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# *Linguistic and Spatial Cognitive Development in Children with Pre- and Perinatal Focal Brain Injury: A Ten-Year Overview from the San Diego Longitudinal Project*

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For the past ten years a group of investigators based in San Diego has been studying the effects of pre- and perinatal focal brain injury on the development of linguistic and spatial cognitive functions. When this project began, even the most basic question concerning whether or not it is possible to identify specific deficits associated with early injury was still a subject of debate. Early studies on the effects of focal brain injury emphasized the "resilience" of young children to the effects of early injury and argued that early available mechanisms subserving a transient capacity for plastic change allow children with early injury to develop normal or near-normal cognitive functioning following injuries to the brain that would leave an adult permanently impaired (Alajouanine and Lhermitte, 1965; Brown and Jaffe, 1975; Carlson, Netley, Hendrick, and Pritchard, 1968; Gott, 1973; Hammill and Irwin, 1966; Krashen, 1973; Lenneberg, 1967; McFie, 1961; Reed and Reitan, 1971). These arguments did not, however, go unchallenged. Other investigators argued that a more fine-grained analysis of behavior showed evidence of persistent cognitive deficit (Day and Ulatowska, 1979; Dennis, 1980; Dennis and Kohn, 1975; Dennis and Whitaker, 1976; Kohn, 1980; Kohn and Dennis, 1974; Rudel and Teuber, 1971; Vargha-Khadem et al., 1983, 1985; Woods, 1980; Woods and Carey, 1979). These apparently contradictory sets of claims suggested a complex interplay between plasticity and specialization of function in the developing brain, but the nature and course of that interaction remained unclear. One limitation of that early work on both sides of the debate was its reliance on retrospective accounts of development in which the outcome of development following early injury is used to infer developmental process.

In order to understand the long-term effects of early neurological insult, it is necessary to investigate processes of recovery and/or compensation as they occur. A prospective approach to the study of development following early injury makes it possible to determine:

(1) whether there is *early* evidence of impairment; (2) whether the profile of impairment in early childhood is the same as or different from that observed in adults with similar injury; and (3) whether there is change in the profile over time.

A small number of investigators have adopted this approach to the study of children with focal brain injury. For example, Aram and her colleagues (Aram and Ekelman, 1986; Aram et al., 1983; 1985; Rankin et al., 1981) have reported data from cross-sectional studies of children under 5 years of age, providing evidence for global linguistic and cognitive deficits in children with early acquired focal brain injury. Longitudinal follow-ups of these children in the school-age period suggest that these early deficits persist with development (Aram, 1988; Aram and Ekelman, 1988; Aram et al., 1985). These studies are important because they provide strong evidence that early focal brain injury does result in significant functional deficits. However, studies by Aram and other investigators also show that the deficits associated with early brain injury are often quite subtle and may require finer-grained measures to be detected. For example, Riva, Cazzaniga, Pantaleoni, Milani, and Fedrizzi (1987) have uncovered grammatical deficits on the Token Test in children with left hemisphere damage (LHD) that are only evident when that test is reanalyzed to extract specific syntactic patterns (see also Dennis and Whitaker, 1976). Similar profiles of early, subtle deficit have been reported for children with frontal lobe injury. Eslinger and his colleagues (Eslinger and Grattan, 1991) report that among younger children the effects of injury to frontal lobe regions may be quite mild. However, they note that with development, patterns of deficit become more pronounced. They suggest that this late emerging pattern may reflect the fact that demands for behaviors mediated by the frontal lobes may become more pronounced as children reach adolescence, thus suggesting a kind of latent deficit profile. This pattern of late emerging deficit has been reported by Levine and her colleagues (Levine, 1993) on measures of IQ. They report a systematic decline in IQ scores beginning in early adolescence. This profile is not confined to children with frontal lobe injury, rather it appears to hold for the focal lesion population as a whole.

One of the most intriguing findings from this longitudinal investigation of the effects of early brain injury on development is that studies of language and visuospatial processing provide different answers to the questions of initial deficit, of mapping to adult profiles of deficit, and of change over time. The differences in these two domain-specific profiles are striking enough that they might have led an individual investigator working in isolation to posit very different theoretical accounts of developmental change following early brain injury. Yet within the context of this project, we are confronted with the fact that the data have been obtained from the same children. One challenge of this work will be the need to reconcile these differences, and provide a single account of development following early focal brain injury that encompasses what is becoming a diverse and challenging set of findings.

## The Population of Children with Early Focal Brain Injury

The studies reported here focus on a group of children with early occurring focal brain injury. The children included in these studies were selected on the basis of the presence of a single, unilateral brain lesion that was acquired prior to, or at birth. Location and size of the lesions were ascertained by obtaining neuro-imaging procedures (MRI or CT scans) on every subject. Individuals were excluded if there was evidence of multi-focal or diffuse brain damage, or if there was evidence of intrauterine drug exposure. Most of the children

were born full-term. Finally, on gross assessment, the children in the population do well behaviorally, both individually and as a group. They do not manifest gross cognitive deficits. In fact, they typically score within the normal range on standardized IQ measures, and attend public schools.

## Language

We based our first round of hypotheses on a handful of relatively uncontroversial claims about adult aphasia that appear in virtually every neurology textbook. For example, there is general agreement that the left hemisphere (LH) is specialized for language in most normal adults (Bryden, 1982; Damasio and Damasio, 1992; Galaburda et al., 1994; Gazzanaga, 1994; Hellige, 1993). It is also generally believed that the perisylvian regions of the LH are especially important for phonological, lexical and grammatical functions (Damasio, 1989; Damasio and Damasio, 1992; Geschwind, 1972; Rasmussen and Milner, 1977). Furthermore, anterior versus posterior damage along the left Sylvian fissure is reliably correlated with the syndromes described by Broca and Wernicke, respectively (Damasio and Damasio, 1992; Goodglass, 1993; Naeser, Helm-Estabrooks, Haas, Aurbach, and Levine, 1984).

More recent research suggests that the right hemisphere (RH) also plays a role in language processing, complementing the functions mediated by the left. Studies have shown that RH injuries have specific effects on the comprehension and production of humor (Brownell, Michel, Powelson, and Gardner, 1983), metaphors (Brownell, Simpson, Birhle, Potter, and Gardner, 1990), and idioms (VanLancker and Kempler, 1986). They also may create problems with cohesion and coherence in narratives (Garner, 1983; Hough, 1990; Joanette, Goulet, and Hannequin, 1990; Kaplan, Brownell, Jacobs, and Gardner, 1990).

Extrapolating from these studies, there was ample reason to hypothesize that the LH, particularly the perisylvian area, may be innately specialized for acquisition of core aspects of language, whereas the RH might come into play as children begin to use language for a variety of discourse functions. We predicted that: (1) children with LHD would develop more severe language impairments than those with right hemisphere damage (RHD) (left-specialization hypothesis); (2) children with damage to anterior regions of the LH, especially the perisylvian area, would develop more severe language production deficits (Broca hypothesis); (3) children with damage to posterior regions of the LH, particularly the posterior portion of the left temporal lobe, would develop more severe language comprehension deficits (Wernicke hypothesis); and (4) children with RHD would demonstrate problems in telling a story or using language to make inferences.

In the sections that follow, we will describe four studies that span the range from 10 months to 12 years of age. The first three studies describe a group of children that was identified prior to the onset of measurable language skills. They were tested longitudinally (in cross-sectional studies) from prespeech through the period when typically developing children begin to regularly use grammatical sentences. The fourth study describes the acquisition of more complex syntax and narrative skills in children from 3 to 12 years.

*Babbling and first words.* The first study was a multiple case study of five infants with focal brain injury who were compared to ten typically developing children matched for level of language development at three data points (Marchman, Miller, and Bates, 1991). Language was measured with the MacArthur Communicative Development Inventory (CDI) (Fenson, Dale, Reznick, Thal, Bates, Hartung, Pethick, and Reilly, 1993) and a

30-minute spontaneous communication sample. Four of the children had LH lesions and one had a RH lesion. Two of the LH lesions were posterior. All of the other lesions were anterior.

All children with focal brain injury were delayed in gesture and word production at all three data points on the CDI. For the children with anterior lesions, however, word production began to move into the normal range at the third data point. The two children with left posterior (LP) lesions, on the other hand, remained below the 5th percentile. The anterior–posterior differences also appeared in the spontaneous communication samples. As a group, the children with focal brain injury did not differ from their typically developing peers on the number or length of vocalizations produced. However, their phonological development paralleled their lexical development: children with focal brain injury produced fewer "true" consonants and a smaller proportion of labial consonants than the age matched normal control children. By the third data point, however, children with anterior lesions had begun to use "true" consonants as frequently as the controls.

These results provided evidence that children with focal brain injury are indeed impaired in the early stages of language acquisition. The profiles of impairment also were different from those of adults with similar lesions, since children with anterior lesions (left and right) began to move into the normal range for word production by 21–22 months of age while those with LP lesions remained significantly delayed.

### *Early lexical development*

Thal et al. (1991) used the CDI to extend these findings to 27 children with focal brain injury who were between 12 and 35 months of age. Results reinforced those of Marchman et al. (1991), indicating delays for the group as a whole in vocabulary comprehension (for the period over which it was measured) and production (throughout the full 12 to 35 month range).

Results also provided evidence that relationships between behavioral profiles and lesion site during development are not the same as those seen in adults. First, significant delays in vocabulary comprehension were found only in children with RHD. Second, particularly severe *expressive* delays were seen in children with LP lesions. Children without LP, on the other hand, moved into normal range. Thus, LP lesions appear to be associated with significant delays in *expressive* language, a pattern that only partially maps onto the profile of adults with focal brain injury.

### *From first words to grammar*

Bates, Thal, Trauner, Fenson, Aram, Eisele, and Nass (1997), further extended these findings in 3 additional studies. In Study 1, children with LHD and RHD were compared using percentile scores on the CDI. Binomial tests indicated that more children than expected by chance were delayed in comprehension and word production and that more children with RHD (but *not* LHD) than expected by chance fell into the delayed range (below the 10th percentile). Even more surprising, *none* of the children with lesions involving the left temporal cortex (+LT), the presumed site of Wernicke's area, were in the risk range for word comprehension. Thus, in the age range from 10–17 months, there is weak evidence for RH specialization for language comprehension and clear disconfirmation

of the Wernicke hypothesis. This is compatible with comprehension results for older children (Eisele and Aram, 1994; Trauner et al., 1996) as well as the earlier study reported by the San Diego group (Thal et al., 1991). Similar analyses of gesture production indicated no significant delays associated with LH or +LT, but support for RH disadvantage in gesture production, comparable to the findings for comprehension.

There was neither a significant left–right difference nor evidence for different effects for +LT on word production percentile scores. Note, however, that this null result is confounded by the surprising finding that comprehension deficits were greater in the RH. Hence it was important to control for the number of words that a child *understands* in order to assess whether there are site-specific effects on *the ability to produce those words*. To control for the confound, Bates et al. examined the proportion of receptive vocabulary that was produced. This analysis did yield a significant disadvantage for +LT. In other words, a LH disadvantage for expressive language was present in 10- to 17-month-old children with focal brain injury when differences in word comprehension were controlled. The fact that this LH disadvantage comes primarily from children with damage involving the left temporal lobe provides yet another challenge to the Wernicke hypothesis.

In Study 2, 19- to 31-month-old children with focal brain injury were compared on percentile scores for word production and two measures of early grammar: mean length in morphemes of the three longest utterances reported by parents (M3L), and the proportion of total vocabulary comprising grammatical function words. These comparisons produced a number of surprises. First, a significant number of children continued to be at risk for delays in expressive language. There were no significant differences overall between LH and RH for word production or grammar, but children with +LT were at a greater disadvantage for both vocabulary and M3L. The left temporal disadvantage appeared to be even stronger when there was also damage to the left frontal lobe, a finding that is compatible with hypotheses based on the adult literature. However, delays were equally serious for children with *right* frontal lobe damage, suggesting that frontal effects are symmetrical during this period of development. There were significant differences between LH and RH on the proportion of grammatical function words in their vocabulary, with LH lower than right, but this reflected a *right hemisphere advantage* rather than a left hemisphere disadvantage.

Finally, in Study 3, free speech samples of 20- to 44-month-old children with focal brain injury were compared on a general measure of grammatical complexity (mean length of utterance [MLU] in morphemes). As a group, subjects were about 4 months behind normal controls on MLU: about 52 percent fell in the lowest 10 percent for their age and LH/RH comparisons did not reach significance. However +LT were significantly lower than children without left temporal damage (−LT). Only 31 percent of the −LT sample fell into the lowest 10 percent for their age while 85 percent of the +LT sample did so (significant by a likelihood ratio, $p < .002$). These results contradict the Wernicke hypothesis, and extend the left temporal findings reported above. However, in contrast with Study 2, frontal involvement did not increase the risk for expressive language delays in this analysis, providing little evidence for the Broca hypothesis.

The series of studies by Bates et al. (1997) does not follow the pattern expected based on lesion site–symptom correlations in adults with focal brain injury. This contradiction may reflect the very different task demands that confront infants and adults in the language domain. Infants and toddlers are learning language *for the first time*, whereas the task for adults is to use that knowledge for fluent and efficient communication. It appears that in the acquisition and development of the linguistic system, children draw on a broader array

of brain structures. However, the Bates et al. (1997) results do provide evidence that the left temporal lobe is of major importance to the emergence of LH specialization for language under normal conditions.

## *Discourse and grammar from 3–12 years*

Our study of older children is based on narratives elicited from 31 children with focal brain injury (13 with RHD and 18 with LHD) between 3 years, 6 months (3,6) and 9 years, 6 months (9,6) and age and gender matched controls (Reilly, Bates, and Marchman, 1998). In our narrative task, the children were asked to look through a wordless picture book, *Frog, where are you?* (Mayer, 1969), and then, while looking at the book, to tell the story to an adult. Our analyses focused on microstructures (lexical types and tokens, morphology and syntax) and macrostructures (narrative components and theme).

Children with focal brain injury produced shorter stories overall than the controls, and they included fewer story components. Their stories contained a smaller number of prepositions, fewer word types and fewer word tokens. There were developmental changes in lexical output that included an increase in both the use and range of evaluative terms, and an increased use of pronouns that are co-referential with a noun in the same sentence, but no clear effects of lesion group. By age 5, the children with focal brain injury were comparable to the controls in vocabulary production during a story-telling task.

Morphological development continued to lag behind in the children with focal brain injury. By age 5, the controls made very few morphological errors; the children with focal brain injury did not reach the same level of proficiency until seven. Similar to our findings for the emergent stages of language development above, this selective disadvantage in the acquisition of morphology before 7 was primarily in +LT.

Syntax was analyzed from two perspectives: frequency of complex sentences and diversity of complex structures. All children used more complex syntax as they got older, but the children with focal brain injury lagged behind the controls across the full age range from 3.5 to 12 years. Among the youngest children (3,6–5,0), children with RHD clustered with controls, whereas children with LHD rarely used complex syntax. Among older children (5,0–9,6), all children with focal brain injury, regardless of lesion site, performed below the controls. If we plot the developmental trajectories across all ages, children with LHD show the same slope as controls but at a significantly lower level, whereas the slope for children with RHD is essentially flat. Since complex syntax is a mechanism for integrating and relating events in a story, this profile may be evidence of a broader RH integrative deficit. Note, however, that there was no evidence of a RH disadvantage in overall level of performance.

Syntactic diversity was measured by applying a scale of 0 to 5 to the number of complex sentence types in each child's story. Among the younger children, +LT produced significantly fewer complex types compared to −LT; however, this profile did not hold after age 5, when both scored significantly lower than controls.

Children with focal brain injury included significantly fewer story episodes in narratives, and the stories tended to focus on local story events rather than tying events together with a theme. This suggests a delay in integrating the macrostructure with individual events as well as in making inferences about the motivations of the characters. The transition from a sequential description of local events to coherent narrative with an integrating theme occurred between age 5 and 6 in controls; it did not appear consistently until 7 to 8 in

the stories from children with focal brain injury. Again, we see no clear patterns relating to lesion side or site.

Overall, we found delays in children with focal brain injury on both linguistic and narrative measures. In addition, +LT appeared to be the most vulnerable for acquisition of new linguistic structures before 5–7 years of age. This profile of delay is reminiscent of the initial delays in language described above in infants and toddlers with focal brain injury. The production delays observed for core linguistic structures do *not* map onto the lesion profiles observed in adults with analogous injuries. However, these findings are compatible with the idea that the left temporal lobe plays an important role in the emergence of the LH specialization for language typically observed in normal adults. Finally, these data suggest that delays in linguistic abilities are not completely resolved by 5 years of age. They may, instead, reassert themselves as children with focal brain injury face new linguistic challenges. What we appear to be witnessing is a dynamic and repetitive process, very much like normal language acquisition, but with a somewhat delayed developmental trajectory.

As we have seen, results from examination of the acquisition of language by children with focal brain injury contradict predictions based on adults with comparable injuries. As we are about to see, the data for spatial cognition in the same population tell another story.

## Spatial Analytic Processing

Spatial analysis is defined as the ability to specify both the parts and the overall configuration of a pattern. Studies with adults have shown that different patterns of spatial deficit are associated with LHD and RHD (e.g., Arena and Gainotti, 1978; Delis et al., 1986; 1998; Gainotti and Tiacci, 1970; McFie and Zangwill, 1960; Piercy et al., 1960; Ratcliff, 1982; Swindell et al., 1988; Warrington et al., 1966). Injury to LP brain regions results in disorders in defining the parts of a spatial array, while patients with RHD have difficulty with the configural aspects of spatial pattern analysis. We have found similar patterns of disorder in our studies of young children with early focal brain injury

### *Spatial classification*

Our study of spatial classification (Stiles-Davis et al., 1985) was the first to explicitly establish a specific disorder of spatial integrative ability in 2- to 3-year-olds with RHD. In this task children were presented with stimulus sets containing two classes of objects (e.g., blocks and small plates, small dolls and wooden rings, cups and spoons) and simply encouraged to play. This procedure elicits systematic class grouping activity in both normal children and children with focal brain injury. The results showed that children with RHD were selectively impaired in their ability to form spatial groupings. Specifically, while RHD children would stack objects or place one object in another, they did not place objects next to one another to extend their constructions out in space. Normal and LHD children regularly placed objects next to each other as early as 24-months.

### *Block construction*

In order to elaborate the spatial classification findings, we conducted a large study using a more structured spatial grouping task. In this study 3- to 5-year-old children were asked

to copy a series of model block constructions (Stiles, Stern, Trauner, and Nass, 1996). This study was designed to allow us to examine both the products of children's construction efforts, and the procedures they used in grouping the blocks. Children in both LHD and RHD groups showed evidence of impairment on these tasks. Children with LHD initially showed delay on the task, producing simplified constructions. By the time they were 4 years of age, they showed an interesting dissociation in performance. Most of the children were able to produce accurate copies of the target constructions, however the procedures they used in copying the forms were greatly simplified. This dissociation between product and process persisted at least through age 6. RHD children were initially delayed. By age 4, they produced disordered, poorly configured constructions. However, at this age the procedures they used to generate their ill-formed constructions were comparable to age-matched controls. However, by the time these children were 6 years of age their profile of performance changed. By that time they were able to accurately copy the target construction, but like their LHD peers, they used simple procedures to generate these constructions. This study suggests that there is indeed impairment in spatial processing following early injury, and there is compensation with development. However, close examination of how spatial constructions are generated suggests persistent deficit. These findings have been replicated in a second study of American and Italian children with localized brain injury (Vicari et al., 1998). This also demonstrated that children with isolated subcortical injury show the same profiles of deficit as children with cortical involvement.

## *Drawing*

Our study of drawing in the focal lesion population has shown that children with RHD initially have considerable difficulty drawing organized pictures (Stiles-Davis et al., 1988). In a simple free drawing task, children were asked to draw a house. By age 3.5 to 4, normal children produce well organized houses, with an outer form representing the building and appropriately positioned inner features representing doors and windows. By age 5, the house drawings of children with LHD are indistinguishable from those of normal controls. However, during the late preschool period drawings by children with RHD are disordered and lack integration. The lack of organization suggests deficits in the ability to integrate parts to form a coherent whole. This is consistent with Swindell and colleagues' (1988) characterization of drawings by adult patients with RHD as, "scattered, fragmented, and disorganized . . . subjects often overscored lines and added extraneous scribblings" (p. 19).

This notable impairment in drawing among the children with RHD does not persist. Our longitudinal studies have shown considerable improvement with age. Improvement in the organization of their drawings is striking, but the drawings also exhibit striking similarity over time. This similarity may reflect the development of graphic formulas. Graphic formulas are common in the normal development course of drawing. Children begin to use graphic formulas from their earliest drawings (Stiles, 1995) and their use persists through adulthood. Thus the development of graphic formulas would not be an abnormal feature of drawing among children with RHD. The development of formulas may, however, offer a useful compensatory strategy by allowing children to represent common objects, while minimizing the spatial processing demands. If the children's improvement on the drawing task is achieved through the compensatory strategy of graphic formula production, then they should be more dependent on formulaic representation than normally developing children. Reliance on graphic formulas was tested using a task

developed by Karmiloff-Smith (1990) in which children are asked to first draw a house, and then an impossible house (Stiles et al., 1997). The most common solution to this task among normal children is to distort the spatial configuration of the house. The drawings of children with LHD are indistinguishable from those of normal controls (see figure 15.1a). However, in our longitudinal sample of 5 RHD children tested every 6 to 12 months for a period from 3 to 6 years, configural distortion was not used (see figure 15.1b). Instead the children derived a number of non-configurational solutions for solving the problem, including verbal descriptions, formula substitution (drawing another formulaic object and asserting it was a house), reduction (putting a dot on the page and saying the house is very

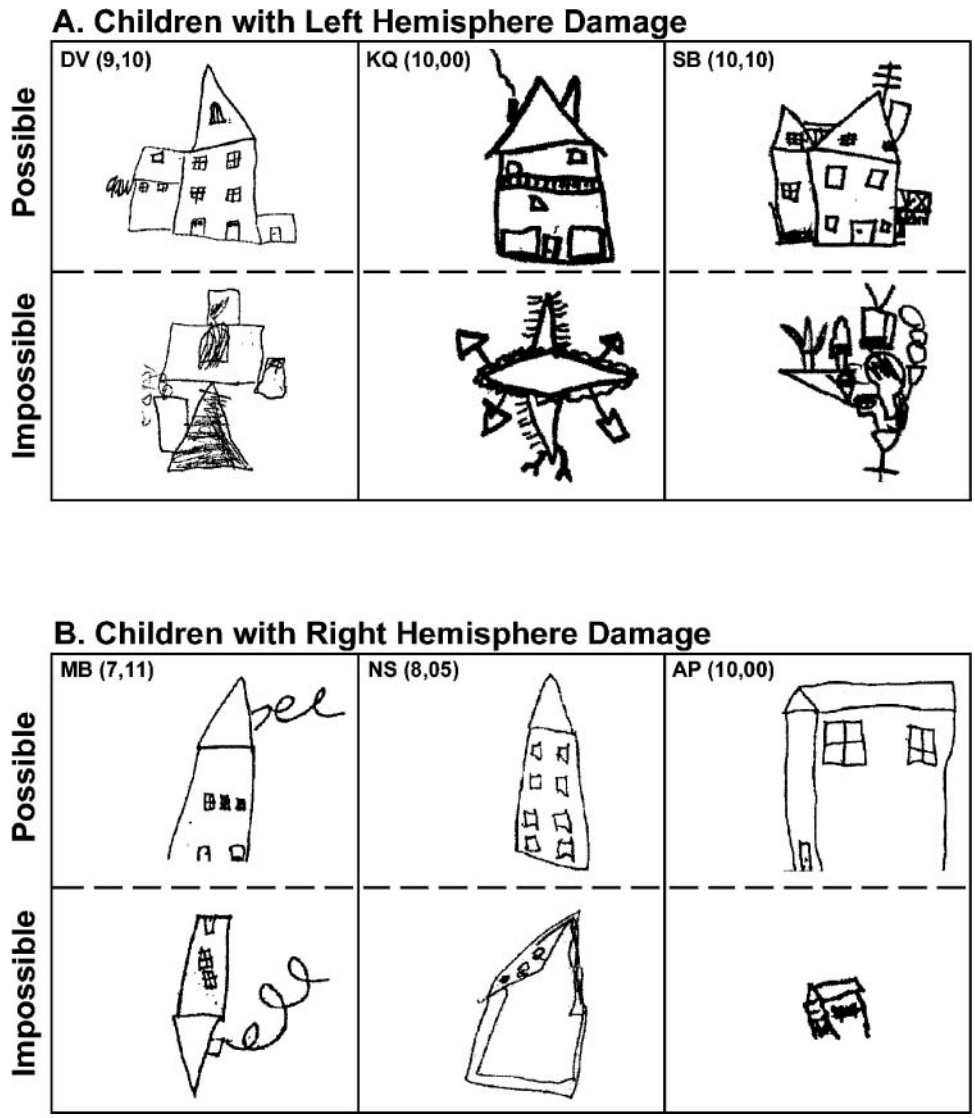


*Figure 15.1* Drawings of possible and impossible houses from children with left (A) or right (B) hemisphere brain injury.

small), and invisibility. Once again, these data indicate that while these children are developing and their performance on specific spatial tasks improves, the processes by which they master these tasks may differ from those of normally developing children. This suggests a pattern of specific, subtle, and persistent deficit in spatial processing.

## *Processing hierarchical forms*

Any visually presented pattern can be conceived of as a structured hierarchy consisting of local level elements and more global level assemblies. One example of a simple pattern hierarchy is the hierarchical form stimulus. It consists of a large letter composed of appropriately arranged smaller letters, such as a large H made up of small Ss. Hierarchical stimuli have been used in studies of normal adults (e.g., Kinchla and Wolfe, 1979; Martin, 1979; Navon, 1977; Palmer, 1980; Palmer and Bucher, 1981) and children (Dukette and Stiles, 1996; 2001; Stiles-Davis et al., 1988). They have also been used successfully to identify differential patterns of spatial deficit in adults with focal left and right posterior brain injury (Delis et al., 1986; 1988; Lamb et al., 1989, 1990; Robertson and Delis, 1986). Specifically, adult patients with RHD have difficulty processing the global level of the form, while patients with LHD have difficulty with the local level.

Data from one study of hierarchical form processing among children with LHD or RHD are consistent with data from adult patients. In this task children were asked to study and remember a hierarchical pattern. After a brief distracter task they were asked to reproduce the form from memory (see figure 15.2 for examples from younger children). Two age groups were tested, 5- to 7-year-olds, and 9- to 11-year-olds. The younger children with LHD had difficulty producing both the global and the local level of pattern structure, while young children with RHD were able to accurately produce the local level elements but had difficulty with the global level. Older children with LHD were impaired only with production of local level stimuli, and older children with RHD continued to show impairment with the global level. For each group the level of impairment was more pronounced

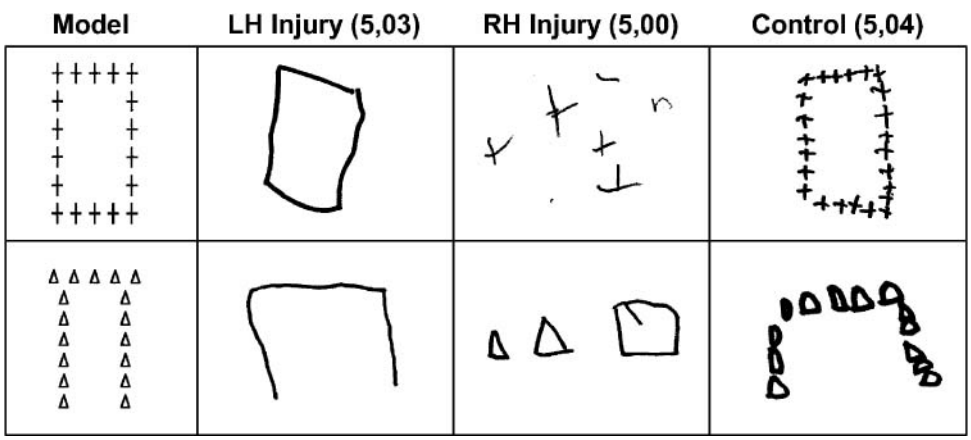


*Figure 15.2* Memory reproductions of two model hierarchical forms by three 5-year-old children, one with prenatal LH brain injury, one with prenatal RH brain injury, and a normal control child.

for the younger than the older children. For the young children with LHD, the severe impairment in ability to produce local elements, also affects their ability to generate a well configured larger form. The reverse profile was not observed for the children with RHD. It was possible for them to accurately generate local level elements, even though they were unable to configure them appropriately.

## Summary and Conclusion

We have provided a brief overview of results from the first large-scale prospective study of behavioral development in children with early focal brain injury. It has taken more than ten years to accumulate a large enough database to justify the (tentative) conclusions presented here. It also goes without saying that all of these findings will need to be replicated in other laboratories. Furthermore, because our findings are still primarily cross-sectional in nature, they must be tested and extended in longitudinal work. With those caveats in mind, we want to underscore that research with this population has yielded a number of surprises, including results that are not always compatible with the view of brain organization that one typically finds in surveys of lesion studies in human adults. We end this chapter with three lessons from research with this population, followed by three questions that are left unanswered by this work.

### *Lesson #1: Against predeterminism*

The idea that the mind–brain is organized into distinct faculties or "modules" goes back to the eighteenth century, to the phrenological proposals of Gall and Spurzheim. This phrenological perspective is often accompanied by a developmental corollary: familiar patterns of brain organization for higher cognitive functions can be found in the mature adult because those patterns were there from the beginning, as innate properties of the human brain. The fact that children in this population outperform adults with homologous injuries can be used to argue against any strong form of predeterminism, predestination, or preformationism. All of the findings that we have reviewed in this chapter point in the same direction: children with early focal brain damage ultimately reach levels of performance well ahead of those observed in adults with homologous injuries. To be sure, brain damage is not a good thing, and children who have suffered some form of focal brain injury typically perform (as a group) reliably below normal controls, sometimes in relatively predictable patterns depending on their site and side of injury. However, our developmental findings suggest that these initial biases are imperfect, indirect, "soft constraints" that can be overcome.

### *Lesson #2: Against equipotentiality*

When studies of children with early brain injury first appeared in the neuroscience literature, they were sometimes used to argue in favor of a *tabula rasa* view of the mind–brain, a view in which cortical tissue is, initially, capable of taking on an infinite number of functions, with no bias toward any particular cognitive domain. In its strongest form, the equipotentiality hypothesis is flatly impossible: if it were true, there would be no way to

explain why familiar forms of brain organization are observed so often in the neurologically intact adult brain. For example, current estimates are that the LH plays a special role in the mediation of language in 95–98 percent of normal individuals. There must be some kind of bias present from the beginning of life in order to explain the well-documented hemispheric specialization for language and spatial cognitive functioning. By carrying out prospective studies of linguistic and cognitive development, we have uncovered subtle but specific patterns of deficit and delay that work against any strong and simple form of the equipotentiality hypothesis. Some form of cortical specialization (or "cortical preference") is clearly there from the beginning of life, although it can give way to an alternative "division of labor" when things go awry. Our challenge for the future is to specify the nature of those early biases, and the developmental processes by which alternative forms of brain organization emerge over time.

## *Lesson #3: Children are not adults*

The patterns of lesion–symptom mapping that we have uncovered in our work differ markedly from the patterns revealed in adult neuropsychology literature. In all the domains that we have studied to date, there are quantitative differences in the effects of homologous injuries on children and adults: the effects on children are generally more subtle (i.e., not as severe, compared with performance by normals in the same age range), and performance improves markedly over time – sometimes to the point where (at least on casual inspection) the deficit seems to have disappeared altogether. These quantitative differences provide further evidence for a conclusion that has emerged in the last 20–30 years of research in developmental neurobiology: the developing brain is highly plastic, and alternative forms of brain organization are possible for the "same" behavioral task (although there is emerging evidence that the processes associated with these alternative forms may differ from those observed with typical organizational profiles).

Within the language domain, the differences are also qualitative. We outlined four simple hypotheses derived from more than 100 years of research on adults with unilateral brain injury: (1) LH specialization for most linguistic tasks, (2) left frontal specialization for expressive language (i.e., the Broca hypothesis), (3) left temporal specialization for receptive language (i.e., the Wernicke hypothesis), and (4) RH specialization for some discourse functions. We did not find unequivocal support for any of these hypotheses, and some of them were flatly contradicted by our results.

For example, none of our infants with left temporal lesions were in the bottom 10th percentile for word comprehension; in fact, although results are probabilistic in nature, there is some reason to believe that RHD is a greater risk factor for comprehension. These findings run against the Wernicke hypothesis, and against LH specialization for basic language functions. In line with the adult literature, we do find evidence that children with LHD are more delayed in expressive language. However, this finding is only evident from 10 months of age (the dawn of language) up to but not beyond 5–7 years. Furthermore, the effect is coming primarily from children with left temporal involvement (against the Wernicke hypothesis). Frontal involvement is an additional risk factor between 19 and 31 months of age, but in this time window it doesn't seem to matter whether frontal damage occurs in the left or the RH (against the Broca hypothesis). Some time after 5–7 years of age, we no longer have any evidence for differences due to side of lesion (left vs. right) or intra-hemisphere site of lesion (frontal or temporal). The RH cases display a flatter

developmental profile in the use of complex syntax for narrative purposes, in line with the idea that the RH may be specialized for discourse. However, there are no significant differences between the left and right hemisphere groups in absolute level of performance after 5–7 years of age. The only firm conclusion that holds in our data for older children with congenital lesions is that brain damage does exact a cost, lowering the group profile below normal controls – though still well within the normal range.

Although we have no ready explanation for these quantitative and qualitative differences in patterning, these findings do remind us of an important point: the children in our prospective studies are encountering language and other higher cognitive functions for the first time. What we are looking at is, in essence, the effect of early focal brain injury on the learning process. Our results suggest that the brain mechanisms responsible for language learning are not the same mechanisms that govern the maintenance and fluent use of language in normal adults. In other words, we do not believe that language literally moves (bags packed) from one brain region to another across the course of development. Rather, the learning process may recruit brain areas that are no longer needed once the learning itself is complete, and the task in question has become a routine part of daily life. This conclusion is, in fact, compatible with recent studies of learning and processing in normal adults using positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) – studies that show differential patterns of brain activity for the same task in novices compared with experts (Raichle, 1994), and differential patterns of activity in the same individuals as a new task is mastered and/or as the same task is administered with increasingly difficult and complex stimuli (Just et al., 1996).

These conclusions are easy to defend, in light of our own work and many other studies in the literature. However, as we have noted, they raise more questions than they answer. Let us end by posing three of the most puzzling questions that we now face.

### *Question #1: Why is language more plastic than spatial cognition?*

Our results for spatial cognition are, as noted, qualitatively similar to the lesion–symptom patterns that have been reported for brain-injured adults. In the spatial domain, RHD seems to be associated with a deficit in the integration of information; by contrast, LHD results in a deficit in the extraction of pattern detail. By contrast, our findings for language development are not at all compatible with the classic aphasia types observed in adults. Deficits in word comprehension and gesture appear to be associated with RHD rather than LHD. Deficits on the production of words and grammar are greater in our LHD sample, as we might expect from adult aphasiology. However, the intrahemispheric patterns observed in children are quite different from those observed in adults, including an asymmetrical LP effect on both vocabulary and grammar, and an additional frontal effect on expressive language that is observed to an equal degree with right frontal and left frontal involvement. More puzzling still, none of these side or site specific effects are observed in our cross-sectional findings after 5–7 years of age, even though our older children have exactly the same congenital etiology as the younger cases. This is not true for our extensive school-age studies of spatial cognition. Why are the findings for language so different from our findings for spatial cognition?

It is possible that language is more plastic than other behavioral functions simply because it is a phylogenetically recent phenomenon. Perhaps there has not been sufficient time for language to evolve into a fixed and irreversible neural system. Although we acknowledge

this possibility, we suspect that this is not the answer. Language is different from the other systems that we have studied to date in a number of crucial respects, with implications for the nature and plasticity of the neural systems that subserve it. First, language is the system that we use to express meaning; indeed, the boundaries of language include semantics as well as grammar and phonology. Because meaning encompasses all of our experience, the system that we have evolved to encode those meanings must by definition include information derived from widely distributed neural systems.

But what about linguistic form, independent of meaning? Could there be a tightly bounded, predetermined region that handles phonology and/or grammar? In principle, this is certainly possible, and to a limited extent it has to be true – at least for speech sounds. The basic input–output architecture used by speech appears to be a universal property of the human brain. As Sigmund Freud pointed out long ago in his seminal book on aphasia (Freud, 1953), it is quite likely that, under default circumstances, the continuous sheets of cortex that subserve the rest of language will organize around these basic input–output "hot spots," leading to the familiar pattern of broad perisylvian specialization for language. At the same time, we now know that this pattern can appear in either hemisphere after early brain injury, and we also know from recent neural imaging studies of normal adults that homologous areas of activation are observed on both sides of the brain in many language tasks, although the activation is typically greater on the left (Just et al., 1996).

This brings us to a central issue in the definition of "language areas": are these regions specialized for speech and language *only* (i.e., as special purpose mechanisms – Fodor, 1983), or is it the case that language "borrows" perceptual and motor systems that also do other kinds of work? At the moment, most of the evidence points to the latter option. For example, a recent fMRI study demonstrated that, in addition to carrying out linguistic functions, the various subcomponents of Broca's participate in the planning and execution of one or more non-speech tasks (Erhard et al., 1996). Similar results have been reported for the left temporal regions that are the putative site of Wernicke's area. In short, although it is possible that some aspects of language processing are carried out in highly localized brain regions, those regions may subserve a wider range of function. This may be one reason why these areas show so much plasticity: language is a problem that the brain solves with a range of different general-purpose tools, and for that reason, a number of different solutions are possible.

This brings us to a related point: if language is a parasitic system, running on hardware that evolved for other purposes, it is fair to ask whether the lesion–symptom patterns that we have observed in language, spatial cognition and affect are related in some way? For example, we have noted that RHD children show a relatively flat profile in the development of complex syntax. Is this language profile related to the information-integration deficits observed in spatial cognition? Do the same integration problems observed in RHD contribute in some fashion to the delays in word comprehension displayed by RHD children in the early phases of development? In fact, learning what a word means for the first time is, by definition, a multimodal integration problem. In the same vein, we may ask whether the deficits in perceptual analysis associated with left temporal lesions are implicated in some way in the expressive language delays that children with such lesions display between 10 and 60 months of age. The evidence suggests that left temporal cortex is especially well-suited to the extraction of pattern detail, temporal as well as spatial. This fact may give left temporal cortex a "competitive edge" in the language learning process. But why should this "edge" appear most clearly in expressive language, rather than comprehension? We have suggested elsewhere (e.g., Bates et al., 1997; Elman et al., 1996; Stiles and Thal, 1993) that

learning-to-produce actually requires a much more fine-grained form of perceptual analysis than learning-to-comprehend, because the child must pull enough detail out of the acoustic signal to permit the construction of an intelligible motor template. In other words, understanding what "giraffe" means in context requires far less analysis of the signal than saying the word "giraffe" for the very first time. Of course these suggestions are still quite speculative, but it is a place to start – which brings us to the next question.

### *Question #2: What is a "bias"?*

We have suggested a compromise between the warring claims of equipotentiality and predeterminism, in which different regions of cortex start out not with innate knowledge, but with "soft constraints," innate predispositions to process information a certain way. This is what Elman et al. (1996), refer to as "architectural innateness," as opposed to "representational innateness." Because of its initial predispositions, a particular region of the brain may be *recruited* to carry out specific aspects of (for example) a linguistic or visual–spatial task, in the same way that a tall child is recruited into the game of basketball. On this view, the division of labor that we see in the adult brain is the product of development rather than its cause. This approach is compatible with findings in developmental neurobiology over the last two decades, suggesting that cortical specialization is driven by activity and experience, in the default situation and in the alternative situations that arise after early brain injury (for reviews, see Elman et al., 1996, ch. 5; Nelson, 1999; Stiles, 1998; 2000). However, we still know very little about the features of different cortical regions that are responsible for these initial predispositions. What do we mean, in concrete neurocomputational terms, when we say that a region is specialized for information integration, or for the extraction of fine-grained pattern detail? Unfortunately, very little is currently known about the neural microcircuitry of the developing human brain. For example, are there concrete, measurable differences from region to region or hemisphere to hemisphere in cell density and cell types within and across cortical layers, the distribution of neurochemicals, and so forth? What are the computational consequences of such differences, if they exist? We know what questions to ask, but there are very few answers available right now, and our conjectures about innate predispositions for learning cannot be turned into testable hypotheses until such information becomes available.

### *Question #3: Why does plasticity sometimes fail?*

We end by pointing out that there are populations of children with deficits in language, cognition and communication that do not display the extraordinary plasticity evidenced by children with early focal brain injury. Examples include children with Specific Language Impairment (SLI), autism, and several different forms of mental retardation including Williams Syndrome and Down Syndrome. All of these populations are currently under study in our San Diego research center, using many of the same behavioral and electrophysiological measures that we administer in our focal lesion studies. On almost every measure, our children with focal brain injury eventually surpass the other clinical groups, even though recent neural imaging studies of SLI, autism, Williams Syndrome and Down Syndrome provide no evidence for frank lesions of any kind. It seems evident from these comparisons that some forms of early brain injury lead to severe and persistent long-term

deficits, without the profiles of recovery and/or compensation that we observe in the group with focal brain injury.

Why does plasticity fail in these cases? There are several possibilities: (1) diffuse, "microlesions" that are invisible in neural imaging studies but are nevertheless so pervasive that they preclude normal development, (2) abnormalities in the cytoarchitecture arising during neurogenesis and/or migration, (3) abnormalities in control of synaptogenesis, apoptosis or other regulatory mechanisms in brain development, and/or (4) neurochemical abnormalities affecting either basic metabolic processes or neurotransmitter production. Although these possibilities are no more than sheer speculation today, they may lend themselves to a rigorous test through the combined application of structural and functional brain imaging techniques.

To summarize, we have raised more questions than we have resolved in this chapter, but some lessons have been learned, and there are good reasons to hope that our new questions will be answered. Interdisciplinary research is difficult, requiring time and patience. But our experience to date suggests that this collaborative approach is well worth the effort.

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