20 Learning Disabilities: Neuroanatomic Asymmetries

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More than 100 years have passed since the first reports of children with learning disabilities appeared in the literature (Bastian, 1898; Hinshelwood, 1900; Kussmaul, 1877; Morgan, 1896). Many of the reports attempted to explain why an estimated 3% to 6% of the school-age population could not learn consistent with what would be expected considering their intellectual ability and repeated attempts at instruction (Gaddes, 1985; Yule and Rutter, 1976). These early reports were often quite rich in clinical detail and provided insights that are remarkably consistent with present research findings.

In fact, in reviewing the early literature, Hynd and Willis (1988) concluded that by 1905 the number of observations that had emerged from the evolving literature was such that a number of tentative conclusions could be offered. Overall, the literature by 1905 supported the following: (1) reading disability (congenital word blindness) could manifest in children with normal ability, (2) males seemed to be more often affected than females, (3) children presented with varied symptoms, but all suffered a core deficit in reading acquisition, (4) normal or even extended classroom instruction did not significantly improve reading ability, (5) some reading problems seemed to be transmitted genetically, and (6) the core symptoms seemed similar to those seen in adults with left temporoparietal lesions.

While no one would contest the idea that learning disabilities may differentially manifest in many areas of learning, including arithmetic, writing, spelling, and so on, there is little doubt that it is with reading disabilities, or dyslexia, where most researchers have concentrated their efforts. For this reason and because so many researchers from neuropsychology, neurology, and neurolinguistics have focused their efforts on reading disabilities, we will examine this literature in an attempt to draw some meaning from the volumes of research that have investigated brain-behavior relationships in this most common of learning disabilities. In fact, an understanding of this literature and the theoretical ideas concerning the meaning of lateralized function and potentially associated deviations in brain morphology may well assist future scholars in their investigation of the neurobiological basis of other forms of learning disabilities.

As the early case studies suggested, learning disabilities have always been thought to have a neurologic origin and present definitions of learning disability reflect this perspective (Wyngaarden, 1987). However, the literature supporting this perspective has generated a great deal of controversy. As Golden (1982) and Taylor and Fletcher (1983) have pointed out, much if not most of the literature through the early part of the 1980s was correlational in nature.

For example, some research indicates that reading-disabled children have an increased incidence of electrophysiologic abnormalities (Duffy et al., 1980) and perhaps differentially so in subtypes of reading disabilities (e.g., Fried et al., 1981). Soft signs are also more frequently found in reading-disabled children (Peters, Romine, and Dykman, 1975) and few would argue that reading-disabled children have a higher incidence of left- or mixed handedness (Bryden and Steenhuis, 1991). Further, reading-disabled children are often inferred to have weak or incomplete laterality, as evidenced on perceptual measures such as dichotic listening (Obrzut, 1991). In fact, volumes summarizing the research in this area have been written (Bakker and van der Vlugt, 1989; Gaddes, 1985; Kershner and Chyczij, 1992; Obrzut and Hynd, 1991), but we are still to a significant degree left with inferential or correlative evidence supporting the presumption of a neurologic etiology for learning disabilities. Typical of such inferential evidence were studies that found that children with learning disabilities performed more poorly than normal children on any given task (cognitive or perceptual) but did better than children with documented brain damage (e.g., Reitan and Boll, 1973). Needless to say, the inference was often made that the learning-disabled children suffered "minimal brain dysfunction" because their level of performance was somewhere between normality and known brain damage. This was clearly an inference and while not without merit theoretically, it did not directly correlate a known neurologic deviation of any kind (e.g., developmental, traumatic) with observed behavioral or cognitive deficits, as we might find in learning-disabled children.

This absence of confirming evidence is certainly not due to a shortage of theories or research, however. Historically relevant is the theory of Orton (1928) who proposed that as children become more linguistically competent, the left cerebral hemisphere becomes progressively more dominant for speech and language. He believed that motor dominance and its evolution in the developing child reflected this process of progressive lateralization. Consequently, according to Orton, children who had mixed cerebral dominance, as might be reflected in poor language skills, reading words or letters backward and inconsistent handedness, were most likely delayed in cerebral lateralization and therefore neither cerebral hemisphere, particularly the left, was dominant for linguistic processes. While decades of research documented that learning-disabled children were indeed deficient in language processes, especially phonological coding, the model of progressive lateralization has not been supported by the research (Benton, 1975; Kinsbourne and Hiscock, 1981; Satz 1991).

Actually, there is a growing body of evidence that indicates that very young children, including infants, are lateralized for language processing (Molfese and Molfese, 1986). Thus, none would refute the notion that in the majority of cases language is lateralized to the left cerebral hemisphere. However, while language abilities clearly develop over the course of human ontogeny, language remains lateralized as it was early in infant development. What may devolve is the capacity for plasticity of function; i.e., the capacity for the other cerebral hemisphere to assume language functions when the dominant hemisphere is severely damaged may decrease significantly with the course of development (Piacentini and Hynd, 1988).

What neurologic structures or deficient neuropsychological systems underlie the behavioral and cognitive symptoms we associate with learning disabilities, particularly reading disabilities? While there are likely many different ways in which one could begin to address this question, we will approach this question from a neurolinguistic-neuroanatomic perspective. We first present a discussion of the lateralized system of language and associated reading processes and then examine its impact and relation to research that employs brain-imaging procedures to investigate morphologic differences in the brains of reading-disabled children and adolescents. In this fashion we hope to directly tie deviations in lateralized brain processes (e.g., language, reading) to potentially associated deviations in brain structure.

NEUROLINGUISTIC-NEUROANATOMIC MODEL

For over a century, those concerned with reading and language disorders have attempted to correlate observed functional deficits with the location of known brain lesions (Bastian, 1898; Dejerine, 1892; Dejerine and Vialet, 1893; Dejerine and Dejerine-Klumpke, 1901; Geschwind, 1974; Head, 1926; Kussmaul, 1877; Wernicke, 1910). These scholars and others interested in the lateralization and localization of language and reading processes contributed to a literature that resulted in a neurolinguistic model of language and reading referred to by some as the Wernicke-Geschwind model (Mayeux and Kandel, 1985). While Wernicke and Dejerine deserve the most credit for the development of this model, it is clear that Geschwind (1974) did much to revive interest in the perspective first proposed in part by Bastian (1898), Liepmann (1915), Marie (1906), and others, whose ideas were controversial even when they were first proposed. As Head (1926) suggested over 60 years ago, "localization of speech became a political question; the older conservative school, haunted by the bogey of phrenology, clung to the conception that the 'brain acted as a whole,' whilst the younger liberals and Republicans passionately favored the view that different functions were exercised by the various portions of the cerebral hemispheres" (p. 25).

Even among the "diagram makers" (Head, 1926) controversy existed. For example, Bastian (1898) argued strongly against the popular perspective

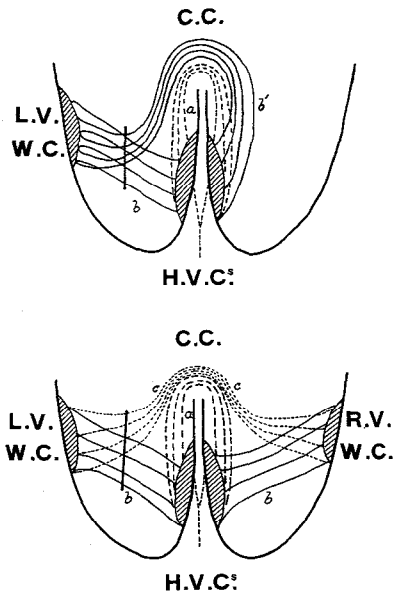

Figure 20.1 A comparison of Dejerine's and Bastain's views on the neuroanatomic basis of "pure word blindness" as presented by Bastian (1898). (*Above*) A simplified diagram representing Dejerine's views as to the mode of production of pure word blindness. The *dark line* indicates the site of a lesion which would cut off the left visual word center (L.V.W.C.) from the half-vision center (H.V.C.) of each side. (*Below*) A diagram representing Bastian's views as to the mode of production of pure word blindness. C.C., corpus callosum.

advocated by Dejerine whose views so influenced Geschwind in his thinking. Bastian proposed that bilateral visual word centers existed in the brain, each of which was involved in visual perception, low-level feature analysis, and cross-modal integration with the central language centers. Dejerine's views prevailed, however, as the accumulation of case studies supported the notion that there was indeed a left-lateralized "word center," most notably, it seemed, in the region of the angular gyrus. Figure 20.1 graphically contrasts Dejerine and Bastian's views on the posterior cortex involved in reading.

Based on the contributions of Broca, Wernicke, and the others noted above, a more complete neurolinguistic model of language and reading evolved. This model presupposes that visual stimuli such as words are registered in the bilateral primary occipital cortex, meaningful low-level perceptual associations occur in the secondary visual cortex, and this input is shared with further input from other sensory modalities in the region of the angular gyrus in the left cerebral hemisphere. This sequential neurocognitive process presumably then associates linguistic-semantic comprehension with input from the region of the angular gyrus; a process which involves the cortical region of the left

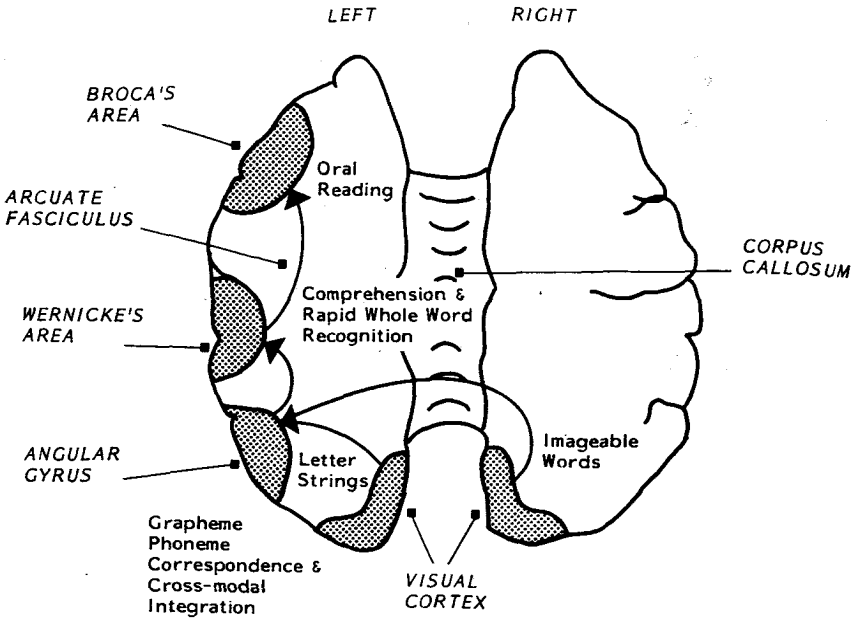

Figure 20.2 The brain as viewed in horizontal section. The major pathways and cortical regions thought to be involved in reading are depicted. Neurolinguistic processes important in reading are also noted.

posterior superior temporal region, including the region of the planum temporale. Then the process is completed when interhemispheric fibers connect these regions with Broca's area in the left inferior frontal region. Figure 20.2 presents this model, and the prevailing view of Dejerine's theory regarding the left lateralized "word center" can be seen in the posterior aspect of the figure.

It was Geschwind (1974), of course, who revived interest in this neurolinguistic-neuroanatomic model. He contributed significantly, however, by focusing attention on the natural left-sided asymmetry of the region of the planum temporale. Reports by early investigators (Flechsig, 1908; von Economo and Horn, 1930) encouraged Geschwind and Levitsky (1968) to investigate asymmetries associated with the region of the planum temporale. They examined 100 normal adult brains and found that the region of the planum temporale (the most posterior aspect of the superior temporal lobe) is larger on the left in 65% of brains, whereas it is larger on the right in only 11% of brains. These findings were taken as evidence of a specialized and asymmetric neuroanatomic region in support of language functions. Studies by other investigators documented the finding of plana asymmetry in both adult and infant brains (Kopp et al., 1977; Rubens, Mahuwald, and Hutton, 1976; Wada,

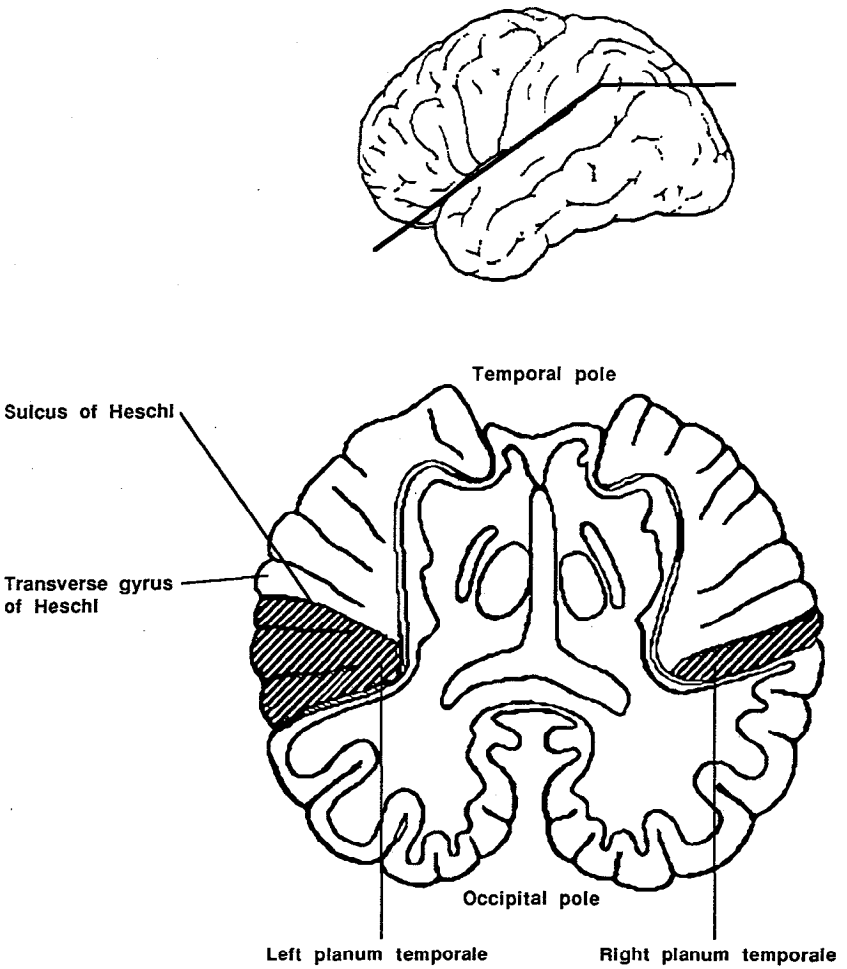

Figure 20.3 A graphic representation (*top*) of a slice up the sylvian (lateral) fissure exposing the posterior portion of the superior temporal region. The planum temporale is shaded bilaterally (*bottom*) and it can be seen that it is generally larger on the left.

Clarke, and Hamm, 1975; Witelson and Pallie, 1973). Figure 20.3 shows the left-sided asymmetry typically found in normal brains that is thought to subserve the evolution of higher-order neurolinguistic processes.

The research that was encouraged by the findings of Geschwind and Levitsky (1968) was significant in that other morphologic asymmetries in the human brain were soon reported. For example, Weinberger and colleagues (1982) found evidence that in approximately 75% of normal brains the right

frontal volume (R) exceeds that of the left frontal cortex (L). Also this pattern of L < R asymmetry seems evident in fetal development as early as 20 weeks. Other documented asymmetries include the left anterior speech region (pars opercularis and pars triangularis of the third frontal convolution) favoring the left side (Falzi, Perrone, and Vignolo, 1982) and cytoarchitectonic asymmetries favoring the left inferior parietal lobe (Eidelberg and Galaburda, 1984), the left auditory cortex (Galaburda and Sanides, 1980), and the posterior thalamus (Eidelberg and Galaburda, 1982).

Based on these as well as other research findings, Geschwind (1974, 1984) and especially Geschwind and Galaburda (1985a–c) argued that these natural asymmetries may be associated in a meaningful manner with language processes and, in cases of reversed asymmetry or symmetry, they may underlie the deficits we observe in severe reading disabilities. While the theory outlined by Geschwind and Galaburda (1985a–c) addresses the possible relations between male gender differentiation, the effects of testosterone on neuronal assemblies, and correlated asymmetries in brain morphology, immune function, and left-handedness, we are especially interested in our research with the idea that deviations in natural brain asymmetries may be related to the deficient linguistic and reading processes we observe in reading-disabled children. Thus, in this context, the remainder of this chapter will address the brain-imaging literature and examine the findings in relation to whether or not evidence exists in support of the notion that deviations in natural asymmetries in the language-reading system in the brain are indeed related in some fashion to the cognitive or behavioral deficits we observe in these children.

BRAIN IMAGING

Many different methodologies have been employed to investigate laterality and asymmetries in human performance. Certainly, visual half-field and dichotic listening experiments have assisted us greatly in better understanding perceptual asymmetries that underly linguistic and visuospatial perception. Dual-task paradigms have helped develop a better understanding of the lateralization of hemispheric attentional mechanisms and handedness–manual preference inventories have likewise helped in documenting variability in human laterality. All of these methodologies rely on the recording of a behavioral response which in turn leads to a measure of laterality.

The documentation of morphologic asymmetries in the human brain that seemed to favor the left hemisphere central language zones encouraged speculation that variability in these patterns of asymmetry might be related to the behavioral deficits we see in such conditions as severe reading disability. Geschwind and his colleagues deserve much of the credit for encouraging this perspective. In this context then, measures of manual preference or perceptual asymmetries might still be of interest but they could not provide a window from which to actually view the brain and its associated morphology.

Table 20.1 Brain imaging studies of subjects with developmental dyslexia

Study	Type	No. of subjects	Mean age (yr)	Diagnostic criteria	Conclusions
Hier et al. (1978)	CT	24	25	Less than 5th-grade reading level on Gray Oral Reading Test or >2-yr delay in reading while in school	Dyslexic subjects with reversed posterior asymmetry had lower verbal IQ 33% had normal L>R posterior asymmetry; 67% had symmetry or reversed (L<R) posterior asymmetry
LeMay (1978)	CT	27 dyslexic subjects[a] 317 controls	NR	NR	33% of dyslexic subjects had normal (L>R) posterior asymmetry compared to 70% of right-handed controls Left-handed controls evidenced more symmetry and reversed (L<R) asymmetry of posterior region
Leisman and Ashkenazi (1980)	CT	8 dyslexic subjects 2 controls	8.2—dyslexic subjects 7.6—normals	NR	100% of dyslexic subjects had symmetry or received asymmetry (L<R) of posterior region
Rosenberger and Hier (1980)	CT	53	6–45 (range)	Two grade levels below actual grade; large verbal-performance IQ discrepancy	42% of dyslexic subjects had reversed asymmetry (L<R) of posterior region Asymmetry index correlated with verbal-performance IQ discrepancy ($r=.38$, $P<.02$)
Haslam et al. (1981)	CT	26 dyslexic subjects 8 controls	11.7—dyslexic subjects 9.8—controls	Reading performance at least 2 yr below expected level based on IQ	46% of dyslexic subjects showed normal (L>R) posterior asymmetry while 87% controls did No relationship between IQ and posterior symmetry or asymmetry
Rumsey et al. (1986)	MRI	10	22.6	Childhood history of reading disability; median Gray Oral Reading Test was 3.7 grade equivalent	90% of dyslexic subjects showed symmetry of posterior regions
Parkins et al. (1987)	CT	44 dyslexic subjects 254 controls	57	Childhood history of reading and spelling disability, psychometrc evidence of dyslexia	Concluded that reversed posterior asymmetries are not characteristic of right-handed dyslexic subjects, but left-handed dyslexic subjects may evidence more symmetry

Table 20.1 (cont.)

Study	Type	No. of subjects	Mean age (yr)	Diagnostic criteria	Conclusions
Larsen et al. (1990)	MRI	19 dyslexic subjects 19 normals	15.1—dyslexic subjects 15.4—controls	Highly significant difference between normals and dyslexic subjects in word recognition; selected prior to study by schools as dyslexic	Measured the patterns of asymmetry in the region of the planum temporale: 70% of dyslexic subjects evidenced symmetry, while only 30% of nondyslexic subjects did All dyslexic subjects with plana asymmetry demonstrated significant phonological coding deficits
Hynd et al. (1990)[b]	MRI	10 dyslexic subjects 10 ADDH children 10 normals	9.9—dyslexic subjects 10.0—ADDH children 11.8—normals	IQ $\geqslant$ 85, positive family history, reading achievement $\geqslant$ 20 standard score points below full-scale IQ on tests of word recognition and passage comprehension	Both dyslexic subjects ADDH children had smaller right frontal widths (more frontal symmetry than normals) 70% of normal and ADDH children demonstrated L > R plana asymmetry, while only 10% of dyslexic subjects did; plana symmetry or reversed asymmetry seems characteristic of dyslexia
Semrud-Clikeman et al. (1991)	MRI	Same as Hynd et al. (1990)	Same as Hynd et al. (1990)	NR	Frontal width symmetry/reversed asymmetry (L $\geqslant$ R) associated with very significant delay in word attack skills Symmetry/reversed asymmetry of plana associated with poor confrontational naming, rapid naming, and passage comprehension
Leonard et al. (1993)	MRI	9 dyslexic subjects 10 relatives 11 controls	15–65 6–63 14–52	Primarily by clinical report and history	Dyslexic subjects had exaggerated left plana asymmetry for the temporal band and right asymmetry for the parietal bank Higher incidence of cerebral anomalies bilaterally

Modified from Hynd and Semrud-Clikeman (1989)

Key: ADDH, attention-deficit hyperactivity disorder; NR, not reported.

[a] LeMay (1981) used all subjects of Hier et al. (1978) adding three of her own in addition to the controls.

[b] Semrud-Clikeman et al. (1991) employed these subjects to examine the relationship between deviations in patterns of brain morphology and neurolinguistic ability in developmental dyslexics.

Computed tomography (CT) and magnetic resonance imaging (MRI) were obviously technologic advances that could help researchers examine directly structure-function relations in living humans. CT, of course, is considered an invasive procedure as there is some limited exposure to radiation, whereas with MRI scans there are no known risk factors. Until MRI became more readily available, CT was the method employed to examine deviations in normal patters of asymmetry in the brains of reading-disabled children and adults. CT studies typically employed a scan between 0 and 25 degrees above the acanthomedial line to examine for posterior asymmetries. With the increased sophistication of MRI scanning procedures it became possible to obtain thinner slices and extreme lateral sagittal scans were used to examine sulcul topography as well. Most scanning facilities now have the capability to obtain three-dimensional volumetric scan data so that later reconstructions can be made on any plane desired. These technological advances have been accompanied by very significant methodologic challenges with regard to head positioning, using a standardized grid system to normalize data acquisition across scans, and other difficulties in defining morphologic boundaries that may have functional significance. Nonetheless, these studies have been revealing and have encouraged increasing interest in using brain-imaging procedures to investigate many issues important to the study of lateralized functioning.

As can be seen in table 20.1, 11 studies using either CT or MRI have been conducted to examine whether or not deviations in normal patterns of asymmetry in brain morphology are associated with the manifestation of reading disabilities. The first such study was reported by Hier and colleagues (1978) who employed CT to investigate posterior asymmetries in 24 dyslexic subjects. They found that only 33% of the dyslexic group had a wider left posterior region while 67% had either symmetry or reversed asymmetry of the posterior region. Since fully 66% of the normal population is expected to show the expected L > R asymmetry, this lower incidence among the dyslexic group was taken as support for Geschwind's (1974) idea that patterns of asymmetry were meaningfully associated with linguistic functioning.

In a further study, Rosenberger and Hier (1980) found that a brain asymmetry index correlated with verbal performance intelligence quotient (IQ) discrepancies, whereas lower verbal IQ was correlated with symmetry or reversed asymmetry in the posterior region in the dyslexic subjects. This study actually was the first to examine whether there was any psychometric or behavioral relationship between asymmetry patterns and performance. In this respect this study was unique and an entire decade elapsed before several new studies also examined behavioral relationships to brain morphology data. Thus, most of the early literature was characterized by examining the rather straightforward issue as to whether there was any deviation from normal patterns of brain asymmetry in subjects with severe reading disability.

In 1981, Haslam et al. found in their sample of dyslexic subjects that 46% had L > R asymmetry similar to the normals, but in contrast to Rosenberger

and Hier (1980), no relationship was found with regard to verbal ability. As Hynd and Semrud-Clikeman (1989) have pointed out, however, the criteria employed by Haslam et al. for defining language delay were less strict than in the Rosenberger and Hier study. Nonetheless, Haslam et al. (1981) did note that fewer dyslexic subjects had the normal L > R posterior asymmetry.

The mid-1980s marked a time of transition in that fewer CT studies were reported with increasingly more studies employing MRI procedures as MRI scanners became more available to the research community. In fact, the last CT study reported was by Parkins et al. (1987) who found that there existed some relationship of handedness to deviations from normal patterns of asymmetry by dyslexic subjects. They found in their older adult sample (mean age, 57 years) that symmetry of the posterior region was characteristic only in the left-handed dyslexic subjects. The results of this study are unusual because previously and in the studies to follow, handedness may have differentiated the normal from the severely reading-disabled sample, but no relationship was ever reported with handedness. The mean age of this sample is also unusual as these were reading-disabled adults who may represent an unusual part of the reading disability spectrum in that their reading disability persisted to such a severe degree well into advanced adulthood. Most other studies typically employed subjects in early adolescence through young adulthood.

The first reported MRI study was reported in 1986 by Rumsey et al. who found in their brief report that 90% of the dyslexic subjects showed evidence of posterior asymmetry. In a sense, this study was typical of the rather unsophisticated methodology that characterized the studies at that time in that determination of asymmetry, symmetry, and reversed asymmetry of the posterior region most often relied on the clinical judgment of a radiologist or other expert in reading scans. Rarely were data presented as to the morphometric measurements that were obtained, if any, and for this reason it was difficult to compare results across studies. About the only conclusion that could reasonably be advanced was that deviations in normal patterns of posterior asymmetry may be found more frequently in the brains of severe reading-disabled persons. Based entirely on the Rosenberger and Hier (1980) study, there was limited but tantalizing evidence that symmetry or reversed asymmetry may somehow be associated with poor verbal-linguistic ability as is often found in dyslexic children.

To this point most studies had focused on posterior asymmetries, but theory had continued to emphasize the region of the planum temporale as being vitally important in verbal-linguistic processes, particularly phonological coding. In fact, Galaburda et al. (1985) summarized their four consecutive autopsy cases and reported that the focal dysplasias clustered preferentially in the left superior posterior temporal region by a ratio of 11 : 1. Thus, there was good reason to shift the attention of researchers away from simple posterior asymmetries toward attempts at measuring asymmetry of the region of the planum temporale. The focal dysplasias, Galaburda and colleagues reported, certainly could not be visualized on MRI scans, but different method could be

employed in attempting to measure either the area or length of this region bilaterally in the brains of persons with dyslexia.

Two studies employed different methodologies aimed at investigating asymmetries in the region of the planum temporale in dyslexic persons. Using MRI to examine the size and patterns of asymmetry in this region in adolescents with dyslexia, Larsen, and colleagues (1990) found that 70% of their dyslexic group had symmetry in the region of the plana in contrast to 30% of the normals. In addition to the importance of this finding, Larsen et al. also found that when symmetry of the plana was present in dyslexia, the subjects demonstrated phonological deficits. They concluded that some relationship may exist between brain morphology patterns and neurolinguistic process, consistent with Rosenberger and Hier's (1980) conclusions.

That same year, Hynd et al. (1990) also reported a study employing MRI in which the relative specificity of patterns of plana morphology were investigated in relation to a population of normal controls and clinic control children. In this case the clinic control group comprised children with attention-deficit hyperactivity disorder (ADDH). For this reason, the study was unique in that of all studies reported previously, none had included a clinic contrast group but rather compared dyslexic subjects only with normal controls. While such an approach has value in determining whether a line of investigation might be productive, the results only suggested differences from normals. There was no way to address the specificity of deviations in brain morphology in relation to the behavioral deficits seen in any one clinical syndrome such as reading. Based on the previous literature, it was hypothesized that if differences existed in the brains of the dyslexic children in the region of the plana, similar differences would not be evident in the brains of the ADDH children who were carefully diagnosed so that this group did not include children with reading or learning disabilities.

Similar to Larsen et al. (1990), Hynd, et al. (1990) found that the dyslexic group was characterized by either symmetry or reversed asymmetry ($L < R$) of the plana. Underscoring the importance of this region scientifically, they found that in 70% of the normals *and* ADDH children, $L > R$ plana asymmetry existed. This is what would be expected according to the normative data provided originally by Geschwind and Levitsky (1968). Fully 90% of the dyslexic children demonstrated symmetry or reversed asymmetry of the plana. In a follow-up study, Semrud-Clikeman and colleagues (1991) reported that symmetry and reversed asymmetry of the planum temporale was associated with significant deficits in confrontational naming, rapid naming, and neurolinguistic processes in general.

If one compares the Larsen et al. (1990) and Hynd et al. (1990) studies, differences seem evident in the way in which the plana were measured. Hynd et al. (1990) measured the length of the plana on extreme lateral sagittal MRI scans. Larsen et al. (1990), however, took measurements from sequential scans so that a measurement of area could be derived. Both studies found that significant indices of symmetry or reversed asymmetry characterized the

brains of dyslexic children even though different methodologies were employed. A point to derive from this discussion is that there are no agreed-upon standardized methodologies, although the method employed by Larsen et al. (1990) most likely provides more reliable data. Further, in examining the literature regarding the neuroanatomic morphology of the plana, one quickly realizes that there may be different sulcul patterns associated with whether or not a parietal bank of the planum temporale exists.

In the most recent study, reported by Leonard et al. (1993), the morphology of the posterior superior temporal region was examined bilaterally including the relative contribution of the temporal and parietal banks to an asymmetry index. The results of this study are particularly revealing in several ways. First, it turns out that nearly all dyslexic subjects and normals demonstrated a natural leftward asymmetry in the temporal bank and a rightward asymmetry in the parietal bank. When they examined intrahemispheric asymmetry, some dyslexic subjects had an anomalous intrahemispheric asymmetry between the temporal and planar banks in the right hemisphere because of an increased proportion of the plana being in the parietal bank. What this suggests is that consideration in future studies must be given to measuring both the temporal and parietal banks of the planum temporale and the relative contribution of both banks bilaterally in deriving asymmetry indexes. To quickly illustrate this issue the reader may wish to refer to figure 20.3 which illustrates the typical fashion in which the plana were described in the literature. By looking at the figure at the top where the slice location is noted, one can see at the end of the sylvian fissure where the slice line cuts horizontally that there is a small ascending ramus that is actually part of the planum. By not including this parietal aspect in lateral measures of asymmetry, the Larsen et al. (1990) and Hynd et al. (1990) studies were incomplete, although at the time they were published they were excellent studies. Finally, the Leonard et al. (1993) study documented that the dyslexic persons were more likely to evidence anomalies such as missing or duplicated gyri bilaterally in the region of the posterior end of the lateral fissure. These cerebral anomalies most likely evolve somewhere between the 24 and 30th week of fetal gestation when gyration occurs and represent a neurodevelopmental anomaly possibly related to a genetic etiology. Figure 20.4 presents sample MRI scans from the Leonard et al. (1993) study showing the anomalous cortex in the dyslexic subjects.

What does this evolving literature suggest about cerebral morphology and lateralized function in reading-disabled or dyslexic children? First, it suggests that asymmetry may indeed be characteristic of most normal brains. Second, in the region of the planum temporale there may be an increased incidence of symmetry or reversed asymmetry if one only measures the temporal bank. If one measures the bilateral temporal and parietal banks in the dyslexic group one may actually end up with these persons having more leftward asymmetry because of intrahemispheric variation in the right hemisphere, at least according to Leonard et al. (1993). As the Leonard et al. (1993) study clearly indicates,

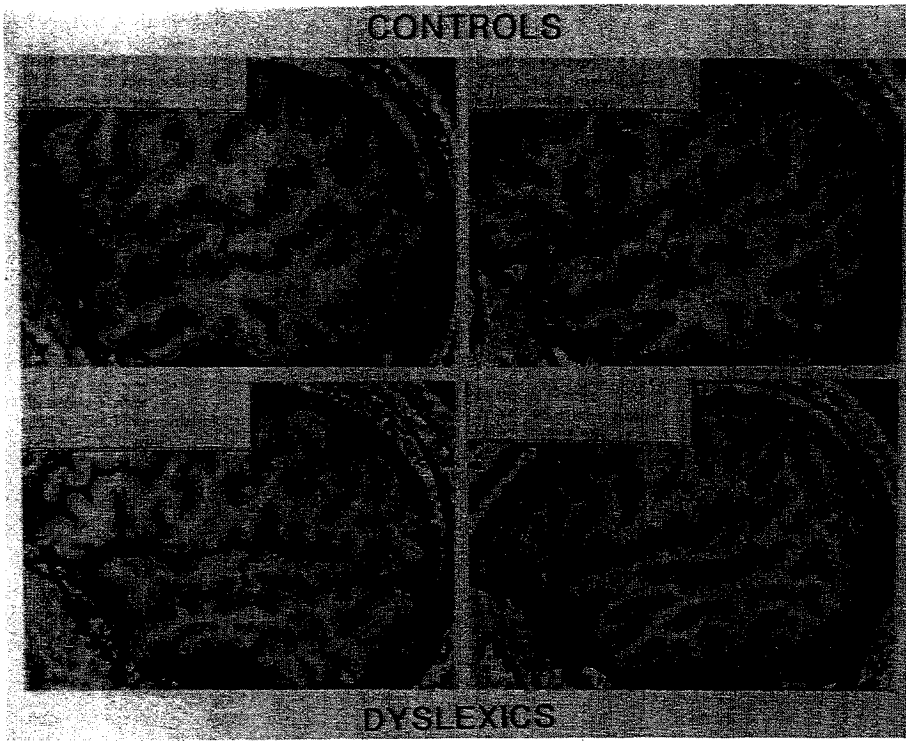

Figure 20.4 Magnetic resonance image of two controls and two dyslexic subjects from the Leonard, et al. (1993) study. The controls have either the typical bifurcation (*left*) or an ascending parietal bank (*right*) that enters a clearly demarcated supramarginal gyrus. Arrowheads mark the borders of the temporal bank of the planum. The two dyslexic subjects have multiple primary sulci and extra gyri in the parietal operculum (stars) and/or multiple Heschl's gyri (stars in lower right), or all of these anomalies.

measuring highly variable brain regions in different subject groups is fraught with complications, and decisions that must be made in terms of what to measure can dramatically influence outcomes. Finally, as Rosenberger and Hier (1980) first suggested, there may indeed be relationships between deviations in brain morphology and neurolinguistic processes. The Larsen et al. (1990) and Semrud-Clikeman et al. (1991) studies provide further support for this important aspect of the theory advanced by Geschwind (1974, 1984).

UNANSWERED QUESTIONS AND FUTURE RESEARCH AGENDA

There should be little doubt that brain-imaging procedures offer much promise in investigating issues related to possible relationships between brain

structure morphology and behavioral observations, whether these observations be clinical or experimental. What needs to be kept in mind, however, is that across all of these studies in which over 200 subjects have been scanned, not one brain of a reading-disabled subject was judged to be abnormal in structure (other than asymmetry patterns). In other words, no evidence of brain damage was found. This should underscore the important findings of Galaburda and colleagues (1985) who find developmental anomalies in the brains of dyslexic persons. The anomalous cortex identified by Leonard et al. (1993) provides further data implicating neurodevelopmental processes as underlying the behavioral symptomatology we see in dyslexia. It appears that reasonable evidence exists implicating unusual developmental processes sometime during the fifth to seventh month of fetal gestation in dyslexia. Clearly, the exact cause of these neurodevelopmental anomalies is one of the most important unanswered questions.

However, with regard to measures of brain asymmetry, it must be pointed out that if 65% of the normal population has $L > R$ asymmetry in the region of the plana, then some 35% have some other pattern ($L \leq R$) that does not seem to put them at risk for exclusion from the normal control population. That is, symmetry or reversed asymmetry is not sufficient to cause or predict deficient linguistic processes or dyslexia since some 35% of normals may likewise have brains with symmetry or reversed asymmetry of the plana.

There are two points to make in this regard. First, as Rosenberger and Hier (1980) suggested, symmetry or reversed asymmetry may not be a sufficient cause of reading or language problems but rather, in the context of a predisposition for language or reading difficulty (e.g., genetically at risk), symmetry or reversed asymmetry may act as an additional risk or potentiating factor. Second, to date no one has examined variability in brain morphology either with regard to asymmetry or sulcul patterns in normal persons for whom extensive neuropsychological, experimental, or behavioral data exist. It may well be that symmetry or less common sulcul topography is related to the significant variability we observe in the normal range of what would be considered individual differences. Investigating this issue should be a clear priority in future studies, especially since by using three-dimensional whole-brain acquisitions, reconstructions can be made in any plane and, if desired, volumetric data can be generated for any region. However, the value of volumetric data at present is questionable since functional, cytoarchitectural, and sulcul-lobe boundaries do not necessarily correlate well. Therefore, dividing the brain up into sections based on qualitative judgments may lead to unreliable and theoretically questionable results. One hopes, however, that future investigators will be able to address this issue in some fashion. For example, it is possible to obtain brain metabolic scans (e.g., positron emission tomography or single photon emission CT) and overlay MRI scans to identify more clearly areas or regions of increased metabolic activity in response to some cognitive task demands. One could then define the functional boundaries in the brain for certain tasks and use this topographically derived area as

a starting point for examining how deviations in morphology (perhaps sulcul patterns) relate to levels and perhaps topographic distribution of functional activation. Integrating multiple sources of brain-imaging data with behavioral data should be a priority, especially in regard to answering some questions about hemispheric laterality.

While research aimed at better understanding the neurobiological basis of learning disabilities has shown significant progress over the past several decades, it is still not known with any certainty why focal deviations in the cellular architecture occur or how they relate to the deficient learning abilities in dyslexic children. Equally, those engaged in MRI work with dyslexic children still do not have a good idea as to how exactly deviations in patterns of expected brain morphology cause or relate to the linguistic deficits we observe in these children. In a rather discouraging sense we are left in the same position as those who inferred brain damage or dysfunction on the basis of psychometric tests; we infer that there is some meaningful relationship between deviations in asymmetry or morphology and learning disabilities. We seem to overlook the fact that these anomalies also occur, perhaps at a lower incidence for some anomalies, in the presumably normal population. This needs to be kept in mind as our enthusiasm grows, in hopes of eventually uncovering the invariably complex interactions that must exist between the morphology of the brain, its many neurotransmitter systems, and the behaviors we deem important enough to study.

REFERENCES

Bakker, D. J., and van der Vlugt, H. (Eds.) (1989). *Learning disabilities*, Vol 1. *Neuropsychological correlates and treatment*. Amsterdam: Swets & Zeitlinger.

Bastian, H. C. (1898). *A treatise on aphasia and other speech defects*. London: H. K. Lewis.

Benton, A. L. (1975). Developmental dyslexia: Neurological aspects. In W. J. Friedlander (Ed.), *Advances in neurology*. New York, NY: Raven Press.

Bryden, M. P., and Steenhuis, R. (1991). The assessment of handedness in children. In J. E. Obrzut and G. W. Hynd (Eds.), *Neuropsychological foundations of learning disabilities: A handbook of issues, methods, and practice* (pp 411–436). New York, NY: Academic Press.

Dejerine, J. (1892). Contribution à l'étude anatomopathologique et clinique des differentes variétés de cécité verbale. *Mémoires de la Societé de Biologie, 4*, 61–90.

Dejerine, J., and Dejerine-Klumpke, A. (1901). *Anatomie des centres nerveux*, Vol 2 (pp 109–114). Paris: Rueff et Cie.

Dejerine, J., and Vialet, P. (1893). Contribution à l'étude de la localisation anatomique de la cécité verbale pure. *Comptes rendus des Seánces de la Societé de Biologie, 45*, 790–793.

Duffy, F. H., Denckla, M. B., Bartels, P. H., Sandini, G., and Kiessling, L. S. (1980). Dyslexia: Automated diagnosis by computerized classification of brain electrical activity. *Annals of Neurology, 7*, 421–428.

Eidelberg, D., and Galaburda, A. M. (1982). Symmetry and asymmetry in the human posterior thalamus: I. Cytoarchitectonic analysis in normal persons. *Archives of Neurology, 39*, 325–332.

Eidelberg, D., and Galaburda, A. M. (1984). Inferior parietal lobule: Divergent architectonic asymmetries in the human brain. *Archives of Neurology, 41,* 843–852.

Falzi, G., Perrone, P., and Vignolo, L. A. (1982). Right-left asymmetry in anterior speech region. *Archives of Neurology, 39,* 239–240.

Flechsig, P. (1908). Bemerkungen über die Hemisphären des menschlichen Gehirns. *Neurologisches Zentralblatt, 27,* 50–57.

Fried, I., Tanguay, P. E., Boder, E., Doubleday, C., and Greensite, M. (1981). Developmental dyslexia: Electrophysiological evidence of clinical subgroups. *Brain and Language, 12,* 14–22.

Gaddes, W. H. (1985). *Learning disabilities and brain function: A neuropsychological approach,* ed 2. New York, NY: Springer-Verlag.

Galaburda, A. M., and Sanides, F. (1980). Cytoarchitectonic organization in the human auditory cortex. *Journal of Comparative Neurology, 190,* 597–610.

Galaburda, A. M., Sherman, G. F., Rosen, G. D., Aboitiz, F., and Geschwind, N. (1985). Developmental dyslexia: Four consecutive cases with cortical anomalies. *Annals of Neurology, 18,* 222–233.

Geschwind, N. (1974). The development of the brain and the evolution of language. In N. Geschwind (Ed.), *Selected papers on language and the brain* (pp 122–146). Dordrecht, Netherlands: D. Reidel.

Geschwind, N. (1984). Cerebral dominance in biological perspective. *Neuropsychologia, 22,* 675–683.

Geschwind, N., and Galaburda, A. M. (1985a). Cerebral lateralization: Biological mechanisms, associations, and pathology, I. A hypothesis and program for research. *Archives of Neurology, 42,* 428–459.

Geschwind, N., and Galaburda, A. M. (1985b). Cerebral lateralization: Biological mechanisms, associations, and pathology, II. A hypothesis and program for research. *Archives of Neurology, 42,* 521–552.

Geschwind, N., and Galaburda, A. M. (1985c). Cerebral lateralization: Biological mechanisms, associations, and pathology. III. A hypothesis and program for research. *Archives of Neurology, 42,* 634–654.

Geschwind, N., and Levitsky, W. (1968). Human brain: Left-right asymmetries in temporal speech region. *Science, 161,* 186–187.

Golden, G. S. (1982). Neurobiological correlates of learning disabilities. *Annals of Neurology, 12,* 409–418.

Haslam, R. H., Dalby, J. T., Johns, R. D., and Rademaker, A. W. (1981). Cerebral asymmetry in developmental dyslexia. *Archives of Neurology, 38,* 679–682.

Head, H. (1926). *Aphasia and kindred disorders of speech.* Cambridge, England: Cambridge University Press.

Hier, D. B., LeMay, M., Rosenberger, P. B., and Perlo, V. P. (1978). Developmental dyslexia: Evidence for a subgroup with a reversal of cerebral asymmetry. *Archives of Neurology, 35,* 90–92.

Hinshelwood, J. (1900). Word-blindness and visual memory. *Lancet, 2,* 1564–1570.

Hynd, G. W., and Semrud-Clikeman, M. (1989). Dyslexia and brain morphology. *Psychological Bulletin, 106,* 447–482.

Hynd, G. W., Semrud-Clikeman, M., Lorys, A. R., Novey, E., and Eliopulos, D. (1990). Brain morphology in developmental dyslexia and attention deficit disorder/hyperactivity. *Archives of Neurology, 47,* 919–926.

Hynd, G. W., and Willis, W. G. (1988). *Pediatric neuropsychology.* Needham Heights, MA: Allyn & Bacon.

Kershner, J., and Chyczij, M. (1992). Lateral preference in six to nine year old children: Relationships to language lateralization and cognitive ability. *Learning and Individual Differences, 4,* 347–367.

Kinsbourne, M., and Hiscock, M. (1981). Cerebral lateralization and cognitive development: Conceptual and methodological issues. In G. W. Hynd and J. E. Obrzut (Eds.), *Neuropsychological assessment and the school-age child* (pp 125–166). New York, NY: Grune & Stratton, Inc.

Kopp, N., Michel, F., Carrier, H., Biron, A., and Duvillard, P. (1977). Étude de certaines asymétries hémisphériques de cerveau humain. *Journal of the Neurological Sciences, 34,* 349–363.

Kussmaul, A. (1877). A disturbance of speech. *Cylopedia of Practical Medicine, 14,* 581–875.

Larsen, J. P., Hoien, T., Lundberg, I., and Odegaard, H. (1990). MRI evaluation of the size and symmetry of the planum temporal in adolescents with developmental dyslexia. *Brain and Language, 39,* 289–301.

Leisman, G., and Ashkenazi, M. (1980). Aetiological factors in dyslexia: IV. Cerebral hemispheres are functionally equivalent. *Neuroscience, 11,* 157–164.

LeMay, M. (1981). Are there radiological changes in the brains of individuals with dyslexia? *Bulletin of the Orton Society, 31,* 135–141.

Leonard, C. M., Voeller, K. K. S., Lombardino, L. J., Morris, M., Hynd, G. W., Alexander, A. W., Anderson, H. G., Garofalakis, M., Honeyman, J. C., Mao, J., Agee, O. F., and Staab, E. V. (1993). Anomalous cerebral morphology in dyslexia revealed with MR imaging. *Archives of Neurology, 50,* 461–469.

Liepmann, H. (1915). Diseases of the brain: Normal and pathological physiology of the brain. In C. W. Burr (Ed.), *Text-book on nervous disease* (pp 445–552). Philadelphia, PA: Blakiston.

Marie, P. (1906). Revision de la question de l'aphasie: La 3 circonvolution frontale gauche ne joue aucun rôle spécial dans la fonction du langage. *Semaine Médicale, 21* (May 23), 241–247.

Mayeux, R., and Kandel, E. R. (1985). Natural language, disorders of language, and other localizable disorders of cognitive functioning. In E. R. Kandel and J. H. Schwartz (Eds.), *Principles of neural science,* (ed. 2) (pp 688–703). New York, NY: Elsevier.

Molfese, D. L., and Molfese, V. J. (1986). Psychophysiological indices of early cognitive processes and their relationship to language. In J. E. Obrzut and G. W. Hynd (Eds.), *Child neuropsychology: Theory and practice.* Vol 1 (pp 95–115). New York, NY: Academic Press.

Morgan, W. P. (1896). A case of congenital word-blindness. *British Medical Journal, 2,* 1978.

Obrzut, J. E. (1991). Hemispheric activation and arousal asymmetry in learning disabled children. In J. E. Obrzut and G. W. Hynd (Eds.). *Neuropsychological foundations of learning disabilities: A handbook of issues, methods, and practice* (pp 179–198). New York, NY: Academic Press.

Obrzut, J. E., and Hynd, G. W. (Eds.) (1991). *Neuropsychological foundations of learning disabilities: A handbook of issues, methods, and practice.* New York, NY: Academic Press.

Orton, S. T. (1928). Specific reading disability—strephosymbolia. *Journal of the American Medical Association, 90,* 1095–1099.

Parkins, R., Roberts, R. J., Reinarz, S. J., and Varney, N. R. (1987). *Asymmetries in adult developmental dyslexics.* Paper presented at the annual convention of the International Neuropsychological Society, Washington, DC, January 1987.

Peters, J. E., Romine, J. S., and Dykman, R. A. (1975). A special neurological examination of children with learning disabilities. *Developmental Medicine and Child Neurology, 17,* 63–78.

Piacentini, J. C., and Hynd, G. W. (1988). Language after dominant hemispherectomy: Are plasticity of function and equipotentiality viable concepts? *Clinical Psychology Review, 8,* 595–609.

Reitan, R. M., and Boll, T. J. (1973). Neuropsychological correlates of minimal brain dysfunction. *Annals of the New York Academy of Sciences, 205,* 65–68.

Rosenberger, P. B., and Hier, D. B. (1980). Cerebral asymmetry and verbal intellectual deficits. *Annals of Neurology, 8,* 300–304.

Rubens, A. B., Mahuwold, M. W., and Hutton, J. T. (1976). Asymmetry of the lateral (sylvian) fissures in man. *Neurology, 26,* 620–624.

Rumsey, J. M., Dorwart, R., Vermess, M., Denckla, M. B., Kruest, M. J. P., and Rapoport, J. L. (1986). Magnetic resonance imaging of brain anatomy in severe developmental dyslexia. *Archives of Neurology, 43,* 1045–1046.

Satz, P. (1991). The Dejerine hypothesis: Implications for an etiological reformulation of developmental dyslexia. In J. E. Obrzut and G. W. Hynd (Eds.), *Neuropsychological foundations of learning disabilities: A handbook of issues, methods, and practice* (pp 99–112). New York, NY: Academic Press.

Semrud-Clikeman, M., Hynd, G. W., Novey, E., and Eliopulos, D. (1991). Dyslexia and brain morphology: Relationships between neuroanatomical variation and neurolinguistic performance. *Learning and Individual Differences, 3,* 225–242.

Taylor, H. G., and Fletcher, J. M. (1983). Biological foundations of "specific developmental disorders": Methods, findings, and future directions. *Journal of Clinical Child Psychology, 12,* 46–65.

Von Economo, C. and Horn, L. (1930). *Zeitschrift für die gesamte Neurologie und Psychiatrie, 130,* 678.

Wada, J. A., Clarke, R., and Hamm, A. (1975). Cerebral hemispheric asymmetry in humans. *Archives of Neurology, 32,* 239–246.

Weinberger, D. R., Luchins, D. J., Morihisa, J., and Wyatt, R. J. (1982). Asymmetrical volumes of the right and left frontal and occipital regions of the human brain. *Neurology, 11,* 97–100.

Wernicke, C. (1910). The symptom-complex of aphasia. In A. Church (Ed.), *Modern clinical medicine: Diseases of the nervous system* (pp 265–324). New York, NY: D. Appleton & Co.

Witelson, S. F., and Pallie, W. (1973). Left hemisphere specialization for language in the newborn. *Brain, 96,* 641–646.

Wyngaarden, J. E. (1987). *Learning disabilities: A report to the Congress.* Washington, DC: National Institutes of Health, Interagency Committee on Learning Disabilities.

Yule, W., and Rutter, M. (1976). Epidemiology and social implications of specific reading retardation. In R. M. Knights and D. J. Bakker (Eds.). *The neuropsychology of learning disorders: Theoretical approaches.* Baltimore, MD: University Park Press.