Developmental Language and Learning Problems

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ABSTRACT

In this article, the much studied issue of children who develop the ability to use language, both spoken and written, slowly and incompletely is discussed from two major points of view: (1) subtyping and (2) lateralization. I take the componential approach to dyslexia, which assumes that anyone or any subset of several processing components essential for the acquisition of language and reading skill can be impaired in its development, yielding corresponding diverse subtypes of these disorders. Subtyping has been attempted both on an empirical (cluster analytic) and a neuropsychologically rooted (syndrome) approach. Methodological issues surrounding these efforts are also discussed in connection with laterality and dyslexia.

INTRODUCTION

The term “learning disabled” designates children who, although of apparently normal intelligence, motivation and educational opportunity, make less academic progress than their age would lead one to expect. The diagnosis by exclusion (Rutter 1978) should distinguish these children from those who underachieve on account of emotional blocks, sociocultural deprivation or alienation, or inadequate instruction. The term implies that the learning-disabled children’s difficulties derive from a lag in development of mental operations that the instructional process relies upon conjecture. Current definitions of learning disabilities cast a wide net, including problems with spoken as well as written language, and are thought to apply to at least 2% of the childhood population.

RELATION BETWEEN COGNITIVE DEFICIT AND ACADEMIC FAILURE

Children may fail to learn because they do not pay or maintain attention to instruction or because, although attending, they do not comprehend or,
though comprehending, they do not remember. The present discussion is limited
to selective learning disabilities and excludes general disorders of attention, task
orientation and conduct, although the latter may also be due to immaturity or
abnormality of brain substrate (Clements 1966). The concepts of “organic
drivenness” (Kahn and Cohen 1934) and of “brain injured child” (Strauss and
Lehtinen 1947) explicitly implicate damaged and therefore ill-developing brain
in the genesis of these conditions. In fact, that early brain damage can be
inferred solely on the basis of the pattern of the child’s disordered behavior
remains, nearly four decades after Strauss’s claim to that effect, unsubstantiated.
(Competing concepts that invoke genetically based brain immaturities are
discussed by Rutter (1982).) I use the term learning disabilities to denote
brain-based school problems which are selective to subsets of the curriculum.
The commonly used term perceptual handicap prejudges the mechanism of the
school problem, and is therefore to be avoided. Concepts of brain injury or
minimal brain dysfunction are relevant only with respect to allegations that they
accompany selective academic problems in some cases (Bannatyne 1966).
Dealing as we are with a substantial segment of the general childhood
population, we cannot assume neurological deficit, but need positive evidence
that it exists.

It is generally and reasonably supposed that particular patterns of academic
difficulties represent failure of development of one or more necessary cognitive
operations. This selective cognitive immaturity relegates the disabled learner to
the status of a much younger normal child with respect to the learning efforts in
question. But there is little agreement in education theory about which cognitive
processes are requisite for learning which material. Nor are there reliable and
valid tests for the presence of individual cognitive operations in either normally
or abnormally developing children. We therefore retreat to surface
phenomenology. Is the child failing in reading and writing, writing alone,
arithmetic by itself or in conjunction with the preceding, or in tasks involving
draftsmanship in some form? Of these, problems of reading and spelling have
been vastly the most studied, and we shall draw mostly upon them in our
illustration of current efforts to elucidate the nature of learning disabilities.

BRAIN–APTITUDE RELATIONSHIPS

It is commonly accepted that people differ in their aptitude for different
academic subjects, that is, in how readily they learn, holding the nature of the
instruction constant. While determinants of aptitude are undoubtedly multiple,
it is widely held that intraindividual differences in the pattern of brain
development play a major role. The approach draws upon the concept of
multiple intelligences (Gardner 1983). Different parts of the brain subserve different cognitive functions, and develop at different rates toward their mature endpoints. These different brain areas are supposed to be under the control of different genes, so that genetic diversity generates much of the existing variability in intellectual profiles. At the extremes of variation, individuals exist whose patterns of intelligence are not conducive to the type of mental work which is rewarded by society. Such maladaptive outcomes may lead to learning disabilities even in the absence of any abnormality or disease process at the neurological level.

Uneven development of intellectual skills can also result from early focal brain damage. This retards the rate at which the function subserved by the damaged part of the brain develops. Because cerebral function is localized, the locus of cerebral damage should determine the pattern of the resulting selective cognitive deficit, and therefore of any consequent academic failure. Although early focal damage in areas subserving higher mental functions can be mitigated by compensatory action of undamaged cortex, such compensation is incomplete. Based on this line of reasoning, an intensive search has been launched for confirmatory evidence for the hypothesized early scattered impairment of developing brain. Drawing on the hypothesized "continuum of reproductive casualty" (Pasamanick and Knobloch 1960), potentially relevant perinatal events and putative results of early damage, such as neurological soft signs and multiple minor congenital anomalies, have been scrutinized. In summary of an extensive literature we may conclude that, whereas such indices of the presence of minimal brain dysfunction have sometimes been shown to be somewhat associated with disordered attention or hyperactivity (Denckla and Rudel 1978; Waldrop et al. 1978; Mikkelsen et al. 1982), there is little evidence of such widespread accompaniment for specific cognitive deficits that cause selective learning disabilities (Nichols and Chen 1980). We therefore draw upon the normative genetic diversity model in our conceptualization, although we cannot exclude the possibility of limited early brain damage as antecedent in some cases.

PSYCHOMETRIC CONSIDERATIONS

The concept of multiple intelligences can be explored concurrently with normally and abnormally developing cases. In the normal population, factor analysis of psychometric test data should reveal the number and identity of separately developing mental operations. The behavioral effects of focal brain damage or limited genetic flaw should validate these results by bringing to attention instances in which a particular mental operation is impaired, whereas the rest develop normally.
At a gross level of analysis there is some support for this view. Analysis of psychometric findings throws up major factors which are labeled verbal comprehension, perceptual organization and freedom from distractability. Deviation quotients for each of these factors can be calculated on the basis of scaled scores from the Wechsler Intelligence Test for Children—Revised (WISC-R), using appropriate formulae (Gutkin 1978). Reading-disabled children taken as a whole are reportedly relatively weak on the WISC subtests of digit span, coding and arithmetic (Naidoo 1972; Rugel 1974). Nevertheless, it is at least plausible to divide pathological cases in the first instances into those implicating visuospatial processing (spatial types of dyslexia and spatial dyscalculia) and those involving attentional deficits (hyperactivity, attention deficit disorder). However, the more tests are included in a psychometric battery, the larger the number of emerging factors and the more learning-disabled children are studied, the greater the inventory of patterns of cognitive insufficiency. Pending convincing alignment between many psychometric factors, and many learning disability subtypes, their presumed correspondence must retain the status of a working hypothesis.

The lack of formal evidence for correspondence between strength and weakness of psychological test performance and strength and weakness in academic achievement has not deterred clinicians in the learning disabilities field from making strong claims about such correspondences even in the individual case. In learning clinic assessments the practice is widespread of administering psychometric and neuropsychological tests not involving the academic skills under scrutiny and deducing from uneven performances on such tests the causes of the learning problems. An instance is the unfounded diagnosis of "perceptual deficits" based on the use of instruments such as the Illinois Test of Psycholinguistic Aptitudes (ITPA) (Kirk et al. 1968), which lays claim to measuring major cognitive processes without any acceptable supporting evidence. For example, "a sequencing disorder" is often generalized from a child's inadequate performance on one or a few tests in which maintaining information in specified sequence is among the requirements. In summary the overriding open question is that of generality. To what extent can failure on a particular test be used to infer broad failure in an underlying cognitive operation, or a particular pattern of failure on such a test be extrapolated to indicate the nature of the child's learning style? Even in the ostensible more clear-cut area of neuropsychology of focal cerebral damage such extrapolations can be made only to a limited extent. Thus, for all the discussion about the "spatial" function of the minor hemisphere, it is still not clear how right hemisphere spatial function is to be operationalized in terms of test performance (De Renzi 1982). The construct "constructional apraxia", as encompassing
spatial productions, copied and remembered, in 2 and 3 dimensions (Critchley 1970) lacks validity (Warrington et al. 1966). Even perceptual closure, the recognition of incomplete figures, is evaluated totally differently depending on the test used (Wasserstein 1980). Given such serious unresolved validity issues in the well-defined cases of focal brain injury, the corresponding problems in defining specific "basic underlying deficits" in the learning disabled are formidable indeed. Current "one-shot" learning disability evaluations do not even guarantee test-retest reliability of the measures which are put to such powerful use. The validity problem has barely been addressed.

The above reservations might not be very serious if the interpretations result in practical outcomes in the form of remedial teaching successes. They do not appear to do so. But little can be concluded, because the teaching options do not even correspond to the diagnostic categories to any useful extent. So the matter has not been properly tested. The recommendations arising from learning clinic evaluations tend either to be operationally useless ("practice sequential skills") or to perseverate on the hackneyed dichotomy of teaching by "whole word" vs teaching by "phonics" (as if reading could be adequately taught by either method alone). The major remedial curricula (Orton-Gillingham, Distar, Stevenson, etc.) deploy a laborious progressive analytic methodology which takes little account of the child's pattern of antecedent psychological test performances. Until evaluation and management are specifically correlated, extrapolations from test profiles will remain unvalidated and also without practical consequences for remedial education.

Against this background of uncertainty, we proceed to consider the best investigated of the learning disabilities, dyslexia, from the point of view of taxonomy and brain basis.

MODELS OF DYSLEXIA AND ITS SUBTYPES

There are two alternative theoretical viewpoints with respect to dyslexia, each of which subdivides into further alternatives. One view regards dyslexia as a unitary disorder. Some proponents of this view construe the disorder narrowly as "pure" exclusively implicating some key process involved only in learning to read and write. Others conceive it more broadly as affecting cognitive processes which, though they are essential for reading and writing, also have observable and measurable effects on the execution of other activities. An opposing view regards dyslexia as comprised of heterogeneous disorders. This diversity view divides into discontinuity theory and continuity theory. According to the discontinuity theory, the dyslexia syndrome incorporates a finite number of discrete disorders or dyslexia subtypes, the sum total of which accounts for the general problem of dyslexia. According to the continuity theory, dyslexic
children differ only in degree from normal readers. They are those in whom one or more of the many mental operations involved in learning to read or write is underdeveloped. This individual difference approach regards dyslexic children as very heterogeneous in the locus of their difficulty, but does not admit to discrete subtypes.

The unitary theory of pure dyslexia was the earliest and is implicit in the strict neurological model of "congenital word blindness" which is alleged to arise from left angular gyrus underdevelopment. It is largely disregarded nowadays. Nevertheless, in the present state of knowledge we cannot exclude this variant. In some children who experience difficulty in learning to read and write, neuropsychological and psychometric measures reveal little or no cognitive inadequacy relevant to reading (Satz and Morris 1981; Lyon and Watson 1981; Watson et al. 1983). Although some would then be inclined to suspect motivational difficulties, these are not apparent either. Perhaps, had one measured the "correct" mental operation, one would have found a more general disorder in such children. Or there might exist a deficit in a mental operation which is so selective to reading and writing that only they reflect its impairment.

Taking the history of dyslexia as a whole, the broad version of the unitary hypothesis has held sway more than any other idea—that dyslexia is unitary, but based on a more general cognitive processing difficulty. The problem was often assumed to be in some way visuospatial or visuodirectional (Orton 1937; Hermann and Norrie 1958). After Benton's (1962) analysis detrimental to that view, the emphasis within this hypothesis has radically shifted toward discriminating a general language difficulty (Vellutino 1979). This viewpoint regards linguistic factors (verbal coding, mastering the phonological structure of the language) as the crucial stumbling block for dyslexic children. It dismisses any other associated test-elicited deficiencies as coincidental and not critical for reading progress. Dramatic evidence for a language deficiency not limited to the use of written language derives from the experimental findings of Spring and Capps (1974) and the results of Denckla and Rudel's (1976) Rapid Automatic Naming Test. Poor readers exhibit prolonged latency when they name, in randomly determined series, stimuli with which they are quite familiar (pictures, colors). This difficulty in verbal retrieval differs in degree only from the extreme word retrieval difficulty prevalent among cases of overt developmental dysphasia (Wolfus and Kinsbourne 1984). It extends beyond naming into verbal decision making (true–false judgement of spoken statements) (J. Kagan 1983). Indirect electrophysiological evidence suggests that the brain detects the anomaly in false statements with normal latency, and that the delay is in the formulation of the response that follows (C. N. Kagan 1983). Other unitary approaches invoke auditory processing limitations (Tallal and Piercy 1973),
temporal order problems (Bakker 1970) and sequential eye movement impairment (Pavlidis 1981) as explanatory principles.

Taylor and co-workers (1979) find little difference in any of many parameters between backward readers who meet and who do not meet exclusionary criteria for dyslexia. A positive diagnosis of dyslexia based on unique manifestations would constitute an advance. Critchley (1970) has described patterns of reading and spelling errors in dyslexia, but these also occur in normal beginning-readers. Boder and Jarrico (1982) restrict the dyslexia diagnosis to those who have significant difficulty in spelling even the words they can read. One possible explanation for the difficulty experienced in finding positive diagnostic criteria for dyslexia is that several subtypes exist. If so, failure to distinguish between them risks assembling a heterogeneous sample in which specific manifestations are hard to detect. Due to sampling variability, some investigators might be impressed by one cognitive profile, others by another, thus resulting in conflicting accounts of the cognitive deficit in “dyslexia”. Actually, several interpretations might be valid, each with respect to a different subtype of dyslexia.

Kinsbourne and Warrington (1963) demonstrated two neuropsychological syndromes plausibly related to the reading difficulty that accompanied them. For some children, language disorder was the limiting factor: these male children were slow to develop language and were also relatively inferior on the verbal subscale of the WISC. They also had a subtle but measurable impairment of verbal comprehension, as measured by the Token Test. Their spelling errors predominantly involved choosing the wrong letters. In contrast, examples of another syndrome were presented in which no evidence of a central language disorder was present. The “Gerstmann” subtype showed a performance deficit on the WISC, notably involving the block design and object assembly subtest. It also featured a problem in arithmetic, both on the WISC and on educational achievement tests (in contrast to the language type in which the difficulties were limited to reading and writing per se). Failure on tests of finger order sense demonstrated a difficulty in identifying body parts based on their sequential position (as opposed to their distinctive individual forms). Right–left discrimination difficulties also exemplified difficulty in making distinctions based on relative position. The children had more difficulty spelling than reading, and made letter order errors, i.e. though they used the correct letters, they would put a letter in too soon, too late, or reverse the position of two letters. In arithmetic, their difficulty was specific to place value: the significance of the digit, not in terms of its shape, but in terms of its spatial relationship to other digits on the same line, i.e. they would mistake 56 for 65 and showed particular difficulty in arithmetic computations in which numbers had to be carried from column to
column. The syndrome was construed as a sequential processing problem (analogous to the Gerstmann syndrome resulting from acquired left parietal injury) and not specifically affecting or limited to verbal material. Indeed, sequences of words or speech sounds seem to be established by a separate processor or arrived at by some method which bypasses the culpable process in the sequential difficulty of the Gerstmann type. More recently, Pirozzolo (1979) has enriched the understanding of this syndrome by discovering that the children also have difficulty in sequential eye movement from left to right (see also Pavlidis 1981) and with the return sweep from the right end of one line to the left beginning of the next one underneath.

An essential component of the above argument is that the processing disorder inferred from neuropsychological test outcomes should be manifest in the qualitative nature of mistakes made in the activities of interest—reading and writing—themselves. While the claim for distinctive spelling error types needs confirmation, the logic it exemplifies could with advantage have been incorporated in subsequent attempts to subtype.

Kinsbourne and Warrington (1963) selected cases exemplifying these two problems from a much larger group of children referred for backward reading. They did not claim to be able to subdivide all children into either one (or a combination) of these 2 categories. They left open the following questions: (1) whether other subtypes also await discovery, and (2) whether these extreme cases represent specific subtypes or the extremes of continua of individual variation in the development of the relevant underlying mental processes.

Boder (1971) pursued the dichotomizing approach with respect to the reading and writing performance. She devised ways, since formalized into a test instrument (Boder and Jarrico 1982), of detecting a selective propensity of children to misapply phonic analysis (dysphonetic errors) or the whole word approach to reading (dyseidetic errors). The classification referred back to Myklebust's (1965) notion of a "visual" weakness on the part of some backward readers and "auditory" weakness in others (see also Bateman 1968; Ingram et al. 1970; but also see Liberman 1985). This subgrouping incidentally lent itself to later attempts to identify dyslexia as relative weakness either of the right or the left hemisphere (whereas Kinsbourne and Warrington's language and Gerstmann subtypes were both based on analogies with adult left hemisphere neuropsychological syndromes).

Boder (1971) and Boder and Jarrico (1982) determine the child's sight vocabulary both for phonetically regularly and irregularly spelled words. Dyseidetics succeed in spelling irregular "unknown" words in phonetically intelligible fashion. Dysphonetics spell unknown words in "bizarre" ways (i.e. they include letters that do not sound right). Thus, two channels for reading
instruction are designated: one, visual, lends itself to "whole word" methodologies, the other, auditory, to "phonics" instruction (see also Marshall 1985). One channel is thought to be "open" in most dyslexic children who could, presumably, be successfully taught by one or other of these methods. There is general agreement that language-delayed dyslexics experience difficulty in applying phonics. That they can benefit normally from whole word instruction has not been shown, and given that phonics and whole word methods are commonplace in regular reading instruction, it is unlikely that simply "switching sensory channels" will remedy many cases.

These efforts notwithstanding, the possibility of subtyping dyslexia attracted little attention until it was rediscovered by Mattis et al. (1975). They described a language and a visual perceptual subtype elicited by different test procedures from those used previously. They introduced an interesting further control. They reasoned that to demonstrate the validity of attributing a reading disability to the associated finding of a neuropsychological deficit on test performance it is necessary to show that any child who has that finding on tests also must have trouble learning to read. They included a brain damaged but not reading-handicapped control group and excluded from consideration those tests on which that subgroup did poorly. They argued that if one could do badly on a test and still learn to read, then the ability underlying that test performance could not be crucial for reading. On this basis, apparently, they excluded the Gerstmann syndrome from consideration, at least as tested by their methodologies. This refinement addresses a crucial issue in subtyping: the evaluation of the negative instance. Subsequent investigators have not checked the concatenations of test results by which they have characterized the dyslexic subtypes for validity. Mattis et al.'s (1975) specific methodology may, however, be questioned. The number of patterns of neuropsychological profiles excluded from consideration because they do occur in normally reading children could simply be a function of how many tests are given to how many children. If one kept on testing children who can read but are otherwise neuropsychologically handicapped, one might well find that virtually every possible combination of test results permits tolerable reading development in some cases. Of course, one could then argue that the children who were able to learn to read in spite of neuropsychological deficits that "should" have stopped them were doing so by some alternative or unusual route.

Mattis et al. (1975) added a third subtype. These children had articulatory and graphomotor deficits. Given their 3 subtypes, they claimed to be able to account for every child in their sample. They did so by using generous criteria for labeling test performance deficient (including even performance only one standard deviation below the population mean). Although on empirical grounds
this cannot be challenged, if it works, it makes one doubt that the subgroups are valid. In a subsequent publication, Mattis (1978) confirmed the subgroups, added a subgroup whose difficulty he interpreted as related to temporal sequencing, but this time did find some children whose reading problem was not accounted for. His classification significantly overlaps that of Denckla (1977).

By the mid-seventies, investigators had become ready to entertain the subtype idea. Interest in dyslexia had greatly expanded, substantial federal funding having become available. Cognitive and educational psychologists were attracted to the field and brought their test armamentaria to bear on the matter. Given these circumstances, the delineation of subtypes became an attractive proposition. Numerous studies have followed. Some involve the concurrent trend toward hemispheric theorizing. In one, dyslexia is dichotomized into right and left hemisphere types (Bakker 1979). In the other, dyslexia attributed to left hemisphere malfunction is contrasted with dyscalculia attributed to right hemisphere malfunction (Weintraub and Mesulam 1983). These lateralizations await confirmation by laterality studies.

Usually, reading and writing difficulty coincide, but some children have difficulty in spelling only. The errors they make are phonetically regular (Nelson and Warrington 1976), witness to their intact language system. Some of these children have mild residual Gerstmann findings, but others do not exhibit an excess of the sequential order spelling errors that Kinsbourne and Warrington (1964) found to characterize Gerstmann cases.

The main departure from the work thus far abandons the clinical approach (which tests the child and scrutinizes the pattern of test outcome for plausible pattern of deficits) for an empirical one. Briefly, it was argued that a more objective procedure would be to administer an extensive neuropsychological test battery to an adequate sample of reading-retarded children and then, by statistical means, determine subgroups that differ in a neuropsychological pattern of findings. The statistical technique that was applied was cluster analysis. The pioneering work was done by Doehring (Doehring and Hoshko 1977; Doehring et al. 1979; Doehring et al. 1981). Petrauskas and Rourke (1979) and Satz and Morris (1981) have also applied cluster analytic techniques to substantial samples of reading-retarded children. Each study emerges with a number of clusters of backward readers.

These sets of cluster are superficially in some agreement. They all include a general language based cluster. Some also have a visuospatial cluster, often associated with finger localization difficulty, suggestive of a sequential (Gerstmann type) deficit. The articulatory–graphomotor subtype (Mattis et al. 1975) and the mixed visual and auditory (Boder 1971) have proved more difficult to confirm. In more intimate detail, it is unclear whether the clusters
superimpose. The statistical methods used differ from study to study, and which is the correct method is controversial (Morris et al. 1981). Cluster analytic technique determines which clusters exist on the assumption that some do. It cannot evaluate the hypothesis that clusters do not exist (Satz and Morris 1981), the alternative possibility being that the data derive from continuous variation along multiple dimensions of performance (in accordance with the individual difference model). There is no inbuilt significance estimation of the clusters that result, and therefore the fact that clusters are derived does not rule out the individual difference approach to subtyping. Thus, two further tests need to be applied to the cluster. One is, does it make neuropsychological sense? Does the cluster characterize children really impaired in mental operations plausibly crucial to reading? With respect to the negative instance, are the relevant test deficits absent from similarly tested children who read normally? The second test is the extent to which the cluster replicates (see for instance, Fisk and Rourke 1979; Watson et al. 1983). At this time, the empirical derivation of clusters remains a superior technique. However, it cannot carry conviction alone but should be bolstered by ancillary findings. Pending such findings, we keep in mind the possibility that the clusters simply represent the full set of cognitive domains pertinent to reading acquisition. If so, they should be discernible among the population of normal as well as of backward readers.

The subtyping of dyslexics remains controversial. Referring back to the concept of multiple intelligences, we note that ultimately the number and nature of subtypes may be found to correspond to the set of distinct mental operations crucial to reading and writing acquisition. Such an outcome is not yet within sight.

A somewhat contrasting view classified disabled readers into those with and those without concomitant problems in spoken and receptive language. The subgroup without language problems is reported not to be distinguishable from controls on a large series of tests (Tallal and Stark 1982).

The individual differences approach is exemplified by the work of Olsen and co-authors (1984). Measuring various component processes of reading, they found these to be continuously (normally) distributed among disabled readers. This continuous variation on a variety of plausibly relevant cognitive parameters explains reading problems on the basis of the affected child's location at particular intersects on multiple relevant dimensions. One would thus speak of dimensions of individual difference rather than subtypes. The relative heterogeneity of children within clusters (Watson et al. 1983) also points in that direction. This approach conforms to the informal belief of many educators that academic difficulties are multifactorial, vary with age (Fletcher and Satz 1983) and call for individualized educational prescription rather than
two or three basic remedial curricula tailored to subtype.

While each of the four major approaches to the understanding of dyslexia has something to commend it, not one of them is conclusively proven to be more applicable than the rest. It thus behoves us to look for supporting evidence from outside children’s reading and neuropsychological test performance. We first pose the question: which of these approaches yields the information most helpful for programming remediation for the child?

Were it that teachers who rely on a particular set of neuropsychological findings experience greater success than those who do not, then, as a practical matter, the model of choice would be clear. In point of fact, no one of these approaches has demonstrably succeeded in closing the gap between the learning clinic evaluation and the actual teaching that then has to follow. It is not clear that any of the above-mentioned approaches generate individualized educational programs that are superior to any others (or indeed that are superior to those formulated in the absence of the type of analysis we have been discussing).

Biological Correlates of Dyslexia

Given the legitimate uncertainties about the appropriate model for dyslexia, as derived from corollary test performance, interest is renewed in the search for brain based correlates (or as it is fashionable to call them, biological markers). Does some bodily or brain based attribute reliably pick out a particular subtype of dyslexia from the rest (or even dyslexia as such from nonspecific reading disorders)? Among possible biological markers, none that are metabolic have been proposed. The recent claim for an abnormality of the short limb of chromosome 15 in certain pedigrees with cases of dyslexia is worth noting (Kimberling et al. 1983). A selective language based reading disability is prevalent among children with 47XXY karyotype (Bender et al. 1983). Eight of 92 children with selective language delays were found to have XXX or translocation chromosomal abnormalities (Friedrich et al. 1982). For the rest, we shall focus on proceeds of the extended neurological and neuropsychological examination.

The various approaches to dyslexia that have been considered leave their brain basis unspecified. A performance deficit can derive from selective brain damage or maldevelopment. It can arise from problems of local neuronal circuitry that currently defy detection. It can result from deviant brain organization, overloading certain brain areas (Levy 1969; Witelson 1977) or rendering certain processes vulnerable to cross-talk interference (Kinsbourne 1980), or it can result from defective or deviant patterns of task-related selective cerebral activation. Any of these concepts could be applied to any of the cited
views of the nature of dyslexia. Indeed, where a heterogeneity hypothesis is adopted, multiple factors could be invoked, e.g. activation deficiency for one subtype, structural damage for another.

The most direct evidence for the brain basis of dyslexia would accrue from examination at autopsy of relevant portions of brain. This was accomplished in four cases by Galaburda et al. (1985), who reported evidence of neuronal developmental abnormality in the language area of the left hemisphere (though also elsewhere and even bilaterally in three cases). If this type of finding were to characterize many or most of the children diagnosed as dyslexics, then this would revolutionize our concept of that disorder (and reveal a prevalence of developmentally based cerebral neuropathology far greater than is currently generally believed). Pending confirmation of the specificity of Galaburda et al.'s (1985) findings, we proceed to consider alternative techniques that might indirectly contribute to the choice between models. These are neurophysiological and radiological methodologies of recent devising.

The use of EEG in dyslexia has not given demonstrably useful information. However, Brain Electrical Activity Mapping (BEAM) has been presented as identifying areas of cerebral cortex, significantly different from control groups with respect to electrophysiological dependent variables in dyslexics (Duffy et al. 1980a, b). Although these might represent structural differences (in the left hemisphere language area and in both supplementary motor cortices), by their nature the dependent variables which were used are not capable of distinguishing structurally abnormal from functionally deviantly utilized brain. We cannot tell from the data thus far whether the abnormalities represent brain-based differences in how the two groups of children approach tasks or even what they are thinking about or feeling during the test situation. These are not insoluble problems. Indeed, a fuller presentation of data already acquired might resolve them. They have not yet been solved, however.

Neuroradiological methods displaying anatomy are generally not subject to such reservations. One study using computerized tomography has claimed a substantial incidence of left lateral ventricular dilatation in a dyslexic group (Hier et al. 1978), but another study has failed to replicate this finding (Haslam et al. 1981). Children come to CT scan for clinical causes, and scanned dyslexics are far from being a random or representative sample of the dyslexic population. Given that developmental abnormality is suspected and lefthandedness is supposed to be a risk factor for dyslexia, it becomes interesting to determine whether “reversed asymmetry”—namely, a reversal of the usual right anterior and left posterior predominance in brain size (LeMay 1977)—characterizes a substantial number of dyslexics. Hier and co-workers (1978) have some evidence for this. Neuroradiological examination of function
might prove capable of identifying abnormal patterns of brain use in dyslexia, but findings to date have been ambiguous. For this population, the methodology of nuclear magnetic resonance, which is free of radiation hazard, is more promising. For purposes of revealing brain function rather than structure (i.e. spectroscopy), however, this method is still in the development phase.

The left-handedness associated with reading failures (Zangwill 1960) attracts much current interest. An association of "immune" disorders, migraine, left-handedness and dyslexia, was proposed by Geschwind and Behan (1982). We have findings supporting the existence of this relationship in families with left-handed members (Kinsbourne in preparation). Even righthanders in such families exhibit more vulnerability than those in dextral families. Whether familial sinistrality indexes a specific subgroup of dyslexics or constitutes a general risk factor is unclear.

A possible association between left-handedness and a dyslexia subtype has recently been explored by Satz and his colleagues. They applied to the problem the concept of pathological left-handedness (Satz 1972): this proposes that a subset of left-handers represents genotypic dextrals who have assumed left-hand preference on account of minor left hemisphere damage involving areas of right-hand motor control. This hypothesized damage could also be responsible for left hemisphere malfunction in the language sphere. They have compiled some intriguing case study material which could be interpreted as relating the developmental Gerstmann syndrome (Kinsbourne and Warrington 1963) to this particular pathogenesis. Satz and his colleagues (1985) have explored the possibility that mild but measurable underdevelopment of right-sided body parts (hands and feet) identify pathological left-handedness.

The pathological left-handedness syndrome is one of several constructs relating at least some subset of dyslexia to an impairment of left hemisphere function. In general, neuropsychological testing that treats these children as a single group finds left rather than right hemisphere disability. Thus Gordon (1980), using a battery of tests validated on patients with lateralized structural brain damage, found his dyslexic group to be relatively inefficient on tests that in the clinical context detect damage of the left hemisphere. Interestingly, he found a similar test profile in the normally reading family members of the dyslexic probands. Those findings favor an origin of dyslexia based on genetic diversity. He even found evidence that dyslexic children were relatively superior on right hemisphere test (Gordon 1980). Whether this apparent trade-off will have to be considered in any valid model of the dyslexic disorder, or whether it represents the results of sampling bias remains to be determined.
Laterality and Dyslexia

The most widely applicable methodology for determining a hemispheric basis for dyslexia involves the use of laterality tests. In most cases, they are applied to unselected groups of dyslexic children. In some cases, the subtype distinction is made.

Laterality testing demonstrates whether one hemisphere is more active than the other during the performance of the cognitive task (Kinsbourne and Hiscock 1983). Thus, in a normally functioning right-hander, hemifield viewing and dichotic listening for verbal messages yield a right-sided superiority. For certain non-verbal materials the superiority is left-sided.

If the expected asymmetry does not arise, this could be because:

1. The function in question is not structurally lateralized in the usual way.
2. The child is not deploying the function in question to an extent sufficient to generate an activation asymmetry capable of influencing the laterality test performance. This could be because he is
   (a) undermotivated;
   (b) unable to activate the relevant cerebral territory (for neurological reasons);
   (c) using a deviant strategy, thus drawing on other parts of brain.

A number of studies have shown no deviation from the expected laterality pattern in samples of reading-disabled children. Some studies have found lack or inconsistency across different presentation conditions of right-side advantage for verbal material. A few have found the opposite—an enhanced right-sided advantage (see review by Satz 1976). The deviant patterns found in these studies will be considered with reference to the following possible neuropsychological models for the dyslexic disability.

A. Structural Models
1. The left hemisphere is structurally inadequate.
2. Language is not lateralized, but bisymmetrically distributed between the hemispheres.
3. Language is right lateralized.
4. Language is left lateralized, but overlapped by bilateral spatial functions.
5. Language is right lateralized and overlapped by right or bilateral spatial functions.
6. Interhemispheric communication is defective.
B. Selective Activation Models

7. Selective activation of the left hemisphere in task-related circumstances is inadequate.

8. Selective activation appropriate to the left hemisphere spreads bilaterally.

9. Selective activation is anomalously diverted to the right side.

Variant 1 currently derives largely from the work of Geschwind and Behan (1982). Variant 2 was hypothesized by Orton (1937) and supported by Lenneberg's (1967) notion of progressive lateralization: the bisymmetric language state in this theory represents a developmental immaturity. Variant 3 is the situation found in some, perhaps pathological, left-handers. Variants 4 and 5 represent "hemisphere" crowding hypotheses as suggested by Levy (1969) and Witelson (1977). The activation models arise from the hypothesis of Kinsbourne (1970), adapted by Kinsbourne (1980) to explain laterality findings and ease of recovery of language function after brain injury in nonright-handers.

As we already remarked, model 1 is feasible but requires further documentation. Most notably it needs to be clarified why an intact cortex does not compensate for the hypothesized left hemisphere deficit, when even total destruction of the left hemisphere is known to leave a child quite capable of learning to speak and read. Perhaps an intact but defective left processor inhibits the gradual development of compensatory functions. Model 2 lacks appeal now that Lenneberg's hypothesis is refuted in favor of the evidence for invariant lateralization (Kinsbourne and Hiscock 1977, 1983). Some findings do show a failure of laterality effects for verbal material to arise, consistent with this model, but also consistent with model 7. Variant 3 lacks support. We also note that when language is right-lateralized in left-handers, this appears not to impede development of language and reading skills. Variants 4 and 5 at this point lack substantial empirical support. Variants 6, 7, 8 and 9 are alternative to structural hypotheses in explaining the diminution of perceptual asymmetries for verbal material found in some studies of dyslexic children and more constantly where there is frank developmental language delay. They might also account for sluggish verbal responding by such children.

Left hemisphere activation difficulty is the best available explanation of the findings of Obrzut et al. (1981) and Kershner et al. (1984) in dichotic listening and of Obrzut et al. (1983) in hemifield viewing. In these studies, learning-disabled children differed from normals in that in the verbal laterality testing the expected right-sided advantage either occurred only when subjects were forewarned of the side of the (visual) presentation, or was lost when subjects were instructed to attend to the left (auditory) message. If perceptual asymmetry does indeed result from selective hemispheric activation (Kinsbourne 1970), we learn
that the learning-disabled children revealed relatively weak left hemisphere activation under verbal conditions, such that the direction of attentional orientation (also based on differential hemisphere activation (Kinsboume 1975) was capable of influencing, or even overriding, the activation relative to the cognitive task. The fact that orienting contralateral to the language hemisphere elicited the best overall task performance in the above studies could have treatment implications. Perhaps orienting to right-sided information sources affords the underactivated left hemisphere additional facilitation (cf. Lempert and Kinsboume's (1982) demonstration of differential head turning effects on verbal memory). If the therapeutic successes of cerebral dominance manipulations in reading remediation claimed by Van den Honert (1977) and Bakker (1981) are confirmed, the above discussed mechanism could apply.

Weakness in maintaining selective activation of the left hemisphere could explain the dyslexic child's problem not only in performing difficult verbal tasks, but also in consistently maintaining a verbal mental set. Continual swings of activation in favor of right hemisphere processes could undermine the consistency of the mental effort called for by the reading instruction. The child's attentional focus is observed and/or experienced continually to drift away from the verbal task. The evidence for deviant cognitive style is consistent with this view. Some evidence for a tendency toward a non-verbal (right hemisphere) style in reading-disabled children has been found (Caplan and Kinsboume 1979; Oexle and Zenhausern 1981).

In two studies, laterality tests were analyzed with respect to subtype. In one, the expected lack of right-sided advantage for verbal material was found in the language subtype, but the usual right-sided advantages prevailed in the sequential (Gerstmann) subtype (Pirozzolo and Rayner 1980). Since the verbal test taps the verbal deficiency in the language subtype, the lack of asymmetry is not unexpected. In the Gerstmann type, specifically linguistic processes are unaffected (Kinsboume and Warrington 1963) and a verbal dichotic test should not yield abnormal results as it is the activation of language areas, generalizing across the left hemisphere which generates the asymmetry in dichotic listening.

Dalby and Gibson (1981) classified their sample of backward readers into dysphonetic, dyseidetic, and nonspecific subgroups. Respectively, these yielded no perceptual asymmetry, asymmetry only for spatial, and only for verbal test. Malateshe and Aaron (1982) found the expected asymmetry for dyseidetics but none for dysphonetics. Bakker and his colleagues (Bakker 1979; Bakker et al. 1980) interpreted their laterality findings as indicating 2 subtypes, one involving left and one right hemisphere deficiencies. Diverse and even discrepant as these findings are, a common thread is a lack of right-sided advantage for verbal material in dyslexics with evidence of language disorder. This is consistent with
the finding of no perceptual asymmetry for verbal processing by children with frank language disorder (Rosenblum and Dorman 1978).

An interhemispheric transfer deficiency would not be expected to yield straightforward laterality abnormality (although Obrzut and colleagues (1981, 1983) have tentatively related certain inconsistencies of laterality outcomes across different testing conditions to such a problem). Tests contrasting successive transfer of tactile information within (as contrasted to between) hands have been proposed as measures of callosal transfer capability (Galin et al. 1977, Galin et al. 1979), and some disproportion between within-hemisphere and between-hemisphere matching appears to obtain in dyslexics (Neff et al. in preparation), although poor readers have been reported to perform relatively poorly in both conditions (Kletzkin 1980).

In summary, the laterality data so far do not resolve the choice between the alternative models for the brain basis of dyslexia. The data that exist are consistent with underutilization of the left hemisphere in some cases (it being unclear whether this is based on defective structure or insufficient use of intact structure). In other cases, the laterality evidence for general hemispheric underusage is absent, suggesting a more limited dysfunction not involving the entire hemisphere.

The choice of model for the brain basis of dyslexia bears on another basic question: does the dyslexic patient function in a manner qualitatively or only quantitatively different from normal? The quantitative alternative can be subdivided into two further hypotheses: the dyslexic functions like an equal age normal who is not trying hard (cf. the underactivation models) or like a younger normal child (cf. the underdevelopment models). Early attempts to underwrite a qualitative distinction by identifying diagnostic error types in reading and writing (Orton 1937; Critchley 1970) have not found support. The most recent claim for a qualitative difference is by Boder and Jarrico (1982) who hold spelling to be disproportionately disadvantaged in both their subtypes of dyslexia. Important as it is, this question is nowhere near resolution. Moreover, even a cognitive imbalance theory such as that of Boder and Jarrico (1982) can be reduced to a quantitative formulation if an individual cognitive system, but not the whole brain, is considered. A selective weakness in a cognitive function, such as phonological analysis, might indeed leave the patient with an imbalance between that and other systems, causing changes in strategy leading to outcomes not found normally at any stage of development. But the nature of the change within the affected cognitive system could still conform to a quantitative model, in one or other of the two possible variants mentioned above. Whether the affected system operates immaturesly, weakly, or abnormally is not resolved by available evidence.
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