A Neurodevelopmental Perspective on Specific Learning Disabilities

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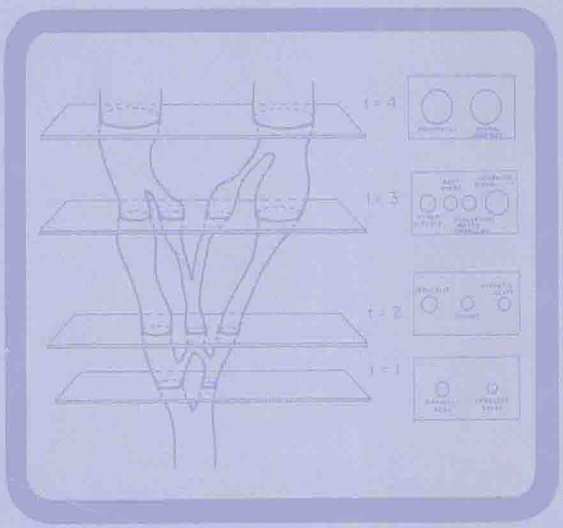

by Pasquale J. Accardo

A NEURODEVELOPMENTAL PERSPECTIVE ON SPECIFIC LEARNING DISABILITIES

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Preface

> But what has been said once, can always be repeated.
>
> Zeno of Elea

> Do not fear to repeat what has already been said. Men need [the truth] dinned into their ears many times and from all sides.
>
> René Laënnec

> Everything that needs to be said has already been said. But since no one was listening, everything must be said again.
>
> André Gide

> What I tell you three times is true.
>
> Lewis Carroll

The field of learning disabilities is one of great confusion for all involved—the children, their parents, and various professionals. A wise pediatrician once said that when the experts in a field are themselves confused, then confusion represents the epitome of knowledge. Indeed one is very skeptical of claims to have discovered *the* answer, the secret remedy, the magic pill, the technical miracle that generations of competent workers failed to uncover. The unknowns (perhaps with some unknowables) far outnumber the knowns. This book will be at fault if it fosters the illusion of a clearly demarcated area with lucid signposts; the state of the art is characterized by confusion and ignorance.

Significant advances are slow in coming. In a standard textbook on reading disorders (Bond and Tinker, 1973), approximately two-thirds of the citations are pre-1960, over 40% pre-1950, and about one-quarter pre-1940. A number of older books have endured (e.g., Money, 1962b; 1966a; P. Wender, 1971); even Huey's (1908) seminal work describes surprisingly current con-

cerns. Probably the greatest single advance in the past half-century has been the recognition that no one discipline has the competence to manage the learning-disabled child. Many edited texts outline a team approach to the diagnosis and treatment of such children: Adamson and Adamson (1979), Flower, Gofman, and Lawson (1965), Newton (1978), and Tarnopol (1969a). The present volume focuses exclusively on the pediatrician's role and attempts to provide him with a general introduction to the entire field. (It is like one of the half-dozen blind wise men expostulating on the nature of an elephant.)

Almost every sensory modality has been implicated in the etiology of learning disabilities; perhaps only olfaction and taste have (thus far) been spared the typical uncontrolled study documenting a statistically significant (but clinically meaningless) correlation with learning problems or hyperactivity. Although cognitive models have not yet yielded significant practical benefits (Schroeder, Schroeder, and Davine, 1978), a rationalist (cf. N. Chomsky, 1973) or linguistic approach seems to hold the greatest promise.

There exists a bewildering array of possible treatments: patterning exercises, dietary fads, yoga, alpha wave conditioning; one of the latest methods spins children around until they vomit. "The list is endless as well as exasperating. We must ask ourselves why some of these remedies (so reminiscent of snake oil) become so widely accepted. It is because the voice of the physician is not heard in the land of education. This very silence is taken as approval" (Newton, 1976). Again, "physician involvement is essential to countermand the multitude of cure-alls advocated by cultists preying upon the sensitivity of distraught parents" (Keys, 1977). Some basic assumptions need to be questioned. Werry's insight remains valid: "The area of learning disorders is beginning to resemble past efforts at curing mental retardation, with enthusiasm outrunning both theory and evaluation of therapy. Just because learning-disabled children are of normal intelligence does not mean that their deficit of learning is necessarily any more treatable than general learning disability (that is, mental retardation)" (Menkes and Schain, 1971).

In the first edition of *Drug Evaluations,* the AMA Council on Drugs (1971) was courageous enough to characterize many drugs as "irrational"; consider the pathos with which future generations will look back on our present folly if we lack a Weyer to fit that most appropriate epithet to even the more accepted therapeutic

modalities in the field of learning disabilities. Sufficient outcome studies exist to suggest that the emperor has no clothes.

Though "inconclusive, trivial or sadly incomplete" (Rourke, 1975), the vast learning disability literature does reflect some measure of clinical insight, which ought not be discarded for theoretically interesting but unproved novelties. The present handling of this literature may sometimes recall the style of Eriugena, who often resorted to authority in support of heterodox positions.[1]

The use of global diagnostic categories, such as minimal brain dysfunction (MBD) and specific learning disability (SLD), is analogous to the use of "cerebral palsy" to encompass a heterogeneous group of motor disorders with a common factor—a central nervous system etiology. If one employs a sufficient number of refined tests and measures, it is possible to define any syndrome out of existence (Dykman, Peters, and Ackerman, 1973). Although replacement of the acronyms MBD and SLD by more meaningful terms is devoutly to be wished and finds almost universal support, there is absolutely no agreement on exactly what to substitute for them. Everyone suffers from his own pet classification scheme of unproved utility.

Karl Popper said that science begins with myths and with the criticism of myths; one might add that science continually creates new myths and that the problem of individual differences may very well be exaggerated by our current scientific mythology. Parents and professionals need to be able to tolerate a high degree of uncertainty. The physician who can foster a loving acceptance of the child who paces to a different drummer is not practicing something called advocacy but rather something called pediatrics.

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[1]E. Gilson, 1955, *History of Christian Philosophy in the Middle Ages*, Random House, New York; cf. J. J. O'Meara, 1969, *Eriugena*, Mercier Press, Cork.

For

Jennifer

who read before kindergarten

Matthew

who refused to read until everyone else did

Claire

who prefers to eat books

and Patricia

who helped with the reading for this book.

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Contents

THE ANALOGICAL CONTEXT

> The doctor said that so-and-so indicated that there was so-and-so inside the patient, but if the investigation of so-and-so did not confirm this, then he must assume that and that. If he assumed that and that, then . . . and so on.
>
> Tolstoy
> *The Death of Ivan Ilyich*

Children fail to learn in school for many reasons. Factors such as impaired general health, poor nutrition, frequent truancy (or school absence for other reasons), and sociocultural deprivation may all play a role. Sensory loss (defective vision or hearing) is investigated intensively, found infrequently, and causally associated with childhood learning problems even less frequently. Emotional disturbances are extremely rare in the etiology of learning disabilities but become increasingly more common with age as secondary manifestations of inappropriate class placement and overly high parent/teacher expectations. Of all those children referred for medical evaluation of school failure, the majority fall into the intrinsic or organic group (Table 1). But it must be remembered that the greater part of school underachievers are never referred for such an assessment. There is a preselection of cases to exclude those children with the more obvious environmental etiologies and motivational problems. When such a child is referred for pediatric evaluation, it is usually because someone has noted that he: 1) has normal ability, 2) is trying his best, and yet 3) persists in being unable to learn adequately with the usual teaching methods.

Paine (1962, 1968) described the brain as exhibiting four major areas of dysfunction, each of which in turn could manifest milder variants of disorder: cerebral palsy is a major motor impairment, the choreiform syndrome or the clumsy child syndrome reflect minor motor involvement; mental retardation is a major cognitive impairment, borderline intelligence (the slow learner) reflects minor cognitive involvement; cortical blindness or central auditory imperception is a major sensory impairment, visual-perceptual disabilities reflect minor sensory involvement; a convulsive disorder is a major electrical impairment, subclinical epilepsy (an abnormal EEG without clinical seizures) reflects minor electrical involvement. The pediatric assessment focuses on these different areas of cerebral function since the cognitive and motor

Table 1. Etiologic factors in a clinic population

Extrinsic (Environmental)		Intrinsic (Organic)	
General	Specific	General	Specific
Sociocultural deprivation, 15%–50%	Emotional block, 1°:1% 2°:25%–90%	MBD, 75%	Dyslexia, 5%

The extrinsic/intrinsic dimensions refer to cause; the general/specific dimensions to effect; the diagnostic labels are examples of entities commonly occurring in each category. This division is not exclusive, and the figures will vary a great deal depending on the nature of the physician's referral practice, the utility of his reports to the multidisciplinary evaluation, and the sensitivity of local teachers to an organic contribution to learning problems.

symptoms of gross brain damage syndromes like mental retardation and cerebral palsy are mirrored in the milder signs associated with minimal brain dysfunction (MBD) (Table 2). The physician's contribution to the multidisciplinary evaluation is to view the child as occupying a point somewhere on the spectrum of chronic neurological handicaps (Figure 1). A careful investigation for evidence of organic involvement should not, however, be taken to imply that *all* cases of school problems with *some* signs of minor neurological dysfunction are, therefore, completely biologically determined; it should, rather, indicate an attempt to give appropriate weight to nonenvironmental causal factors.

While there remains much confusion over diagnostic labels, a basic underlying agreement on the concept of learning disability

Table 2. Brain dysfunction syndromes

	Mental retardation	Cerebral palsy	MBD
Perinatal risk factors	+	+	±
Genetic component	+	−	±
Irritable/colicky infant	−	+	±
Language delay	+ +	−	±
Dysarticulation	−	+	±
Gross neurological findings	±	+ +	−
Soft neurological findings	±	+ +	+
Abnormal EEG	+ +	+ + +	+
Subscore scatter	±	+ +	+
Perceptual deficits	±	+ +	+
Hyperactivity/short attention span	±	+ +	+
Cognitive deficit	+ + +	+ +	+

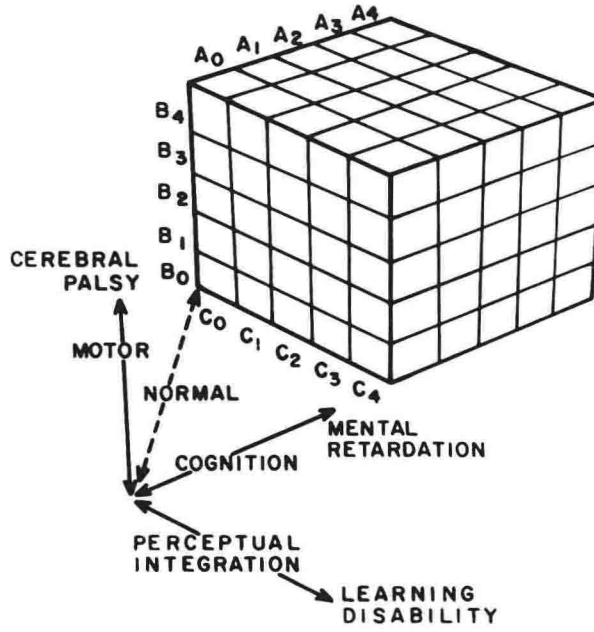

Figure 1. Developmental disabilities matrix. Although the number of variables could easily be multiplied to produce an *n*-dimensional figure, this cubic matrix allows one to approximate the interrelationships among the various developmental disabilities. A fourth dimension (time) is obligatory: the matrix should be visualized as a plastic structure that demonstrates slight distortions in shape as it moves to the right. The location of various chronic neurologic conditions on the matrix may be illustrated by the following examples: $A_0B_0C_0$=normal child; $A_4B_4C_4$=severely retarded, severely motor impaired child with significant perceptual dysfunction; $A_3B_0C_0$=moderate global retardation with no motor disability or perceptual dysfunction; $A_0B_2C_1$=mild cerebral palsy with normal intelligence and minimal perceptual dysfunction; $A_0B_0C_4$=severe dyslexic with normal intelligence and no soft neurologic signs; $A_0B_1C_3$=MBD child with normal intelligence, "clumsy child" syndrome, and moderate perceptual dysfunction; $A_3B_0C_4$=moderate retardation with severe central communication disorder ("autistic" child). (From Accardo and Capute, 1979.)

has been documented (Vaughan and Hodges, 1973). A generally accepted definition of learning disability is lacking, but it appears that most professionals do share a fair degree of common ground once their technical jargon is translated. The extremely large number of terms used to describe children with learning problems reflects different concepts of etiology and symptom primacy. Psychoneurological (D. J. Johnson and Myklebust, 1967) or neurological learning disability (L. B. Silver, 1971b) probably best characterizes children whose general intellectual function is normal but

certain of whose cognitive processes are impaired secondary to brain dysfunction. The child with a significant neurological contribution to his learning problem is called a specific learning disability (SLD).

Attempts to divide SLD children into discrete subgroups have not been very successful. Denckla (1972) found that only 30% of such children fit into specific categories (15% dyslexic syndrome, 10% dyscontrol syndrome, and 5% Gerstmann syndrome), with the other 70% representing a mixture. Owen et al. (1971) defined five groups, into which only 42% of their subjects could be placed without overlap. Thus, although relief from the confusion of global terms would seem to lie in the direction of specific syndrome identification, effective subclassification has hardly begun.

Whether the SLD is secondary to brain damage (the continuum of reproductive casualty), brain dysfunction, such as congenital hypoamphetaminemia (P. Wender, 1971), a biological variant (Werry et al., 1972) made prominent by societal expectations or some other hypothesized etiology, the academic delay is frequently interpreted as a maturational lag. These children's learning and behavior can certainly be attributed to uneven development of cortical functions (de Hirsch, Jansky, and Langford, 1966; Kinsbourne, 1973a; Satz and van Nostrand, 1973), but the misleading aspect of the term *maturational lag* lies in its implication of later catch-up within a predictably short time period (cf. Denhoff, Hainsworth, and Siqueland, 1968). In reality, such "catch-up" is an illusion of perspective. If one uses tests appropriate to early school-age children, the older SLD child will no longer demonstrate the errors characteristic of younger neurologically impaired children; but he will exhibit a persistance of his learning problem when the assessment is geared to age-appropriate cognitive tasks. For example, of learning-disabled children receiving a special class placement (1.5% of the total school population), only one in four was able to be mainstreamed within 5 years. Although they made 1.1 years of reading progress in their first year of special education, this rate dropped to 0.5 years by their fourth year to give them an overall gain of 2.9 years over 4 years (Koppitz, 1971). Those children with fairly mild degrees of impairment may nevertheless benefit from starting school (or at least reading) at a later age, but their ability to integrate well into a regular class after a few years reflects more the wide heterogeneity of grade levels than any resolution of their deficit (cf. Snyder, 1979).