Neuroanatomical Evidence of Dyslexia (I): A Review of Brain Imaging Studies

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Developmental dyslexia is believed to be accompanied by some central nervous system dysfunction. Many researchers have shown anatomical and functional differences between the brains of individuals with dyslexia and those of individuals without dyslexia (Filipek, 1994 ; Galaburda, 1993 ; Hynd & Semrud-Clikeman, 1989 ; Hynd, Marshall, & Gonzalez, 1991).

There are several ways to examine one's central nervous system, and each method has advantages and disadvantages. Previous reviews mainly focused on one method and attempted to depict the characteristics of the brain of the individuals with dyslexia (Filipek, 1994 ; Galaburda, 1993 ; Hynd & Semrud-Clikeman, 1989 ; Hynd, Marshall, & Gonzalez, 1991). However, the results are not always consistent regarding the relationship between specific abnormality and dyslexia. Thus, several different techniques are reviewed in this series of papers in order to provide a better understanding on the relationship between central nervous system dysfunction and dyslexia. The review includes brain imaging techniques, regional cerebral blood flaw studies, and brain potential studies published in 90's.

Brain Imaging Studies

Magnetic resonance imaging (MRI) scan has been used to visualize brain structures in noninvasive way. Recent development in the technology enable to provide clearer images of the brain structures. Researchers have compared the sizes of certain structures of interest between dyslexic and normal group. However, the results are inconsistent partially because of their serious methodological limitations (Hynd & Semrud-Clikeman, 1989). In this section, the issues related to the characteristics of the subjects are discussed first. Then, the research findings on specific brain structures are reviewed.

Characteristics of the Subjects

As summarized by Hynd and Semrud-Clikeman (1989), subject selection process may be one of the sources of the inconsistency. Poor description of the subjects makes it

This is the first part of a series of review articles. The characteristics of subjects appeared in the studies reviewed are summarized in Tables 1 at the end of this article. The summary of results will appear in the second part of this series of articles.

impossible to compare the results across studies. Heterogeneity of the clinical group hurts statistical power in the analyses. The characteristics of the subjects in the studies reviewed in this section are summarized in Table 1. It seems that the characteristics of the subjects are well documented in most of these more recent studies.

Control groups were included in all of the studies reviewed. Their selection criteria are not the same. Since age, sex, intelligence are influential on the relative size of some structures in brain, it is very important to control these variables. If it is impossible to control them during the selection process, researchers should control these variable as covariates to adjust the group difference like Kushch, et al. (1993) and Schultz, et al. (1994) did. Handedness is not usually controlled. Some researchers dealt with this variable as an outcome variable (Larsen, Høien, & Ødegaard, 1990).

Hemispheric Asymmetry

Normal brains have a specific asymmetry patterns in a specific part. For example, about 70% of normal brain shows L > R asymmetry in the posterior region and L < R asymmetry in the anterior region (Hynd & Semrud-Clikeman, 1989). This asymmetry pattern has not be found in dyslexic subjects. Most of the earlier studies reported more symmetry or reversed asymmetry in the posterior region (Hynd & Semrud-Clikeman, 1989). Asymmetry pattern in frontal part was not always reported, and the results did not indicate significant difference from that of the control group when they were reported (Hynd & Semrud-Clikeman, 1989). These studies determine the hemispheric asymmetry by measuring the width of a posterior and/or anterior part of a horizontal slice.

Recent studies examined hemispheric asymmetry in several different ways, which included areas of a horizontal slice, volume, and surface area. Duara, et al. (1991) found that dyslexics showed L < R asymmetry determined by the areas of the middle part of the posterior halves, whereas normal subjects showed a symmetry pattern. Hynd, et al. (1990) did not find the group difference of the asymmetry pattern in the posterior area and width, but reported a different asymmetry pattern in the anterior region (dyslexia : L= R ; normal : L < R). This lack of group difference in posterior asymmetry may be stemmed from the difference in measured parts to determine the posterior asymmetry from the previous studies.

Although previous studies have reported different asymmetry patterns between dyslexics and normals, we need to keep in mind that the asymmetry pattern is not definitive for both groups. Handedness also seems to contribute to the different asymmetry patterns as Hier, LeMay, Rosenberger, and Perlo (1978) and Parkins, Roberts, Reinarz, and Varney (1987) reported (reviewed in Hynd & Semrud-Clikeman, 1989). Recent two studies were not able to add converging results to the previous studies, suggesting either hemispheric asymmetry pattern is not primary characteristic to

differentiate dyslexic group from normal group, or measures used in these studies were not sensistive enough to detect group difference.

Planum Temporale

There is evidence that the left region of planum temporale (PT) is larger than the right PT in majority of the normal brains (Geschwind & Levitsky, 1968). Since the left PT is located in the language processing area, PT asymmetry has been associated with functional lateralization of language processing. Since autopsy studies on dyslexic brains have reported the absence of this asymmetry (reviewed by Steinmetz & Galaburda, 1991), some abnormalities in this area are assumed to play a significant role in developmental dyslexia. However, it is only recently that researchers can use brain imaging technique to study this structure. The earlier scanners could not provide brain images that are clear enough to measure this small structure.

Among four studies reporting the PT asymmetry, Hynd, et al and Larsen, et al. presented data that showed different asymmetry patterns between groups. Reversed asymmetry was reported only in Hynd, et al. (1990), though symmetry and reversed asymmetry were combined and the exact percentage of reversed asymmetry is not clear. Larsen, et al. (1990) administared both phonological and orthographic decoding tasks and found that symmetry PT seems to be associated with phonological deficit in both dyslexic and normal group. On the other hand, Semrud-Clikeman, Hynd, Novey, & Eliopulos (1991) examined the relationship between linguistic performance and PT symmetry pattern and found that PT asymmetry is related with verbal comprehension. They concluded the PT as 'a general language center.' There is not enough empirical evidence to conclude the role of PT in dyslexia. Furher research on the linguistic function and PT is necessary for the function of PT.

The more recent two of the four studies (Leonard, et al., 1993 ; Schultz, et al. 1994) reported no difference between groups. Leonard, et al. reported similar L > R pattern of total PT length and Schultz, et al. showed more than 70% L > R asymmetry among both dyslexic and normal subjects. One common characteristic of subjects in these studies is that all the subjects are right-handed. The dyslexic subjects in these studies may not be a representative of dyslexic population, considering the fact that more left-handed subjects are usually found in dyslexic group in the previous studies.

In sum, we cannot make conclusion about the relationship between dyslexia and PT asymmetry pattern at this point. Again, definition of the structure is a critical factor to contribute to this inconsistency. As Filipek (1994) points out, PT measures in these four studies are not comparable because of different definition of the structure. Because of the variability of its shape, any two-dimensional image cannot provide an accurate measure of the structure.

Steinmetz and Galaburda (1991) argued that PT symmetry or asymmetry alone cannot explain the presence of dyslexia, because 25% of randomly selected individuals have symmetry PT. One of the explanations for the role of PT asymmetry is that it is a manifestation of normal variation that prevents functional compensation for other cortical pathology. Another hypothesis is that the PT symmetry is a pathologic consequence of legion-induced abnormal corticogenesis. There is another piece of evidence that shows PT symmetry is not unique to dyslexia. Petty, et al., (1995) reported the strong association between schizophrenia and reversed asymmetry PT.

Symmetry or reversed asymmetry PT may be one of the characteristics of dyslexia. However, the contribution of the symmetry or reversed asymmetry of PT to the development of dyslexia is not large enough to be detected in the statistical analysis, unless there are large sample size, accurate measurement, and homogeneous dyslexic group and well matched control group.

Temporal Lobe

Uncertainty of the definition of the PT size lead some researchers to measure bigger structure of the brain which includes PT. Kushch, et al. (1993) measured surface area of the temporal lobe reconstructed with 5 mm thick coronal sections. They found that normal subjects showed greater leftward asymmetry than that of dyslexic group in the posterior half of the temporal lobe, but no difference were shown in the anterior half. On the other hand, Schultz, et al. (1994) could not find the group difference in their measures on temporal lobe after removing the effect of sex and age. Although the authors emphasize the importance of the effects of sex and age, Kushch et al. 's results can not be attributed to the age and IQ factors because they control these variables as covariates. Kushch, et al. also did not find the sex effect on any measures in their study. Since Schultz, et al. did not compare the posterior and anterior parts, and age of the subjects are different in these two studies, we can not compare their results directly.

Insular Region

Insular region is one of the parts of the brain that is assumed to get involved in reading process (Mayeux, & Kandel, 1985 ; cited in Hynd, et al, 1990). Hynd, et al. (1990) is the only study which presented the data regarding the size of this structure. They found that bilaterally shorter insular length in dyslexic group than in control group. Considering the variability of the results found in other structure, this single piece of evidence is not enough to make any conclusion. However, insular region should be another structure to be analyzed in the future morphological study.

Corpus Callosum

Corpus Callosum (CC) has become one of the focuses of brain morphology study of dyslexia. Researchers assume that inter-hemispheric dysfunctions underlie dyslexia, and

the dysfunctions may be caused by abnormal CC (Larsen, Høien, & Ødegaard, 1992; Nijiokiktjien, de Sonneville, & Vaal, 1994)

The results are contradictory. Larsen et al. and Njiokitjien, et al. reported no significant difference between dyslexic and control groups. Dyslexic female subjects showed larger genu, splenium, and total CC in Duara, et al. 's (1991) study. Hynd, et al. (1990) presented data which showed smaller genu in dyslexic group.

Different definition of measured area, different criteria for subject selection may have contributed the contradictory results. Nijiokiktjien, et al. (1994) found that 11% of the subjects in dyslexic group showed undersized CC whereas 16% of them showed larger CC. This observation suggests that either directions of CC size abnormality may be associated with dyslexia. Further accumulation of the data is necessary to make any conclusion about the relationship between dyslexia and CC size.

Summary of the MRI Results

We can find relatively stronger evidence of the abnormal size or asymmetry pattern to dyslexia in PT and posterior hemispheric asymmetry. At least, contradictory results have not been reported for these measures. The results are also consistent with autopsy studies (Galaburda, 1993). There is not enough consistent evidence for other structures. Insular region, temporal lobe surface area are promising structures for future studies.

Recent morphological studies on dyslexia failed to add any convergence to the findings in the earlier studies reviewed in Hynd & Semrud-Clikeman (1989). Considering the improvement of the quality of images in newer studies, the association between a certain brain structure and dyslexia may not be so strong as expected. Another interpretation of the inconsistent results is the definition of the area measured. It is not easy to quantify a part of a brain to compare, because a human brain has vary complex structures and does not have clear, straight borderline to separate one structure from another. Researchers have applied different strategies to quantify the size of structures of interest.

Recommendations for Future Studies

One limitation of the studies reviewed is that authors have treated all the abnormality as independent factors. Thus, these are considered as additive model of morphological abnormality, because their assumption is that simply more abnormalities yield more severe forms of dyslexia. However, no one has tested whether the severity of the dyslexia changes depending on the multiple abnormalities. In other words, interaction effects of two or more abnormalities have never been examined. This can be called the interaction model. For example, PT symmetry can be found in subjects both with and without dyslexia, but it may contribute more significantly to the presence of dyslexia when smaller than normal CC exists simultaneously.

We can test the significance of the interaction by including them as explanatory (independent) variables, in multiple regression model (when reading ability or discrepancy score is the dependent variable) or discriminant analysis (when diagnosis is the dependent variable). Probably this kind of analyses have never been done because of the limited sample size. However, accumulation of the database will enable researchers to test this interaction model in the future.

Another recommendation is that breaking the dyslexic group down into more homogeneous group according to their cognitive characteristics. For example, Larsen, et al. (1990) found that PT symmetry is associated more with phonological deficits rather than orthographic deficits. Using reading tests which measure specific reading abilities will help characterizing cognitive ability of dyslexic subjects.

Finally, common definitions for each brain structure of interest should be established. Variability of measurement methods makes it impossible to compare the results across studies. Common definition will enable to develop large database of the subjects, which is necessary to conduct multivariate analysis proposed earlier in this section.

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Table 1 Summary of the Characteristics of Subjects (MRI Studies)

	diagnostic criteria	exclusion of subjects	other neurological disorder	selection of control		N (m / f)	Age	Handed-ness	IQ	Reading
Duara, et al. 1991	1.5 SD discrepancy on reading and/or spelling tests vs IQ, history of childhood reading and spelling problems, family history of dyslexia within 2 generations	neurologic, psychiatric disorders	ADHD	age and years of education matched	Measures			EHI	WAIS-R	(a) GORT-R, (b) WRAT-R, (c) NPRE, (d) NPRT
					Dyslexia	21 (12/9)	39.1	80	FSIQ 114.4	(a) 105.0, (b) 86.1, (c) 62.6, (d) 55.7
					Control	29 (15/14)	35.3	82	NR	(a) 119.5, (c) 102.7, (d) 101.3
Hynd et al. 1995	normal or better intelligence (FSIQ ≥ 85), severe discrepancy between inte IQ and reading achievement (≥ 20 standard score points) positive family history for LD	children who received other primary DSM III diagnoses, mild MR (FSIQ < 70), epilepsy, closed head injury, or other nureologic disorders	ADHD, ADD(2), developmental language disorder, dysthymia, & depression)	age, gender matched	Measures			EHI	WISC-R	(a) WRMT-WA, (b) WRMT-PC, (c) WRMT-T
					Dyslexia	16 (11/5)	9.71	71.88 , 12.5%	FSIQ 104.75 VIQ 103.13 PIQ 102.06	(a) 76.06 (b) 77.37 (c) 77.81
					Control	16 (11/5)	9.91	88.75 , 37.5%	FSIQ 122.19 VIQ 118.88 PIQ 122.00	(a) 107.69 (b) 112.88 (c) 113.63
Hynd et al. 1990	normal or better intelligence (FSIQ ≥ 85), severe discrepancy between FSIQ and reading achievement (≥ 20 standard score points) positive family history for LD, no symptoms of hyperactivity	child ren who received other primary DSM III diagnoses, mild MR (FSIQ < 70), epilepsy, closed head injury, or other nureologic disorders	ovreanxious disorder, major depressive episode, ADD	age and sex matched	Measures			% Right	WISC-R	(a) WRMT-WA, (b) WRMT-PC
					Dyslexia	10 (8/2)	9.91	38	FSIQ 108.00, VIQ 107.00, PIQ 107.70	(a) 73.80, (b) 75.00
					Control	10 (8/2)	11.77	96	FSIQ 125.40, VIQ 122.80, PIQ 124.80	(a) 115.60, (b) 112.30
Kushch, et al. 1993	Discrepancy between IQ and reading measures. Age ≤ 8: 0.5 SD on at least one measure, Age 9-14: 1.0 SD on at least 2 measure, Age ≥ 15: 1.5 SD on at least 2 measures.	no history of congenital, sensory/motor, neurological, or psychiatric problems.	ADD (3)	NR, age, IQ effects were adjusted as covariates	Measures			EHI	WISC & WAIS	(a) GORT-R, (b) WJL, (c) WJP, (d) WJW, (e) NPT, (f) NPE, (g) WRAT-R
					Dyslexia	17 (9/8)	25.8	.63	FSIQ 104.8 VIQ 103.4 PIQ 106.1	(a) 88.4, (b) 87.2, (c) 91.6 , (d) 85.8, (e) 54.4, (f) 62.0, (g) 83.0
					Control	21 (8/13)	33.4	.79	FSIQ 117.8 VIQ 114.8 PIQ 117.0	(a) 122.9, (b) 105.0, (c) 101.2 , (d) 107.5, (e) 95.0, (f) 103.6 , (g) 106.9

Table 2 MRI Result of Hemispheric Asymmetry

	diagnostic criteria	exclusion of subjects	other neurological disorder	selection of control	N (m / f)	Age	Handed-ness	IQ	Reading
Larsen et al. 1992	poor word recognition	poor intelligence, sensory deficits, gross neurological disturbances,poor eduacation, language deviation	NR	age, gender, intelligence, social-cultural factors, and educational environment matced	Measures		NR	Raven	word recognition (a) accuracy (%), (b) RT (sec)
					Dyslexia 19 (15/4)	14.6	--	42.1	(a) 93.9 , (b) 1.1
					Control 17 (15/2)	14.4	--	43.3	(a) 99.2 (b) 0.6
Larsen, et al. 1990	poor word recognition	poor intelligence, sensory deficits, gross neurological disturbances, poor education, laguage deviation	NR	Age, intelligence, SES, educational environment matched	Measures		OHI	Raven	Word recognition (a) accuracy, (b) RT
					Dyslexia 19 (15/4)	15.1	LH: 4, RH: 15	42.1	(a) 93.9, (b) 1.1
					Control 19 (15/4)	15.4	LH: 2, RH: 17	43.3	(a) 99.2, (b) 0.6
Leonard et al. 1993	Clinical interview, family history, results of a test battery	NR	NR	(A) controls: no history of RD within three-degree of relatives, never been referred for diagnosis, (B) unafected relatives: the first or second degree relatives of an individual with dyslexia, but had never been diagnosed as dyslexia	Measures		NR	NR	(a) LAC, (b) WRMT-WA
					Dyslexia 9 (7/2)	36.0 (17.1)	RH: 9	--	(a) 84, (b) 98
					Control (A) 12 (5/7), (B) 10 (4/6)	(A) 37.1 (B) 25.7	(a) RH: 12; (b) RH/LH: 9/1	--	(A)-(a) 99.2, (b) 117; (B)-(a) 91, (b) 109
Njiokiktjien, et al. 1994	(A) dyslexia and dysphasia: FSIQ and PIQ not lower than 85, (B) general mild LD: no significant PIQ/VIQ discrepancies, and FSIQ > 85, (C) general severe LD: FSIQ 50-85	severe epilepsy, cerebra palsy, specific dysmorphy or chromosomal syndromes, and traumati or metabolic encephalopathy, obvious developmental abnormalities revealed by MRI	NR	patients with clinical diagnosis suitable for MRI, who had complaints such as headache seizures, mild head traumas, and precocious puberty	Measures		NR	NR	NR
					Dyslexia (A) 39 (27/12), (B) 24 (20/4), (C) 47 (38/9)	range: 2.5 to 14.0	--	--	--
					Control 42 (22/20)	range: 0 to 20	--	--	--

Table 3 MII Results of Temporal Lobe and Insular Region

	diagnostic criteria	exclusion of subjects	other neurological disorder	selection of control		N (m / f)	Age	Handed-ness	IQ	Reading
Schultz, et al. 1994	regression based discrepancy of 1.5 standard errors between reading achievement and IQ	severe emotional problems, uncorrected vision problems, hearing loss, acquired neurological disorders, non-native English speakers	NR	Age, IQ, handedness matched	Measures			EHI	WISC-R	(a) WJL, (b) WJW, (c) WJP, (d) WJR
					Dyslexia	17 (10/7)	8.68	all RH	FSIQ: 117.6, VIQ: 116.5, PIQ: 113.3	(a) 92.8, (b) 92.5, (c) 97.0, (d) 87.5
					Control	14 (14/14)	8.94	all RH	FSIQ: 121.2, VIQ: 122.7, PIQ: 114.8	(a) 119.0, (b) 112.9, (c) 121.9, (d) 117.9

Note.
ADD = Attention Deficit Disorder without hyper activity, *ADHD* = Attention Deficit Disorder with Hyper activity, *AEP* = Auditory Evoked Potential, *AHQ* = Annett Handedness Questionnaire, *BAS-WRT* = British Ability Scale Word Reading Test *CC* = Corpus Callosum, *CNV* = Contingent Negative Variation, *CPT* = Continuous Performance Task, *DSM-III-R* = Diagno and Statistical Manual of Mental Disorders, Revised Third Edition , *EHI* = Edinburgh Handedness Inventory, *ERP* = Event Related Potential, *FSIQ* = Full Scale IQ, *GFW-RS* = Goldman-Fristoe-Woodcock Reading of Symbols (non-words), *GORT* = Gray Oral Reading Test, *LAC* = Lindamood Auditory Conceptualization Test, *MGN* = Medial Geniculate Necleus, *MR* = Mental Retardation, *MRI* = Magnetic Resonace Imaging, *NPRE* = Nonsense Passage Reading Error, *NPRT* = Nonsense Passage Reading Time, *NR* = Not Reported, *OHI* = Oldfield Handedness Inventory, *PINV* = Post Imperative Negative Varia *PIQ* = Performance IQ, *PNES* = Physical and Neurological Examination for Subtle signs, *PT* = Planum Temporale, *RD* = reading dificulties, reading disabilities, ROI = Region of Interest, VEP = Visual Evoked Potential, *VIQ* = Verbal IQ, *WAIS* Wechsler Adult Intelligence Scale, *WISC* = Wechsler Intelligence Scale for Children, *WJL* = Woodcock-Johnson Letter-Wor Identification, *WJP* = Woodcock-Johnson Passage Comprehension, *WJR* = Woodcock-Johnson Reading Cluster, *WJW* = Woodcock-Johnson Word Attack, *WRAT-M* = Wide Range Achievement Test Revised, Math, *WRAT-R* = Wide Range Achievement Test Revised, Reading, *WRAT-S* = Wide Range Achievement Test Revised, Spelling, *WRAT* = Wide Range Achievement Test, *WRMT-PC* = Woodcook Reading Mastery Test-Revised, Passage Comprehension, *WRMT-T* = Woodcoo Reading Mastery Test-Revised, WRMT-WA = Woodcook Reading Mastery Test-Revised, Word Attack