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THE DEVELOPMENT OF HUMAN CEREBRAL LATERALIZATION

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Introduction

The fact that there are human cerebral lateral asymmetries has been recognized for over 120 years. These asymmetries were first observed with respect to the human language faculty, but have since been found for a variety of higher cognitive functions (e.g., visual closure, arithmetic calculations, face recognition). It is only within the last 20 years, however, that fundamental questions regarding the development of lateralization have been addressed. These are the questions I will consider here.

Structural asymmetries exist in almost all species, so it is no surprise that they exist in humans. The origin of such asymmetries is not yet known, although, in most if not all instances, the ultimate explanation will probably rest in genetics. Are human cerebral asymmetries of the same order as other physical asymmetries, however? After all, cerebral asymmetry is not a species-invariant trait; that is, different patterns of cerebral organization exist in different subpopulations of the species. While species-variable traits can also be under genetic control (14, 50), the causal mechanisms underlying human brain asymmetries are not at all understood, including the basic question of whether the pattern of asymmetry is established by genetic or environmental factors. I will therefore leave aside questions regarding the genetic origins of laterality (but see items 14, 50, 51, and 63 for discussion of these issues). I will instead concentrate on the following questions: When does cerebral dominance set in? Is it present at birth, or is it a developmental process? Is it subject to environmental influences during ontogenesis? What factors determine variations in lateralization?

The Special Role of Language

Cerebral dominance in the human has been most extensively considered in relation to human language, so much so that the phrase "cerebral dominance" (or "lateralization") is often used to mean "cerebral dominance (or lateralization) for language." This association of lateralization with language lateralization is probably a by-product of the fact that language loss is the most obvious or noticeable behavioral consequent of brain damage, and is therefore easier to localize with regard to

cerebral control. To that extent, then, it would seem an accident that language has played such a large role in exploring issues of lateralization. It seems to have been a well-designed accident, however, for there is now reason to believe that language plays a crucial role in both the development and character of cerebral organization.

Lenneberg's Hypothesis

Because of the special place language holds in understanding the development of lateralization, it is fitting that the first set of hypotheses regarding the establishment of cerebral laterality came from Lenneberg's treatise *Biological Foundations of Language* (49). A reanalysis of the data Lenneberg used and a consideration of data unavailable to Lenneberg in 1967 have since challenged his views on the development of lateralization; however, in Lenneberg's treatment of the topic, he raised and addressed so many of the issues still considered to be central ones that his position is useful not only as a starting point but as a framework for our larger discussion.

In his seminal work, Lenneberg argues that at birth the two cerebral hemispheres are unspecialized and have equal potential for subserving language. At about the age of 2, maturationally timed changes in neurochemistry and neurophysiology underlie both the onset of true language acquisition and the onset of the neurological process of language lateralization. These changes involve changes in brain weight, myelination of nerve fibers, the growth of individual neurons, changes in neurodensity, and changes in relative chemical composition of brain tissue. Lenneberg notes that these changes, which unfold along a maturational timetable, parallel milestones of linguistic development and patterns of recovery from brain damage. The establishment of cerebral dominance for language, Lenneberg argues, is therefore a continuing process, nontrivially tied to the process of language acquisition. As more language is acquired, the left hemisphere becomes increasingly dominant for the representation of language knowledge and for the control of its performance. The left hemisphere thus becomes specialized for language. At the same time, having started out participating equally in the early language functions of the child, the right hemisphere becomes less and less involved as the left hemisphere's dominance for language grows. This process continues until puberty, its completion corresponding to the loss of the ability to learn language naturally, which is in turn linked to the endpoint in the brain's organizational plasticity.

There are several theses central to Lenneberg's view: (1) The two hemispheres are equipotential at birth; (2) lateralization is a process of increasing specialization or control (of language) by the left hemisphere alongside a decreasing involvement of the right—a process lasting from age 2 to puberty; (3) recovery of (language) function after brain damage is determined by degree of lateralization (i.e., how well-established or complete cerebral dominance is); (4) cerebral dominance for language is the key brain–behavior relationship to consider in understanding lateralization, because brain lateralization is tied to language acquisition.

Each of these arguments touches on issues fundamental to an understanding of the development of lateralization, and I will consider them in turn. The first two are intimately interwoven and will be considered together.

Equipotentiality and Lateralization as a Process of Increasing Hemispheric Specialization

Lenneberg's contention that at birth the two hemispheres are equipotential for language, and that lateralization for language is a process of increasing specialization of the left hemisphere alongside a decrease in the right hemisphere's role in language, rests on his interpretation of clinical data regarding the incidence of acquired aphasia in children. Relying largely on Basser's (4) study of the effects on language of unilateral lesions in childhood, Lenneberg concludes that, if language acquisition has already begun, children develop transient aphasia regardless of which hemisphere is lesioned, and before that point, they will acquire language regardless of which hemisphere is damaged.

Krashen (46, 47) was the first to reassess these arguments. In his reexamination of the data Lenneberg considered, Krashen notes that in all cases involving right-hemisphere lesions leading to aphasia, the lesion was sustained before age 5. Krashen then postulates a lateralization-by-5 hypothesis. Like Lenneberg, Krashen posits that lateralization is linked to language acquisition, but argues that both are essentially complete by age 5. Krashen also examines the results of dichotic-listening experiments with children. He argues that with the use of an appropriate metric that corrects for performance level, these data reveal that degree of lateralization does not change after 5 and thus support his lateralization-by-5 hypothesis. Krashen further argues that cerebral lateralization (complete by 5) is separate from organizational plasticity, which holds until close to puberty, approximately age 10.

Krashen's work challenges Lenneberg's notions that lateralization continues to puberty. However, Krashen's lateralization-by-5 hypothesis does not address the equipotentiality issue and still leaves us with the possibility that lateralization may be a continuing process from birth to 5 or from 2 to 5. It could be the case, in other words, that initially both hemispheres have equal potential to subserve language, and that the left hemisphere's specialization for language only gradually and progressively sets in sometime before 5. Considerable research argues against both of these logical possibilities.

Clinical Data

To begin with, reexamination of the clinical data Lenneberg used to support his arguments about equipotentiality and the lateralization of language as a progressively increasing phenomenon has shown these data to be unreliable, fragmentary, and difficult to interpret (45). More important, however, Woods and Teuber (87) point out that recent data contradict the early data and show no greater incidence of aphasia after right-sided lesions in children than in adults. They point out that conditions that may earlier have led to aphasia (e.g., systemic infections, unchecked, resulting in diffuse, bilateral encephalopathy) are now treated with antibiotics, restricting recent clinical series to cases that more accurately elucidate the relationship between lateralized lesions and consequent aphasia. The recent data do not

support either the equipotentiality hypothesis or the view that lateralization gradually sets in.

Other clinical data argue perhaps even more strongly against the equipotentiality view. Cases of infantile hemiplegia and hemidecortication demonstrate the unequal potential of the two hemispheres for mediating language (and visual and spatial ability). Hood and Perlstein (42) and Bishop (7), comparing the consequences of left-sided versus right-sided injury in cases of infantile hemiplegics sustaining early damage but not involving hemispherectomy, noted that right-hemisphere damage led only to deficits in articulation and vocabulary acquisition, while left-hemisphere injury led to widespread deficits and delays in language acquisition. Annett (3), whose cases of infantile hemiplegia also involved early damage (before 13 months) without hemispherectomy, demonstrated that subsequent language impairments were far more frequently associated with left-hemisphere damage than with right-sided damage.

These cases involve children whose damaged hemispheres may nonetheless continue to exert an inhibitory influence over their healthy hemisphere during development. Milner (59), for example, reports that early left-hemisphere lesions must be situated in the classic language areas before the right hemisphere's potential for language can be released. Better test cases for true equipotentiality are cases where children develop language with only one extant hemisphere. Dennis and her colleagues (21, 22, 25, 26, 27, 28) have studied three such cases. They are cases where one hemisphere was surgically removed in infancy, before language acquisition. In these cases (and in a similar series of seven additional cases), the two hemispheres are revealed to be unequal substrates for language acquisition, with the left hemisphere outperforming the right across a broad range of linguistic tasks. The right hemisphere is particularly deficient in acquiring what I will refer to as the computational aspects of language (after Chomsky) – phonology, morphology, syntax, and the integration of semantic and syntactic structure – that is, most of the grammar.

There is another aspect of Lenneberg's hypothesis that might yet hold, however. Despite their unequal potential for language acquisition, a process that does not actively begin until 1 to 2 years of age, the two hemispheres may still be equally involved in perceiving and processing linguistic information before that point. There are no clinical data that bear on this possibility, but experimental investigations have produced relevant data.

Experimental Data

Consistent with the clinical data referred to above, experimental data demonstrate no change (increase) in the lateralized response to language and nonlanguage stimuli between ages 2½ and 5. (See 85 for a review of many of these data.) Moreover, experiments on even younger children indicate that at birth, the two hemispheres already differ in their sensitivity and response to language and nonlanguage stimuli. The left hemisphere appears to be preprimed for language stimuli; the right hemisphere, for visual and certain nonlinguistic auditory stimuli. Entus (30) found

that infants aged from 22–140 days display the pattern of lateral asymmetry found in older children and adults for both speech and nonspeech (music) stimuli presented dichotically. Molfese, Freeman, and Palermo (62) demonstrated that infants as young as 1 week of age manifest strongly lateralized electrophysiological responses to speech and nonspeech stimuli. Davis and Wada (19) found a strongly lateralized right-hemisphere response to a visual stimulus in infants as young as 2 weeks. Newborns demonstrate strongly asymmetric motor reflexes and responses as well (e.g., head turning [80]; grasp reflex and grasp duration [9]; stepping reflex [57]; limb movements in response to speech and music [75]), although these responses may be subcortically, not cortically, mediated.

The evidence consistently indicates that the left hemisphere is prepotent for language. And contrary to Lenneberg's view, long before language acquisition has begun, the cortical response to language (and certain nonlanguage) stimulation is clearly lateralized. Functional specialization, then, does not appear to develop in tandem with language acquisition. Functional asymmetries (or their precursors) appear to be present at birth, and at no time from birth on do the two hemispheres appear to be equipotential for language on any interpretation of the term equipotential. Furthermore, neuroanatomical asymmetries that may be mapped onto the functional asymmetries demonstrable in infants and newborns are found in neonatal and fetal brains. With respect to the areas of the two hemispheres in which adult brains have been found to differ reliably – longer left-hemisphere sylvian fissure (in particular, length and area of the planum temporale), and sharper slope of the right-hemisphere sylvian fissure – infant and fetal brains have been found to differ in parallel fashion, and to the same extent, from at least the 29th gestational week (11, 16, 48, 83, 86). Although these neuroanatomical asymmetries do not correlate with functional asymmetries as well as might be expected (see 84 for discussion), lateral asymmetries are present at birth, and the neuroanatomical asymmetries may represent a prewired, neurobiological precursor of functional lateralization.

Recovery of Function as a Reflection of Lateralization

A third of Lenneberg's notions about lateralization is that recovery of function reflects the degree to which cerebral dominance has been established for that function. The data Lenneberg uses are the same clinical data he uses to support his other arguments.

There are several problems inherent in interpreting recovery data. Age is not the only factor that constrains or determines recovery. Size of lesion, depth of lesion, handedness, sex, and the extent and character of the deficit itself all play a role in recovery. In general terms, degree of recovery is greatest in the young child and decreases with age (but cf. 87). This decrease in recovery with age reflects degree of plasticity rather than degree of dominance, however, and both the resulting deficit and subsequent recovery reflect the functional maturity of the lesioned areas at the time the lesion is incurred. The important fact here regarding lateralization is

that with damage to the left hemisphere, language is affected throughout life, even though differently at different ages. This suggests that the left hemisphere's specialization or specialized potential for language is preset at birth – a conclusion consistent with the findings discussed above.

This view of lateral specialization is not incompatible with cerebral plasticity, for, while each hemisphere may be preprogrammed to mediate certain functions, each may also hold the "prospective potency" (30) to subserve functions that are normally under the control of the opposite hemisphere. However, it appears that the degree of innate specification of the language areas, especially for the computational aspects of language, limits the interhemispheric transfer of these language functions such that there is always some residual consequence of the right hemisphere's taking over what was destined to be governed by the left hemisphere, with greater residual effects associated with postacquisition damage. Since plasticity appears to decrease with age, "prospective potency" may be inversely related to the knowledge state of the individual, and potentially to its automatization and degree of innate specification.

To review, I have looked at three of Lenneberg's four central theses about lateralization; it is apparent that each of them fails to be supported. The two hemispheres appear to be unequal substrates for language from birth; lateralization does not seem to set in via a process that gradually and progressively functionally differentiates the two hemispheres, but appears to be preset at birth; and finally, recovery from brain damage reflects brain plasticity, not cerebral lateralization.

What factors, then, affect lateralization? Is lateralization subject to environmental influences? Since lateralization is not a species-invariant trait – that is, different subgroups within the species evidence different patterns of brain organization – what factors influence or determine which pattern of lateralization develops? Two factors appear primary in their relationship to varying patterns of lateralization: sex and handedness.

Sex

Male–female differences in cerebral asymmetry in the human would not be surprising, given increasing data on the important role of sex hormones on brain organization in other species (29) and on the possible relationship between sex chromosomes and both cerebral asymmetry and neuropsychological function in humans (64, 65; but see 43). However, there is considerable controversy over the issue of sex differences in lateralization. Nonetheless, amidst the controversy over how valid and reliable the data are, and how in any event they should be interpreted, the body of clinical and experimental data pointing to sex differences in laterality patterns grows.

McGlone (55) and Sasanuma (73) report a significant sex difference in the incidence of aphasia following insult to the left hemisphere. Both researchers found a less frequent incidence of language breakdown in females than in males after quite similar damage. No incidence of aphasia resulted for either males or females after

right-hemisphere damage. Sasanuma also found a significantly smaller incidence of severe aphasia in females than males. Kimura presents additional data that suggest a male–female difference in the intrahemispheric organization or representation of language (44). These findings, taken together, raise several possibilities: (1) that females have less lateralized, more bihemispheric linguistic function than males; (2) that females have less localized, more diffuse representation of language within the left hemisphere; (3) that females have otherwise different intrahemispheric organization; (4) that females have greater interhemispheric connectivity than males; and (5) that some combination of these holds. Clearly, far more data are needed before we can decide among these alternatives.

There are many more experimental than clinical data on sex differences in language lateralization, including a considerable number of developmental studies on the question (see 56 for review), but here, too, the data are controversial. With exceptions, experimental results generally indicate that males have greater lateralization of verbal, visual, and spatial abilities than females, once again suggesting either greater bihemisphericity of language represented in females, or greater interhemispheric connectivity in the female brain.

Developmental experimental data are at first glance in conflict with the adult data. Of 23 language dichotic-listening studies (see 52 and 81 for reviews), 16 have shown no sex differences, 5 showed sex differences only for children in particular age groups, and only 2 showed more pervasive sex differences (one study indicating a greater right-ear advantage for girls, the other for boys). Of 7 tachistoscopic studies investigating sex differences for language processing, 6 have found no sex differences, and 1 found a greater right-visual-field effect for boys. Of 17 studies looking at the development of motoric lateral asymmetries (i.e., handedness, footedness, etc.), 11 found no sex differences, and 6 found sex differences, in each case indicating greater lateral asymmetry for girls.

The developmental data look as if there is either no sex difference in lateralization or a tendency for females to show greater laterality at an earlier age. However, this apparent conflict with the adult data may be resolvable through consideration of maturational factors. Waber (81, 82) has shown that maturational rate figures critically in the laterality effects evidenced in experimental measures of hemispheric asymmetry. Early maturers show greater laterality effects that late maturers. Since females in general mature earlier than males (15, 67), equivalent laterality effects for girls and boys, or even greater laterality effects for girls, could be expected at least up to puberty.

These developmental data do not, then, contradict the adult data. Sex differences in laterality patterns may exist throughout life, but take different forms at different points in maturation. There is also increasing evidence that sex differences in brain organization do not appear to result from experiential factors. Within the first 2 years of life, male–female differences in hemispheric maturation rate and sensitivity to auditory and visual stimuli have been found (15, 79). In addition, studies of adults exposed prenatally to abnormal levels of sex hormones have been found to display atypical patterns of lateral asymmetries (40, 41). There is little likelihood

that these male–female differences could be the result of gender-related differences in social/cultural experience.

Although the state of the art on sex differences in laterality does not warrant any firm conclusions (see 56 and peer commentary for a review), sex may well turn out to be an important factor governing the pattern of lateralization that develops. As sex is clearly a genetically determined phenomenon, the development of lateralization may be prefixed, at least partially, by genetic factors.

Handedness

There is a greater tendency for human beings to have language lateralized to the left hemisphere than to be right-handed, but the species is nonetheless predominantly right-handed. According to most estimates, only 8–12% of the population is non-right-handed. The hand–brain relationship is a complex matter, and many aspects of this relationship will not be considered here. I will consider only two issues: (1) that handedness is not the result of experience, and (2) that right-handers and non-right-handers have been shown to possess different patterns of cerebral organization.

Handedness is not fully expressed at birth. This fact has led some researchers to look for indices of growth or maturation of handedness. Some have found changes in handedness in the course of growth. Bingley (6), for example, found evidence of a decrease in mixed and left-handedness with age, such that non-right-handers become increasingly right-preferent. Different forms of mixed lateral preferences are common at all ages, but observations of uncertain, ill-defined, or changing preferences in young children have led some scholars to associate ambilaterality with immaturity. In this view, ambilaterality is related to functionally undifferentiated hemispheres (undifferentiated for motor as well as cognitive function). This view assumes an initial equipotentiality for control of handedness. While lateral preference is not fully expressed at birth, there are strong indications that handedness is genetically determined. First, there are clear precursors of handedness (e.g., tonic neck reflex position [36], orientation of the head at birth [12]); second, from early in childhood hand preference remains constant (2, 3, 35); and third, handedness is not the result of experience or social factors (1, 3, 10).

More immediately relevant is the fact that handedness is one of the key factors related to the pattern of lateralization that develops. Most studies report less lateralized representation of function in the non-right-handed. Clinical data reveal that a portion of left-handers have sufficient language governed by both hemispheres so as to be rendered aphasic after damage to either hemisphere (37, 60, 68, 71). These data suggest greater bihemispheric and less lateralized representation of language in the left-hander than in the right-hander. Clinical data also indicate that the initial aphasia in left-handers is generally less severe and recovery more rapid and complete than in right-handers. This fact raises the additional possibility of less inhibitory control of one hemisphere by the other in the left-handed. Experimental data are consistent with the clinical data in that left-handers as a group show consistent-

ly smaller laterality effects than right-handers on both language and visual–spatial tasks.

Left-handers are not a homogeneous group with respect to patterns of lateralization, however. Clinical and experimental data reveal an important difference between familial and nonfamilial left-handers. Nonfamilial left-handers appear to be more like male right-handers; the incidence of aphasia is consistently associated with lesions to only one side of the brain, the left hemisphere. Initial aphasia can be more severe and recovery from aphasia slow and limited as well (53). But familial left-handers and those right-handers with left-handedness in the family appear to have greater bilateral control of language and better prognosis for recovery. In experimental data as well, cerebral ambilaterality is more associated with familial sinistrality, while nonfamilial left-handers show more consistent unilateral left-sided dominance for language (39, 95).

As with sex, handedness is a genetically determined matter, probably preset at birth (but see 34). Thus the factors implicated in variations of cerebral laterality patterns are genetic factors, either preestablished or preprogrammed at birth. This picture is consistent with the neuroanatomical and electrophysiological data reported earlier, suggesting functional lateralization and its possible physiological basis to be prewired. If true, we are led to the view that lateralization is a biologically determined phenomenon wherein at the time of birth each hemisphere is dedicated to specific processes to a prespecified degree. At birth the hemispheres are not yet specialized, but are prepotent for their specialized functions. Their specialization potential is then actualized as specialization for particular knowledge domains, psychological abilities, or information-processing abilities, once they are functional.

Language Acquisition and Lateralization

Lenneberg argued that language acquisition and lateralization go hand in hand, degree of lateralization reflecting degree of language mastery. While in detail his arguments do not hold up, here Lenneberg appears to have been at least partially right. His basic thesis that language acquisition and lateralization are tied does find support.

The specific lateralization pattern an individual is programmed to have is not related to the development of normal language abilities. Normal left-handers, right-handers, females, and males all develop normal ordinary language abilities. Interestingly, though, the development of normal language and the unfolding of an individual's preset laterality pattern do appear to be related. Only a very small body of relevant data exists, but these data suggest a critical tie between language acquisition and the instantiation of the preset pattern of lateralization for language, such that if either one is disrupted, the other will be affected.

The most common subpopulations with disorders of language acquisition—namely, developmentally aphasic children, dyslexics, and autistics—are consistently associated with atypical laterality patterns. Each of these populations comprises more than one subgroup, and the etiology of the disorders is unknown. Yet with each

group, we find developmental language dysfunction accompanied by indications of abnormal or atypical cerebral organization. With both developmental aphasics and dyslexics, there is a higher than normal incidence of non-right-handedness; mixed laterality of handedness, footedness, eyedness, and "visual-fieldedness"; and a family history of mixed laterality and developmental language problems (see 88 for review).

Some research on developmental aphasia and dyslexia is most suggestive of a specific left-hemisphere deficit. Tallal (77) has demonstrated that a substantial portion of language-impaired children are deficient in processing rapidly changing acoustic information of the sort embodied in formant transitions between stop consonants and vowels. Such a deficit has also been demonstrated for aphasics with unilateral left-hemisphere damage and for the disconnected right but not the left hemisphere. In addition, language-impaired children and both the disconnected and isolated right hemisphere of adults evidence similar performance on the Token test (76, 94), implicating an impairment in short-term verbal memory, normally lateralized to the left hemisphere. Recent research on reading in the disconnected and isolated right hemisphere (93) elucidates provocative parallels between patterns of abilities and strategies evidenced by dyslexic children and the right hemisphere. These findings are supported by recent neuroanatomical studies (32, 32a) showing that developmental dyslexia is associated with structural abnormalities of the left hemisphere.

There is no consensus on the origin or cause of autism, a disorder involving pervasive and somewhat unique language-learning impairments, but again, recent research suggests a left-hemisphere dysfunction or abnormal hemispheric dominance and interaction. There is a higher than normal proportion of non-right-handedness (13, 72), an enlargement of the left lateral ventricle (38), a relative increase in the size of the left hemisphere's evoked potential during REM sleep compared to normals (78), and behavioral evidence of right-sided hemiattention or leftward sensory and sensory–motor bias (8, 48).

Direct experimental investigation of cerebral laterality in these groups has led to contradictory or inconclusive results. Thus, no specific conclusions can be drawn other than that each of these groups is associated with developmental language impairments coupled with an abnormality in cerebral dominance. Although which part of this relationship is cause and which effect cannot yet be determined, an intimate relationship between intact language acquisition and the establishment of a normal pattern of cerebral dominance is supported.

Other data provide more direct evidence for the idea that if lateralization is disrupted, language acquisition is adversely affected. This evidence comes from children with congenital or acquired brain disease—specifically, cases of unilateral lesions in childhood and of childhood hemidecortication or hemispherectomy. Unilateral lesions of the left hemisphere in childhood, whether acquired or congenital, interfere with the left hemisphere's prewired specialization for language, and they consistently result in language deficits. Studies of the effects of unilateral lesions in childhood (3, 7, 42) have found that left-hemisphere lesions produce speech delay and disorder. Rankin *et al.* (70), examining language performance in more detail,

found that unilateral left-hemisphere lesions produce particular deficits in comprehension and production of syntax. Dennis (24) also found expressive and receptive deficits in structural linguistic knowledge in a case of left-hemisphere arteriopathy.

In cases of hemidecortication or hemispherectomy, a normal pattern of cerebral organization is prevented by disease and ensuing surgery. Here, too, the inability of the left hemisphere to subserve language (because of its removal) results in consistent and persistent linguistic deficits. Cases of damage and hemispherectomy in childhood *after* early stages of language acquisition reveal that, even before puberty, removal of the left hemisphere results not only in initial global aphasia, but in a preponderance of routinized social speech, inability to correct syntactic errors, and severe deficits in the comprehension and production of many syntactic and morphological structures (21, 93). Even in a female (symptoms at 7–8, hemispherectomy at 10), where we might have expected some residue of linguistic ability due to more bilateral control of language, we find severe, lasting linguistic impairment—telegrammatic speech, limited morphological elaboration, and limited syntactic comprehension (94, 95). These data suggest that once language has been acquired and hemispheric specialization for language established, removal of the language areas of the brain permanently disrupts language function and prevents language from developing normally again, even in childhood.

Hemispherectomy or hemidecortication of the left hemisphere with damage at or shortly after birth has also been shown to result in consistent, though less severe, linguistic deficits (20, 25, 27, 28, 70). In females and males alike, impairments in the processing and production of complex syntax are present in all the left hemispherectomies and hemidecorticates studied. Some of these infant left hemispherectomies have been shown to have semantic and more pervasive syntactic deficits as well. Dennis and her colleagues (22, 23, 25, 27, 28), for example, have found systematic linguistic deficits encompassing most of the computational aspects of the linguistic system—that is, syntax, morphology, phonological manipulations and recodings, and the integration of syntax with interpretive semantic elements.

In addition, in cases of agenesis of the corpus callosum, where the two hemispheres may be healthy but a preprogrammed pattern of cerebral asymmetry may not be established (64, 74), recent evidence suggests that deficits in linguistic function may result. What is more, these deficits seem to parallel in character the deficits found in cases of hemispherectomy and hemidecortication (23).

The kinds of studies that would document clearly the effects of early brain damage on language acquisition have only begun to be done, and the data are consequently sparse and limited. They all point to the same conclusion, however: Disruption of the preset specialization of the left hemisphere for language appears to permanently affect linguistic development, even if it occurs in infancy before the process of language acquisition has begun. Impaired linguistic function contrasts with the generally intact intellectual function of these same children (21, 27).

This part of the relationship between lateralization for language and language acquisition may seem somewhat unsurprising. After all, these cases all involve brain damage; it could be expected that language impairments might result. The uniform-

ity and systematicity of the deficits would not be as predictable, however. What is also less expected is that the relationship should hold in reverse as well. There are even fewer data to consider here, but those that exist suggest that the tie between language acquisition and lateralization is a bidirectional one. Disrupt first language acquisition, and the establishment of a normal pattern of cerebral dominance will be affected. Cases of first language acquisition after the normal and perhaps critical period—that is, after childhood—raise the possibility that language acquisition itself may be the trigger or crucial factor in actualizing the preprogrammed pattern for functional specialization of the hemispheres.

The case of Genie, a case of first language acquisition in adolescence, is one such case (see 17, 18, and 31 for details). Social isolation prevented Genie from acquiring language in childhood, and her language development as a teenager and young adult has been limited primarily to lexical and propositional semantics, with little acquisition of structural (or computational) linguistic knowledge.

Experiments involving dichotic listening and event-related potentials (ERP) were conducted with Genie to assess laterality effects in her processing of language and nonlanguage stimuli. The two experimental techniques produced parallel results. They indicated that Genie uses her right hemisphere for both language and nonlanguage processing (she is strongly right-handed). As illustrated by her dichotic-listening performance presented in Table 1, both her failure to evidence a difference in the direction of laterality effect for language and nonlanguage stimuli and the degree of effect she displayed mark her performance as highly atypical. In terms of ear advantage, Genie's dichotic-listening performance parallels the dichotic-listening performance of subjects with only one hemisphere responding to the task, as illustrated in Table 2. This suggests unihemispheric control of both language and nonlanguage cognitive functions.

TABLE 1
DICHOTIC-LISTENING RESULTS WITH GENIE

Date	Number of Pairs Presented	Stimulus	Number Correct	
			RE	LE
3/27/72	29	Words[a]	6	29
5/10/72	15	Words[a]	1	15
8/16/72	30	Words[a]	5	30
6/3/73	28	Words[b]	0	28
8/2/72	20	Environmental sounds[a]	12	18
8/16/72	20	Environmental sounds[a]	14	19
6/3/73	20	Environmental sounds[a]	14	20
6/3/73	28	Environmental sounds[b]	15	27

[a]Single pair presented.
[b]Two pairs presented.

TABLE 2
GENIE COMPARED WITH SUBJECTS USING A SINGLE HEMISPHERE TO PERFORM THE TASK

Subjects	Stimulus	Percentage Correct	
		Better Ear	Weaker Ear
Genie	Words	100 (L)	16.0
Right hemispherectomized[a]	Consonant–vowel syllables	99 (R)	24.3
Disconnected hemispheres[b]	Digits	90.7 (R)	22.2

[a]Berlin *et al.* (5).
[b]Milner *et al.* (61).

Behavioral data support this interpretation of the experimental data. In level of ability, number of errors, error types, and style of performance evidenced behaviorally, Genie's performance strongly resembles that of the adult disconnected right hemisphere of split-brain individuals on a wide range of tests, including tests of auditory short-term memory, visual short-term memory, visual reproduction, and disembedding (89, 90, 91, 92, D. Zaidel and E. Zaidel, personal communication; see 17 for details).

Genie's failure to learn language in childhood appears to have led not only to abnormal and restricted linguistic function, but to the absence of a normal pattern of cerebral specialization, marked in particular by the failure of the left hemisphere to specialize for language.

A second case of first language acquisition beyond childhood is the case of Chelsea, brought to light by P. Glusker. Chelsea is an individual attempting first language acquisition in adulthood (in her 30s), a severe and undiagnosed hearing impairment having prevented her from acquiring language as a child. Her language to date appears to consist solely of certain aspects of lexical knowledge and to be devoid of the constraints and principles of English grammar. It is thus agrammatic and ungrammatical, and is limited to the somewhat unconstrained concatenation of lexical items (Curtiss, unpublished data).

Preliminary data from Chelsea's performance on visual ERP and tachistoscopic language tasks indicate a lack of lateral specialization for language (H. Neville and N. Dronkers, personal communication and unpublished data). Here too, then, the possibility is raised that without first language acquisition in childhood, not only will language acquisition itself be affected, but a normal pattern of hemispheric specialization will not develop.

Data from congenitally deaf children and adults who are not linguistically proficient in any language (including sign) are consistent with these cases. Examining visual ERPs, Neville (66) found that those deaf individuals who lacked a formal language showed no evidence of hemispheric asymmetries for processing linguistic or nonlinguistic information, while those deaf individuals who had acquired a formal language in childhood showed asymmetries for both. Unexpectedly, Neville (65a)

has also found that the acquisition of sign language as a native language in childhood (and its concomitant lateralization to the left hemisphere) is associated with a specialization of the left hemisphere for certain spatial functions, much as the specialization of the left hemisphere for spoken language may be associated with a specialization for temporal functions.

Neville's data are additionally important because they demonstrate that what is at issue is not the presence or absence of speech, but knowledge of language—spoken or signed. Recent data on the incidence and character of aphasia in fluent signers support Neville's findings (54, 69). The pattern of sign language deficits occurring after left-hemisphere damage or sodium amytal injection is quite parallel to those seen with spoken language. These data provide further evidence that the left hemisphere is specialized (in most individuals) for what linguists refer to as "the grammar," regardless of performance modality. What is most relevant here, however, is that current data indicate that when sign language is learned at the appropriate time (in childhood), signers show functional specialization of the hemispheres; but without language, signed or spoken, individuals show an absence of functional asymmetry.

One particularly striking fact about both the cases involving specific brain damage and those involving the acquisition of a first language past childhood is that the linguistic deficits involved fall within a circumscribed area of linguistic function: the computational modules of language, that is, the grammar minus the lexicon. Those with early left-hemisphere damage are limited in their capacity to acquire the computational modules. Those acquiring a first language after childhood appear even more severely limited in their capacity to acquire the computational component (see also 54a, 66a). Since the left hemisphere seems to be specialized for the computational component and not for all aspects of language knowledge and use, it may not be the entirety of language acquisition, but only the acquisition of the computational component of language that is critically tied to the establishment of a normal pattern of cerebral lateralization. Actualization of hemispheric specialization, either for grammar itself or for the particular abilities the processing and performance of grammar requires in addition to its representation, may be what triggers the establishment of cerebral lateralization. And all of this may depend on first language acquisition at the normal time, by the area of cortex prewired for the task.

Summary and Questions for the Future

In summary, the evidence points to certain conclusions that differ from Lenneberg's—namely, that lateralization is preprogrammed at birth; that lateralization is not a species-invariant trait; and that the particular pattern of lateralization an individual is prewired to develop depends in part on factors such as handedness and sex. But it looks as though Lenneberg may have been right in holding that language acquisition and lateralization are closely related. The data considered here suggest that language acquisition—more specifically, the acquisition of the computational

component—may be a crucial trigger for the development of lateralization. If language acquisition is prevented, lateral asymmetries may never be established.

This hypothesis tying the development of lateralization to the acquisition of the computational component is most speculative at this point, and many questions remain. What is the picture regarding the development of cerebral asymmetries for lateralized abilities aside from language, such as facial recognition, arithmetic calculation, spatial operations, and so on? Since many of these abilities develop later than language, what role (if any) does their acquisition play in the establishment of the final pattern of cerebral dominance? Will systematic limitations or abnormalities in these other systems of knowledge be associated with missing or atypical lateral asymmetries, or will evidence continue to support a special role for language in the development of cerebral lateralization? Definitive answers await more data.

Other questions also remain unresolved and await future research. What is the biological basis of lateralization? Is the basic pattern set by genes or by unknown environmental events? What factors aside from handedness and sex might contribute to variations in lateralization?

What is the precise relationship between neuroanatomical asymmetries and the development of functional asymmetries? There are anatomical asymmetries involving several areas of cortex. Which, if any, lateralized abilities are these asymmetries connected with? Do morphological asymmetries in fact underlie functional asymmetries? The correlation is far from 1.0, so how should this imperfect correlation be interpreted? Perhaps the structures more relevant to understanding the neural basis of functional asymmetries will turn out not to be at the level of gross anatomy, but at a deeper level involving neurons, synapses, transmitter substances, and circuits.

Finally, although lateralization appears to be prewired, there are respects in which cerebral asymmetries may be a changing phenomenon in development. The ontogenetic development of interrelationships between different brain areas (especially between the frontal lobes and other areas) changes with age. This is true for both intrahemispheric and interhemispheric organization. Changes in knowledge states or performance may reflect reorganization of different subsystems of the developing brain and different levels of connectivity both within a single hemisphere and across hemispheres. Understanding the establishment of cerebral organization will require a better understanding of the development of intra- and interhemispheric communication and of the facilitative and inhibitory effects of one cerebral area on another. Since the right hemisphere appears to be dominant or at least indispensable for the performance of particular linguistic abilities (33, 58), the fullest instantiation of the human language capacity is an example of a cognitive system that involves, maybe even requires, an interaction of both the left and right hemispheres.

Only sophisticated studies of the specific cognitive and linguistic capacities of each hemisphere can reveal what it is that becomes lateralized. Only future neurological studies can determine the neural basis for lateralization. A true understanding of the development of lateralization will thus require a serious interdisciplinary effort in which cognitive theories and neurological theories are related to explain this fundamental aspect of the relationship between brain and behavior.

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