
</tr>
<tr>
<td>Amusia</td>
<td>Dysmusia</td>
<td>Musical</td>
<td>Several</td>
</tr>
<tr>
<td>Alexia w/o agraphia</td>
<td>Linguistic</td>
<td>Visual-phonological</td>
<td>Several</td>
</tr>
<tr>
<td></td>
<td>dyslexia</td>
<td>integration</td>
<td></td>
</tr>
<tr>
<td>Alexia w/o agraphia</td>
<td>Visuospatial</td>
<td>Visual perception</td>
<td>Several</td>
</tr>
<tr>
<td></td>
<td>dyslexia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apraxia</td>
<td>Developmental</td>
<td>Praxic</td>
<td>Ajuriaguerra &amp; Stambak, 1969</td>
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<tr>
<td></td>
<td>dyspraxia</td>
<td></td>
<td>Not described yet</td>
</tr>
<tr>
<td>Left convexital</td>
<td>Not described</td>
<td>Verbal fluency</td>
<td>Benson &amp; Geschwind, 1970</td>
</tr>
<tr>
<td>prefrontal</td>
<td></td>
<td>Divergent thinking</td>
<td></td>
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<tr>
<td>Gerstmann syndrome</td>
<td>Dev. Gerstmann</td>
<td>Spatial knowledge</td>
<td>De Renzi &amp; Lucchelli, 1990</td>
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<tr>
<td></td>
<td>syndrome</td>
<td>through language</td>
<td></td>
</tr>
<tr>
<td>Amnesia</td>
<td>Dev dismnnesia</td>
<td>Memory</td>
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Nonetheless, it is a self-evident assumption that a learning-disabled child must present some central nervous system dysfunction, specifically in those brain areas normally involved in the dysfunctional ability (Witelson, 1977). This assumption has led to the search of brain abnormalities responsible for the specific difficulty in learning. Galaburda and Kemper (1979) reported evidence of neural developmental abnormalities (neural migration defects) in the left hemisphere language association areas, of an individual suffering dyslexia. This observation has been further corroborated (Galaburda et al., 1985; Humphreys, Kaufmann & Galaburda, 1990).

Other brain abnormalities have also been demonstrated in dyslexic individuals (Duane, 1992; Galaburda, 1993, 1994; Hier, Lemay, Rosenberger & Perlo, 1978; Hynd et al., 1990; Jernigan, Hesselink, Sowell & Tallal, 1991; Leonard et al., 1993; Schultz et al., 1994). Using magnetic resonance imaging, Leonard et al. (1993) compared three subject groups: dyslexics, unaffected relatives, and controls. They reported that all groups had left-sided asymmetry for the temporal bank and right-sided asymmetry for the parietal bank. The group with dyslexia had exaggerated asymmetries, owing to a significant shift of right planar tissue from the temporal to parietal bank. They also had a higher incidence of cerebral anomalies bilaterally. Activation levels in the thalamus, particularly in the medial geniculate body, have been proposed to be different between normal readers and dyslexic children (Roush, 1995).

Employing brain imaging techniques, it has also been possible to demonstrate in dyslexic subjects some anomalous patterns of brain activation during verbal tasks (Rumsey, Zametkin, Andreason & Hanahan, 1994). Thus, using positron emission tomography, Rumsey et al. (1992) observed that normal readers activated left temporoparietal cortex during a phonological task, but not during a non-phonologic attention task. Dyslexic subjects failed to activate those temporoparietal regions activated in normals during phonological detection, but did not differ from controls in these regions during rest or attentional tests. Flowers, Wood, and Naylor (1991) using measures of regional cerebral blood flow, observed that dyslexics fail to present a normal cortical activation in the left hemisphere during verbal tasks (verbal memory, auditory perception and spelling analysis). Hagman et al. (1992) using positron emission tomography, also demonstrated in dyslexic adults a failure to present normal temporal activation during verbal tasks.

Taken together, all these research studies demonstrate the existence of subtle abnormalities (structural and functional) in dyslexic subjects' brain. Theoretically, similar abnormalities (albeit, in other brain sites), should also be found for other learning disabilities (e.g., dysphasia, dyscalculia, visuospatial disabilities, etc.). These abnormalities would be corroborating that, individual differences in
cognition are correlated with individual differences in subtle brain morphology and activity patterns. As a consequence, a parallel between specific learning disabilities and neuropsychological syndromes is expected (see Table IV).

Geschwind and Behan (1982), and later Geschwind and Galaburda (1987) proposed an integrative theory about learning disabilities. They attempted to relate dyslexia, left-handedness, and immune disorders. They hypothesized that an elevated level of prenatal testosterone, acting independently on the embryonic thymus and the embryonic brain, is the casual link underlying the proposed association among the three conditions. The action on the thymus is responsible for an increased risk of immune disorders, whereas the effects on the brain lead to an increase probability of anomalous lateralization. Left-handedness and dyslexia are thought to result from an altered left-hemisphere development. This hypothesis attracted a great deal of attention and significantly stimulated research in the area (e.g., Bishop, 1990; Hugdahl, Synnevaag & Satz, 1987; Tonnessen, Lokken, Hoen & Lundberg, 1993). The Geschwind-Bechar-Galaburda hypothesis, however, should be regarded as a research proposal requiring further analysis and investigation.

WHAT CAN BE LOCALIZED IN THE BRAIN?

Contemporary neuroimaging techniques have provided valuable information about brain activity during the performance of different cognitive tasks. Based on measures of focal brain metabolism, positron emission tomography (PET) allows one to visualize levels of brain activity and focal involvement during different conditions. Some examples presented as illustrations.

It has been reported that during the performance of a relatively simple task, (such as recognition of syllables) an increase in activation is observed in a very restricted area of the left temporal lobe corresponding to the primary auditory cortex (Black & Behrmann, 1994). During the passive viewing of a black and white checkerboard, the active regions occur along the boundary of the calcarine fissure (primary visual area) (Hirsch, 1994). In contrast, in performing a facematching task a broader regional activation in both occipital lobes is observed, particularly in the right occipital lobe (Chertkow & Bub, 1994). Viewing and hearing nouns, as well as generating verbs activates the left occipital, temporal and frontal areas respectively (Raichle, 1994). In all cases, changes in the level of activation are limited to some rather specific brain areas. It essence, these findings advance the hypothesis that specific brain areas are involved in these relatively simple tasks.

When a "complex" task is utilized, (e.g., reading aloud) a more complex matrix of activated cortical areas is recorded. Different brain areas provide specific
contributions to performance during a reading task: occipital (visual perception), temporal (language decoding), and Broca’s frontal area (language control and production) (Pettersen, Fox, Snyder & Raichle, 1989). For each function, a limited cortical region is specifically activated, whereas other brain areas are only partially active (Price et al., 1994). While speaking, activation not only occurs in the mouth area along the primary motor cortex in the left hemisphere, but also in the superior temporal lobe, and in the supplementary motor area.

Briefly, therefore, it could be stated that: (1) during relatively simple tasks (e.g., listening syllables, watching a checkerboard), brain activity changes are restricted to rather specific brain areas; and (2) during complex cognitive tasks (e.g., reading), a participation of multiple brain areas is observed. Each of these areas makes its particular contribution to the whole system.

Clinical observation supports the assumption that apparently different tasks (e.g., to solve numerical problems and to recognize fingers) can be impaired as a consequence of brain pathology originating from a common neural substrate. Children with difficulties in learning to read, may also present other rather different difficulties (see above). Conversely, acquired brain pathologies of different topographic localization can impair performance in exactly the same task (e.g., the ability to solve numerical problems can be disrupted in cases of quite diverse brain damage locations; Ardila & Rosselli, 1990). A certain “common factor” evidently should be contained in those apparently different, but simultaneously altered, tasks in the case of a single focal pathology (e.g., to solve numerical problems and to recognize the fingers). Broca’s aphasia can serve as an illustrative example.

It is usually recognized that Broca aphasia has two different distinguishing clinical features: (a) a motor component (lack of fluency, disintegration of the speech kinetic melodies, verbal-articulatory defects, etc.); and (b) Agramatism (e.g., Benson & Ardila, 1996; Luria, 1976; Goodglass, 1993; Kertesz, 1985). If both defects are simultaneously observed (i.e., they are very highly correlated), it simply means that both deficits are just two different manifestations of a single underlying defect. It is not easy to understand which central defect could be the single “factor” responsible for these two clinical manifestations (e.g., “inability to sequence expressive elements”). However, a single common factor underlying both defects should be assumed. Most likely, Broca’s area is not specialized in producing language per se, but rather a certain activity in the neural system that is fundamental to the performance of skilled movements required for language production, and also, language syntax. It is interesting to note that deaf-mute subjects (who, as a consequence of never having produced highly skilled verbal articulatory movements) present with a virtually total impossibility to learn, understand, and use language grammar (Poizner, Klima & Bellugi, 1987). Perhaps a lack of
normal verbal articulatory development is inherently associated with a lack of normal grammatical development.

It has been assumed that the cerebral cortex is organized in modules, which probably range in size from a hundred thousand to a few million neurons (Carlson, 1994). Each module receives information from other modules, perform certain processing and then pass the information to other(s) modules (De Valois & De Valois, 1988; Livingstone & Hubel, 1988). It has been proposed that in the primary visual cortex there are some 2,500 modules, each measuring approximately 0.5 x 0.7 mm. The neurons in each module are devoted to the analysis of various features in one specific portion of the visual field. However, it is reasonable to expect that several modules can simultaneously participate in each specific type of information processing, i.e., that modules could be grouped into supramodules, or simply, higher order modules. The activity or information processing performed by a high order module would roughly correspond to a “factor.”

Higher order modules may potentially be useful for apparently different types of abilities (e.g., skilled movements required for language production and language syntax). Potentially, they might be also useful for other nondeveloped or not analyzed yet, activities. The same information processing level can be used in different functions.

Reading can illustrate the complexity of brain organization in any psychological process (functional system). Reading is based in certain fundamental abilities or factors (Luria, 1966, 1973, 1976) (e.g., complex shape perception, cross-modal learnings, etc.) already existing 5,000 years ago, and by nature, existing in illiterate individuals. What might be “localizable” in the brain is not reading per se, but certain basic abilities or factors (information processing levels depending on some specific assemblies or high order modules) required to read, albeit not only to read. Learning to read supposes the existence of the neural activation and long-term potentiation (Levy & Steward, 1983; Perkins & Teyler, 1988) of several higher order modules, functioning as supramodule information processing systems (visual information related with the perception of letter shapes, cross-modal association between letters and language sounds, etc.).

Identical information processing levels, i.e., the same operation, might be useful for apparently different types of cognition. As an example, it has been reported that painting, playing chess, mechanics, and music abilities may be impaired in cases of right hemisphere damage. Damage in those same areas in an Eskimo or Amazonian Indian would imply an impossibility to move around the snow or the jungle (Ardila, 1993). Brain assemblies (“basic circuitry”) or modules able to perform a specific information processing level (“factor”) can be potentially useful for many different types of cognition. However, any type of complex cognition will require the orchestration of multiple factors (multiple brain areas; that is, a
particular “brain system” supporting the corresponding “functional system”)

Focal brain pathology, or brain dysfunction in cases of specific learning dis-
abilities, would imply the abnormal activity of a specific brain area, associated
with a particular information processing level. Pathology affecting a given corti-
cal area can cause the entire functional system (e.g., reading) to fail. Hence, in
complex psychological processes the whole system can fail as a result of pathol-
ogy or dysfunction at different levels. It is not surprising then, to find that there
are different types of alexia and different types of developmental dyslexia.

SOME TENTATIVE CONCLUSIONS

It seems evident that when attempting to explain specific learning disabilities
some assumptions must be taken into consideration:

1. From a psychometric perspective, there is a dispersion in cognitive abilities,
among different individuals (intersubject variability), and also within each indi-
vidual (intrasubject variability). A normal IQ does not imply that homogeneity in
intellectual abilities exists. Learning disabilities would represent an extreme polarity
on a continuum of normal dispersion. Therefore, from a psychometric perspec-
tive, specific learning disabilities are not only comprehensible but also evident.

2. In our current educational system, there are certain highly valuable types of
learning. This is particularly true for language, reading, and mathematics.
Conversely this is generally not true for spatial or musical learning. Devel-
opmental dysphasia, dyslexia and dyscalculia represent important obstacles
for an appropriate academic performance in our contemporary educational sys-
tem. Spatial and musical abilities most often are regarded as less important, or
secondary abilities. In the academic environment, it is not likely that a child will
have to leave school or be referred for professional treatment because of limited
spatial or musical abilities.

3. In dyslexia it has been demonstrated that subtle abnormalities exist in the
brain of these learning disabled individuals. It seems reasonable to assume that
functionally similar abnormalities may be found in other types of specific learn-
ing disabilities.

4. A parallel between specific learning disabilities and neuropsychological syn-
dromes may be proposed. Neuropsychological syndromes represent acquired cog-
nitive defects associated with structural brain pathology; specific learning
disabilities represent developmental dysfunctions of specific brain areas.

5. Simple psychological activities are correlated with the increased activation
of relatively limited brain areas. Complex psychological processes (“functional
systems”) require the participation of multiple brain areas (“brain systems”). Moreover, focal brain pathology or dysfunction may result in the failure of the whole system.

References


