LANGUAGE DEVELOPMENT IN CHILDREN WITH UNILATERAL BRAIN INJURY

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Abstract

Aphasia (defined as the loss or impairment of language abilities following acquired brain injury) is strongly associated with damage to the left hemisphere in adults. This well-known finding has led to the hypothesis that the left hemisphere is innately specialized for language, and may be the site of a specific "language organ". However, for over a century we have known that young children with left-hemisphere damage (LHD) do not suffer from aphasia, and in most studies do not differ significantly from children with right-hemisphere damage (RHD). This result provides strong evidence for plasticity, i.e., brain reorganization in response to experience, and constitutes a serious challenge to the language organ hypothesis. This chapter reviews the history of research on language outcomes in children vs. adults with unilateral brain injury, addressing some discrepancies in the literature to date, including methodological confounds that may be responsible for those discrepancies. It also reviews recent prospective studies of children with unilateral injury as they pass through the first stages of language development. Prospective studies have demonstrated specific correlations between lesion site and profiles of language delay, but they look quite different from lesion-symptom correlations in adults, and gradually disappear across the course of language development. The classic pattern of brain organization for language observed in normal adults may be the product rather than the cause of language learning, emerging out of regional biases in information processing that are relevant for language, but only indirectly related to language itself. If those regions are damaged early in life, other parts of the brain can emerge to solve the language learning problem.

Aphasia, or the loss of language abilities following brain injury, has been studied systematically in adults for over a century, and its existence has been docu-mented since the first Egyptian surgical papyrus more than 4000 years ago (Goodglass, 1993; O'Neill, 1980). There is now a large body of research on adult aphasia, and although there is still substantial controversy regarding its nature and causes, consensus has emerged on at least two points: injuries to the left hemisphere are overwhelmingly more likely to cause aphasia than injuries to the right, which in turn suggests that the left hemisphere plays a privileged role in language pro-cessing by normal adults. The second conclusion has been independently confirmed in the 20th century by methods ranging from sodium amytal (WADA) tests and/or point-to-point electrical stimulation in adult candidates for neurosurgery (Ojemann, 1991), to neural imaging studies of normals, including positron emis-sion tomography (PET), functional magnetic resonance imaging (fMRI), magneto-encephalo-graphy (MEG) and event-related brain potentials (ERP) (for reviews, see Brown and Hagoort, 1999; Xiong et al., 1998).

The privileged status of the left hemisphere for language processing is now beyond dispute (with esti-mates averaging from 95%-98% of normal adults, independent of handedness), but the origins and develop-ment of this specialization are still poorly understood. There must be something about the left hemisphere that makes it especially suited for language -- but what is that "something"? Is it present at birth, or does it develop gradually? Is it possible to develop normal language in the absence of a normal left hemisphere? And if an intact left hemisphere is not required for language development, then when, how, and why does it *become* necessary for language use in adults? Finally, if alternative forms of brain organization for language can emerge in the presence of early left-hemisphere damage, is there some critical period in which this must occur?

The sparse but growing body of evidence on language development in children with left- vs. right-hemisphere damage is relevant to all these points, and it has yielded two very puzzling results: (1) most children with early left-hemisphere damage go on to acquire language abilities within the normal range (although performance is often at the low end of the normal range), and (2) most studies fail to find any significant differences in language outcomes when direct com-parisons are made between children with left- vs. right-hemisphere damage. These unexpected findings in children are hard to reconcile with one of the most popular ideas in neuropsychology: that the left hemi-sphere of the human brain contains an innate and highly specialized organ for language (e.g., Fodor, 1983; Gopnik, 1990; Gopnik and Crago, 1991; Newmeyer**,** 1997; Pinker, 1994; Rice, 1996). The language-organ hypothesis is appealing on many grounds. Aside from its value in explaining left-hemisphere specialization, the existence of a specialized language organ might help to explain why all normal adults are virtuosi in this domain. For example, adult speakers of English produce an average of 150 words per minute, each rapidly select-ed from a pool of 20-40,000 lexical options. As quickly as these words are spoken (often blurred together, without well-marked boundaries), the average listener can parse these unbroken streams of sound into words and phrases, accessing the meaning of each word (from that same large pool), while simultaneously processing all the complex grammatical cues necessary for com-prehension. This is an ability no other species on the planet appears to have, and one that today's largest and fastest computers have yet to master.

Perhaps even more phenomenal than the speed and ease with which we produce and perceive speech is the speed and ease with which we learn how to do it. Most 4-year-olds cannot tie their own shoes, but they can easily ask someone else to help them. In fact, most 4-year-olds have a vocabulary of 6000 words or more, and produce well-formed sentences as grammatically com-plex as those observed in any adult (Bates, in press; Bates et al., in press; Fletcher and MacWhinney, 1995). Children master their native language (or *languages*, for that matter) without formal instruction, without explicit corrections, and, seemingly, without effort. Perhaps we are the only animals on earth that can manage this feat because we have an innate language organ. But the organ metaphor carries a number of assumptions that are contradicted by research on language development in children with early brain injury: (1) the brain in general and the left hemisphere in particular are specialized for language at birth; (2) this specialization involves com-pact and well-defined regions of the left hemisphere that are dedicated to language (and language alone); (3) this specialization is irreversible, so that normal levels of language are precluded if the language organ is severely damaged at birth; (4) even if some degree of language learning does take place (presumably through compen-satory mechanisms), children with early left-hemisphere injuries should display persistent deficits that are not observed with early injuries to homologous areas on the right side of the brain.

All of these assumptions are in peril. Although these issues are not yet settled to everyone's satis-faction, one fact is clear: in the absence of other con-founding factors (e.g., severe and intractable seizures), the language deficits observed in children with early left-hemisphere injury are (if they exist at all) far less pronounced than the aphasic syndromes seen in adults (Bates, 1999; Bates, Vicari, and Trauner, 1999; Eisele and Aram, 1995; Vargha-Khadem, Isaacs, and Muter, 1994; Vargha-Khadem, Isaacs, van der Werf, and Wil-son, 1992). Other conclusions are still controversial, regarding the time course of recovery, the nature of the mechanisms that support it, and whether there are ultimately any significant differences (i.e., mild deficits) between children with left- vs. right-hemisphere dam-age.

Our ability to answer these questions is limited by a number of factors. First, focal lesions in young children are very rare, so that generalizations are sometimes based on samples too small to support them. Second, results across studies are often in direct conflict, due to methodological variations including sample size, etiology (e.g., stroke, tumor, trauma, and conditions that might predispose children to any of these injuries), age of lesion onset, age of testing, the developmental sensitivity (or insensitivity) of the instruments used to evaluate language, and the kinds of statistical comparisons that were made (e.g., whether children with LHD and RHD are compared directly, vs. indirect comparisons in which each clinical group is evaluated against a separate set of normal controls).

Due in part to these troubling methodological factors, research on language outcomes following early brain injury has swung back and forth between two extreme views: *equipotentiality* (site or side of injury do not matter at all in young children, because both sides of the brain are equivalent at birth) and *irreversible determinism* (the left hemisphere is innately and irrever-sibly specialized for language, precluding the possibility of complete and normal language development if it is severely damaged). We will argue that the bulk of the evidence supports a compromise view between these two extremes, in which the two hemispheres are char-acterized at birth by innate but "soft" biases in infor-mation processing that are relevant to language, but not specific to language, permitting both neural and be-havioral reorganization across the course of language development (see also Satz, Strauss, and Whitaker, 1990). On this argument (which we will call *the emergentist view*), we would expect to see left-/right-hemisphere differences early in life, but these differences will decrease with time and may eventually disappear.

We will review the evidence in three partially overlapping phases in the history of this field: an equipotentiality phase, an irreversible-determinism phase, and (after a brief stop to consider the contribution of methodological factors) the current move toward an emergentist view. A summary of evidence involving measures of verbal and nonverbal IQ is presented in Table 1. Evidence based on more specific measures of language is summarized in Table 2.

Phase I: Equipotentiality

Not long after the first 19th-century studies link-ing aphasia to left-hemisphere damage in adults, studies appeared suggesting that children with the same kinds of damage have little or no difficulty with language (Clarus, 1974; Cotard, 1868; both cited in Woods and Teuber, 1978), or that they show temporary deficits that quickly disappear (Bernhardt, 1897).

In the 20th century, Basser (1962) reported on 34 children with severe epilepsy who underwent a radical process called hemispherectomy (removal of the damag-ed side of the brain) to control intractable seizures. Results were consistent with those from the century before: all but one of these children developed speech abilities in the normal range (see also Rasmussen and Milner, 1977). It was Basser's study that led Lenneberg (1967) to his controversial notion that the brain is "equi-potential" at birth, with lateralization determined gradually across the course of development. As a corollary, Lenneberg also argued that this period of equipotentiality and plasticity is brought to an end at puberty, providing the first systematic argument in favor of a "critical period" for language. Lenneberg's views were quite compatible with an earlier proposal by Lashley (1950), who interpreted lesion studies of ani-mals to indicate that loss of learning is predicted by the size of the lesion rather than its location (see also Irle, 1990). Lenneberg's critical-period proposal was com-patible not only with the evidence on recovery from unilateral damage (i.e., the difference between children and adults with comparable injuries), but also with (a) the difficulty that adults display in acquiring a second

language without an accent, and (b) some influential "Wild Child" studies, especially the famous case of Genie (Curtiss, 1977), which seemed to suggest that acquisition of a first language is also precluded if normal input is delayed until late childhood or puberty.

However, Lenneberg's equipotentiality hypothesis did not sit well with some of his contemporaries, who were persuaded by the research of Sperry, Gazzaniga, Geschwind and others that the two hemispheres are too different to support a complete change of roles even early in life (Gazzaniga and Sperry, 1967; Geschwind and Kaplan, 1962; Levy, Nebes, and Sperry, 1971). Equipotentiality was also difficult to reconcile with Noam Chomsky's theory of generative grammar, with all its claims regarding the autonomy, innateness and "unlearnability" of language (Botha, 1989; Newmeyer, 1980). Another round of studies of children with early brain injury rapidly ensued, leading to an entirely dif-ferent view.

Phase II: Irreversible Determinism

In response to Lashley's and Lenneberg's contro-versial ideas about equipotentiality, a number of studies appeared between 1960 and 1980 suggesting that early brain injury does lead to subtle but persistent language impairments, deficits that are more likely following left-hemisphere damage (LHD) than right-hemisphere damage (RHD). For example**,** Woods and colleagues (Woods, 1980; Woods and Carey, 1979; Woods and Teuber 1973, 1978) concluded that LHD in children does lead to speech and language problems, especially if lesion onset occurs *after* one year of age (see below for a more detailed discussion of age of lesion onset), and they attribute earlier evidence for equipotentiality to limitations in medical knowledge at that time (Woods and Teuber, 1978). In the same vein, Dennis and colleagues (Dennis, 1980; Dennis and Kohn, 1975; Dennis, Lovett, and Wiegel-Crump, 1981; Dennis and Whitaker, 1976, 1977) reported that left-hemispherec-tomized children are more likely to have phonological and grammatical problems than children with right hemispherectomies (although the reported deficits were quite subtle).

Although these studies were influential (and are cited in many textbooks), most of them do not include direct statistical comparisons of children with LHD and children with RHD (see Tables 1 and 2). Some looked exclusively at LHD children and controls, while others compared each group to its own set of age-matched controls (a practice followed in many of the studies reviewed below). The latter practice is common, but it is also problematic: authors infer that effects of LHD are quantitatively and perhaps qualitatively different than the effects of RHD, but this supposed difference in patterning assumes an untested statistical interaction (i.e., that the difference between LHD and their controls is statistically greater than the difference between RHD and their controls). As we shall see below, studies that have looked for such statistical interactions (or com-pared LHD and RHD directly) have generally failed to find the predicted effects.

As evidence accumulated, the picture became more complex, and more confusing. For example, Alajou-anine and Lhermitte (1965) reported that children with LHD do have initial difficulty with some aspects of language, especially expressive language, but these difficulties were far less pronounced than those seen in adults, and disappeared within six months to two years after lesion onset. Note that Alajounanine and Lher-mitte did not study right-hemisphere-damaged patients. Riva et al. (1986) found that while left-hemispherec-tomized children performed more poorly than right-hemisphere children on some grammatical comprehen-sion tests, left- and right-hemisphere-damaged children were equally impaired on measures of vocabulary production and comprehension. Similar findings have been reported in a series of studies by Aram et al. (1985, 1986, 1990) and Eisele (Eisele and Aram, 1993, 1994, 1995). While Aram et al (1985) and Eisele and Aram (1993) found that on measures of lexical com-petence, RHD and LHD children were both impaired relative to age-matched controls, it appeared that children with LHD performed worse than their normal controls on a number of other language measures, including tests of both grammatical comprehension and production, phonological discrimination tests, and tests of lexical fluency. By contrast, children with RHD showed no statistical difference from their own controls on nearly all such measures. However, later studies by the same research team reached a different conclusion. For example, Eisele and Aram (1994) report no differ-ences between LHD and RHD on a test on syntax comprehension, although several children from both groups performed at chance. Based on a detailed qualitative examination of lesion data (albeit without a statistical test), the authors conclude that subcortical involvement to either hemisphere may be the most important determiner of failure on this syntax task (Eisele and Aram, 1995).

A similar history can be traced in research by Vargha-Khadem and colleagues. For example**,** Vargha-Khadem, O'Gorman and Watters (1985) reported performance on grammatical comprehension tests was more impaired in children with LHD. However, as they added more cases to their sample, this difference disappeared (Vargha-Khadem et al. 1994). It now appears from studies by this research group that seizure history is the most important predictor of language impairments in brain-injured children, regardless of side or size of injury, or of the age at which the lesion was acquired.

Variations in the tests used to assess language (see Tables 1-2) may be responsible for some of the dis-crepancies seen between studies. However, even when standardized tests of IQ are used, studies differ in factors like age of onset, subcortical involvement, and presence or absence of seizures. When IQ scores are broken down into verbal and nonverbal (performance) quotients, adult LHD patients typically have higher PIQ scores com-pared to their VIQ scores, whereas RHD patients typic-ally show the exact opposite pattern. The extent to which findings for children fit this pattern varies from study to study, due in part to methodological confounds.

In one study, Woods (1980) found that results for VIQ and PIQ depended on both side of lesion and the age at which the lesion was acquired. He found that (1) children with LHD

scored significantly below normal on both VIQ and PIQ, regardless of the age at which the lesion was acquired; (2) children with RHD also scored below normal on both subscales, but only if their lesions were acquired before one year of age; (3) if children with RHD acquired their lesions after the first year, they scored in the normal range for language but below normal on performance IQ. This complex nest of findings led Woods, Teuber and colleagues to propose the "crowding hypothesis": in an effort to salvage language in the presence of LHD, language functions are moved to the right hemisphere, where they interfere with the spatial tasks normally conducted in those areas of the brain.

Riva et al (1986) also report differential effects of age of onset and lesion side, but their results were virtually the opposite of Woods (1980). Children with early LHD were significantly lower than controls on both VIQ and PIQ, but only if their lesions occurred *before* one year of age; children with later lesions did not differ significantly from normal controls on either subscale. Children with RHD scored reliably below normal controls on PIQ, but *not* on VIQ regardless of the age at which damage occurred. More recently, Ballantyne, Scarvie and Trauner (1994) found that brain-injured children as a group performed below controls on all IQ subscales; VIQ was no worse than PIQ for LHD children, but VIQ was better than PIQ for RHD children. Note that none of these studies (Ballantyne et al., 1995; Riva et al., 1986; Woods, 1980) report a direct statistical comparison of LHD and RHD.

Nass, Peterson and Koch (1989) did conduct direct comparisons of children with congenital LHD and RHD, with surprising results: children with LHD actually did *better* on VIQ than PIQ, and they also performed better than children with RHD on the verbal scale. Eisele and Aram (1993) also compared groups of brain-injured children directly. They found the adult pattern for PIQ (with RHD performing worse than both LHD and controls), but there were no effects of lesion side on VIQ (where LHD and RHD were both indistinguishable from controls). Muter et al. (1997) and Vargha-Khadem et al. (1992) found no differences between RHD and LHD groups on either VIQ or PIQ, although children with seizures were more impaired on both scales than children without seizures.

As we move out of the 1990's and into the next millennium, some of the confusion that has charac-terized research in this area has begun to lift. Most investigators now embrace a "third view" midway between equipotentiality and irreversible determinism, a bidirectional relationship between brain and behavioral development in which initial biases and subsequent reorganization are both acknowledged. This consensus is due in no small measure to methodological improve-ments, including the availability of imaging techniques to clarify the relationship between lesion type and language outcomes. But improved neural imaging is not the only relevant factor. Before reviewing a final set of studies in support of this emergentist view, let us consider several crucial methodological factors and their theoretical consequences: timing of lesion onset, lesion type (both site and size), lesion etiology, sample size, and the importance of prospective studies that employ developmentally sensitive measures.

Intermezzo: Methodological Confounds

Time of lesion onset and its implications for plasticity. There is now a large body of evidence demonstrating that the brains of young animals (es-pecially mammals) are quite plastic, and that many aspects of cortical specialization are activity dependent. That is, cortical specialization is determined not by endogenous growth plans under direct genetic control, but by the input that cortical areas receive from the animal's own body (before and after birth) and from the outside world (for reviews, see chapters in this volume by Kolb and by Elbert; Deacon, 1997; Elman et al., 1996; Johnson, 1997; Quartz and Sejnowski, 1994, 1997). For example, if the cortex of a fetal ferret is rewired so that input from the eye is fed to auditory cortex, it has been shown that auditory cortex takes on retinotopic maps (Pallas and Sur, 1993). And if slabs of fetal tissue are transplanted from visual to somato-sensory areas (and vice versa), the transplanted cortex takes on representations appropriate to the input re-ceived in its new home, as opposed to the represen-tations typically seen in their regions of origin (O'Leary and Stanfield, 1985, 1989; Stanfield and O'Leary, 1985). Lesion studies of animals also provide striking evidence for plastic reorganization. For example, Payne (1999) has shown that cats with early bilateral removal of primary visual cortex are virtually indistinguishable from normal on visual tasks; mature cats with the same operation are functionally blind. Webster, Bachevalier and Ungerleider (1995) have shown that infant monkeys with bilateral removal of area TE (the ventral temporal areas that are the final way station of the "what is it?" visual system in mature animals) perform only slightly below unoperated controls on a task that measures memory for new visual objects; mature animals with the same lesions display severe visual amnesia. The accumulated evidence strongly suggests that cortical specialization is (at least in part) driven by cortical input, and that new forms of organization can emerge following early brain injury. Based on this evidence, we should expect to find that early injuries in humans are followed by substantial reorganization, for language and for other cognitive functions (Stiles et al., 1998).

This well-attested finding leads to a prediction that seems, at first glance, to be quite obvious: if plasticity is greater in the young brain, then we ought to find a monotonic relationship between cognitive outcomes and age of lesion onset. Although the shape of this function might vary in a number of theoretically interesting ways (dropping sharply at some point in a nonlinear pattern, or decreasing gradually from birth to puberty), later lesions ought to produce worse outcomes than early ones under any scenario. In fact, the shape of the function governing loss of plasticity in humans is still entirely unknown, and it may not even be monotonic (i.e., plasticity may fall, and then rise again). Many of the studies summarized in Tables 1 and 2 have conflated cases of congenital injury with lesions that were ac-quired at points later in childhood. Other studies have divided age of lesion

onset into broad epochs, with mixed and often contradictory results. For example, Woods and Teuber (1978) conclude that injuries in the first year of life are actually more dangerous than injuries acquired after age one, a finding that seems to fly in the face of accumulated evidence for early plasticity in animal models.

Even more puzzling findings come from Goodman and Yude (1996) and from unpublished data by Vargha-Khadem and colleagues (personal communication, July 1996, cited in Bates, Vicari, and Trauner, 1999). The latter two studies employed relatively large samples (by the standards of this field), and both revealed a result that would not be predicted either by the theory of equipotentiality or the theory of irreversible determin-ism: in the absence of severe seizures (which seem to preclude recovery to normal levels of language in most cases), the best outcomes in both verbal and nonverbal IQ are seen either with congenital lesions (pre- or perinatal) or with lesions that occur between 4-12 years of age! It is of course possible that this U-shaped function is an artifact of other methodological factors, including etiology (e.g., the medical conditions that lead to unilateral injury, including stroke, may be quite different in infants, preschool children and children in the elementary school years) and the developmental status of the child when testing occurs (e.g., grade school children may have more sophisticated behavioral strategies at their disposal, permitting them to perform better on standardized tests in the short run, and to exploit their residual plasticity and recover to higher levels in the long run). It is also possible that this result would not replicate with even larger samples (e.g., according to Vargha-Khadem, personal communi-cation June 1999, the significant U-shaped function reported for her unpublished data by Bates, Vicari, and Trauner, 1999, dropped below significance when the same was expanded to include more than 300 cases). For present purposes, we can only conclude that the limits of plasticity and capacity for recovery in young children are still unknown, and that there is ample reason for families of children with unilateral injury to be hopeful about their children's chances for recovery.

Lesion type: site and size. Earlier studies (in-cluding most of the studies reviewed in Tables 1 and 2) have been restricted to a global distinction between left- and right-hemisphere damage, often established via external neurological signs like hemiparesis. More recent studies have taken advantage of structural brain imaging, and have begun to qualify the crude distinction between LHD and RHD with further distinctions revolving around lesion size, the presence/absence of subcortical damage, and the lobes of the damaged hemisphere that are involved. Nevertheless, the term "focal brain injury" is still defined quite broadly in most studies, referring to a single (contiguous) lesion restrict-ed to one half of the brain, of any size, cortical and/or subcortical.

Variations in lesion size merit consideration, although evidence on the contribution of lesion size to language outcomes is still mixed. Lashley's ***principle of mass action*** (the complement of equipotentiality) predicts that larger lesions will have greater behavioral repercussions, with the less chance for functional recovery. His experiments with adult rats supported this idea. However, Irle (1990) carried out a meta-analysis of over 200 lesion studies in monkeys, and found that while lesion size did affect skill reacquisition, the function was curvilinear; midsized lesions were significantly more likely to cause permanent damage than small lesions or large lesions, with the latter including lesions of up to 60% of total brain tissue. At first glance this result is counterintuitive, but Irle suggests a compelling explanation that she calls “the fresh-start hypothesis”: small lesions have little effect because they are small; midsize lesions are large enough to lead to permanent behavioral impairments, but not quite large enough to precipitate/cause the brain to reorganize; large lesions result in a better outcome, because the animal makes a "fresh start," abandoning the inefficient strategies that an animal with a midsized lesion still struggles to apply. Preliminary evidence by Thal et al. (discussed in more detail below) appeared to provide support for the fresh-start hypothesis, reflected in a significant U-shaped effect of lesion size on early language outcomes (i.e., small lesions or very large lesions were both associated with better language abilities than those observed in children with lesions in the middle range). However, this U-shaped function dropped below significance when the sample was doubled in size (Bates et al., 1997, discussed below), hence the fresh-start hypothesis still awaits confirma-tion, and our understanding of the effects of lesion size on language outcomes is still very slim.

Lesion etiology and its neurological cor-relates. The prospective studies reviewed below have concentrated entirely on children with congenital in-juries (before six months postnatal age) that are usually due to pre- or perinatal stroke (although it is not always possible to make a definitive diagnosis of the cause or timing of congenital injuries). We should not be surprised to find that these studies yield different results from those that have included children with trauma or tumor (Anderson et al., 1999). Results may also differ from studies of children who suffered postnatal strokes secondary to cardiac catheterization (which is often associated with a lifetime of inadequate oxygen intake), and from studies of outcomes following hemispherec-tomy in children who have suffered for many years from intractable seizures. In fact, as Vargha-Khadem and her colleagues have recently reported (see also Ballantyne and Trauner, 1999), seizures are the single greatest risk factor for language and cognitive outcomes in children with unilateral brain injury. We also need to consider when the seizure condition appeared and its subsequent course. For example, no effects of seizure history were found in prospective studies of early language develop-ment (Thal et al. and Bates et al., discussed below). However, such studies necessarily conflate relatively benign neonatal seizure conditions with more severe and persistent forms of epilepsy that may not appear for months or even years after birth.

Sample size. Sample size is a banal but poten-tially powerful factor to consider when evaluating studies with discrepant results. There are massive individual differences in the rate and nature of language development in perfectly

normal children (Bates, Dale, and Thal, 1995). Unilateral injuries are superimposed upon this landscape of variation, which means that single-case studies or small-sample studies must be interpreted with caution. Consider a recent report by Stark and McGregor (1997) on two cases of childhood hemispherectomy, to the left and right hemispheres respectively. These authors report an "adult-like" pattern: selectively greater deficits for language in the case of LHD, compared with a more even profile of delay in the case of RHD, results interpreted to support a mild variant of innate/irreversible determinism. However, these two cases contrast sharply with Vargha-Khadem's case study of Alex, a child with severe LHD and intractable seizures who was virtually mute when he underwent hemispherectomy at 8 years of age (Vargha-Khadem et al., 1997). After an initial delay, Alex went on to attain fluent control over language (with no articulatory problems or specific delays in grammar), commensurate with his mental age. Although case studies can be quite informative in showing us the range of outcomes that are *possible* following various forms of unilateral injury, they should not be used as the basis for generalizations about the correlation between various forms of injury and their linguistic sequelae.

Developmental sensitivity and timing of language testing. There are two related factors at issue here. First, the amount of time that has elapsed since lesion onset may influence how "recovered" a child appears during testing. That is, when children are tested in the middle school years or beyond, those who suffered their lesions earlier in life have also had more time to reorganize and recover. Second, there may be specific effects of lesion type that are only evident in particular phases of development, when children start to come to terms with the demands of a new language task. For both these reasons, studies that focus on the early stages of language may yield qualitative informa-tion about the initial state of the system, and about the processes involved in plastic reorganization of language and other cognitive functions.

Most of the studies summarized in Tables 1 and 2 have been *retrospective* in nature, testing children well after the period in which language is usually acquired and (we presume) after much of the recovery for which this population is so famous has already occurred. For the remainder of this chapter, we will concentrate on developmental studies of children with focal brain injury that take the children's level of development into account, tracking change over time using age- and stage-appropriate language outcome measures. In particular, we will focus on *prospective studies* of children with congenital injuries to one side of the brain, relying primarily on studies by the San Diego group and their collaborators.

Phase III: The Emergentist View

All of the studies that we will consider here involve children with congenital injuries (prior to six months of postnatal age), producing a single contiguous lesion (though often very large) confined to one side of the brain. These lesions are due primarily to pre- or peri-natal stroke, and in all cases have been confirmed by CT or MRI. Children were excluded if the lesion was due to tumor, trauma or arterio-venal malformation, or any form of diffuse or multifocal brain damage, or if they suffered from any serious medical conditions (other than seizures subsequent to the lesion itself). All children come from families in which the predominant language is English, and although they represent a broad socio-economic spectrum, children of middle-class parents tend to predominate (as they do in much of the behavioral literature in developmental psychology).

The San Diego group and their collaborators have conducted cross-sectional and longitudinal studies of this clinical group for approximately 15 years, focusing on many aspects of development including visual-spatial cognition, attention and hemispatial neglect, perception and production of facial and vocal affect. We will concentrate here on studies of speech and language. For reviews of development in other domains, see Stiles, 1995; Stiles, this volume; Stiles et al., 1998. For more detailed reviews of language development in this popu-lation, see Bates et al., in press; Bates, Vicari, and Trauner, 1999; Broman and Fletcher, 1999; Elman et al., 1996.

We will start with results of cross-sectional studies that focus on development after 5 years of age, which largely confirm results of other large-sample studies of language outcomes in this population. Then we will end with studies that have examined the acquisition of language in this population, starting in the first year of life. These studies demonstrate that side- and site-specific biases are present early in life; although the lesion-symptom correlations observed in these studies do not map directly onto the patterns observed in adults, different lesions have different effects on early language learning that must be overcome. The fact that they are overcome (disappearing entirely by 5-7 years of age in the domain of language) provides powerful evidence for the plastic and experience-dependent nature of brain and behavioral development. Furthermore, the evidence sug-gests that language learning itself is the catalyst for this reorganization.

Starting with studies of language outcomes at later stages of development, Bates, Vicari and Trauner (1999) summarize performance by 43 English-speaking child-ren from the San Diego sample (28 LHD and 15 RHD) and 33 Italian-speaking children (18 LHD and 15 RHD) from Rome, tested cross-sectionally between 3 and 14 years of age. Mean full-Scale IQs were in the low-normal range (94-97), although the range was quite broad (from 40 to 140). There were also more cases in the below-80 range (which some investigators use as a cut-off for mild mental retardation) than we would expect if we were drawing randomly from the normal population. However, there were absolutely no differen-ces between LHD and RHD children in full-scale, verbal or nonverbal IQ. For the Italian sample, Bates et al. also summarize performance on several language tests, including lexical comprehension (an Italian version of the Peabody Picture Vocabulary Test), lexical pro-duction (an Italian adaptation of the Boston Naming Test), grammatical comprehension (the Token Test and an Italian version of the Test of Receptive Grammar), and

semantic category fluency. Again, although brain-injured children performed significantly below normal controls on all language measures except the TROG, there was no evidence whatsoever for a difference between LHD and RHD on any measure. Furthermore, when mental age was controlled in analyses of covari-ance, the difference between brain-injured children and normal controls disappeared for every measure except the Boston Naming Test.

These cross-sectional results suggest that the plastic reorganization for which this population is known takes place prior to 5-7 years of age. As a result, children with early focal brain injury recover far better (relative to age-matched controls) than adults with comparable injuries. Although this conclusion has been around for quite a while, and there is a large body of evidence on plasticity from animal research to support it, adults and children have rarely been compared directly, on a common set of measures. More direct comparisons would be helpful in assessing the nature and magnitude of this presumed plasticity. We are aware of only three studies (all by the San Diego group and their collaborators) that have compared school-age children and adults directly on the same measures (other than verbal and nonverbal IQ), using z-scores based on data from age-matched controls.

The first study in this series, by Kempler et al. (1999), compared adults with RHD and LHD to a sample of 6-12-year-old children who had suffered comparable injuries (also due to cerebrovascular acci-dents or CVA) during the pre-/perinatal period. Child and adult patients with LHD vs. RHD were compared directly in an age-by-side-of-lesion design, using age-based z-scores derived from relatively large samples of age-matched controls on the van Lancker and Kempler Familiar Phrases Test. As can be seen in Figure 1, RHD and LHD adult patients display a double dis-sociation on this task. LHD patients have more difficulty on familiar phrases, whereas patients with RHD are significantly worse on idioms or familiar phrases matched for length and complexity. As Figure 1 also shows, child patients displayed absolutely no evidence for a double dissociation; children with LHD vs. RHD both performed significantly below normal controls as a group, but did not differ significantly from each other. Even more important, the child patients performed within the low-normal range on both measures, while the adult patients performed many standard deviations below their age-matched controls on their weakest measure (i.e., novel phrases for patients with LHD; familiar phrases for patients with RHD). In other words, the children were not significantly impaired (i.e., their performance did not reach criteria required to establish the existence of a language deficit) following either right- or left-hemisphere damage, and no selective effects of lesion side were detected.

The second study, by Dick et al. (1999), compared performance by children and adults with unilateral brain injury and their age-matched controls in an on-line auditory sentence comprehension test that contrasts syntactically simple sentences (active and subject clefts that follow canonical word order) with syntactically complex sentences (passives and object clefts that violate canonical word order). All sentences were fully grammatical, and semantically reversible. All groups (including normal controls) displayed the same basic profile of lower accuracy on noncanonical sentences (object clefts and passives). Among the children, group by sentence type interactions were obtained indicating that (1) the youngest normal children were at a greater disadvantage than older children on the more difficult noncanonical sentence types, (2) as a group, brain-injured children showed a greater disadvantage on the difficult sentences than their age-matched controls, (3) however, the brain-injured children were still within the normal range for their age, and most important for our purposes here, (4) there were no significant differences between children with LHD and children with RHD on any of the sentence types. In contrast with these findings for children, adults with unilateral brain injury were severely impaired, especially on the noncanonical sentences. Direct comparisons of adults and children with LHD clearly demonstrate that LHD is associated with receptive agrammatism in adults but not in children.

The third study in this series focused on language production instead of comprehension, based on samples of free speech (Bates, Wulfeck, et al. 1999), collected within the framework of a biographical interview tailored to reflect the different interests of children and adults. Participants included 38 brain-injured children (24 LHD, 14 RHD) between 5-8 years of age, 38 normal controls matched for age and gender, 14 adults with LHD (including 3 Broca's aphasics, 3 Wernicke's aphasics, 5 anomic aphasics, and 3 nonaphasic pa-tients), 7 adults with RHD, and 12 adult controls in the same range of age and education. The structured interviews were videotaped and transcribed following conventions of the Child Language Data Exchange System, and coded into various categories assessing amount of speech (number of word types, word tokens, morphemes, and utterances), length (mean length of utterance in morphemes, or MLU), grammatical com-plexity (number of complex syntactic structures, in both types and tokens), and errors (word omissions, morphological errors, lexical errors). Although it was generally true that children talk far less than adults (including adult aphasics), when proportion scores were used to correct for overall amount of output, results were exceedingly clear: (1) there were absolutely no differences between children with LHD vs. RHD on any measure; (2) in this open-ended free-speech task, there were also very few differences between brain-injured children (combining LHD and RHD) and their controls (the exceptions were small but significant disadvantages for FL children as a group in number of word omission errors and in number of word types); (3) in striking contrast to the child data, there were huge differences between adults with LHD vs. RHD on virtually every measure, in the predicted directions; (4) LHD adults also showed qualitative variations in their symptoms, reflect-ing different aphasia subtypes (e.g., more morpho-logical and omission errors in Broca's aphasics, more lexical errors in Wernicke's aphasics). One small illustration of these results can be seen in Figure 2a, which plots the total number of errors per proposition in children vs.

adults within each lesion group, and Figure 2b, which plots the same data for LHD and RHD children and adults in z-scores based on performance by age-appropriate controls. Figure 2a shows that error rates are certainly higher for children than adults (as we have known for many years), but Figure 2b shows that LHD and RHD children are very close to normal (with z-scores close to zero) while the worst aphasics produce error rates that are orders of magnitude higher than normal controls (whose error rate is extremely small, leading to very small standard deviations). Although these results are not surprising, in view of the accu-mulated evidence for plasticity following early brain injury in humans and in other species, they document this phenomenon with exceptional clarity.

This brings us to a summary of evidence by the same research group looking at the first stages of language development, prior to 5-7 years of age.

In a study focusing on the earliest stages of language development, Thal et al. (1991) describe results for 27 congenitally brain-damaged infants be-tween 12 and 35 months of age, using an early version of the MacArthur Communicative Development Inven-tories, or CDI (Fenson et al., 1993), a parent-report instrument for the assessment of early lexical and grammatical development. Delays in word comprehen-sion in the very first stages of development were actually more common in children with RHD. Delays in first word production occurred for almost all the brain-injured children, regardless of lesion side or site, but tended to be more severe in children with left posterior damage -- an apparent reversal of the expected association between comprehension deficits and damage to Wernicke's area.

Bates et al (1997) followed up on Thal et al. (1991) with a larger sample, using a combination of CDI data and free speech to assess early language development in 53 children between 10 and 40 months of age (36 LHD, 17 RHD), including 18 of the 27 cases from Thal et al. The study was divided into three cross-sectional epochs (although many of the children participated in more than one): a period focusing on the dawn of word com-prehension, word production and gesture (26 children from 10-17 months), a second substudy focusing on word production and the emergence of grammar (29 children from 19-31 months), and an analysis of grammatical development from free-speech samples (30 children from 20 and 44 months). Performance at these various stages of development was evaluated in comparisons based on lesion side, lesion size, and lesion site (i.e., whether or not the frontal lobes or temporal lobes were involved). There were no effects of lesion size in any of these analyses (including a failure to replicate the U-shaped effect of lesion size described by Thal et al., as we discussed earlier). Interesting effects of lesion side and intrahemispheric lesion site did emerge, but in complex patterns that are surprising from the point of view of the adult aphasia literature.

Between 10-17 months, delays in receptive language were particular evident in children with RHD (i.e., more RHD cases than we would expect by chance fell into bottom 10th percentile for word comprehen-sion). By contrast, the LHD children performed within the normal range on word comprehension, even if their lesions involved temporal lobe (the presumed site of Wernicke's area, which is implicated in moderate to severe forms of receptive aphasia in adults). However, there was no significant difference between LHD and RHD on direct statistical comparisons, so the RHD disadvantage is not robust and should be investigated further. There was also a significant RHD disadvantage in the development of communicative and symbolic gesture, and this time the RHD disadvantage did reach significance in a direct LHD/RHD comparison. This result is also surprising, since deficits in the production of symbolic gestures are atypically associated with left-hemisphere damage when they occur in adults (Goodglass, 1993). Finally, Bates et al. do report a selective delay in expressive vocabulary for children with LHD. However, in line with the earlier report by Thal et al., this disadvantage was only evident in children whose lesions involved the temporal lobe.

The second substudy followed children's language development between 19-31 months, when the so-called vocabulary burst is said to occur (e.g., an intense period of development for vocabulary/lexical production), and when children's comprehension is often so vast it is difficult to measure. This is also the period in which children typically start to combine words, followed by the emergence of grammatical inflections and function words. For the 29 children whose scores on this scale were obtained, a selective disadvantage for children with LHD appeared both for expressive vocabulary and the emergence of grammar (with no evidence whatsoever for a dissociation between grammatical and lexical pro-duction). However, this LHD disadvantage was due once again to children with left temporal involvement, in contrast with the typical adult pattern in which expressive deficits (especially nonfluent aphasia) are usually associated with left frontal involvement (i.e., Broca's area and adjacent cortical and subcortical regions). Similar delays in expressive vocabulary and grammar appeared when children with frontal lobe involvement were compared with children whose lesions spared the frontal lobe. However, in contrast with the asymmetrical left temporal disadvantage that we have just discussed, this frontal effect was perfectly sym-metrical: delays were equally severe with left frontal or right frontal lesions.

Curiously, an abnormal proportion of the children with RHD were also producing a higher than normal number of function words for their vocabulary size. As described in some detail by Bates, Bretherton and Snyder (1988) and by Bates et al. (1994), such overuse of function words for children in the early stages of vocabulary development (i.e., under 400 words) is definitely *not* a sign of precocious grammar. In fact, children who overuse pronouns and other function words in the early stages tend to be relatively slow in grammatical development later on. For these children, function words tend to appear in frozen or rote expressions like "I wan dat", a style of early expressive language that has been called "pronominal style", or "holistic style." At the opposite end of the continuum are children who avoid function words in their

first word combinations, producing telegraphic utterances like "Adam truck" or "Mommy sock". This style of early expressive language has been referred to as "nominal style" or "analytic style". Given the terms "holistic" and "analytic", which are often attributed to right- vs. left-hemisphere processing, respectively, one might have predicted that holistic style would be more common in children with LHD (who are presumably relying more on holistic right-hemisphere processes to acquire lan-guage). This prediction is roundly contradicted by the Bates et al. study, where holistic style was robustly associated with RHD (indicating that the overproduction of function words in early speech reflects reliance on the intact left hemisphere). Bates et al (1997) suggest that children with RHD are relying heavily on the more precise acoustic analysis and/or greater acoustic memory available in the left hemisphere, storing up frozen expressions that they are unable to segment or understand beyond a relatively superficial level of analysis (rather like an American who says "Gesund-heit" when someone sneezes, with no idea whatsoever regarding the structure or meaning of that word in German). This would mean, in turn, that right-hemi-sphere processes are very important in the early stages of language learning for the breakdown of acoustic material and its integration into a larger cognitive system. However, once the material has been analyzed, understood and integrated into a larger framework, the contribution of the right hemisphere may be much less important, so that control may shift (in the undamaged brain) to rapid, automatic processes mediated primarily by the left hemisphere.

In the third and final subgroup of children, Bates et al. (1997) collected free-speech samples between 21 and 44 months of age. As their CDI scores predicted, the MLU scores of children with damage that encompassed the left temporal region of their brain were significantly lower than normal, and significantly lower than scores for brain-injured children whose lesions spared this region (including all children with RHD and the subset of LHD children with no temporal involvement). Children with right or left frontal damage also still looked delayed, but this difference was not statistically significant in the 21-44-month subsample.

Vicari et al. (in press) attempted a partial repli-cation of the Bates et al. results for early lexical development, administering an Italian version of the MacArthur CDI to the parents of 43 children between 13 and 46 months of age. Their study differed from the methods used by Bates et al. in two crucial respects: children beyond the age range covered by the MacArthur CDI were included in the study (which means that they could not use age-based percentile scores), and parents were given the Infant or the Toddler version of the MacArthur based not on age but on their child's current level of linguistic ability (children who were still in the one-word stage were assigned the infant form, but children who were starting to combine words were assigned the toddler form). For these reasons, the studies are not entirely comparable, but results replicate and extend the Bates et al. findings in some interesting directions. First, Vicari et al. also report a massive across-the-board delay in early vocabulary development for brain-injured children as a group. Hence, even though the long-term prospects for these children are relatively good, it is obviously hard to get language off the ground when significant damage has occurred to either hemisphere. Second, Vicari et al. report a large and significant interaction between side of lesion (LHD vs. RHD) and stage of language development (single word vs. multiword). Among children who were still in the one-word stage, LHD were significantly slower in vocabulary development than RHD (since 10 out of 12 of the one-word-stage children with LHD had temporal lobe involvement, a specific replication of Bates et al.'s left temporal findings was not possible). By contrast, among children who were now in the multiword stage, the LHD disadvantage had disappeared entirely. In fact, LHD children in the multiword group had a numerical advantage over their RHD counterparts. This advantage was not statistically significant, but it contributed to the robust interaction between language stage and lesion side. Vicari et al. suggest that recovery from this initial delay may begin very early for some children, and may be forced in part by the delay itself. That is, children who are particularly disadvantaged in the first stages of language acquisition (e.g., LHD cases) may be forced to abandon a failing strategy in favor of some alternative approach, leading to earlier and (ultimately) more suc-cessful language learning.

Reilly et al., 1998, conducted a cross-sectional study of 15 RH- and 15 LH-damaged children, between 3 and 12 years if age, using a story-telling format (the well-known Frog Story narratives -- Berman and Slo-bin, 1994) to assess lexical, grammatical and discourse development. For children between 3 and 6 years of age, the now-familiar left temporal disadvantage was ob-served in syntactic complexity and in persistence of morphological errors. However, this effect of lesion site was not observed in children between 6-12 years of age. Among the older children, there were still significant differences between focal lesion children (LHD and RHD combined) and their age-matched controls on a number of measures, but the focal lesion children were nevertheless performing within the normal or low-normal range. Hence the Reilly et al. results for grammar suggest a later variant of the recovery pattern that Vicari et al. observed within the lexical domain.

Because the Reilly et al. and Vicari et al. studies are both cross-sectional, it would be very useful to replicate these results with longitudinal samples. Although their results are still preliminary, based on a relatively small sample, Reilly and colleagues (Losh, Reilly, and Bates, 1996) have tested a longitudinal subgroup across the 5-7-year age range that seemed to be a watershed in their cross-sectional study. They report that children with left temporal involvement do indeed move sharply upward in syntax and morphology across this age range, scoring numerically above children with RHD at the later time point. The general picture seems to be one in which children with LHD display sharper or "steeper" growth functions, while children with RHD show a flatter profile of growth in the language domain.

Summary and Conclusion

Putting these lines of evidence together, we may conclude (or perhaps hypothesize) that the infant brain contains strong biases that, in the absence of early brain damage, guarantee the eventual emergence of left-hemisphere specialization for language. Although (if anything) the right hemisphere seems to play a more important (or at least equally important) role in the emergence of word comprehension and communicative gesture, progress in expressive language (both lexical and grammatical) seems to be delayed with frontal damage (to either side of the brain) and with temporal damage (but, in this case, temporal damage restricted to the left hemisphere). In other words, there is an early bias that predisposes the left hemisphere to "take over" rapid and efficient production of words and sentences, a development which may also result in the emergence of left-hemisphere specialization for many aspects of receptive language as well. In the absence of evidence to the contrary, one might have assumed that this early left-hemisphere advantage for speech/language production (but not reception) has a motor base. And yet several studies by the San Diego group suggest that the source of this left-hemisphere bias lies primarily within the temporal lobe, a region that is supposed to be specialized for perception rather than production.

In this regard, Bates et al. note that some children with severe otitis media (i.e., middle-ear infections) also show selective delays in the emergence of expressive (but not receptive) language. Why would middle-ear impedance have greater effects on language production than comprehension? The answer may lie in a simple fact: language learning is not the same thing as fluent language use. When a child is trying to break into the language system for the first time, the amount of perceptual analysis required to produce her own versions of a new word is greater than the amount of perceptual analysis that she needs to recognize the word (especially if she is asked to recognize that word in a richly supportive social and physical context, which can be integrated with the acoustic signal to achieve compre-hension). If these assumptions are correct, then we can put the story together as follows: left temporal regions may be particularly well suited (perhaps at or before birth) for the extraction of perceptual detail. Indeed, there is ample evidence from visual-spatial processing in adults to support this view, hence the hypothesized "perceptual detail advantage" would not be specific to language, or even to audition. However, such a bias would be particularly relevant in the first stages of language learning, leading (in the absence of injury) to the establishment of left-hemisphere dominance. What these prospective studies do clearly show is that this bias is "soft", and can be overcome. Indeed, by 5-7 years of age the initial disadvantages associated with left-hemisphere damage seem to have disappeared, or at least, fallen below the levels that we are able to detect with the measures that we have developed so far.

Finally, it appears from these studies that the emergence of organization for language (in the un-damaged brain) and reorganization for language (in the damaged brain struggling to overcome initial biases) both occur within the period in which language is acquired, i.e., somewhere between birth and 5 years of age. We may speculate that this correlation between brain and behavioral development is no accident. In fact, we propose that learning itself plays a major role in organizing the brain for efficient language use, as children struggle to find an optimal solution to the challenges associated with language and communica-tion.

REFERENCES

Alajouanine, T. and F. Lhermitte, 1965. Acquired aphasia in children. *Brain*, 88:653-662.

Anderson, S.W., A. Bechara, H. Damasio, D. Tranel, and A.R. Damasio, 1999. Impairment of social and moral behavior related to early damage to human prefrontal cortex. *Nat. Neurosci.* 2:1032-1037.

Aram, D.M., B. Ekelman, D. Rose, and H. Whitaker, 1985. Verbal and cognitive sequelae following unilateral lesions acquired in early childhood. *J. Clin. Exp. Neuropsychol.* 7:55-78.

Aram, D., B. Ekelman, and H. Whitaker, 1986. Spoken syntax in children with acquired unilateral hemi-sphere lesions. *Brain Lang*. 27:75-100.

Aram, D.M., S.C. Meyers, and B.L. Ekelman, 1990. Fluency of conversational speech in children with unilateral brain lesions. *Brain Lang*. 38:105-121.

Ballantyne, A.O., K.M. Scarvie, and D. Trauner, 1994. Verbal and performance IQ patterns in children after perinatal stroke. *Dev. Psychol*. 10:39-50.

Ballantyne, A.O. and D.A. Trauner, 1999. Neuro-psychological profiles of infants and children with early focal brain damage. *JINS* 5:145.

Basser, L., 1962. Hemiplegia of early onset and the faculty of speech with special reference to the effects of hemispherectomy. *Brain* 85:427-460.

Bates, E., in press. On the nature and nurture of language. In *Frontiere della Biologia [Frontiers of Biology]. The brain of homo sapiens*, R. Levi-Montalcini, D. Baltimore, R. Dulbecco, and F. Jacob, series eds. and E. Bizzi, P. Calissano, and V. Volterra. vol. eds. Rome: Giovanni Trecanni.

Bates, E. (1999). Plasticity, localization and language development. In *The Changing Nervous System: Neurobehavioral Consequences of Early Brain Disorders*, S. Broman and J.M. Fletcher, eds. New York: Oxford University Press, pp. 214-253.

Bates, E., I. Bretherton, and L. Snyder, 1988. *From First Words to Grammar: Individual Differences and Dissociable Mechanisms*. New York: Cambridge University Press, 1988. [Paperback edition issued 1991].

Bates, E., P.S. Dale, and D. Thal, 1995. Individual differences and their implications for theories of language development. In *Handbook of Child Language*, P. Fletcher and B. MacWhinney, eds. Oxford: Basil Blackwell, pp. 96-151.

Bates, E., V. Marchman, D. Thal, L. Fenson, P.S. Dale, J.S. Reznick, J. Reilly, and J. Hartung, 1994. Developmental

and stylistic variation in the composition of early vocabulary. *J. Child Lang*. 21:85-123.

Bates, E., D. Thal, B.L. Finlay, and B. Clancy, in press. Early language development and its neural correlates. In *Handbook of Neuropsychology, Vol. 7: Child Neurology* (2nd ed.)., F. Boller and J. Grafman, series eds. and I. Rapin and S. Segalowitz, vol. eds. Amsterdam: Elsevier.

Bates, E., D. Thal, D. Trauner, J. Fenson, D. Aram, J. Eisele, and R. Nass, 1997. From first words to grammar in children with focal brain injury. *Dev. Neuropsychol*. 13:447-476.

Bates, E., S. Vicari, and D. Trauner, 1999. Neural mediation of language development: Perspectives from lesion studies of infants and children. In *Neurodevelopmental Disorders*, H. Tager-Flusberg, ed. Cambridge, MA: MIT Press, pp. 533-581.

Bates, E., B. Wulfeck, M. Opie, J. Fenson, S. Kriz, J. Reilly, N. Dronkers, L. Miller, R. Jeffries, and K. Herbst, 1999. Comparing free speech in children and adults with left- vs right-hemisphere injury (Abstract). *Brain Lang*. 69:377-379.

Berman, R.A. and D.I. Slobin, 1994. *Relating Events in Narrative: A Cross-linguistic Developmental Study* [in collaboration with Ayhan Aksu-Koc et al.]. Hillsdale, NJ: Erlbaum.

Bernhardt, M., 1897. In Nothnagel. *Spezielle Patho-logie und Therapie, 9*, Part 2. Vienna.

Botha, R.P., 1989. *Challenging Chomsky: The Generative Garden Game*. Oxford, UK; New York: Basil Blackwell.

Broman, S., and J.M. Fletcher, eds., 1999. *The Changing Nervous System: Neurobehavioral Consequences of Early Brain Disorders*. New York: Oxford University Press.

Brown, C.M. and P. Hagoort, eds. 1999. *The Neuro-cognition of Language*. Oxford University Press.

Clarus, A., 1874. Ueber Aphasie bei Kindern [Aphasia in children]. *Jahresb.Kinderheilkd* ..7: 369-400.

Cotard, J., 1868. *Etude sur l'atophie partielle du cerveau*. Thèse de Paris.

Curtiss, S., 1977. *Genie: A Psycholinguistic Study of a Modern-Day Wild Child*. New York: Academic Press.

Deacon, T., 1997. *The Symbolic Species: The Co-evolution of Language and the Brain*. New York: Norton.

Dennis, M., 1980. Capacity and strategy for syntactic comprehension after left or right hemidecortication. *Brain Lang*. 10:287-317.

Dennis, M. and B. Kohn, 1975. Comprehension of syntax in infantile hemiplegics after cerebral hemidecortication. *Brain Lang*. 2:472-482.

Dennis, M., M. Lovett, and C.A. Wiegel-Crump, 1981. Written language acquisition after left or right hemidecortication in infancy. *Brain Lang*. 12:54-91.

Dennis, M. and H.A. Whitaker, 1976. Language acqui-sition following hemidecortication: Linguistic superiority of the left over the right hemisphere. *Brain Lang*. 3:404-433.

Dennis, M. and H. Whitaker, 1977. Hemispheric equipotentiality and language acquisition. In *Language Development and Neurological theory*, S.J. Segalowitz and F.A. Gruber, eds. New York: Academic Press, pp. 93-106.

Dick, F., B. Wulfeck, E. Bates, D. Saltzman, N. Naucler, and N. Dronkers, 1999. Interpretation of complex syntax in aphasic adults and children with focal lesions or specific language impairment (Abstract). *Brain Lang*. 69:335-336.

Eisele, J. and D. Aram, 1993. Differential effects of early hemisphere damage on lexical comprehension and production. *Aphasiology* 7:513-523.

Eisele, J. and D. Aram, 1994. Comprehension and imitation of syntax following early hemisphere damage. *Brain Lang*. 46:212-231.

Eisele, J. and D. Aram, 1995. Lexical and grammatical development in children with early hemisphere damage: A cross-sectional view from birth to adolescence. In *The Handbook of Child Language*, P. Fletcher and B. MacWhinney, eds. Oxford: Basil Blackwell, pp. 664-689.

Elman, J.L., E. Bates, M. Johnson, A. Karmiloff-Smith, D. Parisi, and K. Plunkett, 1996. *Rethink-ing Innateness: A Connectionist Perspective on Development*. Cambridge, MA: MIT Press/Bradford Books [paperback edition published 1998].

Fenson, L., P.S. Dale, J.S. Reznick, D. Thal, E. Bates, J. Hartung, S. Pethick, and J. Reilly, 1993. *The MacArthur Communicative Development Inventories: User's Guide and Technical Manual*. San Diego: Singular Publishing Group.

Fletcher, P. and B. MacWhinney, eds., 1995. *Handbook of Child Language*. Oxford: Basil Blackwell.

Fodor, J.A., 1983. *The Modularity of Mind: An Essay on Faculty Psychology*. Cambridge, MA: MIT Press.

Gazzaniga, M.S. and R.W. Sperry, 1967. Language after section of the cerebral commissures. *Brain* 90:131-148.

Geschwind, N. and E. Kaplan, 1962. A human cerebral deconnection syndrome. *Neurology* 12:675-685.

Goodglass, H. (1993). *Understanding aphasia*. San Diego: Academic Press.

Goodman, R. and C. Yude, 1996. IQ and its predictors in childhood hemiplegia. *Dev. Med. Child Neurol*. 38:881-890.

Gopnik, M. (1990). Feature-blind grammar and dys-phasia. *Nature* 344(6268):715.

Gopnik, M. and M.B. Crago, 1991. Familial aggregation of a developmental language disorder. *Cognition* 39:1-50.

Irle, E., 1990. An analysis of the correlation of lesion size, localization and behavioral effects in 283 published studies of cortical and subcortical lesions in old-world monkeys. *Brain Res. Rev.* 15:181-213.

Johnson, M.H., 1997. *Developmental Cognitive Neuroscience: An Introduction*. Cambridge, MA: Blackwell Publishers.

Kempler, D., D. van Lancker, V. Marchman, and E. Bates, 1999. Idiom comprehension in children and adults with unilateral brain damage. *Dev. Neuropsychol*. 15:327-349.

Lashley, K.S. (1950). In search of the engram. *In Symposia of the Society for Experimental Biology, No. 4 Physiological mechanisms and animal behaviour*. New York: Academic Press, pp. 454-482.

Lenneberg, E.H., 1967. *Biological Foundations of Language*. New York: Wiley.

Levine, S.C., P. Huttenlocher, M.T. Banich, and E. Duda, 1987. Factors affecting cognitive functioning of hemiplegic children. *Dev. Med. Child Neurol*. 29:27-35.

Levy, J., R.D. Nebes, and R.W. Sperry, 1971. Expressive language in the surgically separated minor hemisphere. *Cortex* 7:49-58.

Losh, M., J.S. Reilly, and E. Bates, 1996, June. *Language development: Neural plasticity in children with early focal brain injury*. Poster session presented at the 8th Annual Meeting of the American Psychological Association, San Fran-cisco.

Muter, V., S. Taylor, and F. Vargha-Khadem, 1997. A longitudinal study of early intellectual development in hemiplegic children. *Neuropsychologia* 35:289-298.

Nass, R., H. Peterson, and D. Koch, 1989. Differential effects of congenital left and right brain injury on intelligence. *Brain Cogn*. 9:258-266.

Newmeyer, F.J., 1980. *Linguistic Theory in America*. New York: Academic Press.

Newmeyer, F.J., 1997. Genetic dysphasia and linguistic theory. *Journal of Neurolinguistics* 10:47-73.

Ojemann, G.A., 1991. *Cortical organization of language*. *J. Neurosci*. 11:2281-2287.

O'Leary, D.D. and B.B. Stanfield, 1985. Occipital cortical neurons with transient pyramidal tract axons extend and maintain collaterals to subcortical but not intracortical targets. *Brain Res*. 336:326-333.

O'Leary, D.D. and B.B. Stanfield, 1989. Selective elimination of extended by developing cortical neurons is dependent on regional locale: Expe-riments utilizing fetal cortical transplants. *J. Neurosci*. 9:2230-2246.

O'Neill, Y.V., 1980. *Speech and Speech Disorders in Western Thought Before 1600*. Westport, CT: Greenwood Press.

Pallas, S.L. and M. Sur, 1993. Visual projections induced into the auditory pathway of ferrets: II. Corticocortical connections of primary auditory cortex. *J. Comp. Neurol*. 337:317-333.

Payne, B.R., 1999. Immature visual cortex lesions: Global rewiring, neural adaptations, and behavioral sparing. In *The Changing Nervous System: Neurobehavioral Consequences of Early Brain Disorders*, S. Broman and J.M. Fletcher, eds. New York: Oxford University Press, pp. 114-145.

Pinker, S., 1994. *The Language Instinct: How the Mind Creates Language*. New York: William Morrow.

Quartz, S.R. and T.J. Sejnowski, 1994. Beyond modularity: Neural evidence for constructivist principles in development. *Behav. Brain Sci*. 17:725-726.

Quartz, S.R. and T.J. Sejnowski, 1997. The neural basis of cognitive development: A constructivist manifesto. *Behav. Brain Sci*. 20:537.

Rasmussen, T. and B. Milner, 1977. The role of early left brain injury in determining lateralization of cerebral speech functions. *Ann. NY Acad. Sci*. 229:355-369.

Reilly, J., E. Bates, and V. Marchman, 1998. Narrative discourse in children with early focal brain injury. *Brain Lang*. 61:335-375.

Rice, M., ed., 1996. *Toward a Genetics of Language*. Mahwah, NJ: Erlbaum.

Riva, D., L. Cazzaniga, C. Pantaleoni, N. Milani, and E. Fedrizzi, 1986. Acute hemiplegia in childhood: The neuropsychological prognosis. *Journal of Pediatric Neurosciences* 2:239-250.

Satz, P., E. Strauss, and H. Whitaker, 1990. The ontogeny of hemispheric specialization: Some old hypotheses revisited. *Brain Lang*. 38:596-614.

Stanfield, B.B. and D.D. O'Leary, 1985. Fetal occipital cortical neurones transplanted to the rostral cortex can extend and maintain a pyramidal tract axon. *Nature* 313(5998):135-137.

Stark, R.E. and K.K. McGregor, 1997. Follow-up study of a right- and left-hemispherectomized child: Implications for localization and impairment of language in children. *Brain Lang*. 60: 222-242.

Stiles, J., 1995. Plasticity and development: Evidence from children with early focal brain injury. In *Maturational Windows and Adult Cortical Plasticity: Is There Reason for an Optimistic View? Proceedings of the Santa Fe Institute Studies in the Sciences of Complexity, Vol. 23*, B. Julesz and I. Kovacs, eds. Reading, MA: Addison-Wesley, pp. 217-237.

Stiles, J., this volume. Spatial cognitive development.

Stiles, J., E. Bates, D. Thal, D. Trauner, and J. Reilly, 1998. Linguistic, cognitive and affective development in children with pre- and perinatal focal brain injury: A ten-year overview from the San Diego Longitudinal Project. In *Advances in Infancy Research*. C. Rovee-Collier, L. Lipsitt, and H. Hayne, eds. Norwood, NJ: Ablex, pp. 131-163.

Thal, D., V. Marchman, J. Stiles, D. Aram, D. Trauner, R. Nass, and E. Bates, 1991. Early lexical development in children with focal brain injury. *Brain Lang*. 40:491-527.

Vargha-Khadem, F., L. Carr, E. Isaacs, E. Brett, C. Adams, and M. Mishkin, 1997. Onset of speech after left hemispherectomy in a nine-year-old boy. *Brain* 120:59-182.

Vargha-Khadem, F., E. Isaacs, S. van der Werf, S. Robb, and J. Wilson, 1992. Development of intelligence and memory in children with hemi-plegic cerebral palsy: The deleterious consequences of early seizures. *Brain, 115,* 315-329.

Vargha-Khadem, F., E. Isaacs, and V. Muter, 1994. A review of cognitive outcome after unilateral lesions sustained during childhood. *Journal of Child Neurology, 9(Suppl.),* 2S67-2S73.

Vargha-Khadem, F., A. O'Gorman, and G. Watters, 1985. Aphasia and handedness in relation to hemispheric side, age at injury and severity of cerebral lesion during childhood. *Brain* 108:677-696.

Vicari, S., A. Albertoni, A. Chilosi, P. Cipriani, G. Cioni, and E. Bates, in press. Plasticity and reorganization during early language learning in children with congenital brain injury. *Cortex*.

Webster, M.J., J. Bachevalier, and L.G. Ungerleider, 1995. Development and plasticity of visual memory circuits. In *Maturational Windows and Adult Cortical Plasticity: Is There Reason for an Optimistic View? Proceedings of the Santa Fe Institute Studies in the Sciences of Complexity, Vol. 23*. B. Julesz and I. Kovacs, eds. Reading, MA: Addison-Wesley, pp. 73-86.

Woods, B.T. 1980. The restricted effects of right-hemisphere lesions after age one: Wechsler test data. *Neuropsychologia* 18:65-70.

Woods, B.T., 1983. Is the left hemisphere specialized for language at birth? *Trends Neurosci*. 6:115-117.

Woods, B.T. and S. Carey, 1979. Language deficits after apparent clinical recovery from childhood aphasia. *Ann. Neurol*. 6:405-409.

Woods, B.T. and H.L. Teuber, 1973. Early onset of complementary specialisation of the cerebral hemispheres in man. *Trans. Am. Neurol. Assoc.* 98:113-117.

Woods, B.T. and H.L. Teuber, 1978. Changing patterns of childhood aphasia. *Ann. Neurol.* 3:272-280.

Xiong, J., S. Rao, J.-H. Gao, M.Woldorff, and others, 1998. Evaluation of hemispheric dominance for language using functional MRI: A comparison with positron emission tomography. *Hum. Brain Mapp.* 6:42-58.

Figures & Tables

Figure 1. Comprehension of novel and familiar phrases by children and adults with left vs. right hemisphere injury

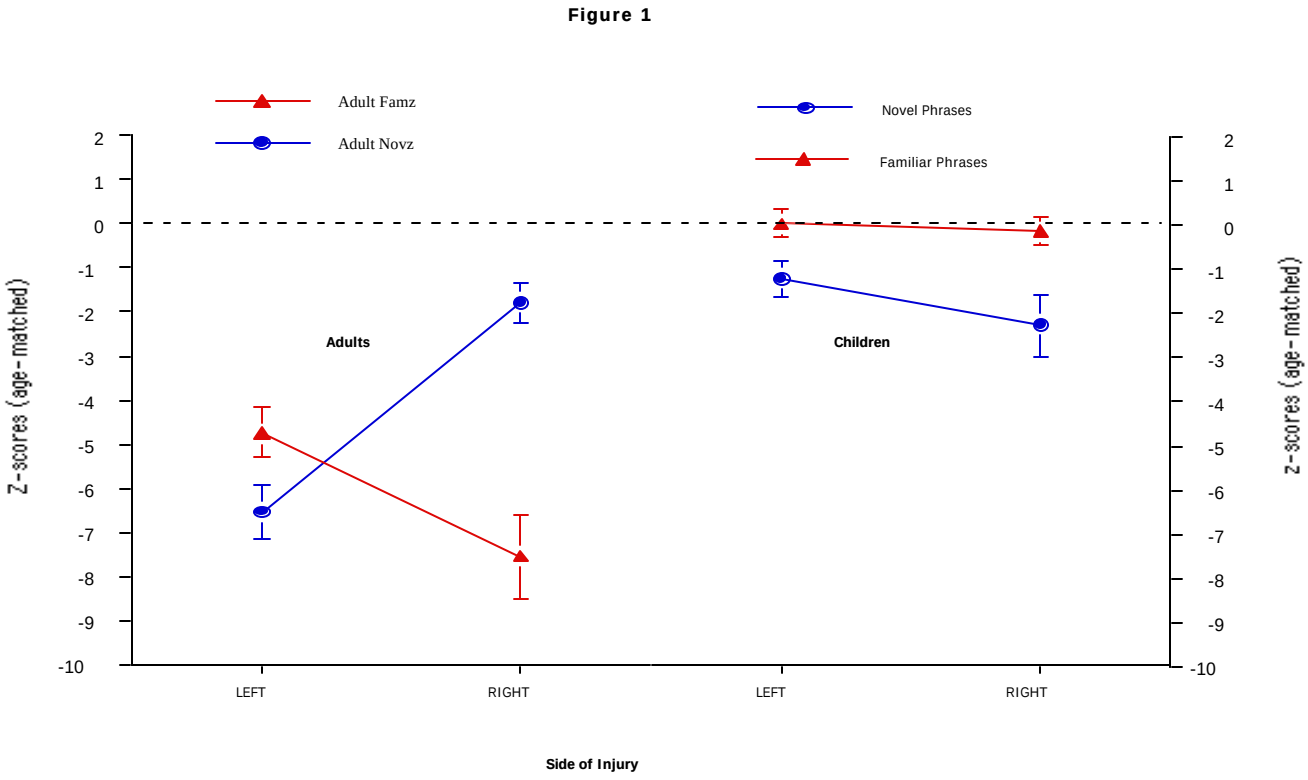


Figure2a. The mean number of errors per proposition for normally developing children, adult controls, and children and adults with right or left hemisphere damage. LHD patients are further sub-divided by aphasia type.

Figure 2a

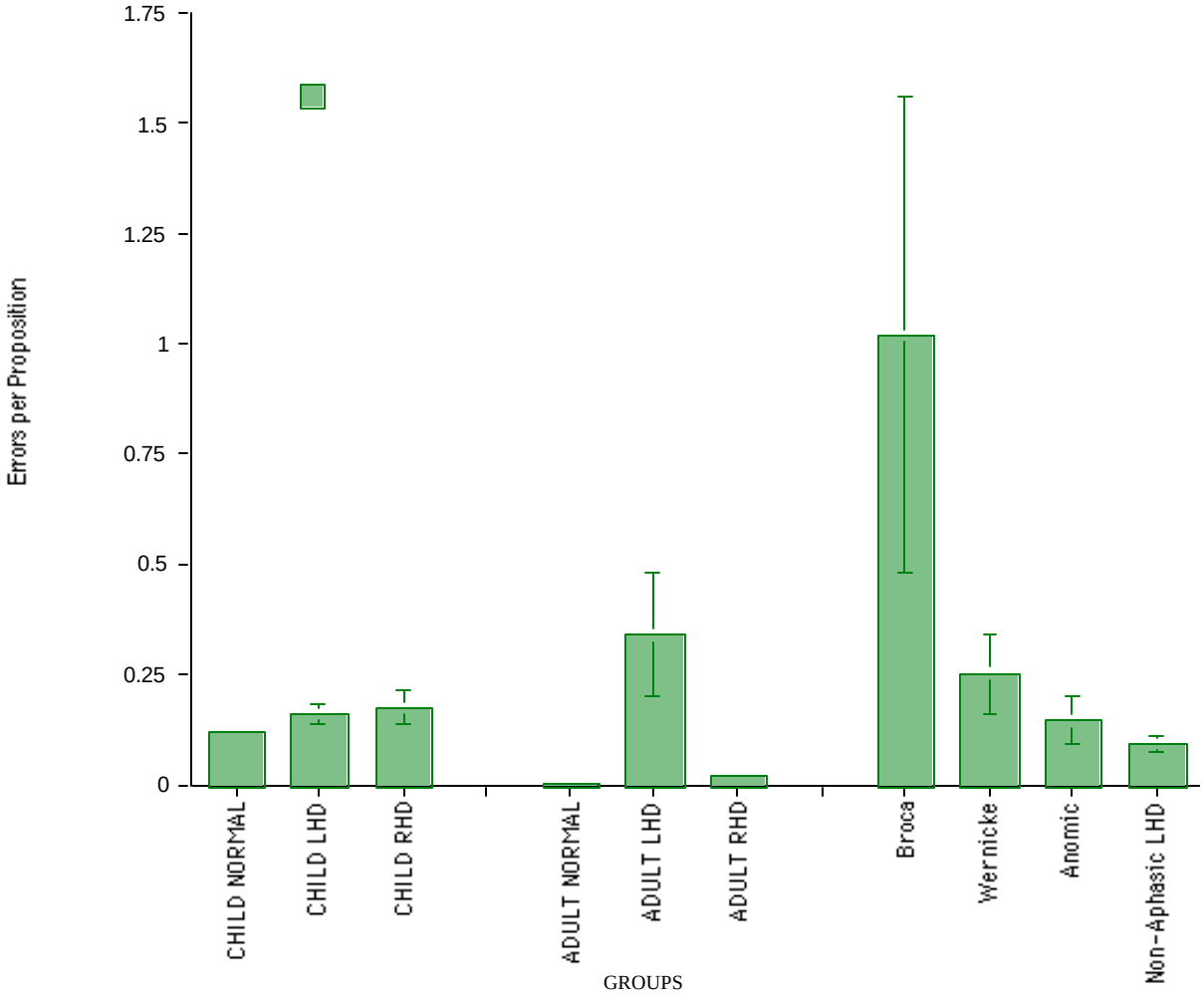
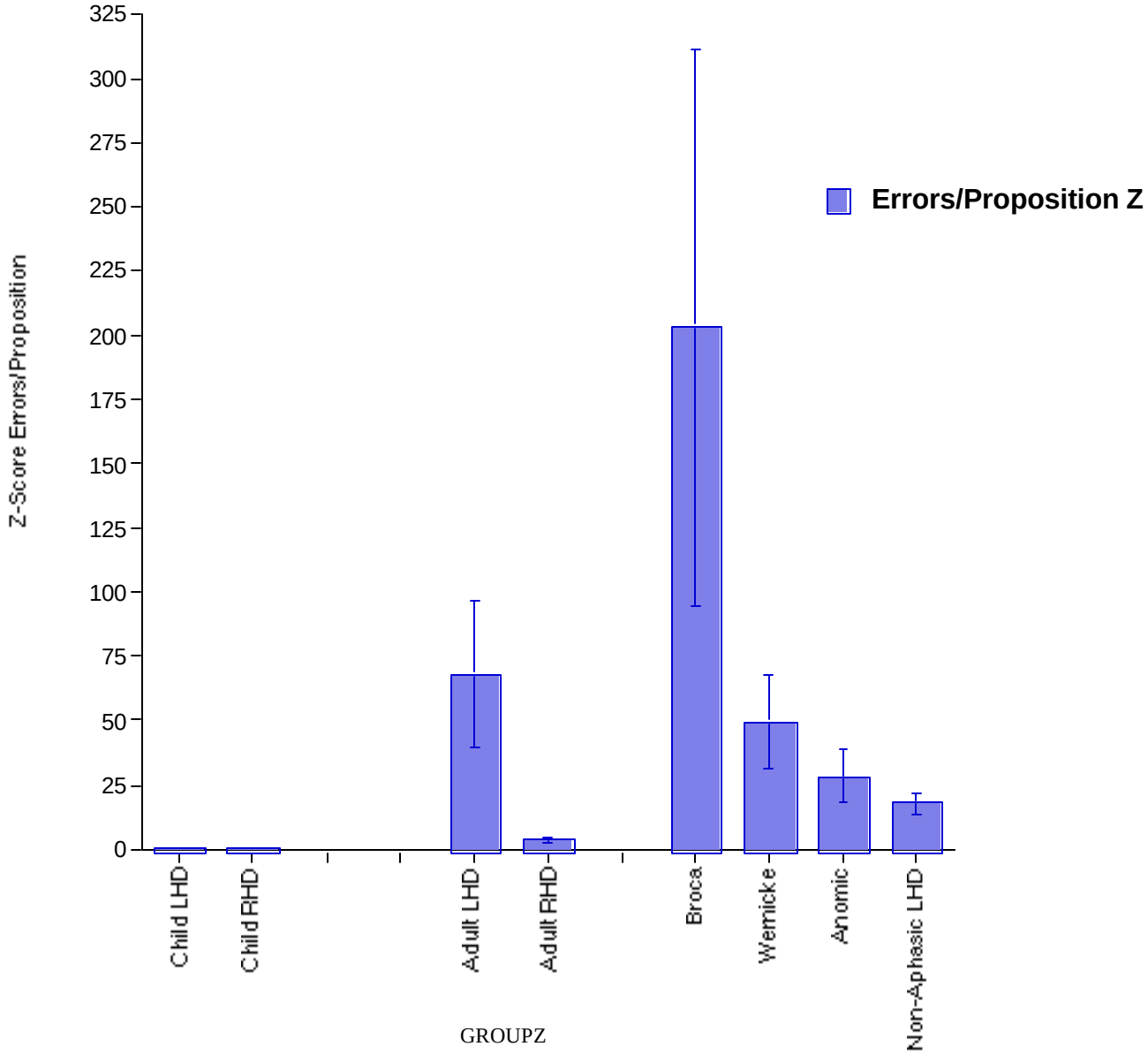


Figure2b. Z score error rates per proposition for children versus adults with right or left hemisphere damage. LHD patients are further sub-divided by aphasia type

Figure 2b


Errors/Proposition Z
Z-Score Errors/Proposition
325
300
275
250
225
200
175
150
125
100
75
50
25
0
Child LHD
Child RHD
Adult LHD
Adult RHD
Broca
Wernicke
Anomic
Non-Aphasic LHD
GROUPZ