PLASTICITY, LOCALIZATION AND LANGUAGE DEVELOPMENT

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The term "aphasia" refers to acute or chronic impairment of language, an acquired condition that is most often associated with damage to the left side of the brain, usually due to trauma or stroke. We have known about the link between left-hemisphere damage and language loss for more than a century (Goodglass, 1993). For almost as long, we have also known that the lesion/symptom correlations observed in adults do not appear to hold for very young children (Basser, 1962; Lenneberg, 1967). In fact, in the absence of other complications, infants with congenital damage to one side of the brain (left or right) usually go on to acquire language abilities that are well within the normal range (Eisele & Aram, 1995; Feldman, Holland, & Janosky, 1992; Vargha-Khadem, Isaacs, & Muter, 1994). To be sure, children with a history of early brain injury typically perform below neurologically intact age-matched controls on a host of language and nonlanguage measures, including an average full-scale IQ difference somewhere between 4-8 points from one study to another (especially in children with persistent seizures -- Vargha-Khadem et al., 1994). Brain damage is not a good thing to have, and some price must be paid for wholesale reorganization of the brain to compensate for early injuries. But the critical point for present purposes is that these children are not aphasic, despite early damage of a sort that often leads to irreversible aphasia when it occurs in an adult.

In addition to the reviews by other authors cited above, my colleagues and I have also published several detailed reviews of language, cognition and communicative development in children with focal brain injury, from various points of view (e.g., Bates et al., 1997; Bates, Vicari, & Trauner, in press; Elman et al., 1996; Reilly, Bates & Marchman, 1998; Stiles, 1995; Stiles, Bates, Thal, Trauner, & Reilly, 1998; Stiles & Thal, 1993; Thal et al., 1991). As these reviews attest, a consensus has emerged that stands midway between the historical extremes of ***equipotentiality*** (Lenneberg, 1967) and ***innate predetermination*** of the adult pattern of brain organization for language (e.g., Curtiss, 1988; Stromswold, 1995). The two hemispheres are certainly not equipotential for language at birth; indeed, if they were it would be impossible to explain why left-hemisphere dominance for language emerges 95%-98% of the time in neurologically intact individuals. However, the evidence for recovery from early left-hemisphere damage is now so strong that it is no longer possible to entertain the hypothesis that language *per se* is innately and irreversibly localized to perisylvian regions of the left hemisphere.

The compromise view is one in which brain organization for language emerges gradually across the course of development (Elman et al., 1996; Karmiloff-Smith, 1992), based on "soft constraints" that are only indirectly related to language itself. Hence the familiar pattern of language localization in adults is the product rather than the cause of development, an end-product that emerges out of initial variations in the way that information is processed from one region to another. Crucially, these variations are not specific to language, although they do have important implications for how and where language is acquired and processed. In the absence of early brain injury, these soft constraints in the initial architecture and information-processing proclivities of the left hemisphere will ultimately lead to the familiar pattern of left-hemisphere dominance. However, other "brain plans" for language are possible, and will emerge when the default situation does not hold.

In the pages that follow, I do not intend to provide another detailed review of the outcomes associated with early brain injury; the reader is referred elsewhere for a more complete catalogue of such findings. What I would like to do instead is to go beyond these findings to their implications for the nature and origins of language localization in the adult, providing an account of how this neural system might emerge across the course of development. With this goal in mind, the chapter is organized as follows: (1) a very brief review of findings from developmental neurobiology that serve as animal models for the kind of plasticity that we see in human children; (2) an equally brief illustration of results from retrospective studies of language development in the focal lesion population; (3) the distinction between prospective and retrospective studies, including a discussion of putative "critical periods" for language development; (4) an overview of prospective findings on language development in children with congenital lesions to one side of the brain; (5) a new view of brain organization for language in the adult, an alternative to the static phrenological view that has dominated our thinking for two centuries, one that takes into account the role of experience in specifying the functional architecture of the brain.

(1) DEVELOPMENTAL PLASTICITY: ANIMAL MODELS

Evidence for the plasticity of language in the human brain should not be surprising in light of all that has been learned in the last few decades about developmental plasticity of isocortex in other species (Bates, Thal, & Janowsky, 1992; Deacon, 1997; Elman et al., 1996, Chapter 5; Janowsky & Finlay, 1986; Johnson, 1997; Killackey, 1990; Mueller, 1996; Quartz & Sejnowski, 1997; Shatz, 1992; PAPERS IN THIS VOLUME). Without attempting an exhaustive or even a representative review, here are just a few of my favorite examples of research on developmental plasticity in other species, studies that provide animal models for the kind of plasticity that we have observed in the human case.

Isacson and Deacon (1996) have transplanted plugs of cortex from the fetal pig into the brain of the adult rat. These "foreigners" (called "xenotransplants") develop appropriate connections, including functioning axonal links down the spinal column that stop in appropriate places. Although we know very little about the mental life of the resulting rat, no signs of pig-appropriate behaviors have been observed.

Stanfield and O'Leary (1985) have transplanted plugs of fetal cortex from one region to another (e.g., from visual to motor or somatosensory cortex). Although these cortical plugs are not entirely normal compared with "native" tissue, they set up functional connections with regions inside and outside the cortex. More importantly still, the transplants develop representations (i.e., cortical maps) that are appropriate for the region in which they now live, and not for the region where they were born ("When in Rome, do as the Romans do....").

Sur and his colleagues (Pallas & Sur, 1993; Sur, Pallas, & Roe, 1990) have rerouted visual information from visual cortex to auditory cortex in the infant ferret. Although (again) the representations that develop in auditory cortex are not entirely normal, these experiments show that auditory tissue can develop retinotopic maps. It seems that auditory cortex becomes auditory cortex under normal conditions primarily because (in unoperated animals) it receives information from the ear; but if it has to, it can also process visual information in roughly appropriate ways.

Killackey and his colleagues have modified the body surface of an infant rat, by removing whiskers that serve as critical perceptual organs in this species (Killackey et al., 1994). Under normal conditions, the somatosensory cortex of the rat develops representations ("barrel cells") that are isomorphic with input from the whisker region. In contrast, the altered animals develop somatosensory maps reflecting changes in the periphery, with expanded representations for the remaining whiskers; regions that would normally subserve the missing whiskers are reduced or absent (Killackey, 1990). In other words, the rat ends up with the brain that it needs, rather than the brain that Nature intended.

Finally, in an example that may be closer to the experience of children with early focal brain injury, a recent study by Webster, Bachevalier and Ungerleider (1995) shows that the "where is it" system (mediated in dorsal regions, especially parietal cortex, including area MT) can take over the functions of the "what is it" system (mediated in ventral regions, especially inferior temporal cortex, including area TE). When area TE is bilaterally removed in an adult monkey, that animal displays severe and irreversible amnesia for new objects, suggesting that this area plays a crucial role in mediating object memory and detection (i.e., the so-called "what is it" system). However, as Webster et al. have shown, bilateral removal of area TE in infant monkeys leads to performance only slightly below age-matched unoperated controls (at both 10 months and 4 years of age). If area TE is no longer available, where has the "what is it" system gone? By lesioning additional areas of visual cortex, Webster et al. showed that the object detection function in TE-lesioned infant monkeys is mediated by dorsal regions of extrastriate cortex that usually respond to motion rather than form (i.e., the "where is it" system). In other words, a major higher cognitive function can develop far away from its intended site, in areas that would ordinarily play little or no role in the mediation of that function.

These examples and many others like them have led most developmental neurobiologists to conclude that cortical differentiation and functional specialization are largely the product of input to the cortex, albeit within certain broad architectural and computational constraints (Johnson, 1997). Such findings provide a serious challenge to the old notion that the brain is organized into largely predetermined, domain-specific faculties, i.e., the phrenological approach. An alternative proposal that is more compatible with these findings will be offered later on.

(2) LANGUAGE OUTCOMES IN CHILDREN WITH EARLY FOCAL BRAIN INJURY: RETROSPECTIVE FINDINGS

As noted earlier, retrospective studies of language outcomes in children with unilateral brain injury have repeatedly found that these children are not aphasic; they usually perform within the normal range, although they often do perform slightly below neurologically intact age-matched controls (cf. Webster, Bachevalier, & Ungerleider, 1995). More importantly for our purposes here, there is no consistent evidence in these retrospective studies to suggest that language outcomes are worse in children with left-hemisphere damage, compared with children whose injuries are restricted to the right hemisphere. Without attempting an exhaustive review, three examples will suffice to illustrate these points.

Figures 1a and 1b (adapted from Bates, Vicari, & Trauner, in press) present idealized vs. observed results for verbal vs. nonverbal IQ scores in a cross-sectional sample of children with congenital injuries who were tested at various ages between 3 and 10 years. Figure 1a illustrates what we might expect if the left/right differences observed in adults were consistently observed in children: higher verbal than nonverbal IQ scores in children with right-hemisphere damage (RHD), which means that these children should line up on the upper diagonal; higher nonverbal than verbal IQ scores in children with left-hemisphere damage (LHD), which means that these children ought to fall on the lower diagonal. These idealized scores were obtained by taking actual pairs of scores for individual children in our focal lesion sample, and reversing any scores that were not in the predicted direction. In contrast with this idealized outcome, Figure 1b illustrates the actual verbal and performance IQ scores for 28 LHD and 15 RHD cases (note that there are no differences between these two groups in gender or chronological age, and no mean differences in full scale IQ). The actual data in Figure 1b illustrate several points. First, in line with other studies of this population, the mean full-scale IQ for the

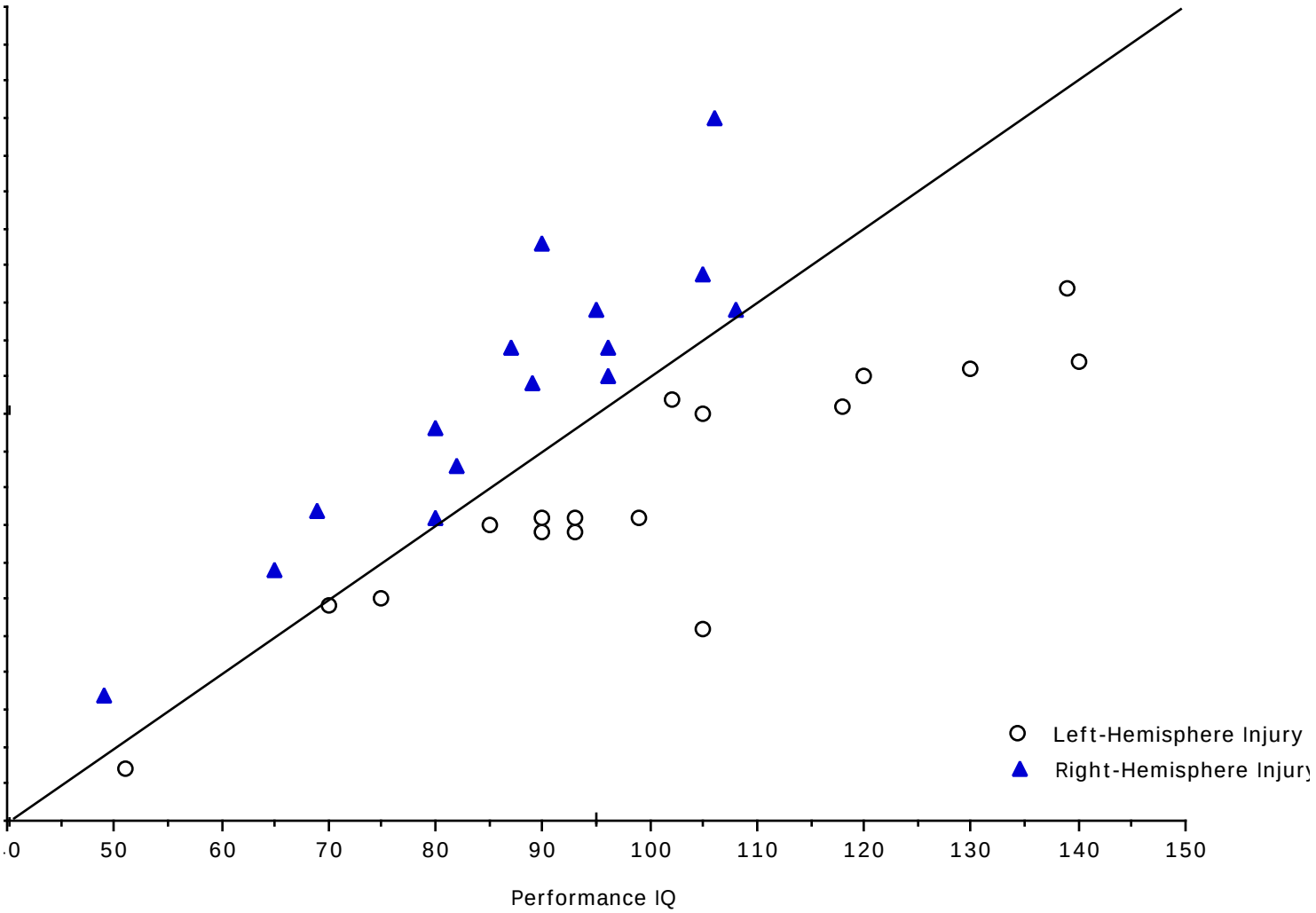


Figure 1a: Idealized Relation between Verbal & Performance IQ in Children with Left- vs. Right-Hemisphere Injury (adapted from Bates, Vicari & Trauner, in press)

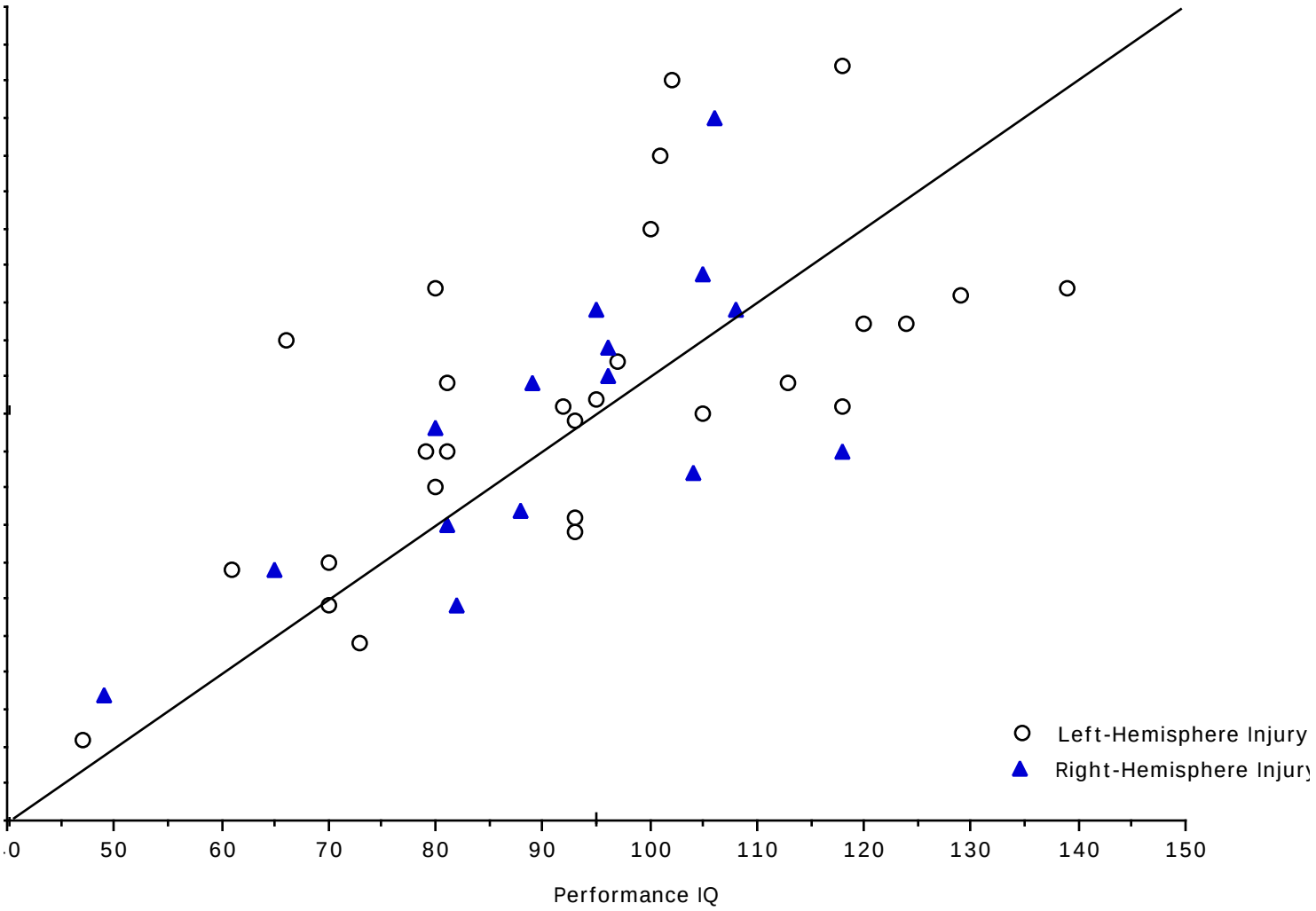


Figure 1b: Observed Relation between Verbal & Performance IQ in Children with Left- vs. Right-Hemisphere Injury (adapted from Bates, Vicari & Trauner, in press)

sample as a whole is 93.2, within the normal range but below the mean of 100 that we would expect if we were drawing randomly from the normal population. Second, the range of outcomes observed in the focal lesion population as a whole is extraordinarily broad, including some children who can be classified as mentally retarded (i.e., 16.3% of this sample have full scale IQs at or below 80), and some with IQs over 120. Third, the correlation between the verbal and nonverbal subscales is relatively strong (+.65, $p < .0001$), which means that verbal and nonverbal IQ do not dissociate markedly in this group. In fact, as we can clearly see from the difference between Figure 1a (predicted outcomes) and 1b (the outcomes actually observed in these children), there is absolutely no evidence in these data for a double dissociation between verbal and nonverbal IQ as a function of left- vs. right-hemisphere injury.

Figure 2 (adapted from Reilly et al., 1998) presents results from a more focused study of grammatical development, illustrating the number of different complex syntactic forms produced in a narrative discourse task by LHD, RHD and neurologically intact controls who were tested between 6 and 12 years of age at testing. This figure demonstrates (once again) that children with focal brain injury perform within the normal range in production of complex syntax, even though they do (as a group) score significantly below neurologically intact controls. In this respect, the Reilly et al. result for grammatical development in human children is remarkably similar to the findings reported by Webster et al., on the relative preservation of memory for novel objects in infant monkeys with bilateral TE lesions (i.e., performance roughly 10% below that of normal controls). In addition, Figure 2 shows that there is no evidence in this age range for a difference in syntactic production as a function of lesion side or site.

Finally, Figures 3a and 3b (from Kempler, van Lancker, Marchman, & Bates, in press) compare results for adults and 6-12-year-old children with LHD vs. RHD on the same sentence comprehension task. The data in both figures are all based on z-scores, with patients at each age level compared with the performance of age-matched normal controls (hence the difference in performance between normal adults and normal 6-12-year-old children is factored out of the results). In this particular procedure, subjects are asked to match each stimulus sentence to one of four pictured alternatives. Half the items are familiar phrases (well-known metaphors and figures of speech like "She took a turn for the worse"), and the other half are novel phrases matched to the familiar phrases in length and complexity. As Figure 3a shows, there is a powerful double dissociation between novel and familiar phrases in adult victims of unilateral brain injury: adults with LHD score markedly better on the familiar phrases, while adults with RHD score better on the novel phrases. This is one example of a growing body of evidence challenging the old assumption that the left hemisphere is "the" language hemisphere, even in adults. The right hemisphere does make an important contribution to language processing, but its contribution is qualitatively different from that of the left hemisphere, involving a number of functions including emotionality, intonation contours and (as this example illustrates) figurative, metaphorical and/or formulaic speech (all forms of speech in which the meaning of the sentence as a whole goes beyond the meaning one would obtain by computing across the separate elements in the sentence). A comparison between Figures 3a and 3b helps to clarify three important points. First, children with focal injuries fare far better than adults with comparable damage, when they are compared with age-matched controls. Second, the powerful double dissociation observed in adults is not observed in children. Third, novel sentences are more susceptible to the effects of brain injury than familiar phrases in the child group, but RHD children actually perform below the LHD group in comprehension of novel sentences (significant by a one-tailed t-test), the opposite of what we might expect if the adult pattern held for children with focal brain injury.

In short, whether we are talking about global measures like IQ or more subtle measures of sentence production and comprehension, children with LHD vs. RHD do not display the profiles of impairment that we would expect based on the adult aphasia literature -- at least not in these and other retrospective studies, with outcome measures at or above six years of age (i.e., beyond the point at which fundamental aspects of grammar and phonology are usually in place -- Bates, Dale, & Thal, 1995).

(3) AGE OF LESION ONSET AND THE PROBLEM OF CRITICAL PERIODS

The distinction between retrospective and prospective studies is related to the controversial problem of "critical periods" for language, with special focus on the age at which a lesion is acquired. By definition, prospective studies focus on children whose lesions are acquired very early, preferably before the point at which language learning normally begins. In contrast, many retrospective studies collapse across children who acquired their lesions at different points across the course of language learning. Our own prospective studies are based exclusively on children with congenital injuries, defined to include pre- or perinatal injuries that are known to have occurred before six months of age, restricted to one side of the brain (left or right), confirmed through one or more forms of neural imaging (CT or MRI). Hence our results may differ from studies of children with injuries acquired at a later point in childhood.

What might those differences be? Unfortunately, there is very little empirical evidence regarding the effect of age of lesion onset on subsequent language outcomes. Only one fact is clear: that the outcomes associated with left-hemisphere injury are much better in infants than they are in adults. This means, of course, that plasticity for language must decrease markedly at some point between birth and adulthood (Lenneberg, 1967). But when does this occur, and how does it happen?

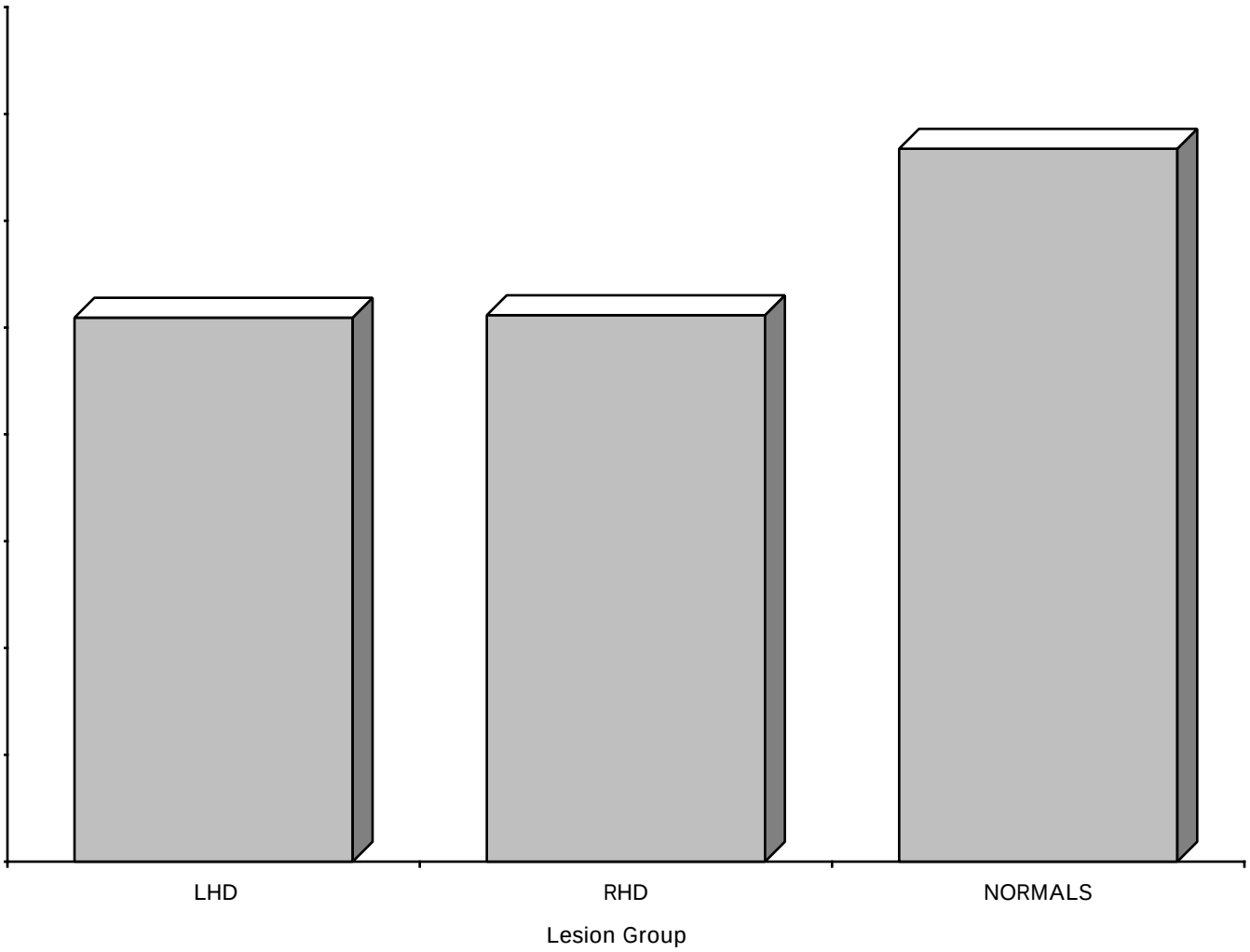


Figure 2: Number of Different Complex Syntactic Forms Produced by Children with Left vs. Right Hemisphere Damage in a Story-Telling Task (age = 6 to 12 years) (adapted from Reilly, Marchman & Bates, in press)

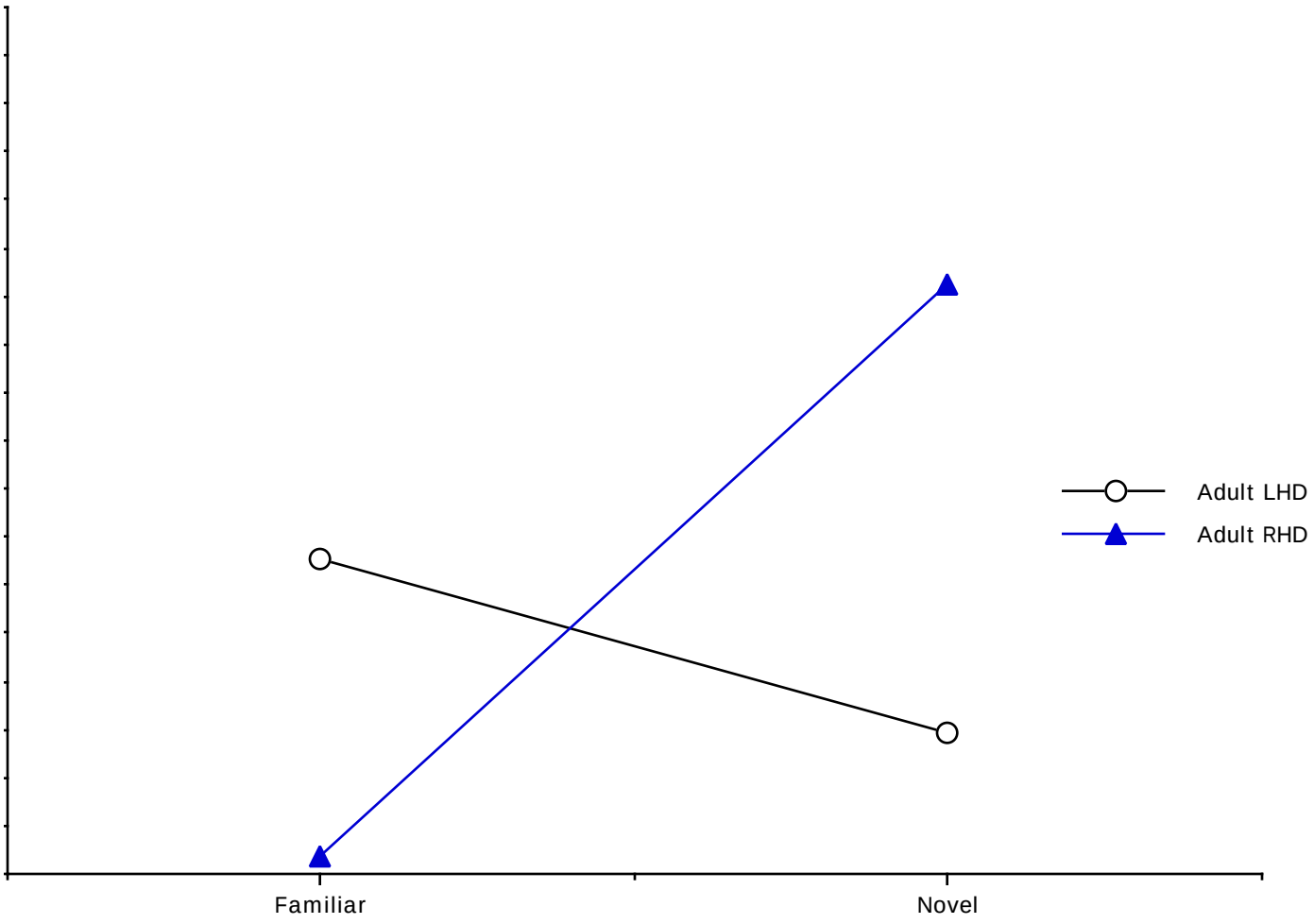


Figure 3a: Performance on Familiar vs. Novel Sentences
in Adults with Left- vs. Right-Hemisphere Injury
(adapted from Kempler, Van Lancker, Marchman & Bates, in press)

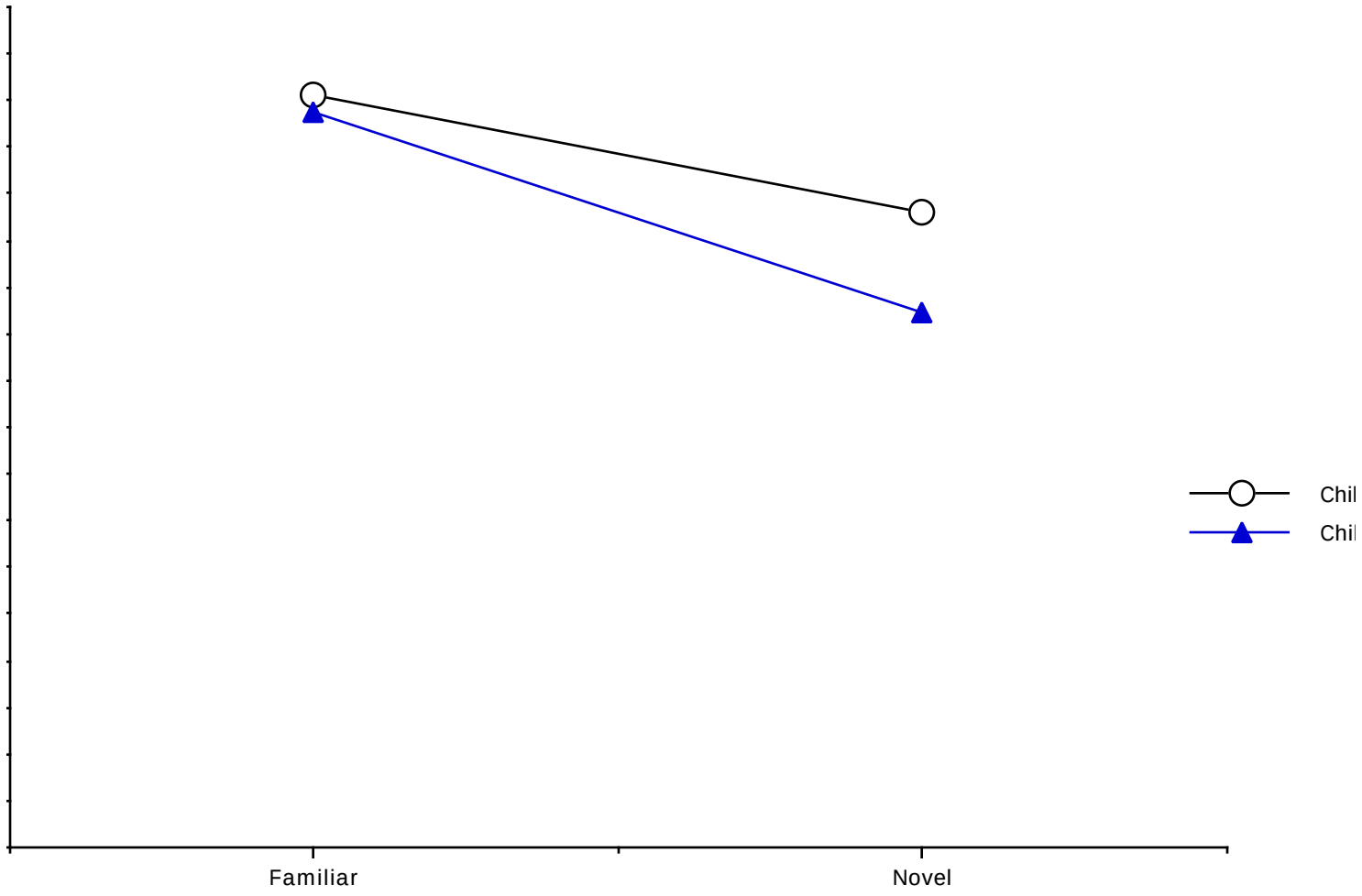


Figure 3b: Performance on Familiar vs. Novel Sentences
in Children with Left- vs. Right-Hemisphere Damage
(adapted from Kempler, Van Lancker, Marchman & Bates, in press)

Many investigators have argued that this decrease in plasticity takes place at the end of a "critical period" for language, a window of opportunity that is also presumed to govern the child's ability to achieve native-speaker status in a second language (for discussions, see Bialystok & Hakuta, 1994; Curtiss, 1988; Elman et al., 1996, Chapter 5; Johnson & Newport, 1989; Marchman, 1993; Oyama, 1993; Weber-Fox & Neville, 1996). So much has been said about this presumed critical period that a newcomer to the field (and many consumers within it) would be justified in assuming that we know a great deal about its borders (i.e., when it begins and when it comes to an end), and about the shape of the learning function in between these points. The very term "critical period" suggests that the ability to acquire a native language and/or the ability to recover from brain injury both come to a halt abruptly, perhaps at the same time, as the window of opportunity slams shut. The fact is, however, that we know almost nothing about the shape of this function. In fact, we are not even justified in assuming that the function is monotonic (i.e., that it gets progressively harder to learn a native language, and progressively harder to recover from injuries to the left hemisphere).

With regard to the presumed critical period for recovery from brain injury, we are aware of only two large cross-sectional studies that have compared language and cognitive outcomes in children who acquired their lesions at different ages, from congenital injuries (at or before birth) through early adolescence (Vargha-Khadem et al., unpublished results, cited with permission in Bates, Vicari, & Trauner, in press; Goodman & Yude, 1996). Figure 4 compares results from both these studies for verbal IQ. As this figure indicates, the effect of age of injury is nonmonotonic in both studies: The worst outcomes are observed in children who suffered their injuries between approximately 1-4 years of age. In support of the critical period hypothesis, better outcomes are observed following congenital injuries. However, in direct contradiction to the critical period hypothesis, better outcomes are also observed in children whose injuries occurred between approximately 5-12 years of age, which means that there is no monotonic drop in plasticity. To some extent, these unpleasant wrinkles in the expected function could be due to uncontrolled differences in etiology (e.g., the factors leading to injury may differ at birth, 1-5 years, and later childhood). At the very least, however, these results ought to make us skeptical of claims about a straightforward critical period for recovery from brain injury.

Similar nonmonotonic findings have been reported in at least one study of second-language acquisition and first-language loss (Liu, Bates, & Li, 1992). To illustrate, compare the results in Figure 5 (adapted from a famous study of second-language acquisition by Johnson and Newport, 1989) and Figure 6 (adapted from Liu et al., 1992). Figure 5 illustrates results from a grammaticality judgment task administered to first- and second-language learners of English, comparing performance of individuals who arrived in the U.S. at different points spanning the period from birth to early adulthood. This well-known figure suggests that there is no single point at which the window of opportunity for second-language learning slams shut. However, it does provide evidence for a monotonic drop in language learning ability from birth to adolescence.

Consider, however, the results in Figure 6, based on a sentence interpretation task administered to Chinese-English bilinguals in both Chinese and English. In this task, subjects were able to use either semantic or word order information to interpret "odd" sentences like "The rock chased the dog". Native speakers of English invariably choose the first noun, using word order to make their interpretation. Native speakers of Chinese invariably choose the second noun, ignoring word order in favor of semantic information. Both these strategies make perfect sense in terms of the information value of standard word order in these two languages (Chinese permits so much word order variation that a persistent word order strategy like the one used in English would not be very useful). Hence this little task serves as a useful litmus test for retention of the first language (L1) as well as acquisition of the second (L2). The interesting point for our purposes is that Chinese-English bilinguals often perform somewhere in between these two extremes, in one or both of their two languages, and these different "weightings" of word order and semantic information vary as a function of age of acquisition. Notice that results for English (L2) are generally in agreement with Johnson and Newport's results for a very different task: although our results asymptote at an earlier point than those of Johnson and Newport, they do provide evidence for a monotonic shift from "English-like" interpretations of English sentences in those who learned their English very early, to "Chinese-like" interpretation of English sentences in those who learned their English relatively late. However, results for Chinese (L1) show a very different function, a nonmonotonic curve in which the best results (movement toward the second language without loss of the first language) are observed in those who are exposed to a second language somewhere between 4-7 years of age.

Although this is a complex result, the point of this comparison for our purposes here is a simple one: There is no single "critical period" for language learning; results depend on many different factors, and the probability of a positive outcome can rise or fall at different points in development, in L2 learning and in recovery from brain injury. This is where prospective studies can be particularly illuminating: By studying children during their first encounters with language and other forms of higher cognition, we can learn more about effects associated with the initial state of the brain, together with the processes of development and (re)organization that lead these children to a normal or near-normal outcome.

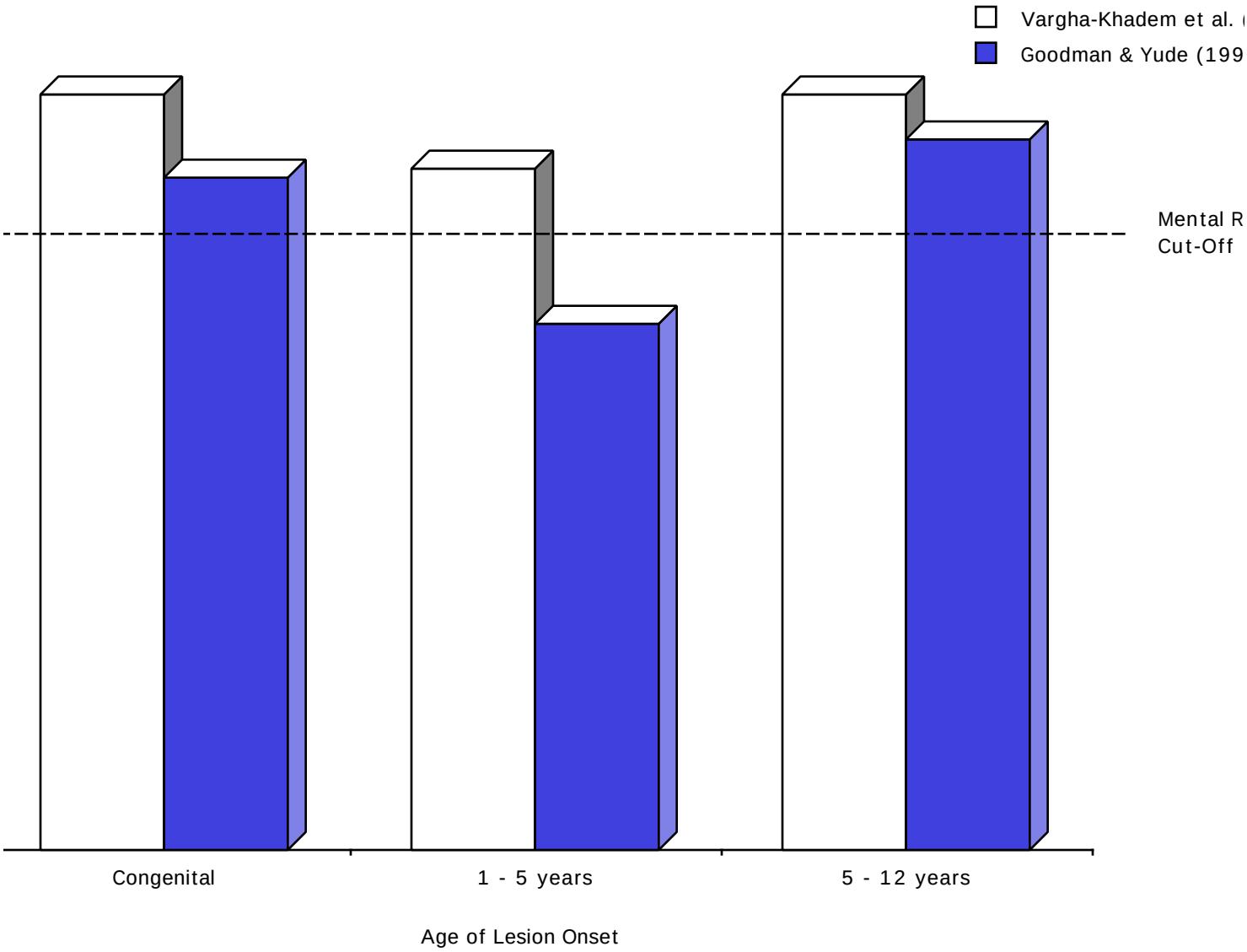


Figure 4: Relationship between Age of Lesion Onset and IQ Scores in Two Samples of Children with Focal Brain Injury

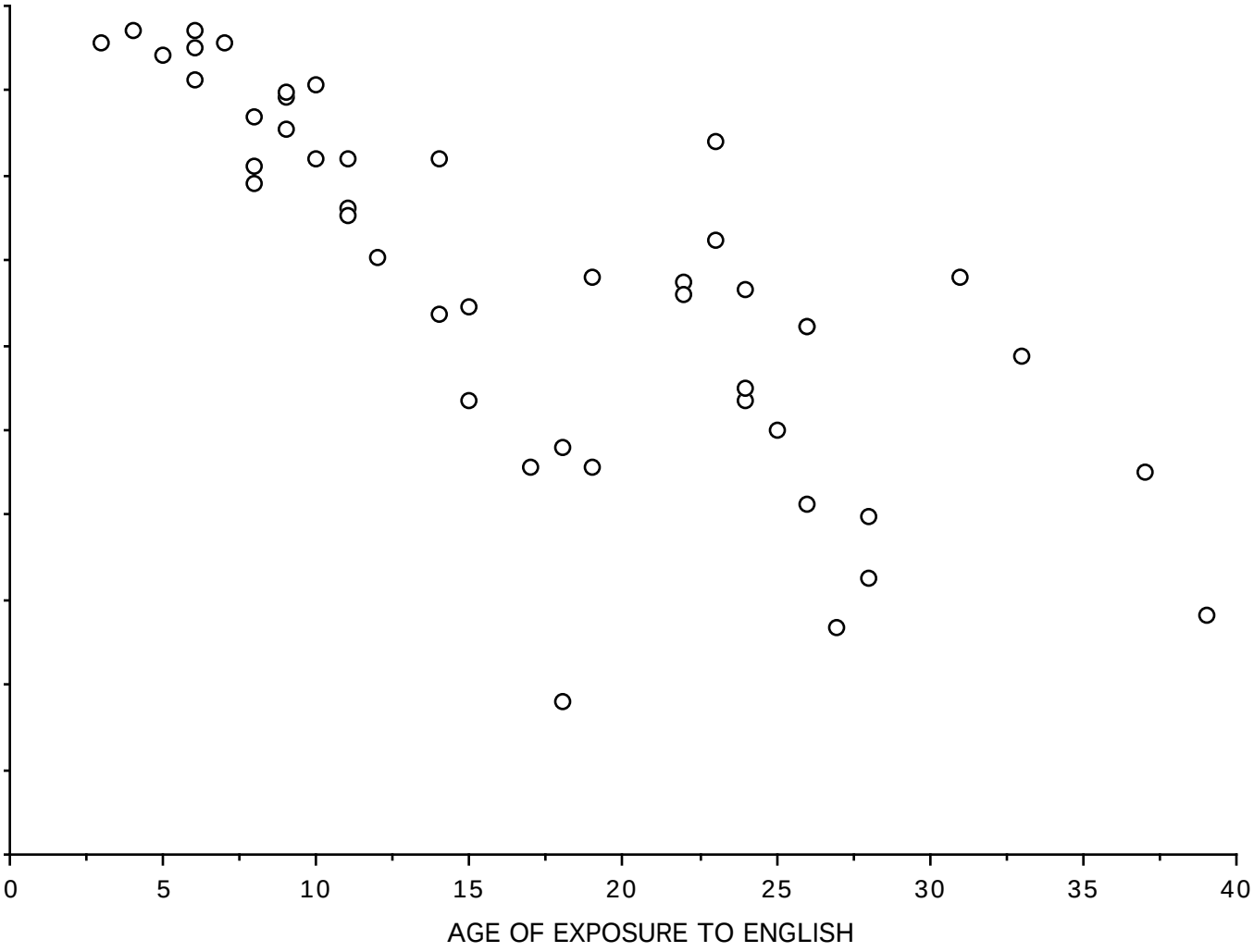


Figure 5: Performance on a Grammaticality Judgment Task in Non-Native Speakers of English as a Function of Age of Exposure to English (adapted from Newport & Johnson, 1992)

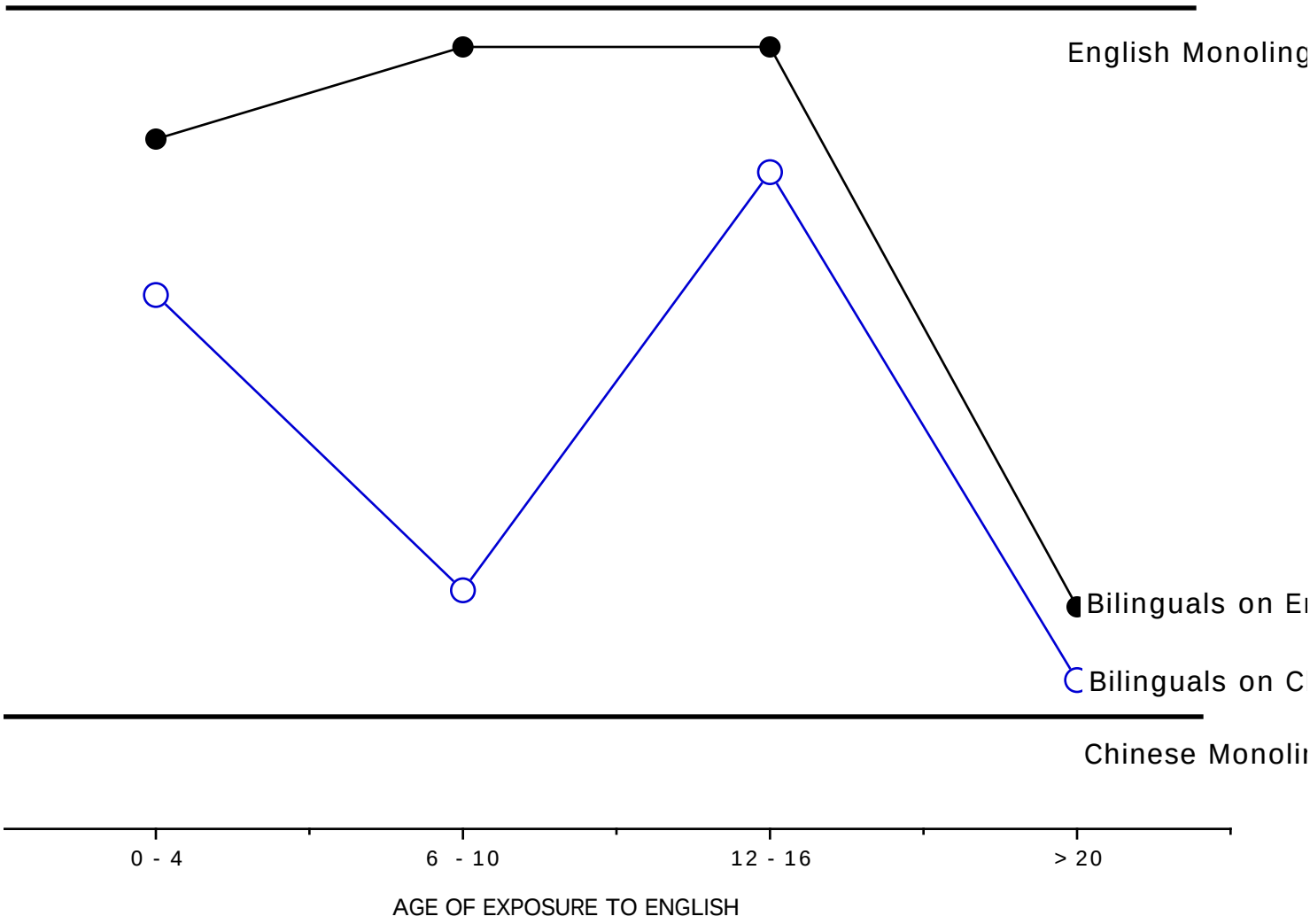


Figure 6: "English-like" vs. "Chinese-like" Grammatical Comprehension as a Function of Age of Exposure to English (adapted from Liu, Bates & Li, 1993)

(4) LANGUAGE OUTCOMES IN CHILDREN WITH EARLY FOCAL BRAIN INJURY: PROSPECTIVE FINDINGS

All theories that take some form of plasticity into account (including theories that assume a critical period) would lead us to expect relatively good outcomes in children with congenital injuries, i.e., the group that we have studied in our laboratory. Evidence for the developmental plasticity of language in this group has mounted in the last few years, due in part to improved techniques for identifying children with early brain injury, including precise localization of the site and extent of damage through neuroradiology. In some cases, we have been able to identify such children in the first weeks of life, prior to the time when language acquisition would normally begin, permitting us to chart the course of language, cognition and communicative development from the very beginning (Bates et al., 1997; Reilly, Stiles, Larsen, & Trauner, 1995; Stiles et al., 1998; Stiles & Thal, 1993), before the point at which alternative forms of brain organization have emerged.

In fact, the prospective studies that we have carried out so far provide compelling evidence for initial deficits and subsequent processes of recovery -- phenomena that are not visible later on, when most retrospective studies take place. For example, prospective studies of nonverbal cognitive development by our colleague Joan Stiles have revealed subtle but consistent patterns of deficit in visual-spatial cognition. For example, children with RHD appear to have difficulty perceiving and/or producing the global or configural aspects of a complex visual array; children with LHD are generally spared at the global level, but they have difficulty with the perception and/or production of local details (Note: I will return to this example later on, relating it to our findings for language). These visual-spatial deficits are qualitatively similar to those observed in LHD vs. RHD adults, although they are usually more subtle in children, and they resolve over time as the children acquire compensatory strategies to solve the same problems (Stiles et al., 1998; Stiles & Thal, 1993).

If a similar result could be found in the domain of language, then we might expect (by analogy to the literature on adult aphasia) to find the following results in the first stages of language development:

-- **Left-hemisphere advantage for language**: Children with LHD will perform below the levels observed in children with RHD on virtually all measures of phonological, lexical and grammatical development, as well as measures of symbolic and communicative gesture.

-- **The Broca pattern**: By analogy to Broca's aphasia in adults, children with damage to the frontal regions of the left hemisphere will be particularly delayed in expressive but not receptive language, and may (on some accounts) be particularly delayed in the development of grammar and phonology;

-- **The Wernicke pattern**: By analogy to Wernicke's aphasia in adults, children with damage to the posterior regions of the left temporal lobe will be particularly delayed in receptive language, perhaps (on some accounts) with sparing of grammar and phonology but selective delays in measures of semantic development.

Our group set out to test these three hypotheses in a series of prospective studies of early language development. In every case, we have uncovered evidence for early deficits, and these deficits do appear to be associated with specific lesion sites. However, in contrast with Stiles' findings for visual-spatial cognition, results for language provide very little evidence for hypotheses based on the adult aphasia literature.

The first study (Marchman, Miller, & Bates, 1991) focused on the emergence of babbling and first words in a small sample of five children with congenital brain injury, two with RH damage, three with LH damage, including one LH case with injuries restricted to the left frontal region. All the children were markedly delayed in phonological development (babbling in consonant-vowel segments weeks or months behind a group of neurologically intact controls), and in the emergence of first words. However, three of the children moved up into the normal range across the course of the study. The two who remained behind had injuries to the posterior regions of the left hemisphere, results that fit with the first hypothesis (LH advantage for language) but stand in direct contradiction to both the Broca and the Wernicke hypotheses.

The second study (Thal et al., 1991) focused on comprehension and production of words from 12-35 months in a sample of 27 infants with focal brain injury, based on a parental report instrument that was the predecessor of the MacArthur Communicative Development Inventories or MCDI (Fenson et al., 1993, 1994). In complete contradiction to Hypothesis 1 (LH mediation of language) and Hypothesis 3 (the Wernicke Hypothesis), delays in word comprehension were actually more likely in the RH group. In line with Hypothesis 1, but against Hypothesis 2 (the Broca Hypothesis), delays in word production were more likely in children with injuries involving the left posterior quadrant of the brain.

A more recent study built on the findings of Thal et al. with a larger sample of 53 children, 36 with LHD and 17 with RHD (Bates et al., 1997), using a combination of parent report (the MCDI) and analyses of free speech. This report is broken into three substudies, with partially overlapping samples. Study 1 used the MCDI to investigate aspects of word comprehension, word production and gesture at the dawn of language development, in 26 children between 10-17 months of age. Study 2 used the MCDI to look at production of both words and grammar in 29 children between 19-31 months. Study 3 used transcripts of spontaneous speech in 30 children from 20 to 44 months, focusing on Mean Length of Utterance in morphemes (MLU). In all these studies, comparisons between the LHD and RHD groups were followed by comparisons looking at the effects associated with lesions involving the frontal lobe (comparing children with left frontal involvement to all RHD cases as well

as LHD cases with left frontal sparing) and the temporal lobe (comparing children whose lesions include the left temporal lobe with all RHD cases and all LHD cases in which that region is spared). Results were compatible with Marchman et al. and Thal et al., but quite surprising from the point of view of lesion/symptom mappings in adult aphasia, as follows.

First, in a further disconfirmation of Hypotheses 1 and 3, Bates et al. report that delays in word comprehension and gesture were both more likely in children with unilateral damage to the right hemisphere, at least in the 10-17-month window examined here. Further studies of gestural development in our laboratory have confirmed that the gestural disadvantage for RH children is still present between 20-24 months (Stiles et al., 1998).

Second, in a partial confirmation of Hypothesis 2 (the Broca Hypothesis), frontal involvement was associated with greater delays in word production and the emergence of expressive grammar between 19 and 31 months. However, in a surprising partial disconfirmation of Hypothesis 2, this frontal disadvantage was equally severe with either left frontal or right frontal involvement. In other words, the frontal lobes are important during this crucial period of development (which includes the famous "vocabulary burst" and the flowering of grammar), but there is no evidence for a left-right asymmetry in the frontal regions, and hence no evidence in support of the idea that Broca's area has a privileged status from the very beginning of language development.

Third, in line with Hypothesis 1 (LH mediation of language) but in direct contradiction to Hypotheses 2 and 3 (analogies to Broca's and Wernicke's aphasia), delays in word production and the emergence of grammar were both more pronounced in children with injuries involving the left temporal lobe. In contrast with the above two findings (which only reached significance within a restricted period of development), this left temporal disadvantage was reliable across all three substudies in Bates et al., from the very first words (between 10-17 months of age) through crucial developments in grammar (between 20-44 months of age). Hence we do have evidence for the asymmetrical importance of Wernicke's area, but that evidence pertains equally to grammar and vocabulary (with no evidence of any kind for a dissociation between the two), and seems to be restricted to expressive language.

Reilly et al. (1998) conducted similar comparisons by lesion side and lesion site in a cross-sectional sample of 30 children with focal brain injury (15 LH and 15 RH) between 3 and 12 years of age; these results were also compared with performance by a group of 30 age-matched controls with no history of neurological impairment. Analyses were based on lexical, grammatical and discourse measures from a well-known story-telling task. For children between 3-6 years of age, Reilly et al. replicated the specific disadvantage in expressive language for children with lesions involving the temporal region of the left hemisphere. However, this effect was not detectable in children between 6-12 years of age -- even though all children in this study had the same congenital etiology. In fact, data for the older children provided no evidence of any kind for an effect of lesion side (left vs. right) or lesion site (specific lobes within either hemisphere). The only effect that reached significance in older children was a small but reliable disadvantage in the brain-injured children as a group, compared with neurologically intact age-matched controls. Figure 7 compares results for younger vs. older children on one grammatical index (mean number of errors in grammatical morphology per proposition), divided into children with left temporal involvement (+LTemp), focal lesion cases without left temporal involving (–Ltemp, combining all RHD cases and all LHD cases with temporal sparing), and neurologically intact normal controls. Although we must remember that these are cross-sectional findings, they suggest that a substantial degree of recovery takes place in the LH group during the first few years of life. In subsequent longitudinal studies, Reilly and her colleagues have followed a smaller group of children across this period of development. These longitudinal findings are compatible with the cross-sectional evidence in Figure 7, suggesting that the crucial period of recovery takes place before the age range covered by most of the retrospective studies in the literature on cognitive and linguistic outcomes in children with focal brain injury.

To summarize, our prospective studies of language development in children with early focal brain injury have provided evidence for specific delays, correlated with specific lesion sites. However, the nature of these lesion/symptom correlations differs markedly from those that we would expect based on the adult aphasia literature. Furthermore, these correlations are only observed within specific windows of development, followed by evidence for recovery and (by implication) reorganization. None of these results are evident in retrospective studies (including our own), where children are tested beyond the point at which this presumed reorganization has taken place.

We are occasionally asked why our results appear to be incompatible with an earlier literature on the effect of hemispherectomy (e.g., Dennis & Whitaker, 1976; but see Bishop, 1983) and/or effects of early stroke (e.g., Aram, 1988; Aram, Ekelman, & Whitaker, 1985; Aram, Ekelman, Rose, & Whitaker, 1985; Woods & Teuber, 1978). Our first answer is that our results are *not* incompatible with the vast majority of studies. However, they do *appear* to be incompatible with a handful of studies that were cited (usually in secondary sources) as evidence in favor of an innate and irreversible role for the left hemisphere in some aspects of language processing. As we have noted elsewhere (Bates, Vicari, & Trauner, in press; see also Vargha-Khadem et al., 1994), apparent inconsistencies between the earlier studies and our more recent work disappear when one looks carefully at the fine print.

First, many of the earlier studies combined data for children whose injuries occurred at different points in development, and they also combined results (usually on rather global measures) for children at widely different ages at time of testing. As we saw in the previous section, there may not be a monotonic relation

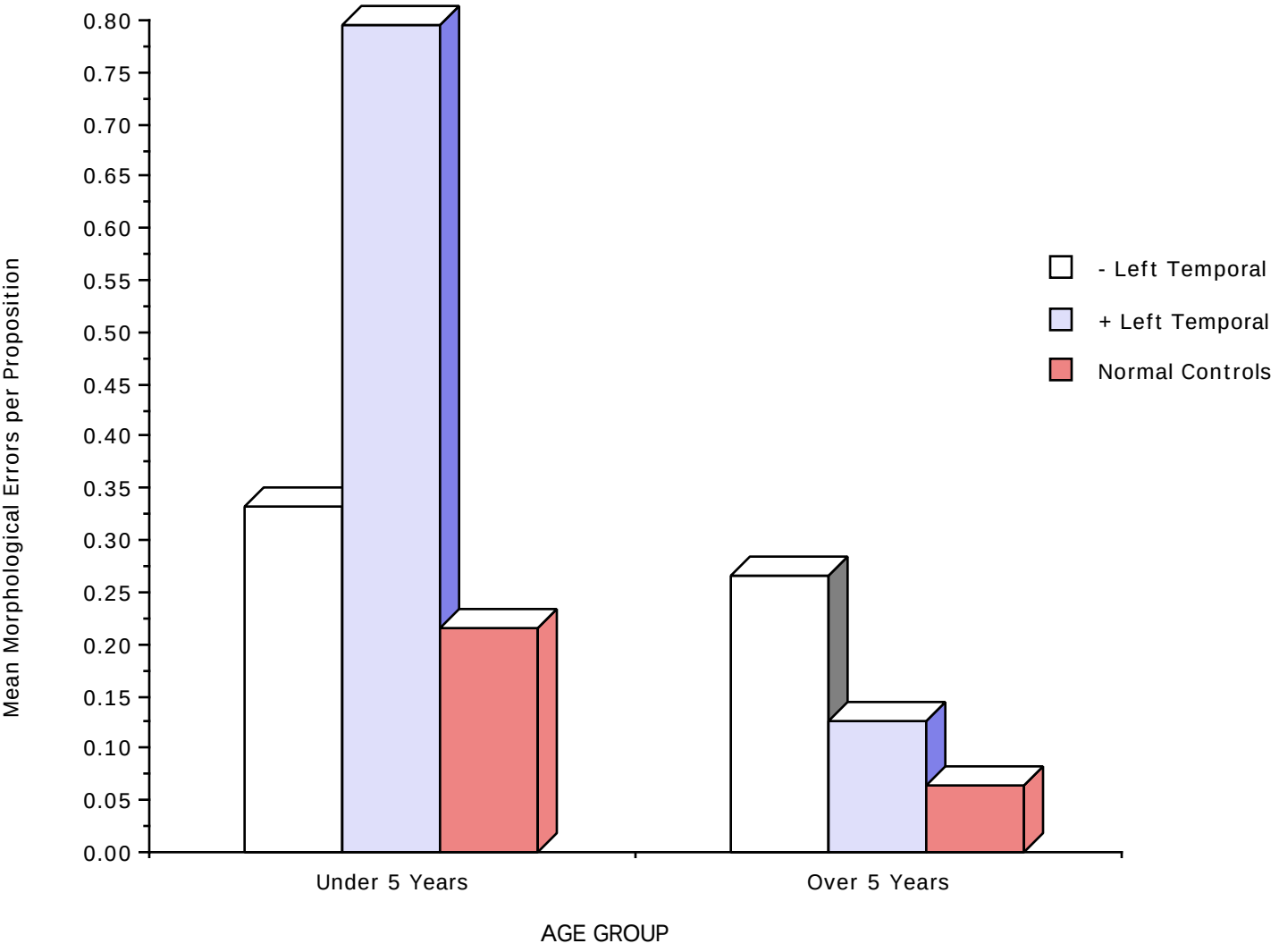


Figure 7: Morphological Errors as a Function of Age and Presence/Absence of Left Temporal Damage (adapted from Reilly, Marchman & Bates, in press)

between age of injury and language outcomes, and the nature of the lesion/symptom mappings that we observe may be quite different depending on the age at which children are tested and the developmental events that are most prominent at that time.

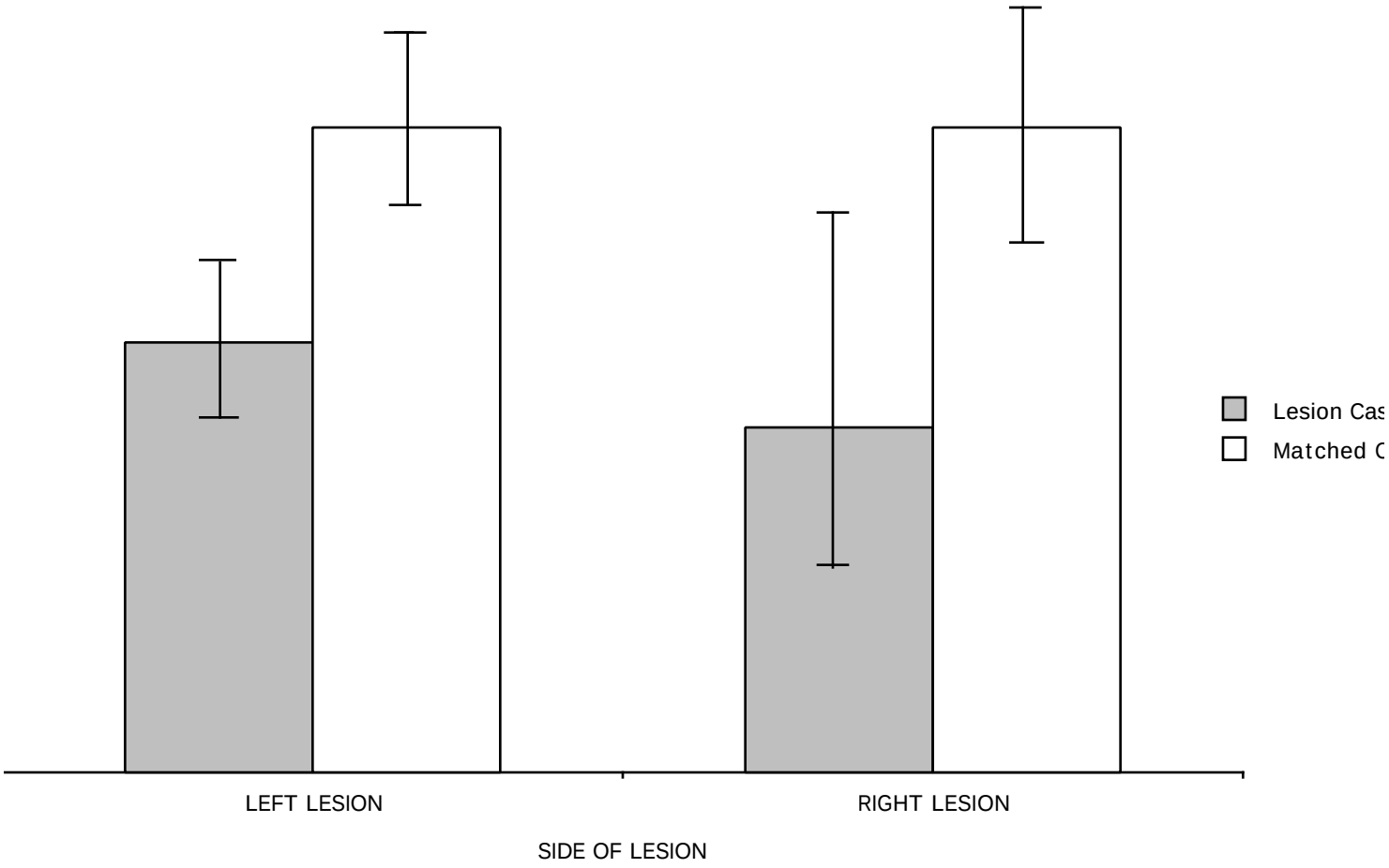
Second, some of the earlier studies had methodological limitations that we have been able to overcome in the studies described above. In particular, a number of well-known studies could not perform direct comparisons of children with LHD vs. RHD, because of uncontrolled differences in age, education and/or etiology. Instead, the RHD and LHD groups were each compared with a separate group of matched controls. For example, Dennis & Whitaker report that their left-hemispherectomized children performed below normal controls on subtle and specific aspects of grammatical processing; no such difference was observed between right-hemispherectomized children and their controls. These results were interpreted as though they constituted a significant difference between the LHD and RHD groups, even though the latter two groups were never compared directly. As Bishop has pointed out in her well-known critique (Bishop, 1983), a careful examination of results for the two lesion groups suggests that this interpretation is not warranted. The general problem that one encounters with the separate control group approach is illustrated in Figure 8, which compares hypothetical data for an LHD group, an RHD group, and their respective controls. As we can see from this figure, performance by the LHD group does fall reliably below performance by their controls (albeit just barely); performance by the RHD group does not fall outside the confidence intervals for their control group. And yet, in this hypothetical example, performance is actually better in the LHD cases! The key to this conundrum lies in the standard deviations for each control group: The standard deviation is larger for the RHD controls, which means that a larger difference between RHD and controls is required to reach statistical significance. Clearly, it would be unwise to draw strong conclusions about left/right differences from a data set of this kind.

Finally, some of the better-known claims in favor of an early and irreversible effect of LH damage have been based on single-case studies or very small samples (including the hemispherectomy studies cited above). This fact limits the generalizability of results, and the same result is often contradicted by other individual-case or small-group studies.

For example, Stark and McGregor (1997) have recently described an interesting contrast between one child with a left hemispherectomy (seizure onset at 1;6, surgery at 4;0), and another with a right hemispherectomy (seizure onset at 2;0, surgery at 5;8). Both children were followed longitudinally with testing at 1-2-year intervals through 9;0 and 9;6 years of age, respectively. Although both children did show substantial development in language and cognition across the course of the study, they fell behind age-matched normal controls at every point. At the end of the study, the LHD case had a full-scale IQ of 71 and the RHD case had a full-scale IQ of 81, well behind the norms for development in children who are neurologically intact. For Stark & McGregor, the most interesting findings lie in the contrasting patterns observed for each child for performance IQ, verbal IQ, and series of more specific language tests. For the LHD case, verbal and performance IQ were both quite low (separated by only four points). However, performance on the specific language tasks followed a profile typical of the pattern observed in children with Specific Language Impairment, i.e., greater impairment in language measures (especially morphosyntax) than we would expect for her mental age. By contrast, the RHD case displayed a sharp dissociation at the end of the study between verbal IQ (95) and performance IQ (70), with scores on most of the specific language measures that were appropriate for her mental age.

This is an interesting and provocative result, and it might indeed reflect evidence for the emergence of some kind of left-hemisphere specialization for language prior to the age at which the surgery occurred. However, our own experience with a relatively large focal lesion sample has made us wary of basing strong results on case studies. Individual differences in language and cognitive ability are immense, even in perfectly normal children with no history of brain injury (Bates et al., 1995; Fenson et al., 1994). A similar degree of variation is observed even within the small cadre of cases that have undergone hemispherectomy.

Evidence for such variation comes from the case of Alex, recently reported by Vargha-Khadem et al. (1997). Alex was nearly mute prior to his surgery between 8-9 years of age, and (to the extent that he could be tested at all) demonstrated levels of language comprehension similar to those of a normal 3-year-old. Soon after his surgery, he demonstrated remarkable recovery in both receptive and expressive language, and continued to make progress into adolescence. Although Alex did suffer some degree of mental retardation (as an adolescent, he has the mental age of a 10-12-year-old child on most measures), his language abilities are entirely commensurate with his mental age. In fact, his level of performance on language measures is superior to both of the cases reported by Stark and McGregor, even though his surgery took place several years later. The contrast between this study and that of Stark et al. underscores two important points. First, it provides further evidence against the assumption that plasticity drops monotonically across a supposed critical period for language. Second, it reminds us that the effects of brain injury are superimposed upon the vast landscape of individual variation observed in normally developing children (for an elaboration of this point, see Bates et al., 1995). Because there is so much variation in the normal population, it is difficult to know in a single-case or small-sample study whether or not the cognitive profiles we observe are statistically reliable. Indeed, they may be no different from the patterns that would be observed if brain damage were imposed randomly on cases selected from the population at large (Bates, Appelbaum & Allard, 1991; Bishop, 1997; see also Basser, 1962, for evidence that the vast majority of cases in a large sample of hemispherectomized children



igure 8: A Hypothetical Example of Comparisons between Left- and Right-Hemisphere Groups with their Respective Controls (LHD-Control Difference is Reliable; RHD-Control Difference is not)

show no evidence at all of a speech/language impairment, regardless of side of surgery).

Despite these concerns, our results for older children are largely compatible with the retrospective literature on language development in the focal lesion population: Children with early injuries to one side of the brain usually acquire language abilities within the normal or low-normal range, with little evidence for effects of lesion side or lesion site (as reviewed in Bates, Vicari, & Trauner, in press; Eisele & Aram, 1995; Vargha-Khadem et al., 1994). Our prospective findings for children under five years of age are qualitatively different, but they are also so new that there is little or no comparable information in the literature, aside from a few single-case or small-sample studies with very different goals (e.g., Dall' Oglio, Bates, Volterra, Di Capua, & Pezzini, 1994; Feldman et al., 1992). Of course it will be important to replicate all these prospective findings with other samples of children, and in other laboratories. In the meantime, we can take some comfort in the fact that these results are based on the largest and most homogeneous sample of children with focal brain injury that has ever been studied in a prospective framework. Although in some cases the same children participate in more than one prospective study, the full sample across our two largest studies (Bates et al., 1997; Reilly et al., 1998) includes 72 cases of children with focal brain injury, from three different laboratories. With sample sizes of 26 or more from one substudy to another, we have been able to use experimental designs and inferential statistics that would not be appropriate in a single-case or small-sample study, revealing new information about the changing nature of lesion/symptom correlations. In short, the findings are solid enough to justify some speculation about the development of brain organization for language under normal and pathological conditions.

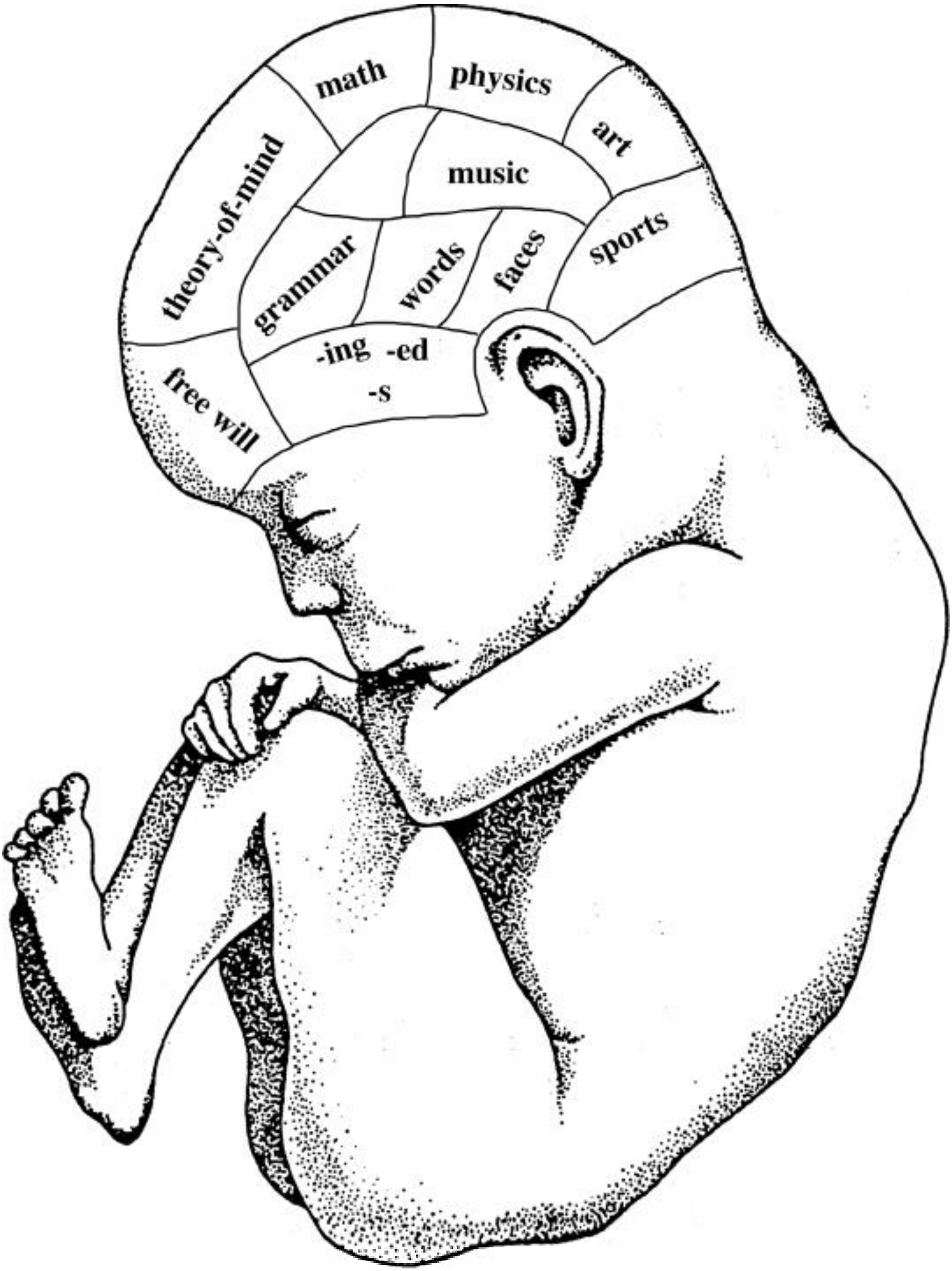
(5) HOW BRAIN ORGANIZATION FOR LANGUAGE EMERGES ACROSS THE COURSE OF DEVELOPMENT

The literature on language outcomes in human children with early unilateral brain injury is quite compatible with the burgeoning literature on neural plasticity in other species. Many of the human results are new, but the information from developmental neurobiology is now well established. Although few neurobiologists would argue in favor of ***equipotentiality,*** i.e., the idea that all areas of cortex are created equal (Lenneberg, 1967), there is now overwhelming evidence in favor of ***pluripotentiality***, i.e., the idea that cortical tissue is capable of taking on a wide array of representations, with varying degrees of success, depending on the timing, nature and extent of the input to which that tissue is exposed (Elman et al., 1996; Johnson, 1997).

This conclusion is well attested in the developmental neurobiology literature, but it has had surprisingly little impact in linguistics, cognitive science and cognitive neuroscience. In fact, the old phrenological approach to brain organization has found new life in the last two decades in various proposals that language is an "instinct" (Pinker, 1994), a "mental organ" (Chomsky, 1980a,b; 1995) or an "innate module" (Fodor, 1983; Pinker, 1997a), with its own neural architecture and its own highly specific genetic base (see also Gopnik, 1990; Pinker, 1991; Rice, 1996; van der Lely, 1994). Indeed, Fodor's 1983 monograph celebrates the contributions of Franz Gall, the original phrenologist, and proudly bears a classical drawing of Gall's subdivided and numbered brain on its cover. The only real surprise is how little the claims have changed across the last two hundred years.

Phrenology in all its reincarnations can be characterized as the belief that the brain is organized into spatially and functionally distinct faculties, each dedicated to and defined by a different kind of intellectual, emotional or moral content. In some of the proposals put forward by Gall, Spurzheim and others in the 18th century, these included areas for hope, combativeness, conjugal love, veneration, cautiousness, calculation, tune, memory, and of course, language. A modern variant of phrenology is represented in cartoon form in Figure 9, which differs from the old version in at least two respects. First, the content of the proposed modules has changed a great deal in the last two centuries: with some exceptions, most of the ethical content is gone (but see Ramachandran, 1997, for a proposed "religiosity module"), replaced by a smaller set of species-specific cognitive and linguistic domains (e.g., music, faces, mathematics, grammar, the lexicon). To be sure, the particular entries and placements in Figure 9 are of my own making, but each one represents explicit claims that have been made in the last 5-10 years in the New York Times and other public outlets. Second, and most important for our purposes here, the modern version of phrenology has a strong nativist component. In contrast with the 19th-century phrenologists (some of whom underscored the role of experience in setting up the functional organiza-tion of the brain -- see especially Wernicke, 1874/ 1977), 20th-century champions like Fodor and Pinker have wedded their theory of modular localization to the doctrine of innateness. In this variant of phrenology, the adult brain is organized along modular lines because the brain came packaged that way, in its fetal form, with specific functions assigned to specific regions by a genetic program (see also Gopnik & Crago, 1991; Rice, 1996; van der Lely, 1994).

In part, the phrenological approach may persist because alternative accounts are difficult to understand. The adult brain is a highly differentiated organ, and the infant brain (though underspecified in comparison to the adult brain) is certainly not a *tabula rasa.* And yet efforts to reintroduce experiential effects on this brain organization (e.g., Bates & Elman, 1996; Elman & Bates, 1997) have been met with great suspicion by those who fear a reintroduction of old behaviorist accounts (Clark, Gleitman, & Kroch, 1997; Jenkins & Maxam, 1997; Pesetsky, Wexler, & Fromkin, 1997; Pinker, 1997b). Some of the heat in this exchange comes from the fact that several logically and empirically distinct issues are conflated in the argument


math
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about mental organs for language. As a result, anyone who opposes the modern doctrine of phrenology in its full-blown form is accused of (gasp!) behaviorism. In order to clarify the difference between old-fashioned *tabula rasa* behaviorism and the emergentist perspective that I am espousing here, we need to break the mental organ doctrine down into a series of separate and separable assumptions about (1) ***innate representa-tions*** (i.e., synaptic connections are determined by a genetic program), (2) ***domain-specific processing*** (each region of the brain is designed to handle a specific kind of content), and three corollaries about localization, (3) ***compact location***, (3) ***fixed location***, and (4) ***universal location***. Table 1 summarizes the five claims of modern phrenology, together with a character-ization of the emergentist alternative on each of these five counts.

Consider first the assumption of innate representations. As my colleagues and I have acknowledged repeatedly, throughout this chapter and elsewhere (Bates, Elman et al., in press; Elman et al., 1996), cortex is not equipotential. There are powerful endogenous constraints in the infant brain that bias the way that brain organization will proceed under normal circumstances. However, claims about the nature of these innate constraints can be made on several different levels: ***innate representations*** (where "representations" are operationally defined as the patterns of cortical connectivity that comprise knowledge), ***innate architecture*** (defined in terms of the global input-output architecture of the brain, and local variations in density, speed and style of information processing), and ***innate timing*** (including variations in length of neurogenesis, and the onset and offset of neurotrophic factors). The mental organ doctrine is deeply committed to the existence of innate representations. The emergentist alternative is committed to the idea that knowledge itself is not innate, but emerges across the course of development, through the interaction of innate architecture, innate timing, and input to the cortex.

In fact, the case for innate representations looks very bad right now. Thirty years ago, representational nativism was a perfectly plausible hypothesis. That is, it was reasonable to suppose that knowledge is built into the infant cortex in the form of detailed and well-specified synaptic connections, independent of and prior to the effects of input to the cortex (what Pinker (1997a) refers to as an innate "wiring diagram"). Indeed, such an assumption is critical for strong forms of linguistic nativism (i.e., the idea that children are born with Universal Grammar -- Chomsky,1980a,b; Pinker,1994; Rice, 1996), because synaptic connectivity is the only level of brain organization with the necessary coding power for complex and domain-specific representations of the sort that would be required to support an innate grammar. However, this particular form of innateness is difficult to defend in the face of mounting information on the activity-dependent nature of synaptic connectivity at the cortical level. Of course the infant brain is certainly not a *tabula rasa.* At other levels of organization, we have ample evidence for endogenous effects that bias the learning game in significant ways. These include constraints on the global input-output architecture of the brain (e.g., the fact that information from the eye usually does end up in visual cortex, in the absence of wicked interventions by Sur and his colleagues), local variations in architecture and style of computation (e.g., primary visual cortex starts out with roughly twice as many neurons as any other area), and variations in timing (e.g., variations from one region to another in the length of neurogenesis, and in the availability of NGF (nerve growth factor). It now seems that the difference between the human brain and that of other primates must be determined primarily by nonrepresentational variations of this kind, controlled by a genetic program small enough to fit into the mere 1-2% difference between the human genome and the genome of a chimpanzee (King & Wilson, 1975; Wilson, 1985).

The second assumption in Table 1, domain-specific processing, is a key component of the mental organ doctrine, i.e., that distinct regions of the brain have evolved to deal with particular kinds of content of compelling interest to our species (Barkow, Cosmides, & Tooby, 1992; Pinker, 1997a). In addition to language (and perhaps to distinct subcomponents of language, e.g., a distinction between grammar and the lexicon), proposed modules or mental organs include a face detector, a theory-of-mind module (that contains algorithms for detecting dishonest behavior by other members of the species), a mathematics module, a music module, and so forth. These systems have presumably evolved to deal optimally with their assigned content, and only with that content. Indeed, Pinker (1997a) has proposed that diverse and specific forms of psychopathology may result if a module is applied to the wrong domain (although it is not entirely clear how this might occur, given the perceptual biases that define a mental organ).

The emergentist alternative to domain-specific processing is that domain-specific knowledge can be acquired and processed by domain-general mechanisms, i.e., by mechanisms of attention, perception, memory, emotion and motor planning that are involved in many different aspects of learning, thought and behavior. In other words, the cognitive machinery that makes us human can be viewed as a new machine constructed out of old parts (Bates, Benigni, Bretherton, Camaioni, & Volterra, 1979). All of the component parts that participate in language are based on phylogenetically ancient mechanisms, with homologues up and down the vertebrate line. The specific functions that make humans different from other species are superimposed on this Basic Vertebrate Brain Plan. Of course it is likely that some and perhaps all of the neural components that participate in human activity have undergone quantitative changes that permit new behaviors like language to emerge, but these components still continue to carry out older and more general functions of object detection, shifting attention, formation of new memories, motor planning, and so forth (i.e., they have kept their day jobs....).

To help us think about the kind of adaptation that would permit the construction of a new machine from

old parts, consider the metaphor of the giraffe's neck. Giraffes have the same number of neckbones that you and I have, but these bones are elongated to solve the peculiar problems that giraffes are specialized for (i.e., eating leaves high up in the tree). As a result of this particular adaptation, other adaptations were necessary as well, including cardiovascular changes (to pump blood all the way up to the giraffe's brain), shortening of the hindlegs relative to the forelegs (to ensure that the giraffe does not topple over), and so on. Should we conclude that the giraffe's neck is a "high-leaf-reaching organ"? Not exactly. The giraffe's neck is still a neck, built out of the same basic blueprint that is used over and over in vertebrates, but with some quantitative adjustments. It still does other kinds of "neck work", just like the work that necks do in less specialized species, but it has some extra potential for reaching up high in the tree that other necks do not provide. If we insist that the neck is a leaf-reaching organ, then we have to include the rest of the giraffe in that category, including the cardiovascular changes, adjustments in leg length, and so on.

In the same vein, our "language organ" can be viewed as the result of quantitative adjustments in neural mechanisms that exist in other mammals, permitting us to walk into a problem space that other animals cannot perceive much less solve. Of course, once language finally appeared on the planet, it is quite likely that it began to apply its own adaptive pressures to the organization of the human brain, just as the leaf-reaching adaptation of the giraffe's neck applied adaptive pressure to other parts of the giraffe. Hence the neural mechanisms that participate in language still do other kinds of work, but they have also grown to meet the language task. In fact, it seems increasingly unlikely that we will ever be in a position to explain human language in terms of clear and well-bounded differences between our brain and that of other primates. Consider, for example, the infamous case of the planum temporale (i.e. the superior gyrus of the temporal lobe reaching back to the temporal-parietal-occipital juncture). It was noted many years ago that the planum temporale is longer on the left side of the brain in the majority of normal, right-handed human adults. Because the temporal lobe clearly does play a special role in language processing, it was argued that the asymmetry of the planum may play a key role in brain organization for language. However, surprising new evidence has just emerged showing that the same asymmetry is also observed in chimpanzees (Hollaway, Broadfield, Kheck, & Braun, 1998). In fact, the asymmetry is actually larger and more consistent in chimpanzees than it is in humans! I don't doubt for a moment that humans use this stretch of tissue in a quantitatively and qualitatively different way, but simple differences in size and shape may not be sufficient or even relevant to the critical difference between us and our nearest relatives in the primate line. In response to findings of this sort, Pinker (1997a) has insisted that the answer lies in the cortical microcircuitry within relevant areas. And yet, as we have seen over and over, developmental neurobiologists have abandoned the idea that detailed aspects of synaptic connectivity are under direct genetic control, in favor of an activity-dependent account. There has to be something special about the human brain that makes language possible, but that "something" may involve highly distributed mechanisms that serve many other functions.

My own favorite candidates for this category of "language-facilitating mechanisms" are capacities that predate language phylogenetically, and undoubtedly involve many different aspects of the brain. They include our rich social organization and capacity for social reasoning, our extraordinary ability to imitate the things that other people do, our excellence in the segmentation of rapid auditory and visual stimuli, and our fascination with joint attention (looking at the same events together, sharing new objects just for the fun of it -- for an extended discussion, see Bates, Thal, & Marchman, 1991). These abilities are all present in human infants within the first year, and they are all implicated in the process by which language is acquired. None of them are specific to language, but they make language possible, just as quantitative adjustments in the giraffe's neck make it possible for the giraffe to accomplish something that no other ungulate can do.

Is there any evidence in favor of this domain-general "borrowed system" view? I would put the matter somewhat differently: Despite myriad predictions that such evidence will be found, there is still no unambiguous evidence in favor of the idea that specific parts of the brain are dedicated to specific kinds of objects, and *only* those objects. For example, there are cells in the brain of the adult primate that respond preferentially to a particular class of stimuli (e.g. faces). However, recent studies have shown that the same cells can also respond to other kinds of content, spontaneously and/or after an extended period of training (Das & Gilbert, 1995; De Weerd, Gattass, Desimone, & Ungerleider, 1995; Fregnac, Bringuier, & Chavane, 1996; Pettet & Gilbert, 1992; Ramachandran & Gregory, 1991; Tovee, Rolls, & Ramachandran (1996). Similarly, certain cortical regions around the sylvian fissure are invariably active in neural imaging studies of language processing, including some of the same areas that are implicated in fluent and nonfluent aphasia. However, each of these regions can also be activated by one or more forms of nonlinguistic processing. This point was made eloquently clear in a recent study by Erhard, Kato, Strick and Ugurbil (1996), who looked at all the proposed subcomponents of Broca's area while subjects were asked to carry out (covertly) a series of verbal and nonverbal actions, including complex movements of the mouth and fingers. Every single component of the Broca complex that is active during speech is also active in at least one form of covert nonverbal activity. In short, even though there is ample evidence for stretches of tissue that participate in language, there appears to be no candidate anywhere in perisylvian cortex for a pure language organ.

This brings us to three key assumptions about the nature of localization, the final three of the five contrasting issues listed in Table 1. On the phrenological account, precisely because of the assumptions

about (1) innate representations and (2) dedicated architecture, it is further assumed that brain organization for language involves (3) a fixed architecture that cannot be replaced and cannot be modified significantly by experience, (4) a universal architecture that admits to very little individual variability, and (5) a compact and spatially contiguous architecture that operates as a coherent and autonomous unit in neural imaging studies, and creates distinct deficits in or dissociations between cognitive functions when it is lesioned ("disconnection syndromes" -- Caramazza, 1986; Caramazza & Berndt, 1985; Geschwind, 1965; Shallice, 1988). By contrast, the emergentist account is more compatible with forms of localization that are (3) plastic and modifiable by experience, (4) variable in form as a result of variations in experience as well as individual differences in the initial architecture, and (5) distributed across stretches of tissue that may participate in many different tasks (including spatially discontinuous systems that can perform separately or together depending on the task). Because of these properties, the emergentist view is much more compatible with all the mounting evidence from developmental neurobiology for the plasticity and activity dependence of cortical specialization, including plasticity for language in brain-injured children.

The emergentist view is also more compatible with the complex and variable findings that have emerged in recent neural imaging studies of normal adults (Courtney & Ungerleider, 1997; Poeppel, 1996). Indeed, new areas for language are multiplying at an alarming rate in language activation studies, including studies using positron emission tomography (PET), functional magnetic resonance imaging (fMRI), magnetoencephalography (MEG) and/or event-related brain potentials (ERP). Although activation is usually larger on the left than it is on the right in language activation studies, and the familiar perisylvian regions of the left hemisphere show up in study after study, there is increasing evidence for participation of homologous regions in the right hemisphere (e.g., Just et al., 1996), although there is substantial variation over individuals, tasks and laboratories in the extent to which this occurs. Language activation studies that involve generation and maintenance of codes and/or a decision between behavioral options seem to result in reliable activation of several different prefrontal regions that were not implicated in older studies of language breakdown in aphasia (e.g., Raichle et al., 1994**;** Thompson-Schill, D'Esposito, Aguirre, & Farah, 1997). New regions that appear to be especially active during language activation have also appeared in basal temporal cortex (on the underside of the brain -- Nobre, Allison, & McCarthy, 1994), in some portions of the basal ganglia, and in the cerebellum (especially on the right side of the cerebellum). Many different aspects of both sensory and motor cortex seem to be activated in language tasks that involve imageable stimuli. More interesting still for our purposes here, these patterns of activation vary as a function of development itself, including variations with chronological age and language level in children (Hirsch et al., 1997; Mills, Coffey-Corina, & Neville, 1997; Mueller, 1996), and varying levels of expertise in adults (Hernandez, Martinez, Wong, Frank, & Buxton, 1997; Kim, Relkin, Lee, & Hirsch, 1997; Perani et al., 1997; Raichle et al., 1994).

The picture that has emerged is one in which most of the brain participates in linguistic activity, in varying degrees, depending on the nature of the task and the individual's expertise in that task. In many respects, this is exactly what we should expect: Language is a system for encoding meaning, and there are now good reasons to believe that the activation of meaning involves activation of the same regions that participate in the original experiences on which meanings are based. Because most of the brain participates in meaning, we should expect widely distributed and dynamically shifting patterns of participation in most language-based tasks. The fact that these patterns of activation change over time is also not surprising, reflecting changes in experience as well as changes in the level of skill that individuals attain in activation and maintenance of both meaning and form.

Clearly, however, there are some important differences in the view of language organization that emerges from neural imaging studies and lesion studies. Neural imaging techniques can tell us about the areas of the brain that *participate* in language. From this point of view, we may conclude that the participation is very broad. Lesion studies can tell us about the areas of the brain that are *necessary* for normal language. The list of areas that are necessary for language (in children or adults) appears to be much smaller than the list of areas that participate freely in a language task. Even in this case, however, improved techniques for structural imaging and lesion reconstruction have yielded more and more evidence for individual variability in lesion/symptom mapping (Goodglass, 1993; Willmes & Poeck, 1993), and for compensatory organization in patients who display full or partial recovery from aphasia (Cappa et al., 1997; Cappa & Vallar, 1992).

There are of course some clear limits on this variability. Some areas of the brain simply cannot be replaced, in children or adults. For example, Bachevalier and Mishkin (1993) have shown that infant monkeys with bilateral lesions to the medial temporal regions (including the amygdala and the hippocampus) display a dense and apparently irreversible form of amnesia that persists for the rest of the animal's life, in marked contrast to the striking recovery that follows bilateral lesions to lateral temporal cortex (Webster et al., 1995). The key lies in the global input-output architecture of those medial temporal regions, a rich and broad form of connectivity that cannot be replaced because no other candidate has that kind of communication with the rest of the cortex. Other parts of the brain cannot be replaced because they are the crucial highways and offramps for information from the periphery (e.g., the insula, which receives crucial kinaesthetic feedback from the oral articulators, or the auditory nerve, which carries irreplaceable auditory input to the waiting cortex -- Dronkers, 1996; Dronkers, Redfern, & Ludy, in press; Dronkers, Wilkins, van

Valin, Redfern, & Jaeger, 1994). These irreplaceable regions form the anchor points, the universal starting points for brain organization in normal children, and they are difficult if not impossible to replace once all the exuberant axons of the fetal brain have been eliminated.

Within this framework, learning itself also places limits on plasticity and reorganization in the developing brain. For example, Marchman (1993) has shown that artificial neural networks engaged in a language-learning task (i.e., acquiring the past tense of English verbs) can recover from "lesions" (i.e., random removal of connections) that are imposed early in the learning process. The same lesions result in a substantially greater "language deficit" when they are imposed later in the learning process. This simulation of so-called critical period effects takes place in the absence of any extraneous change in the learning potential of the network (i.e., there is no equivalent of withdrawal of neurotrophins or reduction in the learning rate). Marchman reminds us that critical period effects can be explained in at least two ways (and these are not mutually exclusive): exogenously imposed changes in learning capacity (the usual interpretation of critical periods), or the entrenchment that results from learning itself. In other words, learning changes the nature of the brain, eliminates some connections and tunes others to values that are difficult to change. Eventually the system may reach a point of no return, a reduction in plasticity that mimics critical period effects without any change in the architecture other than the changes that result from normal processes of learning and development. Marchman does not deny the possibility of exogenous effects on plasticity, but she argues convincingly that there are other ways to explain the same result, including gradual changes in the capacity to learn (and recover what was learned before) that are the product of learning itself -- change that are more compatible with the current developmental evidence than the notion of an abrupt and discontinuous critical period (see also Bates & Carnevale, 1993; Elman et al., 1996, Chapter 4).

Finally, the emergentist view makes room for the possibility of systematic developmental changes in localization, due to a shift in the processes and operations that are required to carry out a function at different points in the learning process. On the static phrenology view, a language area is a language area, always and forever. There may be developmental changes that are due to maturation (i.e., an area that was not "ready" before suddenly "comes on line"), but the processes involved in that content domain are always carried out in the same dedicated regions. On the emergentist account, the areas responsible for learning may be totally different from the areas involved in maintenance and use of the same function in its mature form. In fact, there are at least three reasons why we should expect differences in the patterns of brain activity associated with language processing in children vs. adults.

(1) Early competition. We may assume (based on ample evidence from animal models) that the early stages of development involve a competition among areas for control over tasks. This competition is open to any region that can receive and process the relevant information, but that does not mean that every region has an even chance of winning. In fact, as the competition proceeds, those regions that are better equipped to deal with that task (because of differences in efficiency of access and type of processing) will gradually take more responsibility for the mediation of that function. In prospective studies of language development, we are looking at this process of competition as it unfolds. This leads to the prediction that the earlier stages of development will involve more diffuse forms of processing, a prediction that is borne out by ERP studies of changes in activation across the first three years of language development (from activation to known words that is bilateral but slightly larger in the right, towards activation that is larger on the left and localized more focally to fronto-temporal sites -- Mills et al., 1997).

(2) Expertise. We may also expect quantitative and qualitative change in the regions that participate in a given task as a function of level of expertise. These changes can take three different forms: expansion within regions, retraction within regions, and a wholesale shift in mediation from one region to another. An example of expansion comes from a recent fMRI study of skill acquisition in adults (Karni et al., 1995). In this study, the first stages of learning in a finger-movement task tend to involve smaller patches of somatosensory cortex; with increased skill in this task, the areas responsible for the motor pattern increase in size. Examples of retraction come from studies that show larger areas of activation in the early stages of second-language learning compared with activation in native speakers and in more experienced second-language learners (Hernandez et al. 1997; Perani et al., 1997). Presumably this is because the novice speaker has to recruit more neural resources to achieve a goal that was far easier for a more advanced speaker (equivalent to the amount of muscle a child vs. an adult must use to lift a heavy box). The third possibility may be the most interesting, and the one with greatest significance for our focal injury results. In the earliest stages, areas involved in attention, perceptual analysis and formation of new memories may be particularly important. As the task becomes better learned and more automatic, the baton may pass to regions that are responsible for the reactivation of over-learned patterns, with less attention and less perceptual analysis. A recent example of this kind of qualitative shift is reported by Raichle et al. (1994), who observed strong fronto-cerebellar activation in the early stages of learning, replaced by activation in perisylvian cortex after the task is mastered.

(3) Maturation and "readiness". Finally, the emergentist approach does not preclude the possibility of maturational change. Examples might include differential growth gradients for the right vs. left hemisphere (Chiron et al., 1997), differential rates of synaptogenesis ("synaptic sprouting") from from one region to another within the two hemispheres (Huttenlocher, de Courten, Garey, & van der Loos,

1982), changes from region to region in the overall amount of neural activity (as indexed by positron emission tomography -- Chugani, Phelps, & Mazziotta, 1987), variation in rates of myelination, and so forth. As a result of changes of this kind (together with the effects of learning itself in reshaping the brain -- Marchman, 1993), we should expect to find marked shifts in the patterns of activity associated with language processing at different points in early childhood.

Based on these assumptions, let us return to our findings on the early stages of language development in children with early focal brain injury, to see what these results suggest about the emergence of brain organization for language in normal children.

(1) Right-hemisphere advantage for word comprehension and gesture from 10-17 months. Contrary to expectations based on the adult aphasia literature, we found evidence for greater delays in word comprehension and gesture in children with RHD. This is exactly the opposite of the pattern observed in adults, where deficits in word comprehension and in production of symbolic gesture are both associated with LHD, suggesting that some kind of shift takes place between infancy and adulthood, with control over these two skills passed from the right hemisphere to the left. This result is (as we noted) compatible with observations by Mills et al. on the patterns of activation observed in response to familiar words from infancy to adulthood. There are at least two possible explanations for a developmental change, and they are not mutually exclusive.

On the one hand, the early RH advantage could be explained by hard maturational changes that are exogenous to the learning process itself. For example, Chiron and his colleagues have provided evidence from positron emission tomography for a change in resting-state activation across the first two years, from bilateral activation that is larger on the right to greater activation on the left. Based on these findings, they suggest that the right hemisphere may mature faster than the left in the first year of development. As it turns out, this is the period in which word comprehension and gesture first emerge in normally developing children. By contrast, word production emerges in the second year, and grows dramatically through 30-36 months, the period in which (according to Chiron et al.) the left hemisphere reaches the dominant state that it will maintain for years to come. Hence one might argue that the right hemisphere "grabs" control over comprehension and gesture in the first year, the left hemisphere "grabs" control over the burgeoning capacity for production in the second year, and eventually takes over the entire linguistic-symbolic system (including word comprehension and meaningful gestures).

On the other hand, it is also possible that the right-to-left shift implied by our data reflects a qualitative difference between the learning processes required for comprehension and the processes required for production. The first time that we figure out the meaning of a word (e.g., decoding the word "dog" and mapping it onto a particular class of animals), we do so by integrating the phonetic input with information from many different sources, including visual, tactile and auditory context ("fuzzy brown thing that moves and barks"). It has been argued that the right hemisphere plays a privileged role in multi-modal integration and processing of large patterns (Stiles, 1995 -- more on this below), and for this reason, we may expect the right hemisphere to play a more important role when children are learning to comprehend words for the first time. Presumably, this RH advantage will disappear when words are fully acquired, replaced by a rapid, efficient and automatic process of mapping well-known sounds onto well-known semantic patterns (more on this below). If this hypothesis has merit, then we might also expect to find evidence for greater participation of the right hemisphere in the early stages of second-language learning in adults, a testable hypothesis and one that has some (limited) support.

It is much less obvious how this shift-in-strategy hypothesis might account for the early RH advantage in symbolic gesture. Although this is admittedly a speculative answer, this finding may be related to results for normal children showing that comprehension and gesture are highly correlated between approximately 9-20 months of age (Fenson et al., 1994). One possible explanation for this correlation may lie in the fact that symbolic gestures are acquired in the context of auditory comprehension (e.g., "Wave bye-bye to grandma," "Hug the baby!"). Hence the two skills may come in together in very small children because they are acquired together in real life.

(2) Deficits in expressive vocabulary and grammar with frontal lesions to either hemisphere from 19-31 months. We observed specific effects of lesions involving the frontal lobes in children between 19-31 months of age, a brief but dramatic period of development that includes the vocabulary burst and the first flowering of grammar. Contrary to expectations based on the adult aphasia literature, the delays in expressive language associated with frontal lesions were symmetrical, i.e., there was no difference between frontal lesions on the left and frontal lesions on the right. There are a number of reasons why we would expect to find specific effects of frontal involvement during this important period in the development of expressive language, including contributions to the planning and execution of motor patterns, and contributions from working memory and/or the fashionable array of skills referred to by the term "executive function" (Pennington & Ozonoff, 1996). However, the absence of a left-right asymmetry is more surprising. Nor have we found any evidence for a specific effect of left frontal injury in any of our studies to date, at any age. This difference between infants and adults suggests to us that Broca's area is not innately specialized for language. It becomes specialized across the course of development, after an initial period in which frontal cortex makes a symmetrical contribution to language learning.

(3) Deficits in expressive vocabulary and grammar with left temporal injuries from 10 months to five years of age. This is the most robust and protracted finding in our prospective studies, and it is the only evidence we have for an asymmetry that might be systematically related to a left-hemisphere advantage for language in the adult brain. Note, however, that the effect only pertains to *expressive* language (contrary to the expectation that temporal cortex is specialized for comprehension), and it applies equally to *both* vocabulary and grammar (contrary to the expectation that temporal cortex is associated with semantics while frontal cortex handles grammar -- Zurif, 1980).

We have proposed that a relatively simple bias in style of computation may underlie this left temporal effect, reflecting architectural differences between left and right temporal cortex that are only indirectly related to the functional and representational specializations that are evident in adult language processing. Following a proposal by Stiles and Thal (1993), we note that left and right temporal cortex differ at birth in their capacity to support perceptual detail (enhanced on the left) and perceptual integration (enhanced on the right -- see above). These differences are evident in nonverbal processing, but they may have particularly important consequences for language. For example, a number of recent studies have shown that lesions to the right hemisphere lead to problems in the integration of elements in a perceptual array, while lesions to the left hemisphere create problems in the analysis of perceptual details in the same array (e.g., Robertson & Lamb, 1991). Asked to reproduce a triangle made up of many small squares, adult patients with left-hemisphere damage tend to reproduce the global figure (i.e., the triangle) while ignoring information at the local level. Adult patients with right-hemisphere damage display the opposite profile, reproducing local detail (i.e., a host of small squares) but failing to integrate these features into a coherent whole. Stiles and Thal report that children with focal brain injury behave very much like their adult counterparts on the local-global task, suggesting that the differential contribution of left- and right-hemisphere processes on this task may be a developmental constant. Interestingly, this double dissociation is most evident in patients with temporal involvement, and the special role of left temporal cortex in processing of perceputal details has also been confirmed in an fMRI study of normal adults engaged in the same local-global task (Martinez et al., 1997).

The same left-right difference may be responsible for the lesion/symptom correlations that we observe in early language development. As I noted earlier, the ability to integrate information within and across modalities may be particularly helpful and important during the first stages of word comprehension and (perhaps) recognition and reproduction of familiar gestures. However, the learning task changes markedly when children have to convert the same sound patterns into motor output. At this point, perceptual detail may be of paramount importance (i.e., it is one thing to recognize the word "dog", but quite another thing to pull out each phonetic detail and construct a motor template). If it is the case that left temporal cortex plays a critical role in the extraction, storage and reproduction of perceptual detail (visual and/or acoustic), then children with left temporal injuries will be at a greater disadvantage in this phase of learning (see also Galaburda & Livingstone, 1993; Galaburda, Menard, & Rosen, 1994; Tallal, Sainburg, & Jernigan, 1991). However, once the requisite patterns are finally constructed and set into well-learned routines, the left temporal disadvantage may be much less evident.

(4) No evidence of lesion/symptom correlations after 5-7 years of age. All of the above lesion/symptom mappings seem to have disappeared when we test children with the same congenital etiology after 5-7 years of age. Although this conclusion is based primarily on cross-sectional findings, the few cases that we have been able to study longitudinally across these periods of development are compatible with the cross-sectional results, providing further evidence for plasticity and compensatory organization across the course of language development. Of course it is entirely possible that we will find a new and improved index of efficiency in language processing that yields information about the subtle deficits that remain, e.g., a residual effect of left temporal involvement that shows up in real-time sentence processing and/or in production of complex syntax under certain laboratory conditions. At the very least, however, we may conclude with some confidence that these children have found a form of brain organization for language that works very well, certainly well enough for everyday language use. As a group, children with focal brain injury do tend to perform below neurologically intact age-matched controls. But these differences also tend to disappear when the small group difference in full-scale IQ is taken into account (Bates, Vicari, & Trauner, in press).

If the familiar pattern of left-hemisphere organization for language is not critical for normal language functioning, why does it develop in the first place? To answer this question, we have put forth a "modest proposal" based on the developmental findings and developmental principles listed above, as follows.

Prior to the onset of language development, the infant brain has no innate representations for language, nor does it have a "dedicated language processor" of any kind. However, the initial (prelinguistic) architecture of the infant brain is highly differentiated. The global input-output structure of the brain is well specified (e.g., the retina reports to visual cortex, the cochlea reports to auditory cortex), although there may still be a number of exuberant axons that could (if they are not eliminated in the normal course of development) sustain an alternative form of global architecture if they are needed. There are also innate (experience-independent) variations from region to region in cell density, synaptic density, speed of processing, and the kinds of neurotransmitters that are expressed (Hutsler & Gazzaniga, 1996). Furthermore, even though the infant has little experience in the world, the infant cortex has been inundated with information from the body itself.

As Damasio has noted (Damasio, 1994), the brain is the captive audience of the body, and the body provides the earliest and most reliable input that the growing cortex will ever receive. This includes sensory impressions from the body surface, kinaesthetic feedback from the infant's own movements, and reliable waves of activity from lower brain centers (e.g., bilateral and competitive input from lower level visual nuclei that, we now know, is critical for the establishment of ocular dominance patterns -- Miller, Keller, & Stryker, 1989; Shatz, 1992). Hence, even though there may be no direct genetic control over synaptic connectivity at the cortical level, the newborn infant starts life with a brain that has been colonized by sensorimotor input from its own body, setting down the basic parameters within which all the rest of behavioral development must take place. These facts combine with the regional differences in cortical architecture described above, setting the stage for the post-natal development of cognition and communication, including the development of grammar (MacWhinney, 1999).

As a result of all these forces, the infant comes to the task of language learning with a heavy set of biases about how information should be processed. Some of these biases are symmetrical (e.g., the role of frontal cortex in control of voluntary movements), others are asymmetrical (e.g., the local/global biases described above). Following early focal brain injury, these biases show up in the early lesion/symptom mappings that we have described above, but they are eventually overcome by the competitive pressures that define plasticity and development in both the normal and the abnormal case. However, in healthy children without focal brain injury, these biases shape the development of brain organization for language in some highly predictable directions. In particular, left temporal cortex comes to play an increasingly important role in the extraction of the rapid and evanescent linguistic signal -- first in the construction of motor templates to match slow and dependable inputs, later in the construction of complex meanings for both comprehension and production (events that we would expect to see in both signed and spoken language -- Petitto et al., 1997). In short, under normal conditions (i.e., in the absence of focal brain injury), left temporal cortex wins the language contract. Although there is no asymmetric bias in favor of left frontal cortex in the early stages of development, the left temporal "winner" recruits its partners in the front of the brain, setting up the familiar ipsilateral circuit that characterizes left-hemisphere mediation of language in neurologically intact adults. At this point (and not before), Broca's area has a special job.

This is our proposal for the cascade of events that are responsible for the patterns of brain organization for language that lie behind two hundred years of research on adult aphasia, and hundreds (going on thousands) of neural imaging studies of language activation in normal adults. No doubt this proposal will have to undergo considerable revision as more information becomes available, but we are convinced that the final story will have to be one in which development and experience play a crucial role. Plasticity is not a civil defense system, a set of emergency procedures that are only invoked when something goes wrong. Rather, the processes responsible for reorganization of the brain following early focal brain injury are the same processes that organize the brain under normal conditions. It is time to exorcise the ghost of Franz Gall, trading in the static phrenological view of brain organization for a dynamic approach that reconciles linguistics and cognitive science with developmental neurobiology.

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