

A Theory of Neurolinguistic Development

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This article offers a developmental theory of language and the neural systems that lead to and subserve linguistic capabilities. Early perceptual experience and discontinuities in linguistic development suggest that language develops in four phases that occur in a fixed, interdependent sequence. In each phase of language, a unique ontogenetic function is accomplished. These functions have proprietary neural systems that vary in their degree of specialization. Of particular interest is an analytical mechanism that is responsible for linguistic grammar. This mechanism is time-locked and can only be turned on in the third phase. Confirming evidence is provided by children who are delayed in the second phase of the language learning process. These children store insufficient lexical material to activate their analytic mechanism. Inactivation behaves like damage, shifting language functions to homologous mechanisms in the nondominant hemisphere, thereby increasing functional and anatomical symmetry across the hemispheres. This atypical assembly of neurolinguistic resources produces functional but imperfect command of spoken language and may complicate learning of written language. The theory thus offers a different role for genetics and early experience, and a different interpretation of neuroanatomic findings, from those entertained in most other proposals on developmental language disorders. © 1997 Academic Press

OVERVIEW

This article offers a theory according to which neurolinguistic capacity develops in individual phases that occur in a fixed and overlapping sequence. In each phase, a unique function is accomplished, and each phase has its own commitment of neural resources. The first phase is indexical and affective; the infant is strongly oriented to the human face and voice, and learns caregivers' superficial vocal characteristics. The second phase is primarily

I thank Michael Studdert-Kennedy and Noam Chomsky for helpful comments on the manuscript. Thanks are also due Dorothy Bishop, Leslie Brothers, Marcy Dorfman, Paul Macaruso, Aniruddh Patel, Elena Plante, and Michael Smith for their assistance. I am greatly indebted to Patrick Bateson for discussions of the critical period concept.

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affective and social: its function is to collect utterances, a responsibility that is subserved largely by mechanisms of social cognition sited principally in the right hemisphere. The third phase is analytical and computational. Previously stored forms are decomposed into syllables and segments, a process that facilitates discovery of regularities and is thereby responsible for the child's discovery and subsequent application of grammatical rules. This phase is active for a finite period and is largely served by left hemisphere mechanisms that make possible phonology, morphology, and syntax. The fourth phase is integrative and elaborative. In collaboration with acquisitive dispositions, this phase enables extensive lexical learning. Children who are delayed in the second phase have too little stored utterance material to activate their analytic mechanism at the optimum biological moment, and when sufficient words have been learned, this modular capability has already begun to decline. Inactivation has the same effect as damage. Compensatory use of homologous right hemisphere structures, set in motion by lexical delay, causes increases in functional and anatomical symmetry across the hemispheres. The resulting neurolinguistic resources, not being specialized for phonological operations, are minimally adequate, but not optimal, for development of spoken language and may also disfavor phonological encoding and decoding operations associated with written language.

A theory of neurolinguistic development must deal with several questions about the ontogeny of linguistic capacity. First, what neural mechanisms support the development of language? Second, when and under what neurogenic and behavioral circumstances do these mechanisms become active? Finally, when does the activation period for these mechanisms draw to a close? As the literature on sensitive periods is (indirectly) relevant to these questions, it is necessary first to examine possible contributions of this literature to an ontogenetic theory of language.

A SENSITIVE PERIOD FOR LANGUAGE

The literature contains four types of evidence for a sensitive period (see reviews in Locke, 1993a, 1994b). First, there are the usually dramatic case studies of individuals who were socially deprived in their infancy (cf. Curtiss, 1977). The theoretical impact of these studies has been limited, presumably because there are few well-documented cases and no reports in which significant linguistic deprivation occurred without extreme neglect and abuse. This criticism is inapplicable to a second type of study, of nurturantly reared children who lived for a period with undiagnosed hearing loss and underexposure to both spoken and signed language. Research of this type suggests that there are degrees of sensitivity, with the first few years being most sensitive (Mayberry, 1993).

The third and largest body of research on the sensitive period for language involves the learning of "foreign languages" by children whose families

have emigrated to a linguistically different culture. This evidence suggests that there is a sensitive period for language learning that extends not from 2 to 12 years, as had been thought (Lenneberg, 1967), but from the infant's first directly relevant experience until the age of 6 to 8 years, followed by a transitional decline that extends to adolescence. But even here there is a limitation. This research focuses on linguistic performance—the ultimate success of language learning efforts—rather than the initial activation and build-up of learning systems that enable mastery of language. Because all the essential mechanisms are already at work or in the process of developing when the second language is encountered, there is little that non-native research can do but reveal how well previously developed mechanisms work on the new material. Non-native research also has nothing to say about when native language learning begins, and it says little about any temporal variations, or smaller or specialized intervals, that might occur within the sensitive period.

A fourth type of study has focused on children with unilateral brain lesions. These studies reveal that many children who experience significant damage in the vicinity of language areas in the left cerebral hemisphere go on to acquire a normal or nearly normal command of spoken language, regardless of precisely when the damage occurred. However, while demonstrating the plasticity of the developing brain—including the linguistic potential of “nonlanguage” areas in the brain—these findings say little about which neural mechanisms support the development of language and when these mechanisms become active. Nor do they indicate which neural compensations are responsible for children's post-traumatic development of language (although other studies taken up later do relate to this issue).

Most of the research characterized above takes the instatement of grammatical capacity for granted and only asks when applications of this capacity become less efficient. This may not be the most interesting question. Even a clear answer would tell us little more about how children develop the capacity for language in the first place. It is also a misleading question, for perceptual mechanisms may be usefully, but covertly, at work in the young infant, and later on, there may be a documentable rush of linguistic action that occurs after many of the relevant linguistic capabilities have been in operation for some time. Consequently, I will focus here on the ontogenetic processes and phases through which infants come to take on and use the species-typical neural allocation for spoken language.

NATIVE LANGUAGE DEVELOPMENT

To properly explore the emergence of language, it is necessary to conduct qualitative analyses, and to focus these analyses on the development of native language learning mechanisms. Vocal learning begins as early as the final trimester of pregnancy and is carried over continuously into postnatal life

TABLE 1

Phases and Processing Systems, and Neural and Cognitive Mechanisms, Associated with the Development of Linguistic Capacity, along with the Corresponding Areas of Language

Age of onset	Developmental phases and systems	Neurocognitive mechanisms	Linguistic domains
Prenatal	Vocal learning	Specialization in social cognition	Prosody and sound segments
5–7 months	Utterance acquisition	Specialization in social cognition	Stereotyped utterances
20–37 months	Analysis and computation	Grammatical analysis mechanism	Morphology Syntax Phonology
3+ years	Integration and elaboration	Social cognition and grammatical analysis	Expanded lexicon, automatized operations

(see review in Locke, 1993a). The challenge is to detect *changes* in perceptual learning and the form of utterances when sensitivities within the period for language learning begin, stabilize, and then subside. In principle, this can be done by looking for discontinuities in the normal course of development. Additional, confirming evidence may be obtained by exploring the short- and long-term consequences of variations in the rate of learning from one child to the next, and in the onset of different types of learning.

In the sections immediately following, I will first discuss some developmental precursors to spoken language and then address the development of linguistic capacity by contemporaneous reference to ontogenetic phases, their enabling neural and cognitive mechanisms, and the affected domains of language. A preview of these interweaving considerations is available in Table 1.

ORIENTATION TO SPEECH

Language development does not begin with the child's first efforts to learn material that is linguistic. Rather, it begins with processes that orient the infant to the behavior of talking people, and bias the infant to attend and respond to certain aspects of such behavior (Locke, 1996, 1997). The infant's responsiveness to facial and vocal activity is presumed to be heavily influenced by genetic factors as well as early experience and is supported by specific neural preadaptations. Non-human primates have brain cell assemblies that fire primarily to faces or to facial activity (Desimone, 1991) and to voices or vocal activity (Rauschecker, Tian, & Hauser, 1995); clinical and electrophysiological research reveals that humans have mechanisms that are

similarly dedicated to processing faces and facial activity (Tranel, Damasio, & Damasio, 1988) and to voices and vocal activity (Creutzfeldt, Ojemann, & Lettich, 1989a; 1989b; Ojemann, Berger, & Lettich, 1989; Ross, 1981; Van Lancker, Cummings, Kreiman, & Dobkin, 1988).

This set of neural and cognitive supports constitutes a specialization in social cognition (Brothers, 1990; Locke, 1992, 1993b)—an important component of a pluralistic system of social specializations (Karmiloff-Smith, Klima, Bellugi, Grant, & Baron-Cohen, 1995)—and henceforth I will refer to socially cognitive mechanisms and socially cognitive operations. At the most general level, this specialization supports an affectively oriented developmental growth path that channels infants in the direction of spoken language. More specifically, socially cognitive mechanisms orient infants to linguistic displays—the physical activity of people who are talking—thus providing information about language (Locke, 1993b, 1995). These mechanisms, in turn, regulate social and referential activity that occurs in the context of linguistic displays.

Research in the past several decades has identified many of the socially cognitive operations that seemingly facilitate vocal learning and early word production. These include the infant's disposition to (1) take vocal turns with a partner (Ginsburg & Kilbourne, 1988; Papousek & Papousek, 1989), (2) orient to and mimic variegated prosody (Masataka, 1992), (3) gesture communicatively (Bates, Bretherton, Beeghly, & McNew, 1982; Morissette, Ricard, & Decarie, 1994), (4) assimilate ambient phonetic patterns (Boysson-Bardies, Vihman, Roug-Hellichius, Durand, Landberg, & Arao, 1992), and (5) as they develop a "theory of other minds," seek to interpret and alter the mental activity of interlocutors (cf. Baron-Cohen, Tager-Flusberg, & Cohen, 1993). With relevant perceptual experience and motor development, these operations allow infants to "get by" in their native language, to pass as speakers when their linguistic capacity is still immature. The infant's socially cognitive operations therefore contribute to the first few essential phases in the development of linguistic capacity.

While many more studies need to be done, there is some evidence for facilitative effects by these so-called "precursors" to language. Osterling and Dawson (1994) found a strong inverse relationship between the amount of time infants spent looking at their mother's face in the first year of life and the probability of being diagnosed autistic several years later. Tomasello and his colleagues (Tomasello, Mannle, & Kruger, 1986; Tomasello & Farrar, 1986) observed a positive relationship between the amount of time infants participated in joint attention episodes with their mothers at 15 months and extent of expressive vocabulary at 21 months. Snow (1989) found that vocal imitation at 14 months was related to the number of nouns and verbs produced, the total productive vocabulary, and the ratio of words produced to words comprehended at 20 months. Developments such as these occur inconspicuously—even Lenneberg (1967) missed them, thinking that lan-

guage learning begins at 2 years—but they are a vital part of early language learning.

Our species preadaptation in social cognition is present in varying (usually lesser) degrees in other primates. For example, gorillas appear to use eye movements instrumentally (Gomez, 1990), but seem not to engage in referential pointing (Hewes, 1981). There is some evidence of transient vocal accommodation during dyadic exchanges (Maurus, Barclay, & Streit, 1988), but little or no uncontested evidence of lasting vocal learning (Masataka & Fujita, 1989; Owren, Dieter, Seyfarth, & Cheney, 1992). In the vocal exchanges between monkeys, there is support for a conversation-like temporal structure (Biben, Symmes, & Masataka, 1986; Hauser, 1992), although the evidence is currently limited to mature animals. Studies indicate that some species of monkeys recognize offspring from the voice (Cheney & Seyfarth, 1990; Waser, 1977), and that others use several distinctive call types only or primarily in certain caregiving contexts (e.g., while inspecting or retrieving their infant; Biben, Symmes, & Bernhards, 1989). There is anecdotal evidence of deception (Whiten & Byrne, 1988), which has been challenged (Heyes, 1993). After a largely negative history (Gallup, 1982), there is now some evidence for a concept of self (Hauser, Kralik, Bott-Mahan, Garrett, & Oser, 1995) but there is still little evidence of other minds (Premack & Woodruff, 1978). With appropriate experience (Tomasello, Savage-Rumbaugh, & Kruger, 1993), certain of the apes may spontaneously display an ability to comprehend utterances that equals that of a 2-year-old child, and some disposition to use symbols creatively (Savage-Rumbaugh, Murphy, Sevcik, Brakke, Williams, & Rumbaugh, 1993).

Primate social cognition is subserved by specialized systems that, among other things, convey indexical and affective information. Both human (Etcoff, 1989) and nonhuman primates (Hauser, 1993) control facial affect primarily with their right hemisphere. Likewise for other kinds of information, human and nonhuman primates favor the left hemisphere when responding to meaningful cues that are presented visually or auditorily (Hopkins & Morris, 1993; Petersen, Zoloth, Beecher, Green, Marler, Moody, & Stebbins, 1984). We are left hemisphere dominant for certain classes of manual activity, and so are they (MacNeilage, 1991).

PHASES IN THE DEVELOPMENT OF LINGUISTIC CAPACITY

Although Scott, Stewart, and DeGhett (1974) were interested primarily in canine social development, they said something of particular significance to human language when they wrote that “complex organizational processes involving two or more interdependent subprocesses may show one to several critical periods, depending on the time relationships of the subprocesses” (p. 489). Similarly, it will be argued below that in language development there appear to be four reasonably distinct phases—vocal learning, utterance

acquisition, analysis and computation, and integration and elaboration—each having its own function within the larger period of language development. A brief overview of these phases and functions will be provided here, and additional evidence for their timing will be offered in the section immediately following.

Vocal Learning

Infants learn and respond to properties of the human voice in the first phase in the development of linguistic capacity. During the final trimester of gestation the infant may be aware of, and reactive to, indexical and prosodic cues in the maternal voice (DeCasper & Fifer, 1980; DeCasper & Spence, 1986; DeCasper, Lecanuet, Busnel, Granier-Deferre, & Maugeais, 1994). Prenatal exposure to maternal prosody may explain a postnatal listening preference for the language spoken by the mother during pregnancy (Davis & DeCasper, 1994; Mehler, Jusczyk, Lambertz, Halsted, Bertonicini, & Amiel-Tison, 1988; Moon, Cooper, & Fifer, 1993). In the first few months of postnatal life, infants respond differentially to variations in vocal emotion (Aldridge, 1994; Caron, Caron, & MacLean, 1988; Culp & Boyd, 1975). In the second 6 months of life, infants seem to prefer the sound of their ambient language over a foreign one (Jusczyk, Cutler, & Redanz, 1993; Jusczyk, Friederici, Wessels, Svenkerud, & Jusczyk, 1993) and detect interruptions in the speech stream, enabling them to use prosodic cues to locate linguistic units (Goodsit, Morgan, & Kuhl, 1993; Hirsh-Pasek, Kemler Nelson, Jusczyk, Cassidy, Druss, & Kennedy, 1987; though see Fernald & McRoberts, 1994). During this time, listening experience reinforces some adult-like perceptual categories that are evident in the first few weeks of life and weakens others (Kuhl, Williams, Lacerda, Stevens, & Lindblom, 1992; Werker & Polka, 1993). As for production, infants may imitate intonation contours in the first 6 months of life (Masataka, 1992) and reveal in spontaneous utterances their assimilation of ambient sound patterns in the second 6 months (Boysson-Bardies et al., 1992). In sum, during the vocal learning phase infants become acquainted with vocal cues that identify people, regulate social behavior, and superficially characterize the expression of their native language.¹

Utterance Acquisition

As infants take in information about vocal characteristics of the ambient language, they also store utterances. This may begin as early as 5 months

¹ As infants make their way more deeply into language, meaning is conveyed in parallel by affective and lexical-semantic cues, a fact that can be brought out by artificially placing these types of signals in conflict (Fernald, 1993). But we never lose our interest in indexical and prosodic cues and continue to store this information in adulthood (Schacter & Church, 1992; Speer, Crowder, & Thomas, 1993).

(Mandel, Jusczyk, & Pisoni, 1995). Let us look briefly at the stored utterances that are reproduced and then consider the nature of the storage process. The first recognizable infant speech consists of a single word or what may appear to be a phrase (Peters, 1983). Phrases are usually a rote or nearly rote copy of word sequences that have been heard frequently in the speech of others, e.g., “shoes and socks” and “time to go to bed.” Although they may be said imprecisely (Plunkett, 1993), the length, stress pattern, and intonation contour of these sequences are usually fairly well preserved (Nelson, 1981; Peters, 1977). Often, they contain articles and pronouns, words that are otherwise late to develop, and may not occur elsewhere in the speech of the child (Hickey, 1993). These phrases therefore give pregrammatical children an air of linguistic sophistication.

The word sequences described above are considered *formulaic*, i.e., “holistic phrases not subjected to or requiring grammatical analysis in either comprehension or production” (Van Lancker, 1987, p. 85). They are not rare. Although their frequency seems to vary with children’s learning style (Nelson, 1973, 1981; Weiss, Leonard, Rowan, & Chapman, 1983), all children produce formulaic phrases. The 50-word vocabulary of children usually includes about nine phrases, on average; the 100-word vocabulary typically contains about 20 phrases (Lieven, Pine, & Dresner Barnes, 1992). For some children, nearly half the items in their lexicon are frozen phrases of this type. From a functional standpoint, these units enable young children to produce stretches of precociously grammatical speech even before they know the words—or know that there *are* words—contained therein.

Young children also seem unaware, on any level of consciousness, that the words they do know have constituent elements such as morphemes and phonemes (Echols, 1993; Halle & Boysson-Bardies, 1994; Hickey, 1993), and they appear not to understand the concept of word or lexical meaning, or the taxonomic principle underlying word use (Markman & Hutchinson, 1984). For this reason, it is parsimonious to suppose that most or even all of infants’ utterances are formulaic. The shorter utterances are heard by listeners as words, the longer utterances as multisyllabic words or phrases.

These relatively unanalyzed utterances are *acquired* from the speech of others. Because the infant’s socially cognitive operations take in the vocal material that is supplied by talking people, along with supporting referential information, this interval within the larger period of language development is called the acquisitive or storage phase. The neural mechanisms that support social cognition may be regarded as components of this utterance acquisition system that captures and reproduces prosodic patterns as the infant’s perceptual biases and interests dictate. In adults, the right hemisphere appears to be unusually active in the processing and storage of formulaic material (Van Lancker, 1987, 1990), and the same appears to be true in pregrammatical children whose right hemisphere more robustly activates to speech than their left hemisphere (see section The Anatomy of Delayed Language).

The significance of this second phase is that it gives infants a set of “starter” utterances that can be used appropriately in restricted contexts, and provides infants with the opportunity to participate in adult-like social interactions. Herein they are endowed with the word knowledge needed to produce a number of irregular verb forms (e.g., “run–ran”) that will prove troublesome several months later (Pinker, 1991). However, while this utterance reproduction capability allows pregrammatical children to talk and thereby be regarded as talkers, it will not enable them to achieve a large lexicon. In effect, every utterance they know is an idiom, an irreducible and unalterable “figure of speech.” There are presumed to be absolute storage limitations for material that is prosodically well specified but segmentally underspecified (Lindblom, 1989). Lacking the ability to break utterances into smaller pieces of speech, there exists no possibility of a grammar forming in the acquisitive phase. Nonetheless, this phase does pave the way for other developmental linguistic steps by providing systems still to be activated with an enormous sample of linguistically relevant data.

Structure Analysis and Computation

The third phase in the development of linguistic capacity involves operations—initially the analysis, and then the computation, of structure—that are performed on material that has been acquired in the second phase. These mechanisms are assumed to be experience-dependent; if the infant has no stored utterances, there will be nothing for its analytical mechanisms to work on, and no stored forms to reconfigure by application of computational rules.² The induction of grammatical capability therefore depends upon a certain amount of prior success. Where the second phase is affected by external factors, including the availability of appropriate stimulation, the functions instated in the third phase are affected primarily by internal factors. That is to say, the development of utterance analysis capability is only indirectly affected by exposure, insofar as environmental stimulation is correctly relayed to the structure analyzer by the efficient operation of subordinated processing systems. To understand language development it is therefore not enough to know about the “input” to the child; one must know which utterances were competently perceived, stored, and submitted to segmental analysis by the child. As will be illustrated later, analytical and computational functions appear to engage in a relatively narrow time window.

The structure analysis system locates recurring elements within and across utterances and thereby learns the rules by which utterances are to be synthesized and parsed. In effect, it presents the child with the units needed for

² In using the term “experience-dependent” I am of course referring to the instatement of brain mechanisms that are responsible for linguistic operations, not the learning of particular languages, which obviously depends on experience.

morphology, phonology, syntax, and the lexicon—thereby endowing the propositionally generative child with the capacity to make infinite sentential use of finite phonemic means.

The initial, and perhaps most conspicuous, form of internal evidence that an analyzer is at work comes from children's regularization of irregular *verb tenses and noun plurals*. When this happens, *verbs* like "went" may be temporarily and inconsistently expressed as "goed" (Marcus, Pinker, Ullman, Hollander, Rosen, & Xu, 1992), nouns like foot may be expressed as "foots" (Marcus, 1995). These forms are *generated*, not merely heard and reproduced, by children. There also may be changes at other levels of language, including phrase regressions, as formulaic phrases begin to come apart (Plunkett, 1993); vocabulary-level alterations, as stored words are altered in accordance with recent phonological gains (Macken, 1980); and phonological regressions, as precocious items stored before a system was in place are restructured under the constraints of a simple phonology (Moskowitz, 1980). These discontinuities will be revisited below.

Phase three in the development of linguistic capacity depends on acquisitions in the second phase, and is enabled by a relatively free-standing specialization that typifies our species (Mattingly & Studdert-Kennedy, 1991). This specialization is responsible for grammatical behavior. Its operation is revealed at the morphological level in English when it computes regular past tense verb forms by applying a rule that joins affixes with stem endings, e.g., walk + ed = walked. Irregular forms are handled associatively (Burgess & Skodis, 1993; Pinker, 1991) probably by a different set of neural mechanisms (though see Plunkett & Marchman, 1993).

Whether a rapidly expanding lexicon presses grammatical mechanisms into operation because of storage limitations for segmentally underspecified material, as is claimed here, or because it offers enough of a lexical sample for structural regularities to be discerned, as Marchman and Bates (1994) have suggested, one ought to expect a positive relationship between vocabulary and grammatical measures. And such a relationship does exist: Bates, Bretherton, and Snyder (1988) obtained a .87 correlation between total vocabulary and use of grammatical morphemes at 20 months. These issues will be taken up again in the section Morphology.

Integration and Elaboration

Once analytical and computational capabilities are integrated with acquisitive systems, it becomes possible to achieve a far larger lexicon. For when applied to stored forms, structural analysis produces systemic rules. These rules, in turn, impose organization on incoming utterances, thus expediting the learning of new words (Anglin, 1993; Chafetz, 1994; Maratsos, 1982; Maratsos & Chalkley, 1980). For example, knowing that English nouns are frequently preceded by "a" or "the" and pluralized by adding an /s/ or

/z/ to the end would seem to facilitate sentence segmentation and thus the learning of new nouns (Gerken & McIntosh, 1993; Katz, Baker, & Macnamera, 1974). By particulating speech into commutable elements, structural analysis takes pressure off a holistic type of memory, thereby enabling the creation of larger and larger vocabularies, in which each of the individual entries is merely a unique recombination of a small set of phonemes.³

While lexical capacity is expanding, syntactic processing is also becoming more automatic (Friederici, 1993). With continued fine-tuning of grammatical rules and memorization of irregular forms (cf. Kim, Marcus, Pinker, Hollander, & Coppola, 1994), the child will sound more adult-like. But changes during this phase affect communication as well as language, and mechanisms of social cognition continue to contribute as the child comes to understand the degree to which other individuals' mental activity differs from its own—an advance that should be correlated with general language ability, for reasons identified elsewhere (Locke, 1993a), and is (Jenkins & Astington, 1993).

Up to this point, it has been difficult to avoid mixing ontogenetic phases and systems, their enabling neural and cognitive mechanisms, and the affected domains of language. Table 1 clarifies the relationships among these different phases, mechanisms, and domains.

SCHEDULE OF DEVELOPMENTAL LINGUISTIC MECHANISMS

It should be clear from evidence sampled earlier that the first 10 to 12 months of life, when vocal affect and prosody command a great deal of the infant's attention, are vital to the development of linguistic capacity. Levitt (1993) has speculated that as the first year draws to a close there may be a slight regression in sensitivity to prosody as segmental perceptual boundaries begin to noticeably shift in the direction of ambient stimulation. This is also when lexical meaning begins to compete with affective tone (Fernald, 1993), and differential phonetic experience is initially revealed in infants' productions of segmental forms (Boysson-Bardies et al., 1992). Thus, the first effects of learning on the form or content of infants' utterances, and lexical-semantic interpretation, may occur at or just before the appearance of words, thereby also marking the beginning of the second phase in development of linguistic capacity (Macken, 1993).

Although infants are variable, Phase 2 operations typically accelerate from 5 or 6 months to 18 or 20 months, when new words begin to pile up at a rapid rate (Benedict, 1979). At that time, the third phase commences. In this

³ Gathercole and Adams (1993) obtained positive correlations between performance on word and nonword repetition tasks and receptive vocabulary in 3-year-olds, suggesting that vocabulary expansion may be abetted by improvements in working phonetic memory. Whether working phonetic memory reflects enhanced analytical ability, as I am proposing long-term lexical storage does, independently, is impossible to say at this point.

phase there is activation and probably enhancement of neural systems that analyze and detect recurrent structural patterns, thereby permitting the discovery of important regularities. As these systems provide for efficient storage of linguistic information, and enable generativity, there is little doubt that they facilitate learning. But these systems do not acquire linguistic material. Large volume acquisition is accomplished by a fully integrated, automatic system that allows the child to continue learning lexical items and irregular forms, along with other aspects of language and linguistic communication. The phase that produces this integrated system usually begins when the child is around 3 or more years of age. Let us now look at the morphological and phonological evidence for these estimates.

Morphology

The child's first words and formulaic phrases appear to be stored in a prosodic type of memory (Echols, 1993). As a system that presupposes no discrete, combinable units like the phoneme, prosodic memory is assumed to have a limited storage capacity (Lindblom, 1989). If lexical items continue to accumulate in prosodic form, the system will overload. The issue may be forced by the so-called "lexical spurt," an early quantitative discontinuity at the utterance level. As the term implies, the spurt is a marked quickening of the pace at which new words are comprehended and/or produced (Benedict, 1979). Whatever the reasons for these accelerations, and several have been suggested (Reznick & Goldfield, 1992), expressive lexical spurts tend to occur at about 18 months, when the number of words acquired per week may quadruple (Goldfield & Reznick, 1990). Bates et al. (1988) found that if vocabulary "bursts" at the 50-word stage, then about two-thirds of the children in their sample had experienced their burst by 20 months. At this age, there may be as many as five words comprehended to every word produced (Benedict, 1979). It is hypothesized that this burgeoning store of comprehended words triggers or reinforces the activation of analytical mechanisms.

An appealing variant of the diminishing storage notion discussed above is a sample size hypothesis. According to this hypothesis, children begin to appreciate the structure of their native language when they have a sufficient sample of utterances in storage. For example, as children learn additional words they come across more and more minimal pairs (Lindblom, 1989; Studdert-Kennedy & Goodell, 1992), which helps draw children's attention to the existence of overlapping articulatory gestures. Overlapped gestures point to the underlying presence of the phoneme, a discovery that is critical to generative morphological operations.

Increases in the sample of stored utterances also could help for systemic reasons. It has been suggested that children must have a "critical mass" of words in their expressive lexicon—perhaps as many as 70 verbs, and 400

words overall—before they discover and begin to apply the rules of linguistic morphology (Bates, Dale, & Thal, 1994; Marchman & Bates, 1994; Plunkett & Marchman, 1993). Expressed words are observable and measurable, but it appears to be the number of words *in storage* that counts. The size of a child's receptive lexicon is harder to estimate, but could easily run to several thousand words at this point. Although the distinction between articulatory and systemic factors, and the larger distinction between storage pressure and sample size, are interesting in their own right, the predictions they foster are fundamentally the same: as the child's receptive vocabulary increases, the probability of analysis and computation increases.⁴

The hypothesis offered here is that structure analysis is turned on by maturational advances in collaboration with pressures associated with an expanding vocabulary. As for when this happens, it appears from reported data (Marcus et al., 1992; Marcus, 1995) that children begin to regularize irregular forms between 20 and 37 months of age. Inspection of individual subject data suggests that the earlier portion of that range may be closer to the true onset, for the older subjects produced a lower proportion of overregularizations, as one would expect were the irregular "exception words" being learned. This observation is reinforced by data gathered by the large-sample project that produced the *MacArthur Communicative Development Inventory* (Fenson, Dale, Reznick, Thal, Bates, Hartung, Pethick, & Reilly, 1991). By parent reports, over a fourth of the 18-month-old children had produced overregularizations; by 24 months, the cumulative incidence had increased to half (and by 28 months, the incidence was up to 70%).⁵

There also are utterance-external grounds for placing grammatical capacity at this age: at 2 years, sentence processing and symbolic expression may

⁴ In the interests of completeness, there is another possibility—one that came to mind after reading some of the work on avian imprinting by Gabriel Horn and his colleagues—which is that after some amount of storage there could be a period of time needed to activate neural storage systems, that time being dictated largely by endogenous factors. If this were true of language, the decisive factor might be the amount of stored utterance material present at some point *prior* to grammatical activation rather than the amount of stored material at the time of activation. In this context, it is important to recognize that a matter that has generated some interest elsewhere—whether English verbs are handled by one mechanism or two (cf. Plunkett & Marchman, 1993)—is irrelevant to the issue under consideration here. In the present context, the significance of overregularization is that it demonstrates infants' discovery, through analytic operations undertaken at their own instance, word-final segments in a form that enables suffixing. In connectionist studies, the input is preprocessed into (adult) phonemic units.

⁵ Some studies estimate the size of the average 2-year-old's expressive lexicon at about 185 to 200 words (Nelson, 1973; Paul & Riback, 1993). This is no contradiction of Bates et al. (1988) since analysis and computation may begin, on average, several months later, when more than 200 words would be in use. But regardless, the actual number of words known at the time grammatical capability kicks in will vary from child to child. It therefore seems best to consider that some approximate number or range of stored words acts to trigger or reinforce analytical capability and to resist the temptation to pin down the number more precisely.

still be similar in children and at least one optimally reared ape (Savage-Rumbaugh et al., 1993). But this age is approximately when the complexity of children's spoken language begins to diverge from that of nonhuman primates trained in sign language (Bickerton, 1990). If linguistic ontogeny bears any relationship to phylogeny whatsoever, we should expect some differentiation at that point. Noam Chomsky is apparently more conservative. He asked recently, in reference to his 4-year-old granddaughter: "Does she speak English? What we say in ordinary discourse is that she has a partial knowledge of the language that she will ultimately attain if events follow the expected course, though what she now speaks is not a language at all" (Chomsky, 1995, p. 39).

Phonology

It is assumed that phonemic segments emerge from larger phonetic patterns (Lindblom, MacNeilage, & Studdert-Kennedy, 1983; Studdert-Kennedy, 1987; 1991). The child's first words require a superficial phonetic analysis, but no phonology per se, for the early words contain few if any commutable units (Ferguson & Farwell, 1975). On the basis of present evidence, it would be difficult to pinpoint when this begins to change or becomes adult-like. Perceptual evidence suggests that infants begin to discriminate phonetic segments that distinguish words in their own receptive vocabulary, i.e., develop classically defined phonemic categories, at about 19 months of age (Werker & Pegg, 1992). This advance may be attributable to an analytical mechanism in the left hemisphere—using the same (looking time) paradigm, other researchers have discovered a rightward looking bias that develops between 15 and 16 months and 19 months (Mount, Reznick, Kagan, Hiatt, & Szpak, 1989).

If one goes by gross events in spontaneous speech, one may hear some changes early on. For example, a precocious production of "truck" may regress to "tuck" and the [pr] cluster in "pretty" may be simplified to singleton [p], as though a sound system with constraints were now in effect (Moskowitz, 1980). But isolated examples do not allow one to precisely date the onset of analytical processes.

Phonetic transcriptional analyses suggest that articulatory gestures with similar properties (e.g., alveolar closures and high front vowels) tend to occur adjacently in the babbled syllables of prelexical infants (Davis & MacNeilage, 1994; Locke, Lambrecht-Smith, Roberts, & Guttentag, 1995; Vihman, 1992), a tendency that has also been observed in the speech of 2- to 3-year-old children (Davis & MacNeilage, 1990; Stoel-Gammon, 1983). It seems likely that children of this age may still lack the control needed to move discretely from one phonetic gesture to another and, therefore, to recombine the phonemes expressed by those gestures. Acoustic studies suggest that these larger syllable-sized movements may still be differentiating several

years later (Goodell & Studdert-Kennedy, 1993; Nittrouer, Studdert-Kennedy, & McGowan, 1989), but it is unclear when adult or near-adult levels of gestural independence are reached.

A third class of evidence involves slips of the tongue. Smith (1990) reported a high frequency of segmental exchange errors in 5-year-old children's elicited slips of the tongue. If articulatory gestures are free to exchange with other gestures erroneously, they are presumably capable of expressing recombinable phonological units. There is only a small amount of slip of the tongue evidence on younger children, and a remarkably low rate of non-systematic error in those children on whom data are available. In studies of 2- to 3.5-year-olds, Warren (1986) and Clark and Andersen (1979) encountered spontaneous speech errors, but none involving the exchange of segments. In a diary study, Stemberger (1989) noted some exchange errors in his two daughters, beginning as early as 20 to 24 months, but remarked on the rarity of all spontaneous speech errors. While more studies are needed, it does appear that phonology, while frequently considered to be the most basic level of language, may be the last to develop fully (Locke, 1994c; Studdert-Kennedy, 1991).

Syntax

Fillmore (1979) and Peters (1983) have suggested that the child's acquisition of syntax presupposes the ability to decompose chunks of speech, initially acquired as unanalyzed wholes, into individual words. Since morphology and phonology involve analysis of syllables into component parts, and syntax only requires differentiation of syllables from each other, it is conceivable that syntactic learning—or at least some precursors associated with word order—precedes productivity at the other levels of language. Various investigators have obtained correlations between these three levels of language (cf. Bates et al., 1988, among others).

Although the phases are functionally distinct, each phase grades into the following one. Vocal learning flows continuously into utterance storage, which grades into analysis and computation. The functional value of the first three phases derives strictly from their ability to enable succeeding phases. In the final phase, an appropriately organized grammatical system and lexicon are achieved and used with a high degree of efficiency. The child has, at this point, the mature linguistic capacity that typifies healthy juvenile and adult members of our species, and which operates throughout the lifetime of the normally aging individual. All phases in the induction of linguistic capacity are affected by interactions between neuromaturational events and social stimulation.

Figure 1 depicts the period in which species-typical linguistic mechanisms develop, showing the component phases discussed above. It is assumed that analytical mechanisms develop some readiness to function when the child

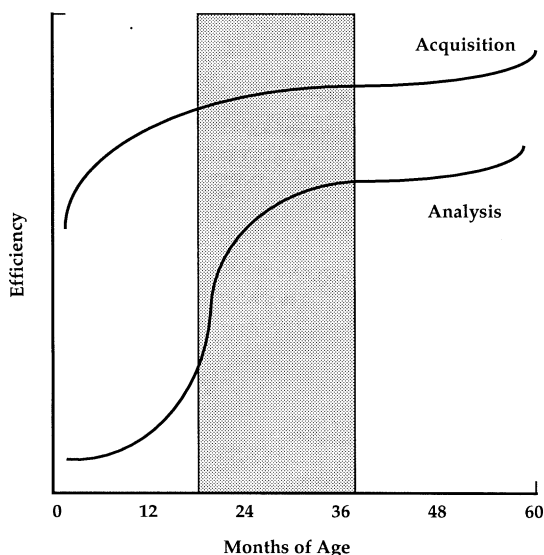


FIG. 1. Phases and systems in which linguistic capacity develops. The stippled area is a critical period in which utterance-acquisition must reach a certain level of demonstrated efficiency in order to fully activate and stabilize an utterance-analytic mechanism that, for its part, is intrinsically "ready" to respond to experience.

is between 2 and 3 years of age, at which time utterances are available to be analyzed, thus encouraging, reinforcing and stabilizing the action of these mechanisms. The figure is based entirely on qualitative analyses of stored utterances by normally developing children but, as we will see below, is confirmable through inspection of the "learning curves" of children whose course of development is atypically timed.⁶

LANGUAGE IN OTHER MODALITIES

If acquisitive and analytical-computational phases of language development are fundamentally different, as is claimed here, it would not be surprising to see this difference revealed in signed as well as spoken languages. Comparative studies by Bonvillian and his colleagues (Bonvillian & Folven, 1987; Bonvillian, Orlansky, & Novack, 1983; Orlansky & Bonvillian, 1984, although see Volterra & Iverson, 1995) suggest that first signs may appear

⁶ Little is known about any children who might make it through the second phase on time, in a perceptibly normal fashion, and show poor progress thereafter. The mere existence of such children would be theoretically informative, for their problems might originate from rather different genetic and neural factors than children who seem to be delayed from the beginning. I am excluding from consideration here children with attested neurological syndromes (cf. Cooper & Ferry, 1978; Landau & Kleffner, 1957).

several months before first words—an advantage that soon disappears—but that morphology and syntax tend to appear in children acquiring spoken languages at about the same time that they develop in signed language learners. Semantic relations, verb agreement, deictic pronouns, and morphologically complex verbs of motion and location all seem to develop at the same ages in both speech and sign.

It is unsurprising that there would be two different ages for onset of speaking and signing since these systems draw upon different perceptual and effector mechanisms. It is also no surprise that there would be just one age for onset of grammatical functions which, unlike perception and production, are presumed to have no inherent sensorimotor constraints. From their comparative studies on the learning of signed and spoken languages, Meier and Newport (1990) suggested that infants have “two (or more) largely independent timing mechanisms . . . one controlling the onset of lexical acquisition and another controlling the acquisition of grammar (that is, syntax and morphology)” (p. 13).

VARIATIONS IN RATE OF LANGUAGE DEVELOPMENT

In animal research conducted in the laboratory, one may exert control over precisely when stimulation is available, and thus detect variations in sensitivity at individual points within the larger optimal period for learning (Bateson & Hinde, 1987). Equal rigor is needed in language research, but of course no one would ever suggest that human infants be deprived of normal social interactions and access to linguistic stimulation at particular times in their development. One must therefore look for whatever sensitivity variations naturally exist in large populations of infants. Data from such populations could enrich, perhaps even confirm or disconfirm, the sequence of cognitive and linguistic functions hypothesized above. Where we looked earlier at variation occurring within individual children, here we turn our attention to variations between children.

Developmental Lexical Delays

There are many normally hearing children who are nurturantly reared by speaking parents but nonetheless denied access to appropriate linguistic stimulation. How can this be? The reason for this seemingly paradoxical circumstance is that such children have utterance processing limitations that *in effect* reduce the stimulation of, and thereby the neurodevelopmental challenge to, experience-dependent language mechanisms in the brain (Locke, 1990). These children's exposure to linguistic behavior may be perfectly adequate, but their experience with it—their *effective exposure*—is not.

Where sensitive periods for learning are concerned, stimulation delayed is stimulation denied. Because the child's stored utterances provide the stimulation that is required by the analytical mechanism, children with a small

mental lexicon are inescapably at risk. For them, a lexicon delayed may be a grammar denied.

Who are the lexically delayed? They are children with small vocabularies that typically are not attributable to poor hearing, low intelligence, brain damage, or primary affective disorder. Most of these children come from nurturant, linguistically stimulating families, but they do not use and may not comprehend the normative quantity of words at expected ages. At 2 years, approximately 7% of these children, who may otherwise appear to be developing normally, will have fewer than 30 words in their expressive vocabularies (Rescorla, 1989; Rescorla, Hadicke-Wiley, & Escarce, 1993)—somewhere between a sixth and a tenth of what they ought to have.

About half of these children will also have a restricted receptive lexicon, and it is these children in particular that end up in the “language impaired” category (Bishop & Edmundson, 1987b; Thal, Tobias, & Morrison, 1991).⁷ This observation fits with some recent findings from a prospective study suggesting that children headed for specific language delays at 25 months fall behind in word comprehension (as well as expression and symbolic play) sometime between 13 and 16 months of age (Ellis Weismer, Murray-Branch, & Miller, 1994). This result converges with other findings (Harris & Chasin, 1993; Plomin, Emde, Braungart, Campos, Corley, Fulker, Kagan, Reznick, Robinson, Zahn-Waxler, & DeFries, 1993; Reznick, 1990) to suggest that lexical delays at about 2 years may be predicted from comprehension performance at least 6 to 12 months earlier.

According to the hypothesis presented here, it is the shortage of stored lexical items that prevents utterance analytical mechanisms from fully or permanently activating, thereby restricting the development of a linguistic grammar. This is a type of higher-order cognitive deprivation that is not different in principle from the sensory deprivation that is more commonly recognized in cases of transient or permanent hearing loss.

If lexically delayed children generally caught up with those who learn words easily, this speculation, and the use of these data, would seem to be pointless. But (1) if they did catch up, it could be because a back-up system took over, thus reinforcing the possibility that there is a critical period for activation of a more primary, species-typical neurolinguistic arrangement; and (2) while they may appear to catch up, many of the children who learn language slowly have residual processing deficits as adults. Although some of these deficits involve overt speaking behaviors of one kind or another

⁷ Although the basic behavioral genetic evidence on specific language impairment is still coming in, one notes that the incidence of language disorders in siblings of expressively delayed 2-year-olds *with normal comprehension* seems not to be elevated (Whitehurst, Arnold, Smith, Fischel, Lonigan, & Valdez-Menchaca, 1991). This finding, along with other evidence presented here, encourages speculation that persisting language disorders arise from genetically determined delays that act at the level of lexical storage.

(Tomblin, Freese, & Records, 1992), the long-term problems of lexically delayed children frequently include reading, writing, and spelling difficulties (Aram, Ekelman & Nation, 1987; Felsenfeld, Broen, & McGue, 1994; Klackenberg, 1980; Lewis & Freebairn, 1992; Tallal, Ross, & Curtiss, 1989).

If language delays were caused by analytical deficiencies per se, we would expect to find few nonlinguistic deficits in such children (except for cognitive deficits that might secondarily reflect the language problem). On the other hand, if there were a general neuromaturational delay, one might well find delays in a wide range of socially cognitive, perceptual, and motoric systems that support language, but no primary structural or functional deficiency in any neural system that is dedicated to language. The literature reveals that lexically delayed children are typically late in a wide range of behavioral developments, but offers no convincing evidence of deficiencies in any particular language learning system (Neville, Coffey, Holcomb, & Tallal, 1993). Let us look at some illustrative studies (also see Locke, 1994a). In one investigation (Tallal, Stark, Kallman, & Mellits, 1981), language delayed children performed inferiorly to mental age-matched normals in their ability to judge the order of briefly presented tones. By itself, this finding might seem to represent the psychophysical basis for perceptual deficits that have been reported in the language-delayed child (Bishop, 1992). But the same children were also inferior in processing briefly presented *visual* stimuli.

The mere existence of visual and other deficits cannot be taken to mean that co-occurring auditory deficits play no causal role vis à vis developmental language disorders. However, the presence of deficits in several modalities makes it necessary to demonstrate that *only* the hypothesized effect is operating, and that the language delay is not the result of a general retardation. The link between language delay and speech processing failure is tenuous in any case; although efforts have been made to correlate perceptual processing deficits with output errors (Tallal, Stark, & Curtiss, 1976), it is well known that language-delayed children—like all normally developing young children—have much more difficulty perceiving and producing the steady-state fricatives than the acoustically transient stop consonants.

In other work (Johnston, Stark, Mellits, & Tallal, 1981), language-impaired children performed inferiorly to controls on a variety of sensory and motor tasks, many of which involved manual movement and shape detection tasks not requiring temporal resolution of rapid events in any modality, or showed a generalized slowness to respond on tasks, whether related to language or not (Kail, 1994). Locke and Goldstein (1973) found that children on the slow end of a normal speech-learning continuum performed inferiorly on an auditory vigilance task, thus throwing open to question the proper interpretation of performance by language-impaired children on any task requiring sustained attention (also see Neville et al., 1993). In what appears to be a reversal of her original position, Tallal (1990) recently criticized several researchers for implying that auditory processing deficits are specifi-

cally related to language processing difficulties or, for that matter, to specific language impairments.

Other studies have found similarly reduced performance on nonlinguistic visual, motor, and tactile tasks by lexically delayed children (Johnston & Ellis Weismer, 1983; Kamhi, 1981; Neville et al., 1993; Powell & Bishop, 1992), who also may be delayed in gestural development (Thal et al., 1991b). The general impression is that these children, as a group, perform poorly not by behaving abnormally, but by scoring as chronologically younger normal children do. This impression is reinforced by the finding that lexically delayed children tend to run late in bowel and bladder control and may even be physically smaller than the average child (Haynes & Naidoo, 1991). It is not surprising, then, that the performance of lexically delayed children tends to improve with age, just as the performance of younger normal children does (Tallal et al., 1981). We should ask, therefore, if these nonlinguistic deficits are correlated with some global factor that is capable of producing language delay.

Neurologists and pediatricians interpret poor performance on certain types of motor coordination tasks to mean that brain development is behind schedule (Denckla, 1974; Wolff, Gunnoe, & Cohen, 1983). Let us look, then, at studies correlating performance on nonvocal motor tasks with language. In several studies by Bishop (Bishop, 1990; Powell & Bishop, 1992), lexically delayed children were given a pegboard moving task along with standard language measures. The children were first shown how to move 10 pegs from a row at the back of the pegboard to the corresponding positions at the front, and then encouraged to do the same as quickly as possible. Analysis revealed that the language-delayed children performed significantly slower than controls. Moreover, there was a correlation over time between the extent of language progress and improvement on the pegboard task. These findings underscore the reasonableness of a maturational hypothesis, for imaging studies suggest that the cognitive processing associated with peg moving involves cerebellar structures distal to those putatively responsible for spoken language (Kim, Ugurbil, & Strick, 1994).

Hughes and Sussman (1983) conducted an experiment to see if in normally developing children right-handed tapping interfered more with concurrent speaking than left-handed tapping, relative to language-delayed children. In the experiment, there was a noncompeting baseline condition in which subjects were simply to tap as fast as possible *without* speaking concurrently. It is of interest that in this control condition, the language-delayed children tapped slower with right and left hands than age-matched children who were developing language at a normal rate. This result is exactly what one would expect were the language-delayed children simply less neurologically mature, for tapping speed regularly increases in normally developing children from 3 to at least 9 years of age (Kamhi & Masterson, 1986). Thus, there are behavioral patterns—performance on pegboard and tapping tasks and

the like—that predict language delay but are not causally linked to language development by any existing theory.

The same probably is true of performance on Tallal's perceptual task. She and her colleagues have witnessed performance to improve steadily with age in her language-delayed subjects, just as it does in normally developing young children (Tallal, 1976), and there is now evidence for a regular progression in the development of tone perception in which intensity discrimination develops first, followed by frequency discrimination and finally duration discrimination (Jensen & Neff, 1993). These and the other studies above give us no reason to suspect primary deficiencies in mechanisms that are dedicated to specific linguistic functions. In fact, in the early stages there is little evidence to support a diagnosis of *language disorder*. What the evidence does support is *lexical delay in association with neuromaturational delay*. These children usually come to the attention of clinicians because they use too few words for their age. They do very little, if anything, that normally developing young children do not do. This holds both for language (Curtiss, Katz, & Tallal, 1992; Leonard, Bortolini, Caselli, & Sabbadini, 1993; McGregor, 1994; Paul & Jennings, 1992; Paul & Shiffer, 1991; Smith-Lock, 1993) and nonlinguistic perceptual and motor behaviors (Powell & Bishop, 1992; Tallal, 1976). However, as we will see later, an unfortunately timed lexical delay could *become* a permanent—and ultimately fairly specific—grammatical deficit.

Lexical Learning in the Lexically Delayed

There is a theoretically interesting paradox about language-delayed children. For all the tasks they cannot perform accurately or quickly, there is little laboratory evidence that they learn new words more slowly than normal children. In one experiment, children with specific language delays learned nonce words—a task that predicts development of receptive vocabulary in older children (Gathercole & Baddeley, 1993)—at the same rate as linguistically normal children (Leonard, Schwartz, Chapman, Rowan, Prelock, Terrell, Weiss, & Messick, 1982). As the subjects in that study ranged from 32 to 50 months of age, we may suppose that the lexical learning of many language-delayed children comes up to normal rates at some time during those ages. Of course this could be optimistic; day-to-day conditions may be less favorable to language learning than those existing in controlled laboratory experiments. But the finding itself is fairly general; numerous investigators have found similar levels of lexical or morphological learning by language-delayed and normally developing children when instructed and observed in the laboratory (Connell & Stone, 1992; Dunn & Till, 1982; Johnston, Blatchley, & Olness, 1990; Kouri, 1994).

If lexically delayed children at these ages can learn new words and morphemes as quickly as normally developing children, why don't they develop

language at the same rate? The answer is that, after their late start, many of them *do* develop language at the normal rate. Gibson and Ingram (1983) reported on a language-delayed boy who comprehended just 20 words at 32 months of age. He was nearly 2 years behind his peers. However, the rate at which the boy acquired new comprehension vocabulary items over the succeeding 4 months was the same as that of a reference group of children with no previous history of delays. Bishop and Edmundson (1987a,b) evaluated the nonverbal motor performance and linguistic progress of 87 seriously language-delayed children, testing them at 4, 4.5, and 5.5 years of age. The investigators obtained a positive correlation between naming vocabulary and syntax at 4 years and found that on most measures, all language-delayed children—even those with general delays—made about 18 months of progress in 18 months of time. They concluded that language-delayed children experience “a constant lag in the timing of different stages of language development, but not . . . unusually long intervals between one stage and the next” (1987a, p. 454). This is not to say, of course, that these individuals eventually catch up with normally developing children.

When lexically delayed children reach 24 months and on neuromaturational grounds their experience-dependent analytical capability should be able to benefit from an internal push, they have fewer than 30 words in their expressive lexicon (Rescorla, 1989). By contrast, normally developing children have 30 words by about 11 months (Benedict, 1979)—long before grammatical behavior emerges—and they have as many as 6 to 13 times that many words at 2 to 3 years, when computational capabilities are first noticeably applied morphologically (Bates, Marchman, Thal, Fenson, Dale, Reznick, Reilly, & Hartung, 1994; Nelson, 1981; Paul & Riback, 1993; Rescorla et al., 1993).

These figures all relate to lexical expression, but it appears that the number of words children *say* is just the more observable face of a more important underlying factor—the number of words they *know*. In a study of late talkers, Thal et al. (1991b) found that the children with small *receptive* vocabularies had made the least progress 1 year later. Although they were between 30 and 35 months of age at follow-up, none produced more words than would be expected of a typical 22-month-old. Receptively delayed children thus provide a test of the functional dependency of the third phase in the development of language learning mechanisms upon the second phase.⁸

In recent years, attention has increasingly been given to the existence of

⁸ The outer limit on activation and computational mechanisms is difficult to estimate on present data. The normally reared kitten responds appropriately to patterned light and has an adult-like visual cortex at 5 weeks, but the critical period for visual development extends to nearly 3 months (Timney, Emerson, & Dodwell, 1979). One can only assume that a decline in grammar-serving mechanisms in the child would not begin until some months after the oldest developmental age at which rule-governed behavior normally appears, at about 36 months.

children with poor utterance acquisition capabilities as a separate subgroup of the larger lexically delayed population. Expressively delayed children with few stored utterances tend to behave differently than equally delayed children with normal receptive lexicons. To date, research has focused primarily on possible differences in relation to social interaction. Craig and her colleagues (Craig & Evans, 1989, 1993; Craig & Washington, 1993) have found that receptively delayed children tend to be less adept at entering ongoing conversations, and at assuming and yielding the floor, than children with similarly small working vocabularies but good lexical comprehension. In a study of late-talking 2-year-olds, Paul, Looney, and Dahm (1991) found that of six children who were also receptively delayed, five were socially retarded on a standardized measure that includes a range of nonverbal social behaviors (e.g., smiling). Moreover, they found that four of seven children with expressive (but no social or receptive) delays at 2 years had measurable social delays by the time they were 3. Together, these findings suggest that social incapacities can both contribute to lexical delay and arise in response to it.

It is important to ask how long it takes for lexically delayed children to acquire as many words as normally developing 2- to 3-year-olds, for this might convey some sense of the outer limits on development of analytical mechanisms. The data on this are sketchy, but it is conceivable that many children with small expressive and receptive lexicons will be 4 to 6 years old before they finally acquire facility with as many words as are used and comprehended by normal 2-year-olds. By that age, the optimum neuromaturational moment for the development of species-typical analytical mechanisms may have already begun to dissipate.

Although the data from Down syndrome can be difficult to interpret, Fowler (1988) found evidence for a distinct plateau ("a virtual halt") in the language development of several children with that syndrome beginning at a chronological age of about six or seven years. Comparative studies (Singer, Bellugi, Bates, Jones, & Rossen, in press) of children with Down syndrome (DS) and Williams syndrome (WS) indicate that in the latter group there is a sharp increase in the number of different parentally reported words produced between about 25 and 45 months of age. By contrast, in DS this period is characterized by very little increase in expressive vocabulary. It is unsurprising that older children with WS demonstrate greater grammatical complexity than DS subjects. However, given their gregarious nature and greater talkativeness, it also could be that WS subjects are more likely to require the services of an utterance analytic system and thus to make more "grammatical hay" out of their lexical knowledge than are DS subjects. An additional logical possibility is that the neural conditions associated with the two syndromes are unequally threatening to critical period shut-down. Lacking knowledge of those conditions in the normally developing child, as well as in various syndromes, it is currently impossible to evaluate this hypothesis.

These arguments encourage the hypothesis that children who are lexically delayed at 2 years are already different—in functions subsumed under social cognition—somewhat before that age. There are very few appropriate data that can be used to support this at present, partly because it is usually too late to look for *any* precursive behaviors by the time lexical delay is documented. However, one thing we might expect to find is diminished use of social vocalization. In that connection, it is interesting that in a prospective study of normally developing children, Nelson (1973) found a relationship between the sheer number of utterances recorded in a session held at 20 months and the age at which a 50-word expressive vocabulary was attained. Number of utterances also was correlated with the age of children at their tenth phrase, and with their rate of lexical acquisition and mean length of utterances. Additionally, it has been found that lexically delayed 2-year-olds initiate fewer of certain types of utterances than normals (Paul & Shiffer, 1991), and it appears that their play and other nonverbal social behaviors are also immature (Paul et al., 1991; Thal, Oroz, & McCaw, 1995; Rescorla & Goossens, 1992).

If at some point lexically delayed children begin to progress at the normal rate, why don't they eventually catch up with those who are never behind? Why the existence of residual deficits in adulthood? The hypothesis offered here is that the critical phase for grammatical analysis, timed by unidentified endogenous factors, expires too soon. When the lexically delayed finally have enough words, neurodevelopmental conditions no longer favor in-statement of grammatical capability. A child who has not realized a sizable lexical increase by about 24 months is therefore at developmental risk, for his analytical-computational capability may not turn all the way on. And even with subsequent advances, it could still be somewhat underdeveloped when the optimum activation period begins to subside.

Figure 2 depicts the neuromaturationally and lexically delayed child who hits a ceiling before linguistic capacity has finished developing. We must assume that there is variation in the timing of the offset, and that the offset is gradual; some lexically delayed 2-year-olds will add significantly to their vocabulary in the year to come (Rescorla, 1989), especially those with good comprehension. But the evidence is that many of the others will continue to lag behind in expressive vocabulary and also in grammatical development (Grimm & Weinert, 1990; Loeb & Leonard, 1991). Several years later, about half of these children will present clinically with highly specific grammatical disorders (Paul & Riback, 1993; Paul & Shiffer, 1991; Paul & Smith, 1993; Rice, Wexler, & Cleave, 1995), with morphology frequently more impaired than syntax (Clahsen, 1989; Smith-Lock, 1993). For example, Smith-Lock (1993) found that SLI children who were 2 years behind their age-matched peers in language, but equivalent on nonverbal measures, were selectively delayed in the acquisition of inflectional morphology and displayed *selective deficits in morphological analysis*.

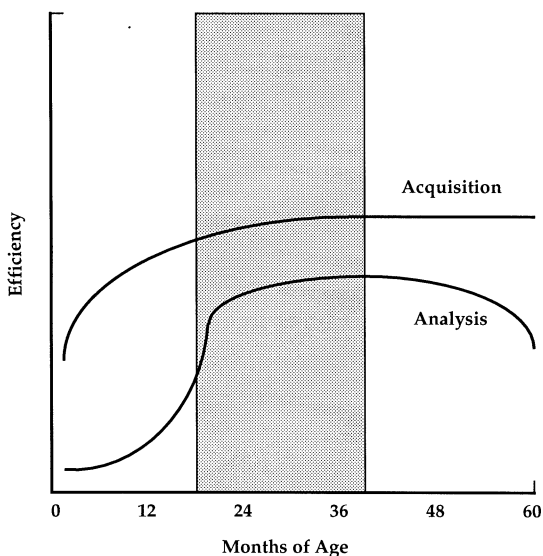


FIG. 2. The plight of the lexically delayed child. Although the nascent utterance-analytic mechanism develops apace, lexical acquisition is behind schedule. By the time there are enough utterances to require a specialized analytical mechanism, trophic conditions are no longer optimal.

NEUROLINGUISTIC EVIDENCE

Developmental neurolinguistic theories must account for, and perhaps are best inspired by, psycholinguistic facts. According to the conception offered here, the problem in developmental language delay originates with delayed development of the child's specialization in social cognition. This primary problem—through the deprived utterance set it produces—then secondarily affects analytical functions. In the section below, I speculate on some possible neuroanatomical consequences of this.

Anatomy of Delayed Language

Language is a species-specific behavior that, like other functional preadaptations, is supported by specialized neural mechanisms. Explicating the neurobiology of developmental language delays might seem to be a fairly straightforward task. Initially one finds out what the linguistic mechanisms are, perhaps by imaging brain activity or lesions in adults, then watches those mechanisms as they fail to develop. But there are problems with this approach. First, children and adults do not necessarily have or use the same neural resources for language production (Ojemann et al., 1989). Second, there is no evidence, at present, of anything intrinsically wrong with the

way lexically delayed children's putative language mechanisms in the left hemisphere are designed or initially might be prepared to operate. Third, the approach described above assumes that our task is to explain the development of neuroanatomical mechanisms that participate in fully competent linguistic behavior. But if we are to avoid "seeing the end in the beginning," i.e., avoid thinking of the infant as an incomplete version of the adult (Studdert-Kennedy, 1991), the task must be to account for the neurology of development. To do this, one must look for brain developments that correspond to behaviors that *lead to* linguistic mastery.

How are neural resources allocated in each of the phases of language development? There are several reasons to impute early vocal learning and utterance acquisition primarily to the right hemisphere in young children. For one thing, that hemisphere is disproportionately responsible for vocal affect and prosody, from which early word-like material emerges (cf. Locke, 1993a).⁹ Second, the right hemisphere appears to be heavily involved in the processing of idiomatic or formulaic utterances of the type that young children typically produce (Van Lancker, 1990). Third, a range of right hemisphere functions, especially those relating to the processing and expression of emotions, develops during the "prelinguistic" period in which prosodically organized utterance material is acquired (Best & Queen, 1989; Cicchetti & Sroufe, 1976; Davidson & Fox, 1988; de Schonen, Deruelle, Mancini, & Pascalis, 1993; Zahn-Waxler, Robinson, & Emde, 1992). Fourth, there is now evidence of temporal coordination between facial affect, a function that is controlled predominantly by the right hemisphere, and early word production (Bloom, 1993). Fifth, electrophysiological recordings reveal that in 20-month-olds in order to measure the difference between known and unknown words activity must be recorded from the right as well as the left hemisphere (Mills, Coffey-Corina, & Neville, 1993). Sixth, of the children who incur unilateral damage in infancy or early childhood, subjects with *right* hemisphere lesions perform significantly worse on lexical comprehension, relative to healthy controls, than do left hemisphere-damaged patients (Aram & Eisele, 1994; Eisele & Aram, 1993; Thal, Marchman, Stiles, Aram, Trauner, Nass, & Bates, 1991; also see Feldman, 1994). The theory offered here accounts for these findings. It also predicts the opposite effect in older subjects—that is, that left hemisphere damaged subjects will do worse on lexical comprehension and expression tasks, and will perform inferiorly on syntactic measures, than right hemisphere damaged subjects. The literature contains support for both of these predictions (Aram, Ekelman, Rose, & Whitaker, 1985; Aram, Ekelman, & Whitaker, 1987; Dennis & Kohn, 1975).

⁹ Although the evidence for this comes mainly from studies of adults, I assume that infants' processing of vocal affect and prosody also takes place primarily in the right hemisphere; within the first few months of life, before any indication of lateralized segmental processing, infants reveal a right-hemisphere specialization for music (Best, Hoffman, & Glanville, 1982; Entus, 1977; Glanville, Best, & Levenson, 1977).

If utterances are acquired by mechanisms responsible for socially cognitive operations, and grammatical analysis attracts material to the hemisphere performing the analysis, then it follows that much of the vocal material that is analyzed and stored in the left hemisphere will previously have spent some time in the right hemisphere in preanalytic, holistic form. This logical scenario is supported by neurophysiological research showing that at 20 months, unknown words selectively activate the right hemisphere, and known and closed class words—the only ones that could have undergone structural analysis—selectively activate the left (Mills et al., 1993).

Much vocal material that is familiar but has not yet been submitted to constituency analyses may continue to reside primarily in quasi- or nonlinguistic storage mechanisms situated in the right hemisphere. This should be unsurprising, since nonhuman primates with limited grammatical capabilities may nonetheless achieve moderately large recognition vocabularies (Savage-Rumbaugh et al., 1993). If the “nonlinguistic brain” can store words, then so, in principle, can “nonlinguistic mechanisms” of the human brain (Locke, 1992), although in actuality there may be several quasi-linguistic areas, or regions that are lexical but not grammatical (Neville, Nicol, Barss, Forster, & Garrett, 1991).

As for the morphology of analytical structures, neuropathological studies reveal that the brain area that is principally implicated in utterance analysis—the left planum temporale—is already larger on the left than the right side in the fetus (Chi, Dooling, & Gilles, 1977; Wada, Clarke, & Hamm, 1975; Witelson & Pallie, 1973). However, morphometric studies show left and right plana to be of similar size in children with developmental language disorders. This departs from the typical adult finding of a larger planum temporale, or perisylvian area, on the left. Because that area is equal to the right in children with developmental linguistic delay, and the left side is where most linguistic analyses are carried out, it would be logical to suppose that language delayed children’s symmetry is due to their left side being smaller than usual. But it is not. *Their right side is larger.*

Some of the evidence supporting these conclusions comes from a magnetic resonance imaging study (Plante, Swisher, Vance, & Rapcsak, 1991) of 4- to 9-year-old boys with developmental language disorders. Volumetric measurements were made of the perisylvian area, which contains portions of the frontal and parietal areas, superior temporal gyrus, and planum temporale, and is active during the conduct of linguistic operations (Petersen, Fox, Posner, Mintun, & Raichle, 1988). In six of the eight subjects tested, the right perisylvian area was either equal to or larger than the left and had a significantly larger absolute volume than in control subjects. The volume of the left perisylvian area did not differ significantly in delayed and normally developing subjects.

Plante (1991) also found that the parents of boys with developmental language disorders generally experienced difficulty with speech, language, or

academic skills when they, themselves, were children. Her morphometric analyses revealed that these parents (and also a sister of one of the probands) had a high incidence of atypical perisylvian asymmetries. Plante concluded that because some individuals without a history of language delay also have atypical perisylvian asymmetry, this particular feature cannot be considered to be specifically linguistic. She also speculated that because the usual perisylvian asymmetry is in place before birth, the symmetries of her language-delayed subjects were set up prenatally. There are alternative ways of viewing both of these issues.

Neurodevelopmental Effects of Lexical Deprivation

If a child cannot achieve its communicative needs with the neural machinery that phylogeny has provided for language, it will use whatever else can be commandeered for that purpose (see below). Symmetries may therefore develop postnatally as an indirect response to general neuromaturational delay. For when lexical development gets off to a late start, and experiences no compensatory surge, there are insufficient words to fully activate the child's utterance analysis system. This system depends on neural mechanisms whose species-typical functions are realized during a circumscribed period and are therefore subject to eventual weakening. There is no reason to suspect, however, that homologous right hemisphere structures that are normally less involved in linguistic encoding and decoding operations are as likely to decline at the same interval in development.

Many have argued for the "equipotentiality" of the two hemispheres, and we need not search extensively for evidence that the right hemisphere can carry out linguistic operations. This possibility is suggested by the fact that a small percentage of neurologically normal subjects are right hemisphere dominant for language, based on carotid-amytal tests (Rasmussen & Milner, 1977), and unilateral left hemisphere damage in childhood can change the hemisphericity of language (Bergman, Costeff, Koren, Koifman, & Reshef, 1984). Carotid-amytal tests with a large number of subjects (Rasmussen & Milner, 1977; Strauss, Wada, & Goldwater, 1992) reveal that lesions of left hemisphere speech areas in childhood frequently shifts manual control and language to presumptively homologous areas in the right hemisphere. A recent electrophysiological study found that in some infants with unilateral left hemisphere damage the right hemisphere takes over the processing of closed class words (Mills, 1994), and there is evidence from adult aphasics to suggest that even the mature right hemisphere can take on linguistic responsibilities that were previously performed by the left hemisphere (Cummings, Benson, Walsh, & Levine, 1979; Kinsbourne, 1971; Papanicolaou, Moore, Deutsch, Levin, & Eisenberg, 1988).¹⁰

¹⁰ This effect is a general one. Damage to relevant nuclei in the left hemisphere of canaries and chicks also shifts functions to the contralateral hemisphere (Horn, 1985; Nottebohm, 1980).

According to the theory of neurolinguistic development espoused here, language areas in the left hemisphere need not be damaged in order to yield to homologous right hemisphere structures. All that is required is lexical deprivation during the interval in which analytical capability normally forms. With time working against them, putative language areas would lose, say, the endocrinological basis for activation or continued operation. Whatever the exact neurotrophic mechanism, one presumes its decline stalls the development of language mechanisms that normally become operational during a circumscribed period of development. In this sense, *inactivation effectively behaves like damage*. This should not surprise us, for it has long been known that when stimulation is not available to an experience-expectant system, structure fails to develop (Wiesel & Hubel, 1963). What is being asserted here is that inactivation also can be caused by *cognitive* understimulation. As would be expected of a competition-based system, other brain areas vie for language. An obvious contender is the right-hemisphere homologue of the hypoactive mechanism. After several years, according to the theory, this "right hemisphere talking" will be revealed functionally and morphometrically. Function studies will indicate that the right side is unusually active during the conduct of linguistic operations, according to the theory, and morphometric studies will reveal decreasing asymmetry over the period from 2 to 6 or 7 years because right-sided structures directly responsible for the enhanced activity enlarge.

Larger Lesions Are Better

At first, there might seem to be a catch. If the scenario described above is accurate, how are we to interpret cases of greater left- than right-hemisphere activity in children who had unilateral left hemisphere damage earlier in their childhood (Papanicolaou, DiScenna, Gillespie, & Aram, 1990)? Why would outright *damage* to the left hemisphere not discourage activity of that hemisphere when, as is claimed here, mere underutilization causes a shift to the right hemisphere? This does not seem troublesome for two reasons. First, unilateral left hemisphere damage may not cause a contralateral shift because the lesion, due to its location, only partially overlaps with grammatically sensitive areas; in such cases, there may be no compelling need for right hemisphere compensation.

A second reason why unilateral damage may not cause a contralateral shift is that the lesion may be small, and there are indications that large lesions may be *functionally less destructive* than small lesions (Irle, 1987). In a study of cats, Irle and Markowitsch (1984) found that prefrontal lesions severely impaired acquisition of a visual reversal task whereas more extensively damaged animals with combined prefrontal cortical, anterior thalamic, mammillary, and subicular lesions learned the task readily. The superior performance of the latter group was attributed to a functional shift to intact brain structures. In a study of infant and juvenile macaques, Carlson (1984a,b) found

that partially lesioned animals required months of training to learn or relearn a tactile discrimination task, whereas totally lesioned animals reached a high level of performance in less than a week. Carlson concluded that "separate processes may be operating to restore function in the case of the partial- and total-lesioned infant . . . function may be more apt to be shifted to another area (whether in the damaged or intact hemisphere) after a total than after a partial lesion of certain cortical areas in immature primates" (p. 113).

In human infants, there appears to be a nonlinear relationship between the size of lesion and degree of resulting linguistic impairment, with some fairly large lesions producing few residual problems (Dall'Oglio, Bates, Volterra, Di Capua, & Pezzini, 1994; Thal et al., 1991a). If large lesions are more likely to discourage species-typical language mechanisms from carrying out their constitutional role too, healthy areas of the brain will be challenged to do that which left hemisphere mechanisms cannot (Mills, Coffey-Corina, & Neville, 1994). For this reason, inactivation may be more likely than unilateral lesions to trigger the use and, compensatorily, growth of homologous right hemisphere mechanisms. If so, 6-year-old children with a history of significant lexical delay may have a larger right perisylvian area than normally developing children. They may also have a larger right perisylvian area than children who sustain focal damage to the language areas of the left hemisphere in their infancy, especially if the damage fails to discourage continued use of ipsilateral mechanisms. In the long run, then, inactivation of a neurolinguistic mechanism could be less injurious to function than outright damage.

Compensatory Hypertrophy

Compensatory hypertrophy of the sort envisioned above is a very common neurodevelopmental process. When normal levels of experience are surgically discontinued, primate brain immediately begins to reorganize itself; neighboring parts of the brain dramatically expand into the now quiescent regions (Merzenich, Nelson, Stryker, Cynader, Schoppmann, & Zook, 1984; Pons, Garraghty, Ommaya, Kaas, Taub, & Mishkin, 1991). Destruction of a structure on one side of the brain causes significant growth of the homologous structure on the other side of the brain (Goldman, 1978; Goldman-Rakic & Rakic, 1984). Unusually heavy use of the brain increases overall brain mass (Diamond, 1988). The frequent exercise of particular behaviors causes elaboration of corresponding control centers (Burnstine, Greenough, & Tees, 1984; Greenough, Larson, & Withers, 1985)—a "glow and grow" type of growth in which structure develops in response to differential activity of the relevant metabolic and electrical systems (Purves, 1994; Stryker, 1994; Wolpaw, Schmidt, & Vaughan, 1991).

Competitive and compensatory effects have also been observed in human brains. Neville, Schmidt, & Kutas (1983) found that in congenitally deaf

subjects, visual stimulation evoked larger responses on the surface of temporal cortex, a region normally associated with auditory processing, than it did in hearing subjects. This is exactly the colonial tendency one would expect to see if an area of the brain, "auditory" cortex in this case, was denied access to stimulation. Using resting EEG with a similar population, Wolff and Thatcher (1990) observed decreased cerebral differentiation, especially in the left fronto-temporal area, and increased differentiation in the right hemisphere, relative to hearing controls. In parallel with Pons et al.'s (1991) deafferentation experiments with monkeys, Ramachandran, Rogers-Ramachandran, and Steward (1992) produced a phantom limb sensation in humans by lightly touching limb amputees' faces on the same side as their missing arm or finger, suggesting that facial sensation areas of the brain may have taken over territory formerly assigned to the limb (a finding confirmed by Yang, Gallen, Schwartz, Bloom, Ramachandran, & Cobb, 1994).

Although demonstrations of lesion-induced hypertrophy have generally occurred at the dendritic level, it is conceivable that the right perisylvian area enlarges when it is challenged to take on, and routinely charged to carry out, levels of linguistic responsibility that are typical for the species but unusual for that hemisphere. The species-typical level of activity is considerable. If a lexically delayed 2-year-old produced *half* as much speech as its normally developing peers, it would still say about 5 to 10,000 words per day (Wagner, 1985). This high level of lexical activity would occur during a period of brisk right hemisphere growth. Indeed, even in children who are left hemisphere dominant for speech, there is continued dendritic branching within the right perisylvian area until at least the age of 6 years (Jacobs, Schall, & Scheibel, 1993; Simonds & Scheibel, 1989), by which time volumetric symmetries may already exist in the language-delayed child (Plante et al., 1991). Moreover, the right hemisphere is not optimally designed for the grammars of language, and it has been speculated that inefficient structures are unusually likely to enlarge with use, presumably because they have to work harder than efficient ones (Kolb & Wilshaw, 1990). It is therefore hypothesized that in children whose left hemisphere language mechanisms have not been fully activated, ordinary levels of linguistic activity during the language learning period induce selective growth of right hemisphere homologues and thus reduce morphometric asymmetry across the hemispheres.

Is there converging evidence from neurophysiological studies of language processing? Language delayed children seem to have something in common with sensorily and socially deprived infants. A part of their brain—a part that analyzes linguistic structure—has been denied the usual input. But the commonality ends where it begins, for in language delayed children the culprit is not an unsupportive environment but an inefficient socially cognitive brain system. One might therefore expect lexically delayed children to use an atypical assembly of neural resources in the conduct of psycholinguistic

operations. Brain function studies, including those that used regional cerebral blood flow (Lou, Henriksen, & Bruhn, 1984) or evoked potentials (Mason & Mellor, 1984), have found reduced activity in putative speech-control areas in children with developmental language disorders, and enhanced activity elsewhere. Recently a right-handed teenager with a well-documented history of a resolved specific language impairment was found, following a gunshot wound, to be right-hemisphere dominant for language (Martins, Antunes, Castro-Caldas, & Antunes, 1995). Although the data base is small at this point, such findings point to functional differences in the language processing of children with developmental linguistic disorders, differences which may have grown up around reduced levels of utterance storage and analysis.

That differences in linguistic experience can differentially affect brain activity is revealed by a recent study indicating that electrophysiological measures of speech perception are influenced by experience. Using EEG, Buchwald, Guthrie, Schwafel, Erwin, and Van Lancker (1994) found that Japanese-reared adults provide no evidence of differential brain activity (and no evidence of differential behavior) during stimulation with /r/ and /l/, whereas English-reared adults do provide evidence of differential brain activity and behavior when presented with tokens of these two phonemes. This result, which undoubtedly reflects different amounts of experience with the /r/-/l/ contrast by Japanese and American listeners, suggests that in other cases atypical brain function may point to nothing more "pathological" than an unusual brain organization that was set up earlier, adaptively, in response to atypical experience. Children experiencing developmental linguistic delays would thus be expected to reveal a deviant pattern of brain activity later on, regardless of their original cause. Unsuspecting researchers may be quick to see the etiological implications of atypical brain activity, but in reality such activity could be an expected manifestation rather than an underlying cause of the linguistic delay.

Over 25 years ago, a landmark postmortem study revealed that in 35 of the 100 unselected brains that were dissected, the left planum temporale was either equal to or smaller than the right planum (Geschwind & Levitsky, 1968).¹¹ As with dyslexic brains, size symmetry was achieved by an absolutely larger than normal right planum rather than a smaller left planum (Galaburda, Corsiglia, Rosen, & Sherman, 1987). At first glance, it may seem unlikely that all cases of symmetry could be due to compensatory hypertrophy, but consider the following. First, virtually nothing is known about the subjects in Geschwind and Levitsky (1968), including their handedness, sex,

¹¹ This is a simply stated outcome, but in actuality there is a continuum of size relationships involving the plana. In Galaburda et al.'s (1987) reanalysis of the Geschwind and Levitsky (1968) sample, there were about as many brains that were *slightly* asymmetric to the right as there were that were slightly asymmetric to the left. Much more substantial interhemispheric differences occurred for extreme asymmetries, where the left planum lead considerably.

developmental history, race, national origin, education, and work history.¹² Second, let us assume that some of the symmetrical brains from Geschwind and Levitsky's sample came from individuals who *never* had a language problem. How do we explain the fact that normally behaving individuals can have abnormally organized brains? This may not be as contradictory as it seems. Successful individuals call upon available neural resources, as needed, in order to meet a range of environmental challenges, but they cannot know *why* their behavior is normal or, indeed, how they use their brains to do any of the things they do. Individuals not appropriately wired for language or its precursors may nevertheless speak, without clinical assistance, as the result of compensations that occur so automatically that no delays are ever observed or documented.

In this context it may be helpful to revisit a finding mentioned earlier—that a linguistically unaffected sister of a language-delayed boy also displayed symmetrical perisylvian areas, just like her affected brother (Plante, 1991). The question here is not why the sister had an atypical brain, but why the proband failed to compensate more effectively for his language learning problem. One might speculate that *both* siblings fell behind in the first or second phase in development of linguistic capacity, and that the unaffected sister, like the proband, relied compensatorily on right hemisphere mechanisms for linguistic operations that normally would be carried out by the left hemisphere. This is not unreasonable—recall that the parents, too, had symmetrical perisylvian areas and histories of developmental language delays, although no contemporaneous communicative disorders were described. For reasons unknown, the boy failed to compensate effectively and thus became “affected.” Why a neural compensation would be more successful in one case than another is unknown, but not at all strange considering the polygenic and epigenetic nature of linguistic capacity.¹³ According to this account, some normally behaving individuals achieve their normalcy not in spite of an atypically organized brain, but because of it, the structural variation serving as a lasting organic record of a successful behavioral adaptation.

Lexical Delay and Developmental Dyslexia

The morphometric findings on children with spoken language delays are congruent with measurements of the planum temporale in adults having a history of difficulty in learning to read (Galaburda, Sherman, Rosen, Aboi-

¹² Recently, this result was replicated with MRI in a smaller group of subjects, but something was known about these individuals—they were right-handed men (Rossi, Serio, Stratta, Petrucci, Schiazza, Mattei, Mancini, & Casacchia, 1994).

¹³ It is possible that males are less able to compensate for developmental language delays than are females. Lefly and Pennington (1991) reported that in their extended family sample, they found 38 male and 19 female adults who were still dyslexic but 7 males and 18 females who had compensated for their dyslexia.

tiz, & Geschwind, 1985). In these studies, too, the left and right plana were of equal size, not because the left was under-sized but because the right was absolutely larger than normal (Galaburda et al., 1987). Later speculation as to the neural developments producing these relationships included most of the logical possibilities. For example, it was recognized that the two plana may be either symmetrical or asymmetrical at birth. If symmetrical, it was considered that this relationship could persist or be altered either by cessation of growth on one side or disproportional growth on the other side. Likewise, initial asymmetry could persist or be altered by cessation of growth of one hemisphere or selective continuation of growth of the other hemisphere (Galaburda et al., 1987; Steinmetz & Galaburda, 1991).

Galaburda and his associates (Galaburda, Rosen, & Sherman, 1990) speculated that in the normal case some volumetric asymmetry exists before birth and becomes even greater due to disproportionate cell death in the right hemisphere. Symmetry is thus attributed to insufficient pruning of cells in the right hemisphere. As presented, it is difficult to evaluate this speculation because it lacks behavioral motivation. The authors do not provide a model of developmental language disorders according to which the typical affected child—who is a strongly right-handed male or female, even if there may be marginal trends toward less than strong right-handedness or maleness—should have a hemispherically selective pruning failure. There is no extant account of behavior development according to which the right hemisphere would be insufficiently pruned.¹⁴

It is not surprising that developmentally dyslexic adults, like children with spoken language delays, would have atypical size relationships in the perisylvian area. After all, many dyslexics experience delays in development of spoken language, and most dyslexics have difficulty in carrying out a range of metaphonological operations even when compared to reading age-matched normals (Pennington, Van Orden, Smith, Green, & Haith, 1990). There is no way to know if Galaburda et al.'s (1985) subjects had spoken language delays because they came to autopsy at advanced ages, having learned to speak before public awareness of developmental communication disorders had developed to present levels. But such a prior history would be unsurprising: in one study, the only remaining sign of developmental language delay between 30 and 60 months was a reading problem at 8 years (Scarborough & Dobrich, 1990).

¹⁴ If I were to offer a behavioral scenario for a pruning failure hypothesis, it would go something like this. Language-delayed children initially appear with small receptive vocabularies, a deficiency that is attributable to generally delayed brain development. This generalized delay initially affects acquisitive functions that are handled by socially cognitive mechanisms situated primarily in the right hemisphere. This underutilization of the right hemisphere results in less than the normal amount of pruning of that hemisphere. I cannot judge on present evidence whether this hypothesis makes good neurodevelopmental sense, but it is a possibility that may be worth further consideration.

These clinical populations appear, then, to share weaknesses in a common underlying area; there are good arguments that lexically delayed children's difficulties in morphology and syntax, and dyslexic individuals' difficulties in segmentation, rhyming and alliteration, originate from the same stratum of the same linguistic domain—the articulated elements of phonology (Leonard, 1989; Mann, Liberman, & Shankweiler, 1980). In this sense, it seems likely that spoken and written language disorders are both “phonology plus” problems. That is, in both types of disorders there may be limits on deeply internal phonological operations that interact with other, unspecified factors. Working in conjunction with these other factors, the phonological deficits would impair performance in one or the other modality, or in both speech and reading.

According to the scenario described above, severe lexical delay causes the right hemisphere to participate in language processing to a greater extent than it normally would (Bryden, 1988), bringing in structures that are not only nonoptimal for spoken language but, as it happens, unsuited to mentalistic phonological operations as well. In that sense, because of the chain of neurogenic developments set in motion by lexical delay, the spoken language problem could indirectly contribute to difficulties in acquisition of written language skills. Lexical delay would promote reliance on structures indisposed to carry out with efficiency the cognitive operations associated with alphabetic reading.

GENETICS

The literature seems to contain no evidence that grammatical capability is slow to activate in children *independent of lexical delay*. Few neurologically normal children acquire utterances easily in the second year of life but freeze when they reach what should be the analytical phase. Such children, if they existed, might be thought to demonstrate a linguistically specific genetic effect. But this still would give us no reason to suspect that humans have a “grammar gene” as some have implied (Gopnik & Crago, 1991).¹⁵ Note the genetic basis of language is not being questioned here, nor is the presence of familial factors in developmental language delays; there is too much evidence to support their existence (Bishop, North, & Donlan, 1995; Lewis, 1990; Lewis, Ekelman, & Aram, 1989; Lewis & Thompson, 1992; Neils & Aram, 1986; Plante, 1991; Tallal et al., 1989; Tomblin, 1989). But these familial, arguably genetic findings do not support the presence of *specifically linguistic* gene action. As Bishop (1987) notes, the rate of overall brain growth also is influenced by genes, so to get a genetically based language

¹⁵ This is underscored by the fact that other investigators later found that the family reported on by Gopnik and Crago had a broad range of previously undocumented cognitive, linguistic, and motor-programming deficits (Vargha-Khadem, Watkins, Alcock, Fletcher, & Passingham, 1995).

deficit one only needs genes that prescribe a delayed onset of neuromaturational development and a species-typical limit on the activation of utterance analysis functions.

For some, the attractiveness of a “grammar gene” may be too tempting to give up easily. That the left planum is already larger than the right in the fetus may suggest that size relationships between the plana are genetically determined and, therefore, that size symmetries in the language disordered also are genetically prescribed. But we must not make the mistake of assuming that “early to develop” means “genetic.” Several investigators (Gottlieb, 1991a; 1991b; Pedersen & Blass, 1981) have demonstrated that prenatal experience can produce species-specific behaviors that are observable at birth, and Weaver and Reppert (1989) have shown that prenatal stimulation can influence early brain structure. What early experience could produce asymmetrical plana? There being some relationship between handedness and asymmetry of the plana (Steinmetz, Volkman, Jancke, & Freund, 1991), the possibility exists that intrauterine manual activity could produce asymmetry in that structure. In an ultrasound study of fetal thumbsucking, it was found that about 90% of fetuses above 15 weeks gestational age suck their right thumb predominantly (Hepper, Shahidullah, & White, 1991). The factors producing manual asymmetries in early fetal life remain unknown, but environmental explanations are not unavailable (Michel, 1983).

DEVELOPMENT OF LANGUAGE LATERALIZATION

At one time, it was believed that neural control of language gradually lateralizes over the preadolescent period (Lenneberg, 1967). This idea has been challenged by presumptive evidence that the left hemisphere is already predominantly responsive to speech at far earlier ages, even before the child begins to speak. The evidence includes studies revealing left-lateralized reactions to consonant–vowel syllables in the neonate and young infant (e.g., Best, Hoffman, & Glanville, 1982; Molfese, Freeman, & Palermo, 1975). How, then, can it be claimed that utterances are initially processed in the right hemisphere, and that an analytical capability housed in the left hemisphere is turned on nearly 2 years later? If language mechanisms in the left hemisphere are already fully functioning in the first 6 months of life, how could there be additional shifts to the left?

Some Asymmetries Develop Gradually

It makes little sense to say that the left hemisphere is already responsible for language during developmental phases in which grammatical capability is not yet developed and, therefore, cannot be actively sited *anywhere* in the brain. Hemispheric priorities may be settled well before adolescence, but there is little reason to suppose that the struggle is over in the first few months or even the first several years of life.

One accepts that volumetric asymmetries exist before birth, when there is no demonstrated linguistic capability, just as one accepts their presence in mature nonlinguistic animals (see review in Galaburda, 1994). However, it is not clear that volumetric measures tell us as much about the expansion of linguistic capacity as dendritic branching. Speech areas of the mature left hemisphere have more distal dendrites than homologous areas of the right hemisphere (Jacobs et al., 1993), but the interhemispheric discrepancy develops gradually over the first 6 years of life, when language is being acquired (Scheibel, 1993). Between 2 and 4 years of age, when there is activation and elaboration of grammatical mechanisms sited principally in the left hemisphere, there is massive development of the brain in general (Chugani et al., 1987; Hashimoto, Tayama, Miyazaki, Fujii, Harada, Miyoshi, Tanouchi, & Kuroda, 1995), and in the left hemisphere in particular (Thatcher, Walker, & Giudice, 1987). At 6 the left Broca's area is still differentiating and still distancing itself from the corresponding area in the right hemisphere (Scheibel, 1993). These neuroanatomical facts would seem to support developmental neurolinguistic gradualism.

On the neurobehavioral side, rightward looking biases—which are associated with a phonetic mode of perception in infants (MacKain, Studdert-Kennedy, Spieker, & Stern, 1983), and correlated with mean utterance length—increase fairly sharply between 15 or 16 months and 19 months (Mount et al., 1989). During this interval, according to the theory presented here, the left hemisphere would be preparing for some serious grammatical business. Inasmuch as the to-be-linguistic areas of the left hemisphere are not more differentiated than homologous areas in the right hemisphere at this age (Simonds & Scheibel, 1989), it is possible that activation of grammatical capacity contributes to the continued development of the left hemisphere.

How, in light of the above, do we explain the greater left hemisphere response to speech at 3 to 4 months of age? One answer is that certain classes of phonetic stimuli (e.g., stop consonant + vowel syllables) are automatically referred to the left hemisphere because of the way its auditory processing mechanisms are wired, not because of any linguistic capability that has already been activated. Experience-expectant properties of mammalian auditory cortex could account for these stimulus biases. Nonhuman primates display greater left- than right-hemisphere activity in response to conspecific calls (Hauser & Anderssen, 1994; Heffner & Heffner, 1984; Petersen et al., 1984), but we do not assume that this finding demonstrates that the monkey's left hemisphere contains mechanisms that are specialized for linguistic grammar.

Early left hemisphere effects may, in an ethological sense, be spurious. In laboratory experiments, stimuli preferentially processed by the left hemisphere typically lack, because they were made to be without, personal, social, or emotional significance to the infant. Indeed, researchers are just beginning to ask how to synthesize vocal emotion (e.g., Murray & Arnott, 1993). When

the stimuli are computer-simulated syllables that have little prosodic contour and emotionality, and appear in no recognizable voice, the right hemisphere has little *reason* to participate. Greater right hemisphere action is hypothesized, other things being equal, where the stimuli are selected for their affective and social significance to the infant (cf. Davis & DeCasper, 1994; Van Lancker, 1991). Even where the stimuli themselves are physically natural, their mode of processing in tightly controlled experiments will generally be unnatural due to instructions that focus the listener's attention on attributes of the stimuli rather than their propositional content (Wray, 1992).

DISCUSSION

That the early lexical learning of infants is distinct from the computational operations that occur later is a subject taken up in Chomsky's (1980) *Rules and Representations*. In his second lecture, he asked readers to consider the possibility that knowing a language "is not a unitary phenomenon, but must be resolved into several interacting but distinct components." (p. 54) The components that were identified included a conceptual system that is responsible for object reference, among other things, and a computational system that is responsible for phonological, syntactic, and semantic patterns. Chomsky also believed it possible, in principle, that a child's conceptual system might be more developed than his computational system, since the two systems would be differently represented in the mind and brain, the products of different evolutionary histories. Others, like Chomsky, have expressed doubts about the linguistic status of behaviors expressed during the one-word stage of language development (Atkinson, 1985; Nelson & Lucariello, 1985).

The neurolinguistically important issue in this is not whether there is an optimal period for learning languages, but whether there is a circumscribed period in which the species-typical preadaptation for language becomes fully operational. Speech areas of the right hemisphere differentiate considerably over the first several years of life (Simonds & Scheibel, 1989). During this interval there are advances in a host of prelinguistic behaviors that are attributed to that hemisphere, e.g., response to vocal and facial affect (Caron et al., 1988). Infants who are delayed in the development of these socially cognitive operations tend to acquire utterances slowly and therefore to feed them slowly to time-locked analytical mechanisms in the left hemisphere. The resulting underactivation of these mechanisms causes homologous areas of the right hemisphere—the best untapped neurolinguistic resource—to assume on a noncompeting and compensatory basis many of the linguistic responsibilities spurned by the left.

Some of this shifting would be avoidable if children with general neuromaturational delays also experienced delayed development of brain systems that normally would analyze linguistic structure, and thereby received an automatic extension of the critical period for induction of species-typical gram-

matical mechanisms. But if these mechanisms are encapsulated from other brain systems, it becomes possible for the developmental neurolinguistic clock to tick for children who are running late just as it ticks for their normally developing peers. Utterance analysis and computation systems would develop on one schedule and other neurobehavioral systems on other schedules.

When the right hemisphere assumes linguistic functions compensatorily, it does not necessarily carry out all the requisite operations flawlessly or in the same way as the left hemisphere. Tomblin et al. (1992) found that 35 adults with well-documented histories of developmental language delay were discriminable from control subjects with regard to spelling, speaking rate, sentence repetition ability, language comprehension, and the grammatical and semantic quality of their spontaneous speech. Measures of sentence comprehension and production were the most discriminating.

Role of Experience

A direct implication of arguments made here is that the development of linguistic capacity requires experience with language—an active involvement in the acquisition and use of language—and not merely exposure to it. Sensory systems develop with physical stimulation alone, but analytic systems require a certain amount of perceptual capture before they can go to work. In discussions of development, however, “genetic factors” are frequently pitted against “the environment.” Putting aside this obvious violation of epigenetic principles, how does one effectively separate some idealized or adultocentric sense of which utterances fall within earshot of the child, or are directed to the child, from the child’s own sense of what exists?

Although most of the work on sensitive periods has been behaviorally oriented, animal behaviorists began to consider the neural correlates of sensitive periods over 30 years ago. Thorpe (1961), for example, observed that “there are rather isolated observations which suggest that there exist specific brain mechanisms ready to be activated only during a particular period of the life span and that if they are not properly activated at the right time subsequent activation is impossible, resulting in permanent disabilities in later life” (pp. 211–222).

In at least one way, the use made of the critical period concept here parallels that of developmental neurophysiologists and anatomists. In the development of vision in the cat, for example, animals unexposed to ambient stimulation during an age-defined period fail to develop experience-expectant neural processing capacity and thus are functionally blind. Histological analyses reveal undeveloped visual cortices. Similar research with primates suggests there may be several partially overlapping critical periods, one for each of several distinct types of visual processing (Harwerth, Smith, Duncan, Crawford, & von Noorden, 1986). In the development of language, lexically de-

layed children are underexposed to endogenous stimulation and thus fail to develop species-typical neural processing mechanisms. But in the case of language, alternative mechanisms are available, and the individual is not therefore denied linguistic capability.

It has been suggested that the term “sensitive period” fits the developmental linguistic facts more closely than the term “critical period” because the early years are an especially good time, but not the only time, one can learn a language (Oyama, 1976). But Oyama was looking at the language learning of individuals who had already acquired their first language. In contrast, the issue here has been the *activation* of language learning mechanisms, something that happens only once in the life of an individual. Attention has additionally been directed to the activation of a particular component—the mechanism that analyzes stored utterances into their constituent elements. As was seen earlier, individuals whose lexical store is understocked during the developmental period in which computational operations normally begin are susceptible to residual deficits in adulthood. Now in this context, one could well ask: in what sense is “critical period” *not* an appropriate term?

Appropriate though “critical period” may be, it is not a critical period for language *behavior*, but for the generation of language behavior by specialized species-typical grammatical mechanisms. This is an altogether different use of the concept, which in the past has involved behavior or the capability for behavior. Any attempt to salvage the conventional concept would have to appeal to either of two types of behavioral evidence. One type of evidence relates to linguistic performance when speakers are pushed past their natural limits or evaluated on subclinical measures such as rate of spontaneous speech, ability to do various types of language games, or to perform on metalinguistic tasks, and the like. In such cases, the measure would be a behavioral one, but may have little to do with some minimal level of functional communicative or representational linguistic adequacy. The other type of evidence relates to mode of linguistic processing, where one investigates the strategies by which individuals carry out linguistic operations associated with expression or comprehension of running speech. Both of these categories of behavioral measure should be pursued in future research, but the outcome of such studies, while enhancing our understanding of the behavioral expression of species-atypical processing mechanisms, would be unlikely to affect the definition of a critical period for language, per se.¹⁶

Invocation of the critical period concept gives a helpful boost to Bishop and Edmundson’s (1987a) “maturational lag” hypothesis of developmental language disorders. Their data reveal that most children with delayed lan-

¹⁶ By extension, one might suggest that the orthodox definition of sensitive periods applies primarily to behaviors having little in the way of effective fall-back mechanisms, since it is in those cases that an animal would be caught not behaving as expected.

guage tend to progress linguistically at the same rate as chronologically younger children of the same linguistic age. But because, as they note, many of these children never do catch up with their peers, "additional explanations" need to be added to the lag hypothesis. A critical period for the in-statement of the optimally efficient species-typical grammatical mechanisms represents such an explanation.

Studies of acquired deafness (in families that do not sign) suggest that the left hemisphere needs about 3 years of initial access to speech if it is to acquire disproportionate responsibility for language (Marcotte & LaBarba, 1987; Marcotte & Morere, 1990). It is interesting in this connection that Genie, the linguistically and socially deprived teenager who heard little speech beyond 20 months, developed a clear right-hemisphere dominance for language, although her manual activity fell predominantly under the control of the opposite (left) hemisphere (Fromkin, Krashen, Curtiss, Rigler, & Rigler, 1974). Once rescued from her isolation, Genie developed vocabulary but made very little progress in grammar. This is not surprising, since by the theory presented here grammar has a narrowly circumscribed activation period—it should have begun almost exactly when her deprivation commenced in earnest—whereas lexical development is more open. The authors speculated that "the left hemisphere must perhaps be linguistically stimulated during a specific period of time for it to participate in normal language acquisition. If such stimulation does not take place during this time, normal language acquisition must depend on other cortical areas and will proceed less efficiently due to the previous specialization of these areas for other functions." (p. 102) This speculation is congruent with claims made here about children who get off to a late start in the development of vocabulary.¹⁷

Underpowered Mechanisms

It seems reasonable to suppose that while utterance analysis is carried out by a modular system, grammatical deficits need not arise from a structural defect in the module and language disorders may not be primarily grammatical. An underpowered acquisition system may produce too few utterances to activate species-typical analytical mechanisms and thereby produce a lasting structural deficit that affects several levels of language. When that happens, one could argue that a transient behavioral delay has produced a permanently limited capacity, at least if neurolinguistic mechanisms in the mature state were underpowered or atypically configured.

This may be the first time that data from lexically delayed children have been used to support a theory about normal language development. And yet,

¹⁷ It should be noted that Genie may have made somewhat more progress in morphology and syntax than has generally been assumed; the degree of her grammaticality at last formal observation remains unresolved (Jones, 1995).

their lack of a circumscribed lesion or period of linguistic deprivation makes them even more interesting than children with such exceptional circumstances. Their slowness to develop may represent natural variation; indeed, some have suggested that lexically delayed children merely represent the lower end of a normal distribution (Leonard, 1991). As a population, they come fairly neatly graded into mild, moderate, and severe delays, satisfying requirements for the study of critical period phenomena set down by Bateson and Hinde (1987), and one can observe them as they reach chronological ages at which something linguistic would normally happen. The data from these children therefore speak in interesting ways to the evolving relationships between neural, cognitive, and linguistic systems.

Although current evidence indicates that language delayed children are generally slow to acquire utterance material—the primary function of the second phase in development of linguistic capacity—it is entirely possible that delays have already occurred in the first phase. This is suggested by the fact that the typically developing 8-month-old gives evidence of comprehending 36 words (Bates, Marchman et al., 1994; also see Harris, Yeeles, Chasin, & Oakley, 1995). To receive credit for lexical comprehension, the infant must respond to a spoken word appropriately and therefore must have stored referential information along with the phonetic form itself and been disposed to point to, look at, touch, or otherwise identify named objects and actions. The comprehension evidence may thus be conservative; the typical 8-month-old may have stored somewhat more than 36 utterances. Unless all utterances are, implausibly, learned the day before the first documentations of comprehension, infants must begin to store utterances in the preceding month or two. During this period, there is evidence that infants' perceptual vowel categories are already being influenced by listening experience (Kuhl et al., 1992).

There may be important differences between partial loss of function when portions of a language structure are damaged and complete loss of function when that same time-locked structure is not activated. Under certain circumstances, inactivation may set in motion neurodevelopmental events that lesions cannot. For example, inactivation may indirectly promote greater growth and activity in right-sided structures in lexically delayed children than in controls or children with lesions “in the vicinity of” language mechanisms in the left hemisphere.

Crowding

If evolution is a tinker (cf. Jacob, 1982), then brain structures that used to do one thing now do several things, and a similar phenomenon can occur ontogenetically as multiple functions compete for a limited number of synaptic sites (Bishop, 1981). It has been suggested that ontogenetic crowding can reduce efficiency of the new, and now overburdened, mechanisms. Woods

and Teuber (1973) thought that, following left hemisphere damage, verbal functions might recover at the expense of nonverbal functions. Milner (1984) suggested that when there is extensive damage to putative language areas in the left hemisphere, control may shift to the right hemisphere, negatively affecting general intelligence. "There is always an intellectual price to pay for such plasticity," she argued, for "people with most of their cognitive abilities 'crowded' into one hemisphere are likely to be lower in general intelligence" (p. 87).

If linguistic responsibilities are absorbed by mechanisms in the right hemisphere, these mechanisms would have been doing something else beforehand. One of these responsibilities is visuospatial processing (Henry, Satz, & Saslow, 1984; Wendt & Risberg, 1994). Stiles and Nass (1991) demonstrated that right hemisphere damage impairs performance on spatial integrative tasks—the performance of their recovered right hemisphere-damaged patients was qualitatively different from that of normals. Is there reduced performance on visuospatial tasks by individuals recovering language following left hemisphere damage? The evidence suggests that there is, at least if the damage occurs before the age of 5 years (cf. Liederman, 1988; Mancini, de Schonen, Deruelle, & Massoulier, 1994; Witelson, 1977). We might therefore expect that when lexically delayed children tinker, they begin to carry-out linguistic operations with right hemisphere mechanisms. This may cause the rate of visuospatial development to decline with progress in language, providing indirect evidence of right-hemisphere compensation.

Competitive Exclusion

Recent reinterpretations of the work on imprinting suggest that the offset of sensitivity is caused by the preemption of structure by specific experience—a theory of competitive exclusion—rather than the nonspecific stimulation that occurs with the passage of time—the so-called "clock model" of behavior development (Bateson, 1987). If few stored utterances are available when analytical mechanisms become "ready" to operate, it is not clear what would cause these mechanisms to become "unready" for phonetic analysis. This is because little is known about whatever nonlinguistic functions might be carried out by neural systems otherwise dedicated to analytical operations. However, it seems reasonable to suppose that when underutilized, language-dedicated systems, like other specialized brain systems, are usurped by competing functions.

To assume that language is domain-specific, as many now do, is to assume that language mechanisms carry out only linguistic operations (see Bates, 1994, for an analysis of the relevant arguments). Lexically delayed children provide an opportunity to ask some pertinent questions about this assumption. For example, when analytical-computational functions are taken up by the right hemisphere, does something else move with it? If language areas

of the left hemisphere are also responsible for hierarchical organization (Greenfield, 1991; Grossman, 1980), when the child turns compensatorily to the right hemisphere for the control of language, does organizational ability move with it? If so, how efficient are right hemisphere mechanisms in these areas?

Regulation

If brain-damaged and severely lexically delayed children are able to achieve normal or near-normal linguistic competence with less than species-typical levels of participation by the left hemisphere, one must assume the presence of a regulatory or coordinative function, a set of "rules for changing the rules" when the linguistic game plan is thrown off by a lesion or delayed maturation (Bateson, 1976). Since such accidental or natural variations do not necessarily prevent infants from reaching approximately the same endpoint as their peers, one must assume that there is an internal guidance system for language development that contains a self-correction system. Thus, the ultimate linguistic success of lexically delayed children may reflect the availability of back-up systems and the efficiency of a central oversight mechanism in deploying and coordinating those resources.

QUESTIONS REVISITED

It is time to revisit the three questions asked at the outset. The first question pertained to the neural mechanisms that support language development. Although one cannot say precisely what these mechanisms are, it appears that there are several, as discussed, and not just one system that becomes increasingly empowered. As for when, and under what neurogenic and behavioral circumstances, neurolinguistic mechanisms become active, it seems possible that although linguistic capacity emerges gradually, individual mechanisms may activate on individual schedules that relate both to neuromaturational factors and to specific behavioral pressures provided by capacities developed previously. As for when the activation period for neurolinguistic mechanisms draws to a close, here one must hazard a guess. In the typical case, the totality of mechanisms needed for adult-like levels of linguistic control are all functioning at high levels at 5 or 6 years. There would be no benefit in keeping the activation period for these systems open much beyond this age (Hurford, 1991; Pinker, 1994).

TESTS OF THE THEORY: STRUCTURAL CONSIDERATIONS

Earlier the phases of language development were seen as discontinuities in observable speaking behaviors, and we observed findings on lexically delayed children that seemed to confirm the temporal envelope and functionality of these phases. The developmental neurolinguistic theory offered here

can and should be tested. An initial indication of how this might be done is available in Diamond's (1991) guidelines for testing brain-behavior relationships. For example, to obtain a measure of convergent validity, Diamond suggested that data be gathered from a range of tasks, all showing improvement in performance at some point in development, linked to a particular neural system. For the present proposal, this would require evidence from a range of naturalistic behaviors and experimental tasks to indicate that analytical and computational capabilities are present, and that utterance analyses are taking place in the vicinity of the left perisylvian area.

Diamond suggested several measures of divergent validity. One measure requires evidence that other brain systems are unrelated to the observed improvement in performance. This could be done by showing that other areas of the brain are relatively less active than the left perisylvian area during the conduct of analytical and computational operations. In a second measure of divergent validity, the investigator would obtain evidence that performance on tasks linked to other neural systems is unaffected by disruption of functioning in the neural system of interest. To follow this guideline, one needs evidence that performance on tasks associated with other areas of the brain does not decline, except as might be produced by crowding, when putative language areas in the left hemisphere lose species-typical levels of control over language.

One can of course go beyond the guidelines suggested by Diamond, which were offered in the context of testing frontal lobe development. The following falsifiable predictions are offered additionally:

1. Children with small vocabularies and poor grammar will reveal levels of brain activity (as evaluated, e.g., by Thatcher et al., 1987) and brain maturation (as evaluated, e.g., by van der Knapp, Valk, Bakker, Schooneveld, Faber, Willemse, & Gooskens, 1991) that are characteristic of chronologically younger children.

2. Children with small vocabularies and poor grammar who subsequently make significant linguistic progress will—during the time of, and in proportion to, this progress—experience slow-down in the development of right hemisphere cognitive and socially cognitive functions. This will be revealed by reduced performance, relative to appropriate controls, on tasks associated with a fully operational right hemisphere, e.g., those tapping visuospatial functions, interpretation of vocal and facial affect, and possibly the concepts and processes associated with calculation.

3. Following unilateral left hemisphere damage, there is a greater incidence of right hemisphere control of language in individuals also experiencing a change from right- to left-handedness. A weakening of right-handedness also may occur as children develop language following serious delays in development of expressive vocabulary and analytical-computational functions. A corollary of this prediction is that right-handedness may be weaker in resolved than in unresolved cases of developmental language delay.

4. In the children in (2) above, right hemisphere mechanisms responsible for linguistic progress will appear absolutely more active during MR-imaged language processing and relatively more active than homologous mechanisms in the left hemisphere, than is the case with appropriate control subjects.

5. In children described in (2) and (4), atypically active right hemisphere areas will increasingly elaborate and expand during the period in which language capacity develops, reaching or exceeding the complexity and, perhaps, size of homologous mechanisms in the left hemisphere.

To be comprehensive, developmental neurolinguistic theories must include cognitive accounts of the many behavioral changes that precede and lead continuously to a full command of language. They also must identify a consortium of neurolinguistic mechanisms, each with different degrees and types of functional responsibilities. But success in these efforts seems unlikely unless theorists also recognize the existence of variation in linguistic material and populations of speakers. Utterances distribute along a creativity continuum extending from the formulaic and overlearned to the generative and novel. These different types of material are produced and perceived with dissimilar allocations of cognitive and neural resources (Swinney & Cutler, 1979; Van Lancker, 1990). Likewise, some children are more creative than others (Nelson 1973), and there is every reason to assume that they process language with differing configurations of neurocognitive resources. Characterizing research populations by their gender or handedness seems to make no more sense, and may prove less helpful, than categorizing subjects by their language processing style.

Future theorizing in developmental neurolinguistics will most productively occur within a framework that jointly values descriptive linguistic and psycholinguistic data on the one hand, and neurodevelopmental findings on the other. Morphometric and functional brain measures must be performed in conjunction with fine-grained qualitative linguistic and cognitive analyses of a type that are addressed by few if any standardized tests. Without such analyses, one misses discontinuities of behavior that point to important transitions between emerging informational systems. These transitions, in turn, imply shifting challenges to nascent brain mechanisms.

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