

EARLY LANGUAGE DEVELOPMENT AND ITS NEURAL CORRELATES

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Most children master the basic structures of their native language by the age of four, together with an array of cognitive and social accomplishments that appear to be necessary for language learning to take place. As a result of all this rapid development, the 4-year-old is a very sophisticated being: indeed, we have met children as young as three who can use their new linguistic tools to engage in dialogues about life after death, and the existence or nonexistence of God. How does all this happen in such a short time, in all normal children, in every culture? It is difficult to escape the conclusion that language is part of our biological heritage, an achievement that depends upon the unique characteristics of the human brain. To the extent that this is true, developmental psychology and cognitive neuroscience are faced with a great opportunity: by studying the co-development of brain and language in the first few years of life, we may be able to identify some of the neural mechanisms that permit the emergence of language in our species.

Unfortunately, this is easier said than done. There is a discouraging disparity between our knowledge of language development and our knowledge of the brain mechanisms that support it in humans, a disparity so great that it is difficult to formulate a coherent theory of the neural events that make language possible. In fact, as we will point out later, current evidence suggests that the relationship between brain and language development is far more complex than previously believed, with causation running in both directions—including effects of learning itself on the developing nervous system.

On the behavioral side, a vast and detailed body of information about language acquisition has been gathered in the forty years since Roger Brown and his contemporaries launched modern research in this field (Kessel, 1988). This includes information from dozens of different languages (Slobin, 1985-1997), in situations of poverty and privilege (Hart & Risley, 1995; Wells, 1985), in normal children and in a range of clinical populations (Beeghly & Cicchetti, 1987; Bishop & Mogford, 1993; Broman & Fletcher, 1999; Broman & Grafman, 1994; Leonard, 1998; Tager-Flusberg, 1994, 1999; Thal & Reilly, 1997). Although the process of data collection and transcription is tedious and expensive, a computerized archive of child language data has been established that puts these hard-won products at the disposal of interested researchers around the world (MacWhinney, 1992, 1995; Mac-

Whinney & Snow, 1985, 1990). As a result, detailed records of language acquisition are now available to linguists, computer scientists, neuroscientists and other investigators who do not have the necessary time or expertise to gather such data for themselves. To be sure, controversies abound in this field, and there is no consensus about the nature of language learning or the amount of innate knowledge that is necessary for such learning to take place. There are still many proponents of a strong nativist view, in which language is viewed as an “instinct” (Pinker, 1994) or a special-purpose “mental organ” (Fodor, 1983), with its own neural architecture and its own genetic program (Gopnik, 1997; Rice, 1996). However, evidence and arguments have mounted for a more epigenetic perspective, in which the human capacity for language emerges, phylogenetically and ontogenetically, from quantitative changes in mental/neural systems that humans share with other species (Bates, Bretherton, & Snyder, 1988; Bates, Thal, & Marchman, 1991; Deacon, 1997; Elman et al., 1996; Lieberman, 1998; MacWhinney, 1999; Quartz & Sejnowsky, 1997; Tomasello & Call, 1997). Some of these controversies could be resolved if we had a better understanding of the relationship between brain and behavior during the language-learning years. The comparative study of language and brain development has been held back not by a lack of information about behavioral change, but by a paucity of information about the changes that take place in the human brain during the years in which most children acquire their first language. There are two related explanations for this disparity.

First, for ethical reasons many of the most important tools of modern neuroscience (e.g., single-cell recording) cannot be used with human beings of any age. A handful of noninvasive methods can be applied in the study of human adults (e.g., magnetic resonance imaging; positron emission tomography; event-related brain potentials), but there are additional ethical and/or practical constraints that limit the use of these methods with human children. In other areas of cognition (e.g., memory, attention, visual perception), some of these ethical and practical constraints can be circumvented through the use of animal models. For obvious reasons, we cannot build a convincing animal model of language use or language learning—although, as we will outline in more detail below, it may be possible to formulate useful animal models of the nonlinguistic

mechanisms on which our capacity for language is built.¹

The second problem follows from the first. Much of our information about the neural bases of language has come from studies of brain-damaged adults, based on well-known correlations between site of lesion and forms of language breakdown (i.e., aphasia). However, these brain-behavior correlations are far from perfect, and there are a number of serious logical and empirical problems that limit the interpretability of lesion data (Bates, McDonald, MacWhinney, & Appelbaum, 1991; Caramazza, 1986; Shallice, 1988). These problems multiply when the lesion method is extended to studies of brain-injured children, because the effects of focal brain injury in childhood are much less severe and even less consistent than the effects of a homologous injury in an adult. Although this plasticity is not total, and some residual effects of early damage can be seen in young victims of either left- or right-hemisphere injury (Aram, 1988; Aram, Ekelman, & Whitaker, 1985; Aram, Ekelman, Rose, & Whitaker, 1985; Dennis & Whitaker, 1976; Eisele & Aram, 1995; Vargha-Khadem, Isaacs, & Muter, 1994; Vargha-Khadem, Isaacs, Watkins & Mishkin, in press), it seems clear that the human brain can organize or reorganize in ways that we do not yet understand. In fact, *most* children with early focal brain injury achieve what appear (on the basis of current measures) to be normal or near-normal levels of language ability, despite damage to regions of cerebral cortex that are thought to be crucial for normal language functioning in an adult. This phenomenon provides an interesting and important challenge to neurolinguistic research. As we will point out later, our understanding of the effects of early focal brain injury has improved markedly in the last few years, as researchers move their focus from retrospective studies (looking at the sequelae of early injury, long past the period in which language is normally acquired) to prospective studies (following children with congenital injuries from the very beginning of language development, observing the plastic reorganization for language for which these children are famous *as it occurs*).

Despite these positive signs, many aphasiologists believe that research on language development in brain-injured children is too complex, and should be postponed until we make more progress in understanding

¹The ongoing debate about "ape language" remains unresolved. On the one side, it is claimed that chimpanzees are merely skilled imitators, and that none of their signs are symbolic (Seidenberg & Petitto, 1979). Yet work with the pygmy chimpanzee (*pan paniscus*, Savage-Rumbaugh, 1986) has provided support for linguistic abilities in that species. She has reported generative use of signs and convincing evidence of comprehension of spoken English

aphasia in the adult "steady state".² Is research on the brain bases of language development too difficult to undertake at this time? Although this concern is understandable, we propose a more optimistic view, dubbed the *structure-from-motion principle*: The outer boundaries and internal structure of a physical object are often easier to see when that object starts to move; so too, the relationship between brain and language may emerge more clearly when we observe its construction over time, under a range of normal and abnormal conditions.

Our goal in this chapter is to promote much-needed research on the neural substrates of language development, by describing milestones and variations in the behavioral domain that parallel developments in the human brain. We should stress from the outset that such correlations do not imply *any* simple form of cause and effect. Language development necessarily reflects a complex bidirectional interplay of maturation and experience: although some level of brain organization is necessary for learning to take place, it is now clear that experience helps to shape the architecture of the brain throughout development (Elman et al., 1996). Hopefully the correlations that we will point out here will ultimately be replaced by a more subtle theory of the brain and the mechanisms responsible for language learning in our species, one that can accommodate the bidirectional cascade of causes that are responsible for these events.

The remainder of this chapter is organized as follows:

Part I: Component Parts of Language.

In this section we will define those linguistic terms (e.g., phonology, semantics, grammar, pragmatics) that are typically used in studies of language and language development.

Part II: Adult Aphasia. Traditionally, neuropsychological research with adults is used as the starting point for neuropsychological studies of children. In this section, we will review efforts in the adult aphasia literature to establish a one-to-one mapping between components of language and components of the brain. We will show why this effort has failed, and why most aphasiologists are searching for a new framework to characterize language breakdown in adults. We end this section by suggesting a reversal of the traditional approach: developmental research may help adult aphasiology to shape the new framework by providing a new view of brain/mind architecture based on the mechanisms that are used to get language off the ground in the first place.

²The term "steady state" is used here because it is a commonly used term to describe the asymptotic levels that are reached in many structural and functional systems by adulthood. However, we do not mean to imply that there are no changes in the adult brain.

Part III: Prerequisites to Language. This section offers a summary of the developments in the first year of life that lead up to the emergence of meaningful speech. The summary is divided into five areas of cognition and communication that come together in the child's first efforts to map sound onto meaning around 8-10 months of age, including (1) the ability to analyze and produce **speech sounds**, (2) the ability to **recognize and categorize objects** (the obvious prerequisite to naming), (3) the development of **imitation** (i.e., the ability to translate auditory and/or visual input into a motor analogue), (4) the development of those forms of **intentionality** (including social intentions and means-end analysis) that are necessary for the deliberate use of sound to express meaning, (5) basic changes in **memory** that make it possible to store and reproduce sound-meaning pairs.

Part IV: Language Milestones. Here we will describe basic milestones within "language proper", including first signs of **word comprehension** (9-10 months), first signs of **word production** (12-13 months), first **word combinations** (20 months), and the burst of **grammatical development** that follows soon thereafter (20-30 months). We will also characterize some relatively late changes in linguistic ability, from 3 years of age through puberty.

Part V: Variations and Dissociations. This section is a supplement and corrective to Parts III and IV, focusing on the range of variations that are possible at each of these developmental milestones: variations in rate and style that have been observed within a single language, variations that occur across drastically different language types, and certain robust dissociations between linguistic milestones that have been reported in normal and abnormal populations. This will include a survey of dissociations (or lack thereof) in children with congenital lesions to the left or right hemisphere, an important contrast to our review of adult aphasia in Part II.

Part VI: Neural Correlates of Language Development. After our survey of milestones and variations in language development, Part VI will contain an overview of current information about pre- and postnatal development in the human brain, focusing on similarities and differences between those events in humans and other species, with the hope of deriving some neural factors that may play a role in the emergence of language in our species. If our reader is hoping for a neat match between linguistic milestones and maturational events in the human brain, s/he will be very disappointed. We will show instead that the search for simple one-to-one correlations between neural and behavioral events must be abandoned in favor of dynamic theories that can encompass the complex bidirectional interplay of brain and behavior that occurs during development.

I. THE COMPONENT PARTS OF LANGUAGE

The study of speech sounds (phonetics and phonology). The study of speech sounds can be divided into two subfields: *phonetics* and *phonology*. Phonetics is the study of speech sounds as physical and psychophysical events. This includes a huge body of research on the acoustic properties of speech, and the relationship between acoustic features and speech perception (Ohala & Jaeger, 1986; Pickett, 1985). It also includes the detailed study of speech as a motor system, with a combined emphasis on the anatomy and physiology of speech production (Levelt, 1989; Levelt, Roelofs, & Meyer, 1999). Within the field of phonetics, linguists work side by side with acoustical engineers, experimental psychologists, computer scientists and biomedical researchers (Blumstein & Stevens, 1981; Jakobson, Fant, Gunnar, & Halle, 1952; Kent, Weismer, Kent, Vorperian, & Duffy, 1999; Perkell et al., 1997).

Phonology is a very different discipline, focused on the abstract representations that underlie speech perception and production, within and across human languages. For example, a phonologist may concentrate on the rules that govern the voiced/voiceless contrast in English grammar, e.g., the contrast between the unvoiced "-s" in "cats" and the voiced "-s" in "dogs". This contrast in plural formation bears an uncanny resemblance to the voiced/unvoiced contrast in English past-tense formation, e.g., the contrast between an unvoiced "-ed" in "walked" and a voiced "-ed" in "wagged". Phonologists seek a maximally general set of rules or principles that can explain similarities of this sort, and generalize to new cases of word formation in a particular language. Hence phonology lies at the interface between phonetics and the other regularities that constitute a human language, one step removed from sound as a physical event.

Some have argued that phonology should not exist as a separate discipline, and that the generalizations discovered by phonologists will ultimately be explained entirely in physical and psychophysical terms. Others maintain that phonology is a completely independent level of analysis, whose laws cannot be reduced to any combination of physical events. A unification of these two disciplines is limited by the fact that training in phonology takes place entirely within the field of linguistics while training in phonetics usually takes place in departments of psychology, cognitive science, computer science or acoustic engineering. As a result, phonologists and phoneticians are rarely active consumers of each other's research. Nevertheless, there are reasons for optimism. First, there have been a number of theoretical advances in phonology, including "auto-segmental phonology" and "optimality theory" (Menn & Stoel-Gammon, 1995; Stemberger & Bernhardt, 1999) that bring the field closer to an understanding of the physical substrates of the sound system. Second,

there is a complementary trend within experimental phonetics, as researchers make use of ideas and methods from computer science and neural network modeling) (Dell, Schwartz, Martin, Saffran, & Gagnon, 1997; Plaut, 1994) that are much more compatible with these new phonological theories. These new ideas have just begun to appear in research on phonetic and phonological development, with great promise for the future).

The study of meaning (semantics). The study of linguistic meaning takes place within a subfield of linguistics called *semantics*. Semantics is also a subdiscipline within philosophy, where the relationship between meaning and formal logic is emphasized. Semantics can be divided into two areas: *lexical semantics*, focused on the meanings associated with individual lexical items (i.e., words), and *propositional* or *relational semantics*, focused on those relational meanings that we typically express with a whole sentence.

Lexical semantics has been studied by linguists from many different schools, ranging from the heavily descriptive work of lexicographers (i.e., “dictionary writers”) to theoretical research on lexical meaning and lexical form in widely different schools of formal linguistics and generative grammar (Fauconnier, 1985; Goldberg, 1995; Jackendoff, 1983; Lakoff, 1987; Langacker, 1987; Newmeyer, 1998; Tomasello, 1998). Some of these theorists emphasize the intimate relationship between semantics and grammar, using a combination of lexical and propositional semantics to explain grammar; others argue for the structural independence of these linguistic domains.

The study of lexical processing is one of the busiest subfields in psycholinguistics, because it is now possible to study the “temporal microstructure” of word comprehension and word production, in and out of context, using computer-controlled, “on-line” techniques (both behavioral and electrophysiological) that can track these events with 1-10-millisecond sensitivity (Gernsbacher, 1994; Grosjean & Frauenfelder, 1996; Kutas & Van Petten, 1994; Marslen-Wilson, 1989; Small, Cottrell, & Tanenhaus, 1988). The major issues of concern within this field are similar to the ones that divide linguists: some view lexical access as an independent mental activity, a kind of reflex that is not influenced by higher levels of knowledge (although the products of lexical access are rapidly passed on to these higher mental processes—Friederici & Frazier, 1992; Levelt, Roelofs, & Meyer, in press; O’Sheaghda, 1997; Swinney, 1979); others view lexical access as a process that is deeply penetrated by sentence meaning and other sources of information, a process that may actually begin before the very first portion of a target word is presented (Allopenna, Magnuson, & Tanenhaus, 1998; Altmann, van Nice, Garnham, & Henstra, 1998; Elman, 1990; Elman & McClelland, 1986; Grosjean, 1980; MacWhinney & Bates, 1989; Marslen-Wilson & Tyler, 1981, 1987; MacDonald, Pearlmutter, & Seiden-

berg, 1994; McRae, Spivey-Knowlton, & Tanenhaus, 1998; Spivey-Knowlton, Tanenhaus, Eberhard, & Sedivy, 1998; van Petten, Coulson, Rubin, Plante, & Parks, in press; Vu, Kellas, Metcalf, & Herman, in press; Vu, Kellas, & Paul, 1998). This split in psycholinguistics between “modularists” and “interactionists” reflects the split in theoretical linguistics between proponents of “linguistic autonomy” (e.g., Chomsky, 1982) and cognitive linguists who emphasize the interactions between grammar and semantics (e.g., Bates & Goodman, 1997; Langacker, 1987).

In contrast to the feverish empirical work on lexical semantics and lexical processing in the field of psycholinguistics, propositional semantics has been dominated primarily by philosophers of language. The primary issues here revolve around the relationship between the “natural logic” that underlies natural language, and the range of possible logical systems that have been uncovered in the last two centuries of research on formal reasoning. A *proposition* is defined as a statement that can be judged true or false. The internal structure of a proposition consists of a *predicate* and one or more *arguments* of that predicate. An argument is an entity or “thing” that we would like to make some point about. A one-place predicate is a state, activity or identity that we attribute to a single entity (e.g., we attribute beauty to Mary in the sentence “Mary is beautiful”, or we attribute “engineerness” to a particular individual in the sentence “John is an engineer”); an n-place predicate is a relationship that we attribute to two or more entities or things (e.g., we predicate an asymmetric relationship of “kissing” to two entities in the sentence “John kisses Mary”, or we predicate an asymmetric relationship of “giving” to three entities in the sentence “John gives Mary a book”). Philosophers tend to worry about how to determine the truth or falsity of propositions, and how we convey (or hide) truth in natural language and/or in artificial languages. Linguists worry about how to characterize or taxonomize the propositional forms that are used in natural language. Psychologists tend instead to worry about the shape and nature of the mental representations that encode propositional knowledge, with developmental psychologists emphasizing the process by which children attain the ability to express this propositional knowledge.

The study of how signals are combined (grammar). The subfield of linguistics that studies how individual words and other sounds are combined to express meaning is called *grammar*. The study of grammar is traditionally divided into two parts: *morphology* and *syntax*.

Morphology refers to the principles governing the construction of complex words and phrases, for lexical and/or grammatical purposes. This field is further divided into two subtypes: *derivational morphology* and *inflectional morphology*. Derivational morphology deals with the construction of complex

content words from simpler components, e.g., derivation of the word “government” from the verb “to govern” and the derivational morpheme “-ment”. Some have argued that derivational morphology actually belongs within lexical semantics, and should not be treated within the grammar at all. However, such an alignment between derivational morphology and semantics describes a language like English better than it does richly inflected languages like Greenlandic Eskimo, where a whole sentence may consist of one word with many different derivational and inflectional morphemes. Inflectional morphology refers to modulations of word structure that have grammatical consequences, modulations that are achieved by *inflection* (e.g., adding an “-ed” to a verb to form the past tense, as in “walked”) or by *suppletion* (e.g., substituting the irregular past tense “went” for the present tense “go”). Some linguists would also include within inflectional morphology the study of how free-standing function words (like “have”, “by”, or “the”, for example) are added to individual verbs or nouns to build up complex verb or noun phrases, e.g., the process that expands a verb like “run” into “has been running” or the process that expands a noun like “dog” into a noun phrase like “the dog” or prepositional phrase like “by the dog”.

Syntax is defined as the set of principles that govern how words and other morphemes are ordered to form a possible sentence in a given language. For example, the syntax of English contains principles that explain why “John is kissing Mary” is a possible sentence while “John is Mary kissing” sounds quite strange. Note that both these sentences would be acceptable in German, so to some extent these rules and constraints are arbitrary. Syntax may also contain principles that describe the relationship between different forms of the same sentence (e.g., the active sentence “John hit Bill” and the passive form “Bill was hit by John”), and ways to nest one sentence inside another (e.g., “The boy that was hit by John hit Bill”).

Languages vary a great deal in the degree to which they rely on syntax or morphology to express basic propositional meanings. A particularly good example is the cross-linguistic variation we find in means of expressing a propositional relation called *transitivity* (loosely defined as “who did what to whom”). For example, English uses word order as a regular and reliable cue to sentence meaning (e.g., in the sentence “John kissed a girl”, we immediately know that “John” is the actor and “girl” is the receiver of that action). At the same time, English makes relatively little use of inflectional morphology to indicate transitivity or (for that matter) any other important aspect of sentence meaning. For example, there are no markers on “John” or “girl” to tell us who kissed whom, nor are there any clues to transitivity marked on the verb “kissed”. The opposite is true in Hungarian, which has an extremely rich morphological system but a high degree of word order variability. Sentences like “John kissed a girl”

can be expressed in almost every possible order in Hungarian, without loss of meaning, for at least two reasons. First, the Hungarian language provides case suffixes on each noun that unambiguously indicate who did what to whom. In addition, Hungarian puts special markers on the verb that agree with the object in definiteness. Hence the Hungarian translation of our English example would be equivalent to “John-actor indefinite-girl-receiver-of-action kissed-indefinite”.

Some theoretical linguists are interested in developing a theory of **Universal Grammar**, defined as a set of innate constraints on the forms that a grammar can take in any natural language. Proponents of linguistic autonomy (e.g., Bickerton, 1981; Chomsky, 1980, 1988, 1995; Lightfoot, 1989) or “linguistic modularity” (Fodor, 1983; Levelt, 1989) believe that this Universal Grammar will prove to be quite arbitrary in form, based on innate principles that evolved for grammar and nothing else—a kind of “linguistic algebra” (Marcus, 1999; Pinker, 1997, in press). These theorists further argue that human beings have evolved a “mental organ” for grammar, an innate and hard-wired neural system that is unique to our species, in the same way that echo location may be unique to bats (Pinker, 1994; Pinker & Bloom, 1990). Others believe that grammars look the way they do for a reason, and that any universals we might discover across natural languages will ultimately prove to reflect universal meanings and/or universal constraints on information processing. Members of the second school (called “functional grammar” or “cognitive grammar”) believe that grammar is a way of solving communication problems that takes a particularly well-developed form in our species; nevertheless, the mechanisms used for grammar have their roots in more ancient neural and cognitive systems that we share with other species (Bates & MacWhinney, 1989; Bates, Thal, & Marchman, 1991). This controversy has colored a great deal of research in linguistics and psycholinguistics, and has played a major role in modern research on aphasia, as we will see shortly.

The study of language as a communicative system (pragmatics). The various sub-disciplines that we have reviewed so far reflect one or more aspects of linguistic form, i.e., the set of signals that human beings use to convey meaning. *Pragmatics* is defined as the study of language in context, a field within linguistics and philosophy that concentrates instead on language as a form of communication, a tool that we use to accomplish certain social ends (Birner & Ward, 1998; Cole, 1981; Givón, 1989; Prince, 1981). Pragmatics is not a well-defined discipline; indeed, some have called it the wastebasket of linguistic theory. It includes the study of *speech acts* (a taxonomy of the socially recognized acts of communication that we carry out when we declare, command, question, baptize, curse, promise, marry, etc.), *presuppositions* (the background information that is necessary for a given speech act to work,

e.g., the subtext that underlies a pernicious question like “Have you stopped beating your wife?”), and *conversational postulates* (principles governing conversation as a social activity, e.g., the set of signals that regulate turn-taking, and tacit knowledge of whether we have said too much or too little to make a particular point).

Pragmatics also contains the study of *discourse*. This includes the comparative study of discourse types (e.g., how to construct a paragraph, a story, or a joke), and the study of *text cohesion*, i.e., the way we use individual linguistic devices like conjunctions (“and”, “so”), pronouns (“he”, “she”, “that one there”), definite articles (“the” versus “a”) and even whole phrases or clauses (e.g., “The man that I told you about....”) to tie sentences together, differentiate between old and new information, and maintain the identity of individual elements from one part of a story to another (i.e., *coreference relations*). Within a new and growing branch of linguistics called *information structure*, linguists are analyzing the relationship between specific grammatical forms and the discourse functions that they serve. From this point of view, the traditional boundary between grammar and pragmatics (treated as different kinds of content) is giving way to a unified view in which pragmatic factors serve as the motivation for grammatical form (Lambrecht, 1994; Langacker, 1987; Newmeyer, 1998; Tomasello, 1998).

It should be obvious that pragmatics is a heterogeneous domain without firm boundaries. Among other things, mastery of linguistic pragmatics entails a great deal of sociocultural information: information about feelings and internal states, knowledge of how the discourse looks from the listener’s point of view, and the relationships of power and intimacy between speakers that go into calculations of how polite and/or how explicit we need to be in trying to make a conversational point. This is one area where social-emotional disabilities could have a devastating effect on language development and language use (Butterworth, Harris, Leslie, & Wellman, 1991; Cicchetti & Carlson, 1989; Gopnik & Meltzoff, 1997; O’Connell & Bretherton, 1984; Sodian, Taylor, Harris, & Perner, 1991; Tomasello & Call, 1997).

II. ADULT APHASIA

The term “aphasia” refers to the breakdown of language in adults following an acquired insult to the brain. This is one of the oldest fields in cognitive neuroscience (extending back as far as 3000 B.C. in the first Egyptian Surgical Papyrus), and one of the most interesting, because it is clear that language can break down in a variety of different ways depending on the nature and location of the injury. The problem is, however, that there is still no consensus about the nature or location of the mechanisms responsible for different kinds of aphasia. We can distinguish three periods in the history of the field, starting with relatively simple accounts and moving forward to the

grand confusion that characterizes our understanding of aphasia today.

(1) Sensorimotor accounts of aphasia.

When the basic aphasic syndromes were first outlined by Broca, Wernicke and their colleagues, differences among forms of linguistic breakdown were explained along sensorimotor lines, rooted in rudimentary principles of neuroanatomy. For example, the symptoms associated with damage to a region called Broca’s area were referred to collectively as *motor aphasia*: slow and effortful speech, with a reduction in grammatical complexity, despite the apparent preservation of speech comprehension at a clinical level. This definition made sense when we consider the fact that Broca’s area lies near the motor strip. Conversely, the symptoms associated with damage to Wernicke’s area were defined collectively as a *sensory aphasia*: fluent but empty speech, marked by moderate to severe word-finding problems, in patients with serious problems in speech comprehension. This characterization also made good neuroanatomical sense, because Wernicke’s area lies at the interface between auditory cortex and the various association areas that were presumed to mediate or contain word meaning. Isolated problems with repetition were further ascribed to fibers that link Broca’s and Wernicke’s area (resulting, if lesioned, in Conduction aphasia); still other syndromes involving the selective sparing or impairment of reading or writing were proposed, with speculations about the fibers that connect visual cortex with the classical language areas (for an influential and highly critical historical review, see Head, 1926). This sensorimotor characterization of the various aphasias is appealing in its simplicity and its correspondence to known facts about the sensorimotor organization of the brain. Indeed, it is the account that we still find in some medical school textbooks. Unfortunately, it does not account for detailed facts about the sparing and impairment of language in different forms of aphasia, which brings us to the next phase.

(2) *Linguistic accounts of aphasia.* In the period between 1960 and 1980, a revision of the sensorimotor account was proposed (summarized in Kean, 1985). Psychologists and linguists who were strongly influenced by generative grammar sought an account of language breakdown in aphasia that followed the componential analysis of the human language faculty proposed by Chomsky and his colleagues. This effort was fueled by the discovery that Broca’s aphasics do indeed suffer from comprehension deficits: specifically, these patients display problems in the interpretation of sentences when they are forced to rely entirely on grammatical rather than semantic or pragmatic cues (e.g., they successfully interpret a sentence like “The apple was eaten by the girl”, where semantic information is available in the knowledge that girls, but not apples, are capable of eating, but fail on a sentence like “The boy was pushed by the girl”, where either

noun can perform the action). Because those aspects of grammar that appear to be impaired in Broca's aphasia are precisely the same aspects that are impaired in the patients' expressive speech (namely, morphology and complex syntax), the idea was put forth that Broca's aphasia may represent a selective impairment of grammar (in all modalities), in patients who still have spared comprehension and production of lexical and propositional semantics. This led to the proposal that Broca's area may be the neural home of a modular "grammar processor" (Caplan & Hildebrandt, 1988; Zurif & Caramazza, 1976).

From the same point of view, the problems associated with Wernicke's aphasia were reinterpreted to reflect a selective impairment of semantics (resulting in comprehension breakdown and in word-finding deficits in expressive speech), accompanied by a selective sparing of grammar (evidenced by the patients' fluent but empty speech). Hence Wernicke's area could be viewed as a "lexical semantic processor" (Ullman et al., 1997). Similar reinterpretations were made of Conduction aphasia, with researchers noting that (in addition to problems with repetition) these patients display a host of phonological problems that could be explained if we assumed that Conduction aphasia arises from damage to a separate "phonological processor".

Finally, other investigators working within this "linguistic module" approach to brain and language pointed out that right-hemisphere damage seems to lead to specific deficits in understanding the point of a joke, or telling a good story, which could mean that the right hemisphere is the natural home of some kind of "pragmatics processor". For example, Gardner and his colleagues have presented some evidence to suggest that aspects of linguistic pragmatics are selectively impaired in patients with right-hemisphere damage. These patients reportedly demonstrate difficulties in following the point of a joke, telling a coherent story (Gardner, Brownell, Wapner, & Michelow, 1983) and interpreting idioms (VanLancker & Kempler, 1986)—all domains that require the ability to relate a sentence to its verbal or nonverbal context. They also demonstrate problems with some of the paralinguistic skills that are sometimes ascribed to pragmatics (i.e., prosody or tone-of-voice phenomena that convey emotion and distinguish one speech act from another (Ross, 1981; Ross & Mesulam, 1979).

By the mid-1980s, many investigators were convinced that the brain is organized into separate and dissociable modules, one for each major component of language. It was never entirely obvious how or why the brain ought to be organized in just this way (e.g., why Broca's area, the supposed seat of grammar, ought to be located near the motor strip), but the lack of a compelling link between neurology and neurolinguistics was more than compensated for by the apparent isomorphism between aphasic syndromes and the components predicted by linguistic theory. It looked

for a while as if Nature had provided a cunning fit between the components described by linguists and the spatial representation of language in the brain. Unfortunately, evidence against this attractive theory has accumulated in the last 15 years, leaving most aphasiologists in search of a third alternative to both the original modality-based account (i.e., motor vs. sensory aphasia) and to the linguistic account (i.e., grammatical vs. lexical deficits).

The current crisis in aphasiology. To understand why the linguistic approach to aphasia has fallen on hard times, consider the following arguments against the neural separation of words and sentences (for more extensive reviews, see Bates & Wulfeck, 1989; Bates & Goodman, 1997).

(1) Deficits in word finding (called "anomia") are observed in all forms of aphasia, including Broca's aphasia (Goodglass, 1993). This means that there can never be a full-fledged double dissociation between grammar and the lexicon, weakening claims that the two domains are mediated by separate brain systems. In fact, it now looks as though lexical deficits accompany any and all linguistic symptoms, in both children and adults (Bates & Goodman, 1997).

(2) Deficits in expressive grammar are not unique to agrammatic Broca's aphasia, or to any other clinical group. English-speaking Wernicke's aphasics produce relatively few grammatical errors, compared with English-speaking Broca's aphasics. However, this fact turns out to be an artifact of English! Nonfluent Broca's aphasics tend to err by omission (i.e., leaving out grammatical function words and dropping inflections), while Wernicke's err by substitution (producing the wrong inflection). Because English has so little grammatical morphology, it provides few opportunities for errors of substitution, but it does provide opportunities for function word omission. As a result, Broca's seem to have more severe problems in grammar. However, the grammatical problems of fluent aphasia are easy to detect, and very striking, in richly inflected languages like Italian, German or Hungarian. This is not a new discovery; it was pointed out long ago by Arnold Pick, the first investigator to use the term "agrammatism" (Pick, 1913/1973).

(3) Deficits in receptive grammar are even more pervasive, showing up in Broca's aphasia, Wernicke's aphasia, and in many patient groups who show no signs of grammatical impairment in their speech output (Bates, 1991; Dick, Bates, Wulfeck, Utman, & Dronkers, 1999). In fact, it is possible to demonstrate grammatical symptoms very similar to those observed in aphasia in normal college students who are forced to process sentences under various kinds of stress (e.g., perceptual degradation, time-compressed speech, or cognitive overload—Blackwell & Bates, 1995; Dick et al., 1999; Kilborn, 1991). Under such conditions, listeners find it especially difficult to process inflections and grammatical function words, and they also tend to

make errors on complex sentence structures like the passive (e.g., “The girl was pushed by the boy”) or the object relative (e.g., “It was the girl who the boy pushed”). These aspects of grammar turn out to be the weakest links in the chain of language processing, and for that reason, they are the first to suffer when anything goes wrong.

(4) One might argue that Broca’s aphasia is the only “true” form of agrammatism, because these patients show such clear deficits in both expressive and receptive grammar. However, numerous studies have shown that these patients retain knowledge of their grammar, even though they cannot use it efficiently for comprehension or production. For example, Broca’s aphasics perform well above chance when they are asked to detect subtle errors of grammar in someone else’s speech (Devescovi et al., 1997; Linebarger, Schwartz, & Saffran, 1983; Lu et al., 1999; Wulfeck & Bates, 1991). It is hard to understand how a patient who has lost her “grammar organ” would perform so well in detecting detailed and language-specific grammatical mistakes.

In the wake of all this evidence, we must reject the appealing idea that language breaks down along the lines laid out by linguists (and reviewed in Part I). The effects of brain injury appear to cut across traditional linguistic boundaries, and although there are indeed qualitative differences in the symptom patterns associated with particular aphasic syndromes (i.e., fluent Wernicke’s aphasia and nonfluent Broca’s aphasia), a characterization of the mental/neural mechanisms responsible for these patterns still eludes us. Aphasiologists are hard at work on alternative accounts of the different forms of language breakdown that have been observed to date. For example, many investigators are now pursuing the idea that fluent and nonfluent aphasia represent differential disruption of *strategic* or *slow* components of language processing (impaired in fluent aphasia) versus *automatic* or *fast* components (impaired in nonfluent aphasia)—a difference that may cut across phonetic, semantic and grammatic boundaries (Milberg, Blumstein, & Dworetzky, 1988; Zurif, Swinney, & Garrett, 1990). Other investigators still seek an account that honors the structural details of generative grammar, although their proposals are much more subtle and restricted in scope than the original notion of “central agrammatism” (Caplan & Waters, 1999; Grodzinsky, in press). Still others have rejected any attempt at all to map linguistic symptoms onto separate components of the brain (for a critical review, see Shallice, 1988). At the moment, there is absolutely no consensus regarding the nature of the neural mechanisms that are responsible for linguistic symptoms in brain-injured adults.

Given the current disarray in research on adult aphasia, it seems most unwise to apply the same well-worn taxonomies to research on the neural bases of language development in children. If adult language

does not break down along traditional linguistic lines, we should not expect it to build up along those lines either. The linguistic terms that we have introduced here are still useful, but they apparently do not stand in a one-to-one relationship to brain organization. For example, although children babble before they speak, and they produce single words before they produce sentences, such parallels do not go beyond this broad description. The developmental literature reveals lexical effects on phonology and vice-versa in the first stages of word production (Locke, 1983; Menn & Stoel-Gammon, 1995; Vihman, Ferguson, & Elbert, 1986; Vihman & Greenlee, 1987), semantic effects on grammar and vice-versa that begin even before the child is able to produce a single sentence (Bates & Goodman, 1997; Caselli et al., 1995; Caselli, Casadio, & Bates, 1999), and pragmatic effects on the entire process of language learning (Bamberg, 1988; Bates, 1976; Berman & Slobin, 1994; Ochs & Schieffelin, 1979). To account for basic milestones of language learning in neural terms, we need a more dynamic model, based on the skills that a small child needs to break into language during the first few years of life—which brings us at last to normal language development and its cognitive, perceptual and social prerequisites.

III. PRESPEECH AND PREREQUISITES TO LANGUAGE ACROSS THE FIRST YEAR

An overview and quick summary of the many behavioral developments in the first year of life that prepare children for language can be found in Table 1.

Perception and production of the speech signal. Research on the development of speech sounds can be divided into two parts: perception and production. Considerable progress has been made in both these areas in the last thirty years, particularly in the domain of speech perception.

Speech perception. A series of clever techniques has been developed to determine the set of phonetic/phonemic contrasts that are perceived by preverbal infants. These include High-Amplitude Sucking (capitalizing on the fact that infants tend to suck vigorously when they are attending to an interesting or novel stimulus), habituation and dishabituation (relying on the tendency for small infants to “orient” or re-attend when they perceive an interesting change in auditory or visual input), and operant generalization (e.g., training an infant to turn her head to the sounds from one speech category but not another, a technique that permits the investigator to map out the boundaries between categories from the infant’s point of view). For reviews of research using these techniques, see Aslin, Jusczyk, & Pisoni (1998), Eimas, Miller, & Jusczyk (1987), Haith & Benson (1998), Kellman & Banks (1998), and Kuhl (1986).

After many years of experience with these procedures, it now seems clear that human infants are capable of perceiving virtually all of the speech

contrasts used in natural language, at birth and/or within the first few weeks of life. There is even a certain amount of (controversial) evidence suggesting that infants may acquire a preference for the speech sounds of their native language during the last few weeks *in utero* (DeCasper & Fifer, 1980; Jusczyk, Friederici, Wessels, Svenkerud, & Jusczyk, 1993; Mehler et al., 1988). Because the acoustic ability of human infants is so impressive, some investigators have argued that we possess an innate and highly specialized “speech detector”, an ability that is unique to our species (Eimas, 1985). Alas, current research on speech perception in other species suggests that this conclusion was premature (Kuhl, 1986). The capacity to perceive speech contrasts has now been found in several different species, including evidence that chinchillas and other mammals perceive consonant boundaries in a categorial fashion! The mammalian auditory system is a splendid device that is capable of many fine-grained auditory discriminations; these discriminations apparently include speech, whether or not the species in question will need the particular auditory contrasts used by human language.

Of course human infants do a number of things that we are very unlikely to observe in a chinchilla. For example, Kuhl and Meltzoff (1988) have presented 2-3-month-old infants with visual displays of mouths making different speech sounds (e.g., a face making the sound “eeee” on the left screen; the same face making the sound “oooo” on the right screen). When one of these two sounds is played from a speaker located midway between the two visual displays, infants look significantly longer at the display that matches the speech sound, suggesting that they are capable of a very early form of “lip reading”. Not only that, these infants also struggle to reproduce the sound themselves—but only when the sound and visual display are presented together (i.e., they do not make the same mouth movements to either the sound or the visual image presented alone).

Assuming that no one observes a similar result in chinchillas (a most unlikely prospect), should we conclude from this result that human babies have an innate and domain-specific device for mapping speech sounds onto mouths? Although we cannot rule this out at present, this possibility has to be balanced against recent evidence that human infants are extraordinarily fast learners. For example, Saffran, Aslin and Newport (1996) exposed 8-month-old infants to a series of drab and uninteresting speech sounds, presented by a disembodied artificial voice that was played in the room in which the infants were happily playing with toys on the floor. The sounds were made up of strings of meaningless syllables like “BA DI CO RA BI” in which some syllables were presented in random combinations while others were always presented together, as if they represent the ordered components of a word (e.g., “BADICO”). After only two minutes of exposure to

these unattended sounds, the infants were given an opportunity to listen to the same stimuli, or to exactly the same syllables played in different orders (breaking up the statistical structure of the original “word-like sounds”). In this “preferential listening task” (in which infants displayed their preferences by turning to the relevant speaker), the 8-month-olds showed a reliable preference for the “new and unusual” strings. In other words, two minutes of exposure to a boring and repetitive stimulus were sufficient for these babies to induce the statistical regularities in the input, without reinforcement and without paying much attention. When this result first appeared, some investigators suggested that it was only possible because humans have a “special-purpose speech acquisition device.” However, the same result has now been demonstrated with auditory tones, and with visual stimuli that have no speech content of any kind. The bottom line is that human babies are very good at statistical induction, with minimal exposure. Furthermore (as we will point out in more detail below), they also have a very strong interest in social stimuli, coupled with the ability and the drive to reproduce those actions for themselves. Hence it is entirely possible that they have learned the mapping between speech sounds and mouth movements in the first days or weeks of life.

In fact, the major message from the speech perception literature in the last few years has been the extraordinary speed and power of learning. In addition to the finding that some learning has taken place *in utero*, Kuhl and colleagues have shown that infants rapidly acquire a preference for language-specific vowels (called “vowel magnets”), with clear differentiation evident by six months of age among Japanese, English and Swedish infants (Kuhl, 1993; Kuhl, Williams, Lacerda, Stevens, & Lindblom, 1992). Jusczyk and his colleagues have published a comprehensive series of studies showing how and when English children acquire a host of language-specific phonological regularities (called “phonotactics”—Jusczyk, 1997; Jusczyk & Houston, in press; Santelmann & Jusczyk, 1998; Tincoff & Jusczyk, 1999). For example, between 6-10 months of age English-learning infants seem to be tuned into the statistical fact that English words tend to have stress on the first syllable (in contrast with languages like French in which stress is more likely to occur on the final syllable).

In view of all this evidence for language-specific learning, research on infant speech perception has shifted in a new direction, attempting to identify when infants *lose* the ability to hear all the phonetic/phonemic contrasts that are used in languages other than their own. For example, Japanese adults find it very difficult to hear the English contrast between “ra” and “la”; Japanese infants can hear the same contrast perfectly well. When does the child begin to “shut down” or inhibit unnecessary contrasts? Current studies suggest that the selective loss or inhibition of non-

native consonants is already underway by 10-12 months of age (Lalonde & Werker, 1990; Werker & Tees, 1984). This is the point at which children begin to understand meaningful speech (see below), to recognize the intonational contours that are typical of their own language (Hirsh-Pasek et al., 1987), and to produce the particular sounds of their native language in their own prespeech babble (Boysson-Bardies, Sagart, & Durand, 1984). In other words, children begin to inhibit perception of sounds that are *not* in their native language at the same point that they begin to understand and produce the sounds that *are* in their language. Hence, even though children may start out as “citizens of the world” (Kuhl, 1985), ready to learn any human language, they swim upstream against the flood of sound coming toward them from the very beginning, working doggedly, persistently and successfully to carve up all those sounds into the ones that they will need. This is an impressive accomplishment.

Speech production. The literature on speech production is considerably older than the literature on speech perception, perhaps because the study of speech output is less dependent on new technologies. Nevertheless, the pace of research on phonological development has increased markedly in the last few years. We will restrict ourselves here to an overview of the basic milestones summarized in Table 2 (for details see Locke, 1983; Menn, 1985).

In the first two months, the sounds produced by human infants are reflexive in nature, “vegetative sounds” that are tied to specific internal states (e.g., crying). Between 2-6 months, infants begin to produce vowel sounds (i.e., cooing and sound play). So-called canonical or reduplicative babbling starts between 6-8 months in most children: babbling in short segments or in longer strings that are now punctuated by consonants (e.g., “dadada”). Research by Boysson-Bardies et al. (1984) and others suggests that babbling “drifts” toward the particular sound patterns of the child’s native language between 6-10 months; that is, native speakers are able to discriminate at above-chance levels between the babbling produced by Chinese, Arabic, English or French infants. However, we still do not know what features of the infants’ babble lead to this discrimination (i.e., whether it is based on consonants, syllable structure and/or the intonational characteristics of infant speech sounds). In fact, several investigators have argued that there are hard maturational limits on the infant’s ability to control the detailed gestures required for speech production. Hence, even though intonation may “drift” in language-specific directions, the production of consonants seems to be relatively immune to language-specific effects until the second year of life (Eilers et al., 1993; Oller, 1980; Roug, Landberg, & Lundberg, 1989).

Whether or not their consonants match the specifics of their native language, most children begin to produce “word-like sounds” around 10 months of age, used in

relatively consistent ways in particular contexts (e.g., “nana” as a sound made in requests; “bam!” pronounced in games of knocking over toys). From this point on (if not before), infant phonological development is strongly influenced by other aspects of language learning (i.e., grammar and the lexicon). There is considerable variability between infants in the particular speech sounds that they prefer. However, there is clear continuity from prespeech babble to first words in an individual infant’s “favorite sounds” (Vihman, 1985). This finding contradicts a prediction by Jakobson (1968), who believed that prespeech babble and meaningful speech are discontinuous. Phonological development has a strong influence on the first words that children try to produce (i.e., they will avoid the use of words that they cannot pronounce, and collect new words as soon as they develop an appropriate “phonological template” for those words—Schwartz, 1978). Conversely, lexical development has a strong influence on the sounds that a child produces; specifically, the child’s “favorite phonemes” tend to derive from the sounds that are present in his first and favorite words. In fact, children appear to treat these lexical/phonological prototypes like a kind of base camp, exploring the world of sound in various directions without losing sight of home (Leonard, Newhoff, & Mesulam, 1980).

Phonological development interacts with lexical and grammatical development for at least two years beyond this point (Vihman, 1985). For example, children who have difficulty with a particular sound (e.g., the sibilant “-s”) appear to postpone productive use of grammatical inflections that contain that sound (e.g., the plural—Camarata & Gandour, 1985). A rather different lexical/phonological interaction is illustrated by many cases in which the “same” speech sound is produced correctly in one word context but incorrectly in another (e.g., the child may say “guck” for “duck”, but have no trouble pronouncing the “d” in “doll”).

After 3 years of age, when lexical and grammatical development have “settled down”, phonology also becomes more stable and systematic: either the child produces no obvious errors at all, or s/he may persist in the same phonological error (e.g., a difficulty pronouncing “r” and “l”) regardless of lexical context, for many more years. The remainder of lexical development from 3 years to adulthood can generally be summarized as an increase in fluency, including a phenomenon called “coarticulation”, in which those sounds that will be produced later on in an utterance are anticipated by moving the mouth into position on an earlier speech sound (hence the “b” in “bee” is qualitatively different from the “b” in “boat”).

The basic facts of speech production and its development have not changed very much in the last 10-15 years. However, there have been some major changes in the theoretical frameworks used to describe and explain these events. This includes proposals

couched in phonological theories like Autosegmental Phonology and Optimality Theory (Menn & Stoel-Gammon, 1995; Stemberger & Bernhardt, 1999), as well as proposals based on connectionist or “neural network” models of acoustic and articulatory learning (Plaut, 1994). Because this is a relatively “hard” physical domain, permitting a clear and unambiguous test of competing hypotheses, phonological/phonetic development is an area in which it may be possible to make serious progress regarding the interplay of maturation and learning during the first three years of life.

To summarize so far, the development of speech as a sound system begins at or perhaps before birth (in speech perception), and continues into the adult years (e.g., with an increase in fluency and coarticulation). However, there is one point in phonetic and phonological development that can be viewed as a kind of watershed: 8-10 months, marked by phonological drift, the onset of canonical babbling, the first signs that nonnative speech sounds have been inhibited, and the first signs of word comprehension—which brings us to the next domain.

Object recognition and categorization.

To understand the idea that things have names (i.e., *reference*), a human child must be able to recognize and categorize objects and events (Case, 1998; Haith & Benson, 1998; Mareschal & French, 2000; Rogoff, 1998). There has been a real explosion in our knowledge of object recognition and categorization in infancy, using some of the same techniques described in the section on speech perception (high-amplitude sucking, habituation and dishabituation, operant generalization), together with techniques that are unique to the visual modality (i.e., preferential looking, eye movement monitoring—for detailed reviews, see Bertenthal & Clifton, 1998; Gibson & Spelke, 1983; Haith, 1990; Osofsky, 1987). It is now clear that infants under six months of age are much more competent than we previously believed, capable of surprisingly fine-grained discriminations of object boundaries and three-dimensional space. This includes at least some ability to perceive the cross-modal characteristics of objects and events. One example will suffice (Meltzoff & Borton, 1979): very young infants are placed before a two-choice visual array, with a nubbly-textured nonsense form on one side and a display with smooth contours on the other. If a nubbly-textured pacifier is placed in the infant’s mouth, s/he tends to look longer at the nubbly display; if a smooth-textured pacifier is placed in the infant’s mouth, more visual attention is directed to the display with smooth contours. It appears that pioneers like Piaget (1954, 1962) underestimated the extent of the infant’s innate preparation for visual exploration of the physical and social world—innate skills that also help to prepare the child for those cognitive categories that lie at the core of every natural language.

However, it is also clear that the child’s ability to form concepts and categories undergoes marked development from 0-9 months of age. These include changes between 2 and 5 months in the infant’s ability to predict or anticipate changes in a moving display (Haith, 1990) changes between 3 and 9 months in the ability to synthesize a whole pattern out of local details (Bertenthal, Campos, & Haith, 1980; Bertenthal, Proffitt, Spetner, & Thomas, 1985; Spitz, Stiles, & Siegel, 1989), and changes between 6 and 10 months in the ability to recognize objects as members of a category (Cohen & Younger, 1983; Reznick & Kagan, 1983). If we were asked to choose a point at which object categories are clearly established, with sufficient stability and flexibility to serve as the basis for acts of labelling or reference (e.g., “Where’s the doggie?”), we would (again) choose 8-10 months of age as a watershed.

Imitation. The development of speech production presupposes a well-developed capacity to imitate, i.e., the ability to transform an auditory input into a motor output. Piaget’s original stage model of sensorimotor development postulated six stages in the development of imitation: from no imitation at all (Stage 1, 0-2 months), through various levels of “pseudo-imitation” (repetition of adult models that are already present in the child’s own motor repertoire—Stages 2-4, 2-8 months), to the first signs of “true imitation” (reproduction of novel motor patterns—Stage 5, 9-18 months), to a final stage of “deferred imitation” (reproduction of novel motor patterns from memory—Stage 6, beginning somewhere between 12-18 months). Current evidence suggests that Piaget was wrong about at least two of these points. First, human neonates apparently can reproduce a small set of innate motor patterns in response to an adult model (e.g., sticking out the tongue—Meltzoff & Moore, 1979). Hence “pseudo-imitation” is present at Stage 1. Second, deferred imitation has now been demonstrated in children as young as 9 months of age (Meltzoff, 1988). However, Piaget’s observations about the transition from pseudo-imitation to true imitation appear to be correct. Furthermore, the ability to reproduce novel vocal and gestural patterns appears around the now familiar 8-10-month turning point (e.g., the onset of gestures like “bye-bye” and “pattycake”, and the onset of prosodic contours and consonant-vowel babbling sounds that start to approximate patterns present in the child’s linguistic input).

Imitation is an aspect of early cognitive development in which human infants are real “stars”, outperforming any other primate by orders of magnitude. In fact, as Tomasello and Call (1997) note in their book on primate cognition, there is so little evidence for systematic imitation of novel models in other primates that expressions like “monkey see, monkey do” or the verb “to ape” are quite misleading. However, the bare beginnings of a capacity for imi-

tation are visible in the primate line, and recent discoveries in primate neurophysiology have led to some tentative conclusions about the neural basis of imitation (Rizzolatti, Fadiga, Gallese, & Fogassi, 1996). Rizzolatti and his colleagues have uncovered regions of monkey prefrontal cortex in which neurons tend to fire when the animal is planning a particular hand movement, or a movement of the arm in a particular direction. Interestingly, the same neurons also fire when the monkey observes a human being making the same movements! These "mirror neurons" constitute a powerful demonstration of "analysis by synthesis", in which the actions of another animal are analyzed by internally reproducing a version of the same act. Because human infants outperform all other primates in imitation of novel acts, it seems quite likely that this kind of process has undergone powerful selection in our species.

Joint reference and intentionality. Language acquisition is an active process. No child would ever crack the code that maps meaning onto sound without the will and the ability to try those mappings out for herself. First, the child needs a strong motivation to communicate with others, and to be like (sound like) other people. Without this motivation, it is unlikely that any child would ever spend the necessary hours attending, imitating, practicing and contemplating the linguistic input. Second, every human child must understand the means-end relationship between sound and meaning. Symbols constitute a form of tool use: we use them as instruments for social interchange, and, through the use of symbols, other human beings can be moved to act on our behalf. There are important changes in both these aspects of intentionality across the first 8 months of life, developments that prepare the child for entry into a linguistic system.

Social motivation, at least in some form, is innate in our species. However, there are also important changes in the nature and complexity of social intentions across the first year. The newborn infant is responsive to touch, and s/he can differentiate the human voice and face from other auditory and visual stimuli, showing an early preference for these species-specific patterns. Face-to-face interaction begins in the first hours of life, and increases in complexity across the next few weeks. Back-and-forth games of "vocal tennis" are also common by three months. By five months of age, infants have learned to follow the parent's line of visual regard, resulting in a "joint reference" to the same objects and events in the world (Butterworth, 1990; Butterworth & Jarrett, 1991). However, infants do not take an active role in the establishment of joint reference until 8-9 months of age, when they begin to show, give and eventually point to objects as a form of social exchange.

This 8-9-month transition toward the active use of objects in social exchange occurs in two directions: use of objects as a means of obtaining adult attention (e.g.,

giving, showing, pointing), and use of adults as a means of obtaining a desired object (e.g., reaching, eye contact, pointing and request sounds used interchangeably in a deliberate and persistent request sequence). Bates, Camaioni and Volterra (1975) have referred to these two forms of human tool use as "proto-declaratives" and "proto-imperatives". Interestingly, these two forms of "human tool use" coincide with a third, nonsocial form of tool use: the use of one object as a means of obtaining another (e.g., pulling on a cloth support to bring a desired toy within reach). All three forms of tool use (object-to-object, object-to-person, person-to-object) are highly correlated in a sample of 9-month-old children (Bates, Benigni, Bretherton, Camaioni, & Volterra, 1979); and all three forms are also correlated with the subsequent emergence of meaningful speech (see below).

Tomasello and Call have pointed out that joint reference is another arena in which human infants excel, outperforming other primates of any age. It is perhaps for this reason that human infants go on to achieve "secondary reasoning", computing the intentions of other human beings and acting on them. Hence joint reference can be viewed as the basis of what has been called "theory of mind", referring to our ability to reason about the contents of other people's minds (Baron-Cohen et al., 1996; Charman & Baron-Cohen, 1995; Leslie, 1994). This kind of ability is crucial for the computations that are involved in telling a good story, or getting one's point across by taking the other person's point of view into account (e.g., the difference between "He hit me" vs. "That boy I told you about yesterday hit me again!"). Hence joint reference (established in the first year) and secondary reasoning (established a year or so later) can be viewed as critical phylogenetic and ontogenetic inputs into our ability to acquire and use a grammar (Tomasello, 1998; Tomasello & Call, 1997).

Memory: Finally, to achieve mastery of a system that maps meaning onto sound, human children must have the ability to store, recognize, and recall signals in the appropriate context. This will necessarily involve advances in memory, including recognition memory for speech comprehension, recall for speech production, and the working or planning memory required to stage a novel utterance. This is yet another area where infancy researchers have made a great deal of progress (Haith & Benson, 1998; Harris, 1983; Mandler, 1998; Schneider & Bjorklund, 1998). For example, we now know that 2-month-old infants can learn to recognize a sequence of simple events (e.g., moving lights) well enough to anticipate the next move (Haith, Benson, Roberts, & Pennington, 1994). There are also marked shifts in the ability to remember and/or retrieve a hidden object after a short delay between 7 and 10 months of age (Baillargeon & Graber, 1988; Piaget, 1954), as well as the length of time the location of the object can be held in memory (Diamond, 1985). Therefore there is

considerable evidence for some forms of visual recognition memory very early in life (see Rovee-Collier, 1984; Rovee-Collier, Lipsitt, & Hayne, 1998). Some form of recall memory has also been shown in infants as young as 9 months. For example, 9-month-old infants are capable of reproducing a novel action after a 24-hour (Meltzoff, 1988) and a 1-month delay, suggesting that at least one form of recall is also available within the same 8-10-month window (see imitation above). Putting this evidence together, we may conclude that 9-month-old infants have some aspects of memory that are necessary to hold in mind a sound or word while retrieving from memory an object category (word comprehension). They may also have the ability to retrieve and produce a sound from memory in the presence of an associated class of objects or events (word production).

In all of the cognitive and communicative domains that we have just reviewed, important changes take place between 8-10 months of age, providing the child with the basic skills required to initiate language learning. Language acquisition cannot get underway until some threshold level is reached in all these domains, and maturational changes in any of these domains may influence the nature and timing of language acquisition beyond the first stage. In addition, we propose that limitations in one or more of these areas may be responsible for the array of developmental language pathologies described in this volume (see chapters by Nass; van Hout; Rapin, Allen, & Dunn; Evrard—this volume). In the next three sections, we will use this framework to analyze basic milestones in language development, from babbling to the acquisition of complex discourse skills.

IV. MILESTONES OF LANGUAGE DEVELOPMENT

We have reviewed the early stages of normal language development in a number of different places, to make a number of different points: on the role of language within the broader framework of infant development (Bates, O'Connell, & Shore, 1987), on individual differences in style and rate of development across the normal range (Bates et al., 1988), on continuity of individual differences from infancy to the childhood years (Bates, Dale, & Thal, 1995; Thal & Bates, 1989), on the cognitive correlates of language learning (Bates & Snyder, 1987), on relations between linguistic and gestural development (Bates, Thal, Whitesell, Fenson, & Oakes, 1989; Iverson & Thal, 1997; Shore, Bates, Bretherton, Beeghley, & O'Connell 1990; Thal & Bates, 1990; Thal & Tobias, 1992, 1994; Thal, Tobias, & Morrison, 1991), on relations between language development and language evolution (Bates et al, 1979; Bates, Thal, & Marchman, 1991), on similarities and dissimilarities between adult aphasia and the dissociations observed in normal and abnormal language development (Bates & Thal, 1991; Reilly, Bates, & Marchman, 1998), and on norms of language devel-

opment from a practitioner's point of view (Thal & Bates, 1989; Thal & Katich, 1996; Thal, Tobias, & Morrison, 1991). Readers are referred to those sources for details. Here we will restrict ourselves to a brief review of major events in language, in enough detail to support claims about (a) how things come together (basic milestones and their cognitive correlates), and (b) how things come apart (variations and dissociations under normal and abnormal conditions), providing enough information to consider how milestones and variations in language acquisition map onto major events in human brain development.

Word comprehension. The first systematic evidence for word comprehension is generally found between 8-10 months, usually in response to specific, contextually supported sounds (e.g., responding appropriately to "no no", to his/her own name, or to a few routines such as patty-cake or waving bye-bye). However, many children display a rapid spurt in comprehension after this point. Indeed, most parents can no longer keep track of their child's receptive vocabulary beyond 16 months of age, because it has become too large. For example, in a recent study of approximately 1800 children in San Diego, Seattle, and New Haven (Fenson et al., 1994), parents estimated that their children comprehended an average of 67 words at 10 months, 86 words at 12 months, 156 words at 14 months, and 191 words at 16 months.

Word production. True word production typically begins between 11-13 months. As with comprehension, it usually starts with a few contextually supported vocal routines such as producing animal sounds in a ritualized game, or consistently using a specific sound when requesting an object or activity. Those ritualized "words" turn into what looks like true naming of objects by 12-13 months in most children, but the words remain limited in scope, and they are unstable (coming and going from the child's repertoire) until the child has established a repertoire of about 10 consistently produced words. From that point, stable new words are added gradually until the child has a production vocabulary of approximately 50 to 75 words (Goldfield & Reznick, 1990). In the first part of the "one-word stage", single words are used primarily to label and or to ask for objects and people. That is, except for a few idiosyncratic nonnominal terms like "bye-bye" and "up", they are used to communicate referential rather than predicative information.

For many children the 50-75-word point is pivotal in development, as it coincides with what has been called the "vocabulary burst", i.e., a rapid acceleration in the rate at which new words are learned. In our recent tri-city norming study (Fenson et al. 1994), parents reported that their children produced an average of 10 words at 12 months, and 64 words at 16 months. By 24 months, the average production vocabulary has reached 312 words, and by 30 months it is 534 words. In many individual cases it is even larger (see variations, below).

This vocabulary burst is accompanied by changes in vocabulary composition; specifically, there is a *proportional* increase in verbs, adjectives and other words that serve as predicates (i.e., relational terms) in the adult language. For example, verbs typically comprise less than 2% of the first 50 words produced by English-speaking children; by the time cumulative vocabulary reaches 100 words, verb ratios have risen to 10-12%. It is tempting to conclude that there has been a qualitative shift in the way that words are acquired and used, a shift from reference (single-word meaning) to predication (relational meaning). This shift may be implicated in the next major milestone, the onset of word combinations.

Word combinations. Word combinations typically emerge between 18-20 months. However, the correlation between word combinations and vocabulary size tends to be considerably stronger than the correlation between word combinations and age. In fact, most children begin to combine words when their cumulative vocabulary falls between 50-100 words (Bates et al., 1988; Fenson et al., 1993, 1994). Interestingly, this is also the point at which children begin to produce verbs, adjectives and other predicate terms, suggesting that the move into "sentencehood" does depend (at least in part) on the emergence of predicative or relational meanings.

The form and content of first word combinations have been studied in detail in many languages (e.g., Braine, 1976). Although there is considerable variability from one language to another in the forms that children use to communicate relational meanings (see Part III), these studies show that the same basic stock of relational meanings are encoded by 20-month-olds all around the world. Those meanings revolve around existence (e.g., appearance, disappearance and reappearance of interesting objects or events), desires (refusal, denial, requests), basic event relations (agent-action-object, possession, change of state or change of location), and attribution ("hot", "pretty", etc.).

Grammatical development. A rapid "burst" of grammatical development typically occurs between 20-36 months of age, a kind of high-level repetition of the vocabulary burst that occurred earlier in the second year. At this point we see rapid growth in the language-specific means available to encode the stock of meanings which have previously been encoded with single words. At the same time, children also produce language-specific contrasts whose meanings may be quite opaque. For example, take the issue of gender marking in German: what child *or* adult can make sense of the fact that the word for "bottle" in German is feminine, but the word for "little girl" is neuter? And yet before age 3-4, normal children manage to acquire most of their grammar, including many apparently arbitrary and abstract contrasts (e.g., grammatical gender), together with some fairly complex syntactic devices (e.g., passives, relatives). This is why most 3-

year-olds sound like competent speakers of their native language, despite serious limits in vocabulary and continuing limits in speech fluency.

Changes after 3 years of age. Although the basic structures of grammar are laid down before 4 years of age in most languages (see cross-linguistic variation, Part III below), there are still some significant changes in the nature of language use after this point (besides, of course, changes in the content of vocabulary that continue across the life span).

First, there are changes in the extent to which children use language for *discourse cohesion* purposes. Indeed, Karmiloff-Smith (1979) has suggested that there is a complete reorganization of language between 4-6 years of age, from "intrasentential grammar" (grammar that is used to express simple single-sentence meanings) to "intersentential grammar" (use of the same basic grammatical contrasts to express the relationship between sentences). Returning to the terminology we introduced earlier, this means that children are learning to use grammar for discourse and/or text cohesion purposes. For example, the child learns that use of a pronoun "he" requires prior establishment of the referent (i.e., the entity to which "he" refers) in the information shared by speaker and listener. It should be obvious why this is not a purely linguistic skill: it requires considerable knowledge of the listener, a "theory of mind" that takes many years to construct. It is also likely that this move from sentence-level grammar to discourse-level grammar is encouraged by entry into the school system, where children receive much more experience with connected discourse, in oral and written forms.

Second, there are changes in the *accessibility* of forms that have been there for a considerable period of time. This point is perhaps best illustrated by a recent study in our laboratories examining changes in the probability and nature of grammatical passives in children between 3 and 18 years of age (Marchman, Bates, Burkhardt, & Good, 1991). We set up a situation that could be viewed as the "ecological niche" for passive forms: presented with a series of short cartoons in which one animal acts on another (e.g., a horse bites a goat), children were asked to describe the scene from the point of view of the receiver-of-the-action (e.g., "Tell me about the goat" or "What happened to the goat?"). In this situation, adults produce a passive form more than 80% of the time (e.g., "The goat was bitten by the horse"). In the same situation, most children are capable of producing at least one passive by 3 years of age. However, it is also clear that small children prefer not to produce the passive at all, using a range of alternative forms that avoid the difficult passive form but accomplish the "discourse goal" of focusing on the object (e.g., "The goat was sitting there and then the horse bit him"). In other words, even though almost all the children in the study possess **knowledge** of how to produce at least one

passive (the top line in Figure 1), there is consistent growth in the **accessibility** of passives in real-time language use (the bottom line in Figure 1). There are also gradual changes in the nature of the passive forms that children prefer to produce (e.g., a gradual shift from "get" passives, as in "The goat got bitten" to "be" passives, as in "The goat was bitten", even though both forms co-exist within individual children for many years). For obvious reasons, it would be inappropriate to choose any single point in development from 3-18 years as "the moment" at which adult-like use of the passive is acquired. Many of the other late-onset language functions that we have investigated in our laboratories show a similar linear shift from age 3-4 to adolescence. For these reasons, we are persuaded that grammatical performance changes gradually over time from 3 years to adulthood. There is very little evidence for sudden or discontinuous change in linguistic abilities after 4 years of age. We will return to this point later, when we discuss aspects of brain development that parallel major milestones in the development of language.

Are these milestones specific to language? Probably not, at least not the early ones. At each of the early stages noted above, researchers have found correlates in nonlinguistic cognition. These include intentional communication and means-end understanding between 9 and 11 months (Bates et al., 1979), production of single words and "recognitory gestures" or "enactive names" around 12 to 13 months (Bates et al., 1979; Escalona, 1973; Werner & Kaplan, 1963), and a correlation between word combinations and gesture combinations around 20 months of age (Brownell, 1988; Fenson & Ramsay, 1981; McCune-Nicolich, 1981; McCune-Nicolich & Bruskin, 1982; O'Connell & Gerard, 1985; Shore, 1986; Shore, O'Connell, & Bates, 1984—See Table 2). There is also evidence for a link between later grammatical development (around 28 months of age) and the ability to reproduce arbitrary sequences of 5 gestures (Bauer, Hertsgaard, Dropik, & Daly, 1998). Interestingly, this grammar/gesture link is not observed if only 3 gestures are used, or if the sequence that binds these gestures is causal and meaningful. Hence the later link seems to have something to do with memory demands that language shares with nonlinguistic systems. All of these language/cognition correlations are interesting, because they suggest that language development is paced by mechanisms outside of language proper. However, our interpretation of these language/cognition correlations must be tempered by the variations documented below.

V. VARIATIONS & DISSOCIATIONS IN LANGUAGE DEVELOPMENT

Variations

We can divide our discussion of variations in normal language development into three parts: cross-linguistic variations, variations in rate of development within a single language (in this case, English), and a

new and puzzling literature on variations in "style" of language learning.

Cross-linguistic variations

There are marked variations from one language to another in the nature and timing of all language milestones. For example, contrary to an influential "universalist" theory of phonological development proposed years ago by Roman Jakobson (1968), recent cross-linguistic studies of phonological development by Ferguson, Vihman and their colleagues (Vihman, 1985; Vihman et al., 1986; Vihman & Greenlee, 1987) have shown that the content and nature of babbling varies a great deal from language to language. Although there are some consistent tendencies (e.g., fricatives tend to develop late in every language), there is little evidence for the kind of lawful unfolding of phonological contrasts that Jakobson envisioned. Jakobson also suggested that there is a "silent period" between babble and speech, and discontinuity in the forms that children use in babble and speech. Current evidence suggests instead that there is a great deal of continuity in the forms that children use in their prespeech babble and their first words. Because these "favorite sounds" vary from one language to another, this suggests that cross-language differences in the content of phonology begin by 8-10 months of age! Furthermore, rare forms like clicks in Bantu, vowel harmony in Turkish or Hungarian, or tones in Chinese come in surprisingly early. Hence cross-linguistic differences in "markedness" (a continuum from common to rare forms) fail to predict the order in which children acquire the speech contrasts in their language.

With regard to cross-language variation at the one-word stage, it is fair to say that acquisition starts with something like a one-word stage in every language. But there are variations in the form of this "one-at-a-time" phase of development. For example, one-year-old infants may start out by producing little pieces of a complex word in languages like Greenlandic Eskimo (where many whole sentences consist of a single word plus many inflections). And in languages with a very rich and salient morphological system (e.g., Turkish), children sometimes begin to produce verb or noun inflections late in the one-word stage, i.e., before they have produced any word combinations (Slobin, 1985-1997).

These variations are now uncontroversial, but there are others that have been hotly debated in the last few years. For example, Gentner (1982) wrote a very influential paper arguing that nouns must always precede verbs in early development, in every language in the world, for several reasons: because verbs convey more complex and evanescent concepts (compared with the solid and bounded simplicity of the objects conveyed by common nouns), and because verbs tend to carve up reality in much more variable ways from language to language. In contrast, Choi and Gopnik (1995) and Tardif (1996) presented evidence from Korean and

Chinese, respectively, suggesting that nouns do not always appear before verbs. They argued that verbs are acquired earlier in these languages because they are more salient (e.g., Korean is an SOV language where verbs appear at the end, in an easily remembered position, and Korean and Chinese are both languages that permit extensive omission of nouns, so that a sentence is often composed of a single naked verb!). Indeed, Gopnik and Choi even went so far as to suggest that these language differences feed back on nonlinguistic cognition, and as a result, Korean children perform better on means-end tasks (which are more verb related) while English children perform better on object permanence tasks (which are more noun related). This was an exciting idea, and it set off a lively round of research. However, in the end the issue has come down to one of methodology. When researchers use diary or parental report methods that provide a comprehensive estimate of all the words in the child's vocabulary, then the familiar pattern of "nouns before verbs" seems to be the rule in every language, including Korean (Pae, 1993; for a review, see Caselli et al., 1995). Furthermore, when one-year-old Korean and American children were exposed to novel nonsense nouns and verbs in the same experimental task, nouns were learned more easily than verbs in both groups (Au, Dapretto, & Song, 1994). Hence, when we use methods that tap into what the child knows, the noun-verb sequence seems to be a cross-linguistic universal. However, when we look at free-speech records that tell us what forms children prefer to use (statistically speaking), cross-linguistic differences in the proportional use of nouns and verbs may be observed.

The idea that language-specific variations can change the way we think (including nonverbal cognition) is an old one in linguistics, proposed in its strongest form by the 20th-century linguist Benjamin Whorf. According to the "Whorf hypothesis", languages cut up reality in a variety of ways, and native speakers of those languages will tend to view reality in ways that are predictable from their language. So, for example, in languages that have noun classifiers marking the shape of objects, children are (or so it was proposed) more sensitive to variations in shape at a very young age. Evidence in favor of this interesting hypothesis has been largely disappointing. However, there has been a recent surge of interest in a modified version of the Whorf hypothesis, in which language does not change basic perceptual and cognitive processes, but it can draw our attention to aspects of reality that we might not have noticed without it. As a case in point, Choi and Bowerman (1991) have built on another difference between English and Korean: both languages have prepositions to contrast "in" and "out", but Korean also makes a contrast between "in-close-fitting" and "in-loose-fit". Young Korean children seem to pick this up quite easily. Furthermore, McDonough, Choi, Bowerman and Mandler find that English- and Korean-

speaking children begin to be responsive to terms for containment and support by about 18 months, and respond appropriately to the distinctions their own language makes (McDonough, Choi, Bowerman, & Mandler, in press). If more evidence of this kind can be found, it will provide strong support for a strongly interactive theory of the development of language and cognition in the first years of life.

The most compelling evidence for cross-language variation begins after 20 months of age, when grammatical development is well underway. Here we see so much variation as a function of linguistic input that it is difficult to maintain the belief in one "universal stage" of grammatical learning (Bates & Marchman, 1988). For example, the whole system of case morphology appears to be completely mastered in Turkish by 2 years of age; this early mastery reflects the fact that Turkish morphology is exceptionally regular (i.e., very few exceptions) and phonologically salient (with clear stress-bearing inflections occurring at the end of every noun). By contrast, Russian and Serbo-Croatian children take much longer to learn their "messy" case system (i.e., systems which involve a large number of irregular forms and several arbitrary contrasts, including gender). There are also large variations from one language to another in the acquisition of word order: from very early display of word order regularities in a "rigid" language like English, to a near-absence of word order regularity in a flexible language like Turkish. Finally, many so-called complex forms appear quite early in a few languages, if they are very frequent and used for common pragmatic purposes (e.g., relative clauses in Italian, which are five times as common in Italian 3-year-olds than they are in their English counterparts—Bates & Devescovi, 1989; passives in Sesotho, used very frequently by adults and acquired before 3 years of age by Sesotho children—Demuth, 1989).

There is a sense in which this had to be true. For example, adult native speakers of Italian have to produce approximately three times more morphological contrasts than English speakers to convey the same idea. Consider, for example, the sentence "Wolves eat sheep", which contains three words (wolf, eat, sheep) and four morphemes (where wolves = wolf + plural marker). In Italian, the corresponding sentence would be "I lupi mangiano le pecore", in which articles are obligatory, both the article and the noun are marked for number and gender, and the verb is obligatorily marked for third person plural. For all these reasons, the Italian sentence contains five words, and fourteen morphological contrasts! This leads to two logical possibilities for cross-linguistic differences in grammatical development: (1) Italian children will take three times as long to acquire their language, or (2) Italian and English children acquire their language at the same rate, but at any given point in development, Italians will produce roughly three times as much morphology as their English

counterparts. Evidence to date suggests that the latter hypothesis is correct.

Individual differences within English: Variations in rate

Even if we restrict ourselves to the acquisition of English, we find marked variations in rate of development among normal children throughout the first stages of language learning, as follows.

Babbling. Individual children vary markedly in the content (i.e., preferred sounds) and onset of pre-speech babble (Cruttenden, 1970; Ferguson, 1984; Kent & Miolo, 1995; Locke, 1983, 1988; Vihman et al., 1986; Vihman & Greenlee, 1987). Normal children typically begin to produce canonical babble somewhere between 6 and 10 months—a substantial range of variation, considering how many changes occur in the first year of life. There is also variation in the course of phonological development after this point. Some children stick with a very small set of phonetic contrasts for a number of weeks or months while others attempt a much larger array from the very beginning.

Word comprehension. Although systematic evidence of word comprehension is usually available by 8-10 months, there is enormous variability after this point in the rate at which receptive vocabulary grows (Bates et al., 1988; Benedict, 1979; Reznick, 1990). For example, in the tri-city norming study for the MacArthur Communicative Development Inventories (Fenson et al., 1994), parents reported a mean comprehension vocabulary of 67 words at 10 months of age; but the standard deviation was very large (60.19), with a range from 2 to 280 words. At 12, 14 and 16 months, scores varied similarly: a mean of 86 at 12 months (SD 49.23, range 7 to 242), a mean of 156 at 14 months (SD 77.95, range 11 to 343), and a mean of 191 at 16 months (SD 87.58, range 40 to 396). Of course one can always question whether the outer extremes are valid in a study based on parental report (especially for comprehension, which may be more difficult for some parents to assess—Tomasello & Mervis, 1994) but there are good reasons to believe (based on laboratory validations of the CDI—Bates & Goodman, 1997; Jahn-Samilo, Goodman, Bates, Appelbaum, & Sweet, 1999; Reznick, 1990; Ring, 1999; Thal, O'Hanlon, Clemmons, & Frailin, 1999) that the means and standard deviations are a faithful reflection of reality.

Word production. There is also large variation in the age of onset and course of development of expressive vocabulary. In our tri-city study, parents reported vocabulary onset in individual children from as early as 8 months. As with comprehension, production vocabularies across individual children varied widely. Some examples follow: at 12 months mean production vocabulary was reported as 10 (SD 11.20, range 0 to 52), at 16 months mean production was 64 words (SD 70.27, range 0 to 347), at 24 months the mean number of words produced was 312 (SD 173.67, range 7 to 668) and at 30 months the mean was 534 (SD 116.65, range

208 to 675). The impressive array of variation that can be observed from 8 to 30 months is illustrated in Figure 2 (redrawn from Fenson et al., 1993, using percentiles rather than standard deviations).

We reported above that many children show a vocabulary burst at the point where cumulative vocabulary falls between 50 and 75 words. However, in a longitudinal study of language development in the second year, Goldfield and Reznick (1990) were the first to report that there are also individual differences in the shape of vocabulary change over time. Some children did show the typical "burst", but others showed a more even rate of change at every point. Yet another group appeared to develop in a series of small bursts, each followed by a small plateau. In a recent longitudinal study by Goodman and colleagues (Goodman et al., 1999; Goodman & Bauman, 1995), 28 children were followed monthly from 8-30 months of age; parents filled out the CDI monthly, and children came into the laboratory to participate in language assessments monthly from 12-30 months. There were strong positive correlations between laboratory and CDI assessments of language growth, not only in vocabulary size but also in the shape of the growth curves that individual children display across this period of development. We may conclude with confidence that the variation in rate of vocabulary growth illustrated in Figure 2 is real, and can be observed in both cross-sectional and longitudinal designs, in both parent report and laboratory observations. The fact that this much variation is observed within the normal range provides an important lesson for clinicians: risk for language disorders must be evaluated against the full range of variation that we can expect to see in perfectly healthy, normal children.

Word combinations. Although 20 months is reported as the mean age for production of novel word combinations, the range of normal variation around this milestone is wide. Novel combinations have been reported as early as 14 months of age (Bates et al., 1988). At the same time, many normal children do not produce combinations of any kind before 24 months. There is also a certain amount of variation in the relationship between word combinations and vocabulary size: approximately 20% of the sample in our tri-city norming study were reported to produce at least a few word combinations with vocabularies under 50 words; and another 15% with vocabularies between 100-300 words were still not producing any word combinations at all. Hence, although the relationship between lexical development and word combinations is very strong (see "dissociations" below), the appearance of first word combinations is not locked to a single vocabulary size.

Early grammar. A widely used index of early grammatical development in English is Mean Length of Utterance in morphemes (MLU). This is a count that includes content words, function words, and inflections like the plural "-s" or the past tense "-ed". Brown

(1973) used MLU to break development down into epochs: MLU ranges from 1.05-1.50 in Early Stage I (single words to first combinations), from 1.5-2.0 in Late Stage I (first inflections), from 2.0-2.5 in Stage II (productive control over grammar begins), 2.5-3.0 in Stage III (grammatical development well underway), from 3.0-3.5 in Stage IV (complex sentences begin), and so on. Chapman (1981) has provided norms for middle-class children using Brown's stages, and they are expressed as ranges: 19.1-23 months is the average for Early Stage I, 23.8-26.9 months for Late Stage I, 27.7-30.8 months for Stage II, 31.6-34.8 months for Stage III, and 35.6-38.7 months for Stage IV. However, in videotaped laboratory sessions, Bates et al. (1988) found cases across the full range from Stages I to IV in a sample of 27 healthy middle-class children at 28 months of age.

In the tri-city norming study for the MacArthur Communicative Development Inventories, parents were asked to provide three written examples of the longest sentences they had heard their child say recently (Fenson et al., 1994). A mean was computed from these three utterances, providing a measure that we have called maximum sentence length (MSL). Although this is an upper-limit measure (rather than a mean length) and it is calculated from three examples (rather than the typical 50-100 used for MLU calculations (Miller, 1981), MSL correlated highly and significantly ($r = .77$ and $.74$, $p < .01$, for 20- and 24-month-old children, respectively) with MLU (Fenson et al.). Like MLU, we also found wide variation across individual children. Examples at selected ages follow: at 16 months mean MSL was 1.48 (SD .88, range 1-4.7), at 20 months mean MSL was 2.78 (SD 1.59, range 1-7.7), at 24 months mean MSL was 4.69 (SD 2.66, range 1-12.3), and at 30 months mean MSL was 8.18 (SD 3.45, range 3-19). It is fair to conclude, then, that the variation in grammatical development is substantial.

Individual differences within English: Variations in style

In addition to these well-documented variations in rate, there is also a sizeable literature on variations in "style" of language learning (Bates et al., 1988; Bloom, Lightbown, & Hood, 1975; Dore, 1974; Horgan, 1979, 1981; Nelson, 1973; Peters, 1977, 1983) which has shown an interesting dissociation within language. Briefly summarized, children may be described as using "analytic" versus "holistic" learning styles. So-called "analytic children" enter into language development by breaking the input down into small units, and struggling to understand those units before attempting a synthesis. This pattern shows up at every level of development: from babbling (where short and consistent consonant-vowel segments predominate), to first words (where the child concentrates on object naming), to first word combinations (telegraphic speech with function words and inflections eliminated). In contrast, so-called "holistic children" seem to enter into language de-

velopment from the opposite extreme: they start by using relatively large, global chunks of speech in familiar contexts, giving their speech a more adult-like sound while they gradually break speech units down into their component parts. This style can also be found at every level of language development: from babbling (where sporadic consonants are nested within long streams of sentence-like intonation), to first words (with heterogeneous vocabularies that often include formulaic expressions like "wannit"), to first word combinations (where inflections, pronouns, and other function words may be present from the beginning, in frozen expressions and/or in formulae with limited productivity).

Because these variations cut across age levels and content domains (from babbling through grammar), most investigators agree that individual differences in "linguistic style" reflect the differential operation of two fairly general learning mechanisms: an analytic mechanism that serves to break linguistic input down into smaller segments, and a holistic mechanism that makes it possible for the child to remember and reproduce relatively large segments of speech before those segments have been fully analyzed or understood (Bates et al., 1988; Nelson, 1973; Peters, 1977; Thal, Bates, Zappia, & Oroz, 1996). Both mechanisms are necessary for normal language learning to take place, but children may differ in the degree to which they rely on one or the other. The causes of such a differential preference are still unknown, and there is considerable controversy concerning the relative contribution of environmental factors (e.g., maternal style), child temperament (e.g., reflective vs. impulsive approaches to solving a problem), and/or individual differences in the rate at which the different neural mechanisms responsible for language start to mature (see below). In fact, these explanations are not mutually exclusive: any and all of them may serve to encourage differential reliance on analytic/segmenting vs. holistic/suprasegmental mechanisms in language learning. Later we will look at recent evidence from children with focal brain injuries, suggesting that the differential contribution of left- vs. right-hemisphere processes may play some role in the determination of stylistic variation.

Having described milestones and variations in the way that language "comes together" in normally developing children, we can now turn to the ways that language can "come apart". We will start by looking at dissociations within the normal range, and then present a summary of variations and dissociations in children with congenital injury to the left or right side of the brain. Taken together, these associations and dissociations provide clues to the component parts (linguistic or nonlinguistic) that constitute our faculty for language learning, with implications for how and where these various aspects of language are acquired and mediated in the brain.

Dissociations within the normal range

Comprehension and production. One of the most striking disparities in the second year is that found between comprehension and production. In the lexical domain, comprehension almost always exceeds production; furthermore, comprehension appears to mark an *upper limit* on the number of words a child can produce. For example, children with receptive vocabularies under 50 words rarely produce more than 10 words, those with receptive vocabularies under 100 words usually have production vocabularies in the 0- to 50-word range. Very large expressive vocabularies are rarely seen in children with reported comprehension vocabularies under 150 words. However, comprehension clearly does not set a *lower limit* on word production: at every point across the comprehension range from 0 to 200 words, we have found at least a few children who produce little or no meaningful speech.

As described by Thal and her colleagues (Thal & Bates, 1989; Thal, Tobias, & Morrison, 1991), these dissociations are particularly marked in a population of "late talkers", i.e., children between 18 and 24 months of age who are in the bottom tenth percentile for expressive vocabulary. Some of these children are equally delayed in comprehension and production, but others appear to be normal for their age in receptive language despite their expressive delays (as established by laboratory testing as well as parental report). In a follow-up study of the same children (Thal, Marchman, et al., 1991), spared comprehension discriminated between those children who ultimately caught up with their peers in expressive language (i.e., so-called "late bloomers"), while the children with both receptive and expressive delays between 18 and 24 months fell even farther behind their age mates, qualifying for a clinical diagnosis of specific language impairment. Similar results were found for children classified at a younger age. Specifically, Thal (1999) used the MacArthur Communicative Development inventory (CDI) to identify children with delays in production but normal comprehension, and those with delays in both comprehension and production, at 16 months of age. At 28 months, vocabulary production and grammatical complexity scores on the CDI were well within the normal range for the late producers who had normal comprehension at 16 months. Scores for the children with delays in both comprehension and production, on the other hand, were in or close to the delayed range on both measures of expressive language. These results could lead one to conclude that a dissociation between comprehension and production is associated with positive outcomes. However, such a conclusion requires qualification. In a study focused on the effects of intervention on language learning in language-delayed toddlers, Olswang and Bain (1996) examined children who were delayed in both comprehension and production who had varying degrees of difference between comprehension and production. In that study, the children

with greater comprehension/production gaps were least likely to make the transition from single- to multiword speech during the period of intervention. Thus, a dissociation between comprehension and production, with production lower than comprehension, is associated with a positive outcome when comprehension is in the normal range, and with a more negative outcome when comprehension is delayed.

Comprehension/production disparities are also observed at the other end of the developmental spectrum, i.e., in children who are "early talkers" (in the top tenth percentile for expressive vocabulary between 12-21 months of age). Many of these children are equally advanced in comprehension and production, but we also find children who meet our "early talker" criterion despite receptive vocabularies in the normal range (i.e., within one standard deviation of the mean). At first glance, this seems like a startling finding: how can a child move into the front ranks in word production without achieving comparable status in word comprehension? The reason is that most individuals (children and adults) typically produce only a small proportion of their receptive vocabulary (think of the words that you produce in everyday conversation, compared with all the words that you recognize when filling out a crossword puzzle). The early talkers who display a Production > Comprehension profile apparently do so by producing an abnormally large *proportion* of their receptive vocabularies; in essence, they are trying to tell us everything they know! These cases prove that comprehension and production can be dissociated in either direction, a double dissociation which most neuropsychologists would accept as evidence for the existence of two distinct mental/neural mechanisms (e.g., Shallice, 1988).

Analytic/holistic style. The comprehension/production dissociation is also related (albeit indirectly) to the analytic/holistic style distinction described earlier. Within the second year of life, children at the analytic extreme tend to be high comprehenders, and more precocious overall; children at the holistic end tend to be less advanced in comprehension, and slower to develop overall. However, our recent work with early talkers suggests that this association is not necessary. This can be seen best in a case study of two children who were extraordinarily precocious in early expressive language. SW was 21 months old and had an expressive vocabulary of 627 words; MW was 17 months old and had an expressive vocabulary of 596 words. Both children also produced a wide array of verbs and adjectives as well as nouns, a development that typically signals the onset of grammar. And both had begun to master the rudiments of English grammatical morphology, producing contrasting endings on at least a few nouns and verbs (e.g., walk" vs "walking"). The one clear difference between these two exceptional children revolves around sentence length: MW had a Mean Length of Utterance (MLU) in

morphemes of 2.39, equivalent to a 30-month-old child; SW had just begun to combine words, with an MLU of 1.19, within the range of what we would expect for a child of her age. Because these children are not measurably different in their mastery of noun and verb endings, we would not want to conclude that they demonstrate a dissociation between vocabulary and grammar. Nor would we want to conclude that MW is advanced in syntax, since her long sentences contain very little evidence for transformation, extraction, inversion, or any of the operations that define and characterize the syntactic component of grammar. We suggest, instead, that SW and MW vary markedly in the size of the unit that they are able to store and produce at any given time. This interpretation is supported by the fact that MW had a repertoire of idioms like "No way, José!" and "You little monkey!". Her ability to manipulate, and even blend, these large units is illustrated by the expression "No way, you monkey!", which she produced for the first time in our lab. In other words, we suggest that SW and MW represent an analytic/holistic dissociation in two children who are both quite advanced, but without differences in levels of comprehension and/or expressive vocabulary.

Grammar vs. vocabulary. Inspired by claims in the adult neurolinguistic literature, we have also looked carefully for a third type of dissociation, between grammar and semantics (with special reference to vocabulary development). To our surprise, the relationship between grammar and vocabulary development has turned out to be one of the strongest associations that has ever been observed in any aspect of human development (with the possible exception of height and weight!). Figure 3 (from Bates & Goodman, 1997) illustrates the powerful nonlinear developmental function that governs the relationship between grammatical complexity and vocabulary size. These data are taken from the MacArthur parent report forms, but several studies in our laboratory have shown the same strong relationship in laboratory measures as well. Notice that the relationship holds at every point from 50-600 words (covering the period from 16-30 months of age). One certainly might have expected a relationship at the lower end of the distribution, simply because one cannot combine words until there is something to combine. We might also have expected some kind of "trigger" or "threshold" relationship between vocabulary and grammar, e.g., a critical number of words that need to be acquired for grammar to get off the ground. What we find instead is a continuous and accelerating function that holds at every point across this period of development. To be sure, there is some variation around this curve, but we do not find extreme dissociations of the sort that clearly are observed for comprehension and production (e.g., children who understand more than 200 words but produce virtually nothing). This powerful relationship holds for very late talkers, and very early talkers as well.

In short, we have found very little evidence to support the idea that grammar and lexical semantics can "come apart" in the early years of language learning (Bates et al., 1988). There is some evidence for a *temporary* dissociation between vocabulary size and the onset of word combinations in a small number of children (e.g., the cases of SW and MW, described above), but as we have just noted, these observations may be a by-product of analytic vs. holistic style (i.e., the ability to extract, store and reproduce relatively short units, vs. the ability to record and reproduce long but underanalyzed phrases). For the moment, we conclude that the comprehension/production and analytic/holistic dissociations observed in our work to date represent the most robust and natural "fault lines" in the human language processor. By contrast, dissociations between grammar and vocabulary are not observed in healthy, normal children, suggesting that these two aspects of language are governed and acquired by the same mental/neural mechanisms. This brings us to our final consideration in this section: variations and dissociations among linguistic milestones in children with congenital brain injury.

Variations and dissociations in infants with focal brain injury

The neural bases of the dissociations we have just described are still unknown, but our research on early language development in infants with focal brain injury has provided a few clues, permitting us to reject some of the more obvious hypotheses suggested by the adult aphasia literature.

All of our studies to date have focused on infants with unilateral injuries sustained before six months of age. There has been a great deal of controversy about this population since the 1930s. Early studies suggested that early unilateral injuries have no effect at all on long-term language outcomes, and that the two hemispheres of the brain are equipotential for language at the beginning of life (Basser, 1962; Lenneberg, 1967). Later studies (in the 1970s and 1980s) reported subtle deficits in language in left-hemisphere-damaged children (e.g., Aram, 1988; Dennis & Whitaker, 1976; Riva & Cazzaniga, 1986; Riva, Cazzaniga, Pantaleoni, Milani, & Fedrizzi, 1986), and concluded that the left hemisphere is innately and irreversibly specialized for language. However, it is important to point out that none of these later studies actually conducted a direct statistical comparison of children with left- vs. right-hemisphere injury (for a critical review, see Bishop, 1997), nor did they provide evidence for anything like a true childhood aphasia following early unilateral injury.

More recent reviews of the literature suggest a compromise between equipotentiality and irreversible determinism (Bates, Vicari, & Trauner, 1999; Eisele & Aram, 1995; Stiles, Bates, Thal, Trauner, & Reilly, 1998; Vargha-Khadem et al., 1994; Vargha-Khadem et al., in press). First, it is now widely agreed that early unilateral injury does not lead to clinically significant

language disorders in the vast majority of cases, if children with extraneous complications are excluded from the sample. To be sure, children with a history of brain injury tend to perform below neurologically intact age-matched controls on a host of linguistic and nonlinguistic measures, but their performance is usually in the normal to low-normal range, corresponding to an average drop in verbal and nonverbal IQ of about 5-7 points. Second, when left- and right-hemisphere-damaged children are compared directly (with sample sizes large enough and sufficiently well matched to permit a statistical test), and measured after 5-6 years of age (when language acquisition is virtually complete), there is no evidence to date for a difference in long-term language outcomes as a function of lesion side (left vs. right), lesion site (e.g., anterior vs. posterior) or even lesion size (large vs. small).

In contrast with the results of retrospective studies, prospective studies of development prior to 4-5 years of age in this population demonstrate moderate to severe delays in all the early language milestones. These include delays in the onset of babbling and preverbal communication (Marchman, Miller, & Bates, 1991), and delays between 1 and 5 years of age in lexical development and grammar (Bates et al., 1997; Reilly et al., 1998; Thal, Marchman, et al., 1991; Vicari et al., in press). Most important for our purposes here, we find more dissociations than we would expect by chance in this period of development, and we find correlations between these dissociations and specific lesion sites.

Comprehension vs. production. Based on the adult aphasia literature, we might expect a profile of delayed production with normal comprehension to occur more often in children with left anterior involvement (by analogy to adult Broca's aphasia). Conversely (by analogy to adult Wernicke's aphasia), we might expect children with left posterior damage to display a profile in which comprehension vocabularies fall below the levels that are normally observed in children at the same level of production. This issue has been investigated by Thal, Marchman, et al. (1991) and more recently by Bates et al. (1997). Results of both studies were quite surprising: it seems that the development of word comprehension is *not* selectively affected by lesions to left posterior cortex. Instead, the Wernicke-like profile was actually more common in children with right-hemisphere damage, a finding that has no obvious parallel in the adult aphasia literature. However, this finding is compatible with electrophysiological studies of normally developing children (Mills, Coffey, & Neville, 1993; Mills, Coffey-Corina, & Neville, 1997), which show that the difference in the brain's response to familiar vs. unfamiliar words is bilateral (but somewhat larger on the right) prior to approximately 18 months of age. After that point (and strongly correlated with the "vocabulary burst"), there seems to be a reorganization in the brain's response to familiar words, with a larger difference between familiar and unfamiliar words

observed in the left hemisphere, primarily across frontal and temporal sites. To explain these findings, Bates et al. (1997) suggest that the right hemisphere plays a larger role in the first stages of word comprehension because that hemisphere appears to be particularly important for integration of information across multiple sources (Stiles et al., 1988). For adults who already know their language (and also for older infants), this kind of multimodal integration may not be necessary in order to understand a familiar word. But for infants who are struggling to "crack the code", right-hemisphere resources may play a particularly important role.

Analytic vs. holistic style. As we noted earlier (see variations in style), children who are acquiring English tend to deal initially with pronouns and other function words in one of two ways: analytic/referential-style children tend to leave those forms out of their speech altogether; holistic/pro-nominal-style children tend to produce those forms from the very beginning, but only in rote, "frozen" expressions. Applying these definitions to our focal lesion sample, Thal et al. reported a significantly higher incidence of holistic/pronominal style across the sample as a whole than we would expect if the sample were drawn randomly from the normal population (based on norms from Fenson et al., 1993). This finding is reminiscent of a report by Johnston and Kamhi (1984), showing that language-impaired children tend to "pad" their utterances by extensive use of a handful of grammatical function words. There were, however, several cases of extreme referential style in the focal lesion data as well. These contrasting extremes provide us with an opportunity to examine two different hypotheses that have been offered to explain the analytic/holistic dissociations observed in normal children (see Bates et al., 1988, for a discussion).

The **interhemispheric hypothesis** is based on the claim that the left hemisphere is specialized for fine-grained analytic operations, while the right hemisphere is specialized for holistic/configurational operations (e.g., Bradshaw & Nettleson, 1981). By this argument, an analytic/segmenting approach to language learning should be blocked if the left hemisphere is damaged, while a holistic/configurational approach should be blocked if the right hemisphere is damaged. This prediction is reasonable, but it is not borne out by our studies of infants with focal brain injury. In fact, Thal et al. report a significantly *higher* incidence of pronominal/expressive style in children with right-hemisphere damage, with proportionally more referential/telegraphic speech in children with left-hemisphere damage. This finding suggests that left-hemisphere processes may play an important role in the early production of pronouns and other function words—even when those words are used in rote or "formulaic" expressions. In short, although the right-hemisphere account of holistic style in normal children

is an appealing one, it receives no support from the patterns observed in children with focal brain injury.

The **intrahemispheric hypothesis** is based on an analogy between Broca's aphasia and analytic/referential style (i.e., telegraphic speech with a high proportion of content words, especially nouns), and on an analogy between Wernicke's aphasia and holistic/pronominal style (i.e., fluent but "empty" speech with a high proportion of pronouns and other function words). By this logic, analytic style should be more common in children with left anterior damage, while holistic style should occur more often in children with left posterior injuries. This hypothesis is also disconfirmed by the Thal et al. findings. In fact, the holistic/pronominal pattern was significantly less common in children with left posterior injuries, suggesting that regions of left posterior cortex may play a particularly important role in the production of pronouns and other grammatical function words—even when (or particularly when) those forms are largely restricted to memorized fragments of speech.

There is a third possibility which could account for all these findings, although it has not been explored in the literature on individual differences in normal children. We know from research on visual-spatial pattern analysis by Stiles and others (see below) that children and adults with LH damage demonstrate deficits in the *extraction of local detail*, while children and adults with RH damage experience difficulty with *overall configuration*. The interhemispheric hypothesis described above assumes an equation between formulaic speech (including unanalyzed use of grammatical function words) and holistic/configurational analysis. However, this assumption may be incorrect. Instead, it is possible that "expressive style" children produce a relatively high proportion of pronouns and function words in their first word combinations because they have extracted a higher-than-normal proportion of "local detail" from their linguistic input; they reproduce these "little words" in a rote fashion, and may be slightly delayed in the long-term mastery of the rules that govern those forms (see Bates et al., 1988, for a discussion) because they have failed to integrate those forms into the larger semantic-grammatic framework that motivates use of pronouns and other function words in the adult language.

This **local-detail hypothesis** has several advantages for our purposes here. First, it suggests that "expressive style" should be minimal in children with left posterior damage—the same lesion type that is associated with problems in extraction of local detail in our visual-spatial tasks (see below). Second, this proposal could account for the other expressive language problems displayed by infants with left posterior damage, from babbling through onset of grammar. By analogy with the adult literature, we tend to equate comprehension with sensory processing, and production with motor abilities. However, during the period in

which children are learning to produce speech, they have to analyze the speech stream in sufficient detail to permit the construction of a motor analogue. **It is possible that the selectively greater problems in expressive language displayed by children with left posterior damage derive not from motor problems but from limitations on the kind of sensory analysis that is required for precise sensory-to-motor mapping.** Once that phase of learning is over, there may be a corresponding reduction in the role played by left posterior cortex in expressive language—particularly for "overlearned" aspects of production, i.e., phonology and grammar.

So which hypothesis is correct? Bates et al. obtained a rather clear conclusion in their investigation of children between 19-30 months of age: expressive/holistic style was significantly more common in children with right-hemisphere damage. In other words, the pattern of "running off at the mouth" that is characteristic of expressive/holistic style seems to be more common when the right hemisphere is unable to perform its modulating/integrative role in early language learning.

Grammar vs. vocabulary. In the same study by Bates et al., it was also possible to ask whether grammar and vocabulary dissociate at any point during the first stages of language learning. The answer was very clear: they do not dissociate, at least not in this period of development. They do report that some lesions have a greater effect on expressive language development than others, but these lesions have equally serious effects on both vocabulary and grammar. Specifically, children whose lesions involved the left temporal lobe tend to suffer greater delays in both vocabulary and grammar across the first years of language learning, an effect that is still visible as late as 5 years of age (but not beyond that point). In addition, they report that children who have frontal damage to *either* the left *or* right hemisphere also tend to move more slowly in expressive language development. However, this bilateral effect only reached statistical significance between 19-30 months of age (the most intensive period of language development, encompassing both the vocabulary burst and the first wave of grammaticization in normally developing children). Most important for our purposes here, the delaying effects of frontal involvement were equally evident in grammar and vocabulary. It appears that the same laws that govern co-development of grammar and vocabulary in children who are neurologically intact are also operating in the focal lesion sample, suggesting that the acquisition of grammar and vocabulary may be mediated by the same neural mechanisms during this period of development.

Are there any populations in which we do see a dissociation between grammar and vocabulary? Selectively greater delays in grammar (compared with voca-

bulary and other aspects of pragmatics and semantics) have been reported for much older children in several different populations, including Specific Language Impairment (Johnston & Kamhi, 1984; Leonard, 1998), Down Syndrome (Chapman, 1995; Fowler, 1993; Singer Harris, Bellugi, Bates, Jones, & Rossen, 1997; Vicari, Caselli and Tonucci, 1999) and even in deaf children who are trying to acquire an oral language (Volterra & Bates, 1989). The reasons for the selective vulnerability of grammar have been hotly debated (Bishop, 1992, 1994; Rice & Wexler, 1995; Rice, Wexler, & Cleave, 1995). Some investigators attribute this selective delay to deficits in an innate grammar module, controlled by a specific gene that is deficient in children with Specific Language Delay. Other investigators have argued instead that children with SLI suffer from some kind of perceptual impairment (Bishop, 1994, 1997; Tallal, Stark, Kallman, & Mellits, 1980, 1981; Tallal, Stark, & Mellits, 1985a,b), one which has greater effects on grammatical morphology than any other aspect of language precisely because those “little words” and endings are low in perceptual salience.

We are sympathetic to the latter position, for two reasons. First, studies in our laboratory have shown that selective deficits in grammatical morphology can be induced in perfectly normal college students when they are forced to process sentences under perceptually degraded conditions, through compression, low-pass filtering, or some combination of the two (Blackwell & Bates, 1995; Dick et al., 1999; Dick, Bates, Wulfeck, & Dronkers, 1998; Kilborn, 1991; see also Miyake, Carpenter, & Just, 1994). This result lends plausibility to the hypothesis that a subtle congenital deficit at the perceptual level could have serious repercussions for language development in general, but for grammatical development in particular. Second, the fact that this profile of grammatical delay is also observed in deaf children and in children with Down Syndrome lends support to the idea that such a delay reflects weaknesses in auditory processing (an area that is known to be especially vulnerable in Down Syndrome (Chapman, 1995). In this regard, a recent study by Singer Harris et al. (1997) compared the first stages of vocabulary and grammar in children with Down Syndrome (for whom auditory deficits are a frequent problem) and a matched sample of children with Williams Syndrome (who are known to have particularly acute hearing). Both groups of children were equally and massively delayed in early vocabulary, suggesting that language cannot get off the ground until children reach the same cognitive level (or mental age) at which language appears in normal children. Indeed, there were no significant differences between the Down and Williams populations—until the point at which grammar begins to flourish. At this point, a significant difference emerged. For children with Williams Syndrome, the growth of grammar was tied quite closely to vocabulary size (following the curve in Figure 3), but children with Down Syndrome

displayed levels of grammar below what we would expect for their vocabulary size. It is worth noting that there were strong correlations between grammar and vocabulary in both groups (i.e., the two domains do not completely dissociate), but the slope of the function for Down children was very low, as we would expect if grammar had to be acquired through some kind of noise filter.

Returning for the moment to evidence from the focal lesion population, a comparison between the substantial delays observed in infancy and the normal to low-normal performance in older children leads to a hypothesized “window of recovery” between 1 and 5-6 years of age, a period in which children with focal brain injury find alternative ways to solve the problem of language acquisition. This finding permits us to cast the old problem of brain/behavior correlations in a different light, and provide a solution to the debate between proponents of equipotentiality and proponents of irreversible determinism. On this compromise view, there are initial biases in the regions of the brain that are likely to take over the language task, such that lesions to these areas create specific patterns of delay during language acquisition. However, these biases can be overcome, and alternative forms of brain organization for language can emerge across the course of language learning. Of course much more evidence is necessary before we can draw firm conclusions about the neural factors that underlie normal and abnormal variations in language learning. Only one conclusion seems clear so far: **The brain regions that mediate language acquisition in the first years of life are not necessarily the same regions that mediate processing and maintenance of language in the adult.** This brings us to the next and final section, on human brain development and its relationship to the language milestones and variations that we have discussed in such detail.

VI. THE NEURAL CORRELATES OF LANGUAGE MILESTONES

We have shown that the course of language development is exceedingly complex, characterized by massive variability across children, and by multiple bursts and plateaus within individual children. We have also seen that learning plays an extremely important role throughout this process, starting as early as the third trimester of pregnancy. Children start to pick up language-specific preferences *in utero*, and they continue to “tune” the language processor in various directions depending on the nature of their input. This is not the view of language development that Lenneberg (1967) had in mind when he laid down a series of observations and predictions about the maturation of language. Lenneberg paid more attention to means than variations in language acquisition by English-speaking children, and he proposed that these milestones may be timed by a biological clock that also governs motor milestones like crawling and walking. In his 1967 book, he

provided a famous table comparing milestones in language and motor development (e.g., first steps and first words around the first birthday) in defense of this maturational view. This oft-cited table is based on average onset times across different samples of children, but it implies a set of correlations that ought to hold within individual children as well. As it turns out, there is little evidence for such a lockstep process when milestones are compared within the same sample of healthy, normal children. For example, Bates et al. (1979) looked for such correlations in their longitudinal study of language and communication from 9-13 months. There were no significant links between motor and language milestones, and if anything the non-significant correlation between walking and talking seemed to run in the wrong direction, as if there were a slight tendency for children to make some kind of choice about where to invest their energies among the various skills that are starting to emerge around this time. And yet we know that the nervous system continues to develop after birth in our species. Surely it ought to be possible to find neural correlates (and perhaps neural causes?) for the dramatic changes that characterize language development in the first few years of human life.

In an earlier version of this chapter (Bates, Thal, & Janowsky, 1992), we joined the search for neural correlates of language development, and pointed out two likely candidates for such a relationship. First, we noted (see above) that the period between 8-10 months is a behavioral watershed, characterized by marked changes and reorganizations in many different domains including speech perception and production, memory and categorization, imitation, joint reference and intentional communication, and of course word comprehension. We speculated that this set of correlated changes (and they are correlated within individual children) may be related to the achievement of adult-like patterns of connectivity and brain metabolism, with particular reference to changes involving the frontal lobes. Second, we noted (see above) that the period between 16 and 30 months encases a series of sharp nonlinear increases in expressive language, including exponential increases in both vocabulary and grammar. We pointed out a possible link between this series of behavioral "bursts" and a marked increase in synaptic density and brain metabolism that was estimated to take place around the same time. We produced our own table comparing estimated/average onset times for behavioral and neural events across the human lifetime, and we speculated (based on average onset times across different children) that some kind of causal relationship may be involved. That table was very popular, and has been cited frequently since the 1992 chapter appeared (though not as frequently as its ancestor in the Lenneberg volume). Hence some readers may be disappointed to find that the table has disappeared, replaced by a much more complex and challenging story. It is undoubtedly

the case that brain maturation plays a causal role in language learning and in many other aspects of behavior development. But we have learned a number of lessons that mitigate this claim, and make us wary of summary tables that imply any simple form of cause and effect. Here are three lessons that have led us to remove the old table of correlations.

First, it has become increasingly clear that learning plays a massive role in language development. Of course this has to be true in some trivial sense, because we know that English children learn English and Chinese children learn Chinese. However, there has been a long tradition of skepticism about learning in the child language literature, because language development is characterized by so many "funny-looking" events, including long plateaus interrupted by exponential shifts, with occasional steps backward (e.g., the child who produces error-free versions of the past tense for several weeks or months, and then suddenly starts to make mistakes like "goed" and "stooded-up"). These nonlinearities and nonmonotonocities have led many investigators to underplay the role of garden-variety learning in favor of a maturational view in which apparent discontinuities at the behavioral level are caused by discontinuities in the nervous system (Pinker, 1994; Wexler, 1996). Although this is a plausible scenario, and it might be true for some subset of events, it rests on a mistaken assumption: that "garden-variety learning" is a simple linear process, and cannot produce nonlinear and especially nonmonotonic functions of the kind that are so often observed in language development. As it turns out, that simply isn't true. In the last decade, we have seen many examples of nonlinear and nonmonotonic learning in artificial neural networks (Elman et al., 1996). These multilayered networks are relatively simple in their structure (and certainly must be viewed as abstract toys, in comparison with real nervous systems at any level of phylogeny), but they are very good at pattern perception and learning, and they have been used to simulate in considerable detail many of the "funny-looking" learning functions that characterize language development. They are able to do this because, despite their simplicity, they constitute nonlinear dynamical systems, which are notorious for their unpredictability and for the wide range of growth functions that they can display. In other words, the linear view of "garden variety learning" is wrong. At the same time that we have learned to appreciate the emergence of complex learning in simple systems, evidence has also mounted showing that very young infants are capable of rapid and powerful forms of statistical learning, inside and outside of the linguistic domain (e.g., Bates & Elman, 1996; Elman & Bates, 1997; Saffran et al., 1996). Putting these trends together, we can no longer assume that all discontinuities in behavior reflect discontinuities in the developing nervous system. Even within a structurally stable learning device, funny-looking things can

happen—including the discontinuities that characterize language and communicative development at the 8-10-month watershed, and the exponential bursts that are observed in vocabulary and grammar between 16-30 months.

Second, we know much more than we knew ten years ago about human brain development, before and after birth. Ten years ago there was an appealing story in the air that we incorporated into our chapter on language and its neural correlates: prenatal development is characterized primarily by “additive events” (e.g., neural tube formation, cell proliferation and migration, and the first wave of connectivity); postnatal development does include some further additive events (especially synaptogenesis and myelination), but it is characterized primarily by “subtractive events”, including the whittling away or subtraction of cells, axons and (above all) synapses under the careful guidance of experience. This general view of exuberant growth in the first wave (more cells, axons and synapses than the organism will ever need) followed by subtraction is still true, but not in the two-stage form in which we presented it the first time around. Instead, our current view of brain development acknowledges a host of both additive and subtractive events, before and after birth, in multiple dimensions with multiple gradients. In fact, the picture of human brain development that we will try to present here is one that is quite compatible with the burgeoning literature on early (even prenatal) learning, because so many of the events required to create a learning machine take place within the first two trimesters of prenatal human life. Everything that happens after that is really a matter of degree—maturational changes at every level of the system, in multiple overlapping gradients. These postnatal changes do have interesting consequences, but one is hard pressed to find anything that changes in a punctate fashion. Above all, there is little evidence for the old-fashioned notion of modular brain systems that “turn on” at a particular time, like successive levels in a computer game.

Third, it has become increasingly clear that the relationship between brain development and behavior is bidirectional. That insight was already present in our earlier chapter, where we underscored the role of experience in synaptic elimination and other subtractive events, yielding the metaphor of experience as a sculptor working away in the studio of life. However, recent research in developmental neurobiology has shown that the bidirectional dance between brain development and experience occurs at many more levels of the system, including additive events throughout the lifetime of the organism. To be sure, a huge wave of synaptogenesis that takes place in the first year of postnatal life is never seen again, and the early waves of neurogenesis and migration have no postnatal parallels. Nevertheless, it is now known that complex learning in adulthood induces synaptogenesis and other striking

morphological changes in brain regions related to the challenging new task (Kleim, Lussnig, Schwarz, Comery, & Greenough, 1996; Kleim et al, 1997). In a few privileged areas of the brain, like the dentate gyrus of the hippocampus, new brain cells can be formed throughout life and the rate can be modified depending on experience (Kempermann, Brandon, & Gage, 1998; Kornack & Rakic, 1999). As a result of all this new information, it is no longer advisable to assume (without further evidence) that correlated changes in brain and behavior reflect a causal flow in one direction. It could just as easily be the other way around.

With these lessons in mind, we will provide an overview of basic events in human brain development that precede, prepare, parallel and (perhaps) participate in the language-learning process. We will review neural events globally, concentrating mainly, but not exclusively on the *isocortex* (a synonym for *neocortex* that neuroanatomists prefer because it does not make false assumptions about how “new” in phylogeny the cortex is), and not only on those areas conventionally viewed as “language areas” in the adult. Unquestionably, neurological disturbances in these areas will produce language-related deficits (at least in the adult), but it may be more accurate to think of language acquisition and production as an interactive process involving auditory, visual, somatosensory, motor, memory, emotional and associative functions. The neural areas governing these functions are located in widespread regions of temporal, parietal, frontal, and prefrontal cortices, and do not develop in isolation.

We will address attention to three issues.

1) *Prenatal Neural Events: Fundamental Brain Scaffolding*: What is the state of the brain at or before birth when the rudiments of language learning begin?

2) *Postnatal Neural Events*: What types of neurodevelopmental events take place after birth and across the period in which languages are learned? We will focus here on synaptogenesis, the process through which neurons receive their connections, as the key changing component of brain organization during this period, and on the postnatal elaboration of gradients of various maturational processes initiated prenatally.

3) *Interactions of Neural Patterns and Events with Language Learning*: Do any neurodevelopmental events seem placed or ordered in such a way as to constrain when events in language learning might occur? Alternately, does language learning itself alter the course of brain development?

1) Prenatal Events: Fundamental brain scaffolding

Fixing the timing of events. There are no experimental studies directly relating language and cognitive development to brain maturation, and there are only a handful of studies that have tried to relate disorders of brain and behavioral development to fundamental cellular processes. As a result, our estimates of maturational timing in the human brain

must be based on correlational and comparative approaches. Fortunately, the literature on perinatal brain development in other mammals has grown so rich in the past decade that our basis for correlation and inference is extremely strong. We are also aided by recent investigations showing that the schedule of human brain development can be mapped with some precision onto the maturational schedules of other animals (Clancy, Darlington, & Finlay, in press; Darlington, Dunlop, & Finlay, in press; Finlay & Darlington, 1995). In fact, the order and relative spacing of early neural events is remarkably stable across all mammalian species, permitting use of a log/linear equation to generate a sequence of predicted dates for corresponding developmental events in humans. This model captures the statistical relationships among 92 mammalian developmental events obtained from 9 different mammalian species, including some from humans. With a model that is initially derived from nonhumans, we are able to predict known dates of human developmental events with considerable precision, and as a result we can also predict the dates for events that have not yet been empirically measured in our species. Although there are many striking similarities across species, this approach has also shown that primates (including humans) differ systematically from other mammals in the timing of neurogenesis (which refers to the process in which new neurons are produced) in two key neural regions, the limbic system and the isocortex. The limbic system is a circuit of widely distributed neural structures that includes the hippocampal formation, associated with memory and spatial learning, as well as neural regions associated with olfaction and emotion. Neurogenesis of the limbic regions is abbreviated in primates, resulting in uniformly smaller limbic structures when compared to similar areas in nonprimates. In contrast, the isocortex in primates has a relatively protracted neurogenesis, and a consequently increased relative size (Clancy et al., in press; Finlay & Darlington, 1995). A very simple principle underlies this difference in the relative size and shape of brain systems: if a species gains extra cycles of neurogenesis across the course of evolution, the greatest relative enlargement occurs in the parts of the brain that develop relatively late.

With this fact about primate variability factored into the Finlay and Darlington (1995) statistical model, we are able to produce reliable predictions for the dates of a number of uninvestigated human neural developmental events, including aspects of neurogenesis, pathway formation, and various regressive events across functional brain systems which would typically require invasive procedures for accurate determination (discussed in more detail in Clancy et al. in press). Unless indicated, all statements in the following text about the time of occurrence of maturational events in human neural development are drawn from data produced using this comparative mammalian model.

First trimester. It is startling to realize how much of fundamental brain morphology and organization is already laid down by the end of the first three months of life (before many mothers even know that they are pregnant). Approximately 10 days after conception, the developing embryo, as yet without much tissue differentiation, has implanted itself into the uterine wall. There it quickly flattens into three distinct layers (ectoderm, mesoderm and endoderm); the skin, sense organs and the rest of the central nervous system will all develop out of the ectodermal layer. Within the ectoderm, rapid proliferation of cells on either side of the midline along the length of the entire embryo pushes up edges of tissue which meet and form the neural tube, enclosing a fluid-filled ventricle. The neural tube gives rise to the entire brain, forebrain at one end, and spinal cord at the other. All neurons in the brain are generated from stem cells on the inside of the tube, called either the ventricular zone because it adjoins the ventricle, or the proliferative zone. Neurons migrate out from the ventricular zone to the overlying mantle along radial glial guides, with some number of their mature neuronal features already specified and others to be picked up as they migrate and settle into their terminal regions by communication with other cells and the general cellular environment. By six weeks, the form of a human embryo is recognizable.

Virtually every neuron in the nervous system is generated in the first trimester, with the exception of the tail of the distribution of the last layer of the isocortex, and the external granular layer of the cerebellum. Two other exceptions are the hippocampal dentate gyrus and the olfactory bulb, which are (as far as we now know) the only regions in which neurons are generated throughout life (albeit at a very low rate) in all mammals studied, including primates (Bayer, 1982, 1983; Kornack & Rakic, 1999; Kuhn, Dickinson-Anson, & Gage, 1996; Luskin, 1998). The first activity the early-generated neurons engage in is to lay down the basic axonal pathways of the brainstem (Easter, Ross, & Frankfurter, 1993). An interesting point to note here is how variable (in different parts of the nervous system) the sequence of neural differentiation can be—there is no simple lockstep plan for all neurons like “Migrate; become electrically excitable; produce axon; produce dendrites; make neurotransmitter, fire away.” To take the case of axons alone, axons can be produced while neurons are migrating; not produced until the terminal site is reached; may show growth of multiple stages and types (branching or not, for example); may be produced and then retracted; or may show prolonged periods of no growth (“waiting periods”).

Two more critical processes are virtually complete by the end of the first trimester: the differentiation of cells into different subtypes (also called “cell specification”) and the migration of cells from their birthplace in the ventricular zone to their ultimate destinations in

cortical sites. In fact, these two events (specification and migration) are functionally intertwined. The “type” of a neuron includes many aspects—what shape it has, what information it receives, what transmitters and receptors it produces, and so forth. Some of these features can be specified by location, so that the path taken by a cell as it migrates and its ultimate arrival in a certain brain region will fix some aspects of its “type” while others are set on, or immediately after, generation in the ventricular zone (Cepko, 1999). For example, cells begin to express various complements of signaling chemicals (neurotransmitters and neuromodulators) before migration, as soon as they are born in the ventricular zone (Lidow & Rakic, 1995). The neurotransmitters and neuromodulators that any particular class of cells exhibits (including both receptors and the metabolic machinery for making and degrading these substances) continue to develop in the following months. Although there are many different kinds of neurochemicals within and across cell types, there seems to be a general developmental principle at work: in early stages of development, neurons will often co-express multiple transmitters and modulators whereas single cells in the mature brain exhibit much less diversity.

Second trimester. This is the period in which the basic wiring of the brain takes place, i.e., the large patterns of connectivity develop between neural regions, including the isocortex. This picture is confirmed in humans by looking for molecular markers that reflect the activity of building axonal and dendritic arbors (Honig, Herrmann, & Shatz, 1996). From a developmental point of view, one of the most important events is the establishment of connections from the thalamus to all regions of the isocortex. The thalamus is a subcortical structure that transmits virtually all sensory input from the body surface and special sense organs (except olfaction) to the isocortex. Developmentally, the thalamus maintains the “packaging” that separates one kind of input from another (e.g., visual, auditory, somatosensory). These connections are set up in the second trimester in a pattern that very much resembles the adult pattern from the start, with animal studies showing that visual, somatosensory, auditory and limbic areas of cortex all receive projections fairly exclusively from those thalamic nuclei that will project to them in adulthood (Miller, Chou, & Finlay, 1993; Molnar, Adams, & Blakemore, 1998; O’Leary, Schlaggar, & Tuttle, 1994). This is particularly important for theories of development, because it means that the brain is “colonized” by the body long before birth, with boundaries between major brain regions determined by their input well before the outside world has a chance to instruct the brain. Intracortical pathways (i.e., connections from one cortical region to another) also begin to establish their mature connectivity patterns in the second trimester. The corpus callosum makes its first appearance around postconceptional day 90 and lays down a pattern of homotopic connections over the

following month, that is, connections between the area of cortex and its corresponding cortex on the other side (reviewed in Innocenti, 1991). The long-range axonal connections start to produce synapses in their target structures in short order, although the bulk of synaptogenesis will occur later (Antonini & Shatz, 1990; Bourgeois & Rakic, 1993).

As we noted earlier, neural development is characterized at many levels and at many points in time by exuberance or overproduction of elements (an additive event), followed by a large-scale “shake-down” or elimination of the same elements (a subtractive or regressive event). A particular kind of regressive event called apoptotic neuronal death occurs in the second trimester (“apoptosis” is a morphologically distinct kind of cell death associated with an orchestrated death program, not a disorganized dissolution of the cell). This kind of developmental cell death usually occurs in close association with the establishment of major axon pathways between regions, and can contribute to removal of errors in axonal connections and numerical matching of connecting populations of cells (Finlay, 1992). Apoptosis can be quite extensive and rapid, often resulting in the loss of the majority of the neurons originally generated. For example, the retina establishes its connections with subcortical targets in the third month post conception in humans, and reaches the peak number of axons in the optic nerve about a month later. By the end of month 5, retinal ganglion cell loss is over, removing as much as 80% of the originally generated cell population (a process that has been directly demonstrated in humans—Provis & Penfold, 1988; Provis, van Driel, Billson, & Russell, 1985). Such cell loss also occurs in the isocortex, particularly in the subplate and the upper cortical layers (Shatz, Chun, & Luskin, 1988; Woo, Beale, & Finlay, 1991). Though subplate loss is prenatal, isocortical death in the upper layers may extend into the first couple of postnatal months (O’Kusky & Collonier, 1982). Overall, early neuronal death seems to serve to grossly fix cell numbers in interconnecting populations and to fine-tune topographic projections between structures, but does not contribute to the kind of fine-tuning of connectional anatomy associated with learning from the extra-uterine environment in the isocortex.

The second trimester is also the period in which something akin to learning or “self-instruction” begins, a process of activity-dependent self-organization of the nervous system. While the physiological and cellular consequences of this phenomenon have been best studied in the visual system, it seems like such a useful developmental mechanism for organizing spatially distributed systems that it is likely it will be discovered elsewhere. For example, the first motor activity of the fetus begins at 2-3 months post conception and continues through intrauterine life, and although the neuroanatomical consequences of this activity are not known, the pattern of activity that it generates in the

nervous system is structured and phasic (Robertson, Dierker, Sorokin, & Rosen, 1982). In the retina, “waves” of activity begin to be propagated across the retinal surface, generated by amacrine cells, beginning (in cats and ferrets) after basic connectivity is established and stopping before eye opening, corresponding to second trimester in human development (reviewed in Wong, 1999). This organized activity can be the basis for a kind of primitive categorization, a process in which similar (correlated) inputs hang together while dissimilar (uncorrelated) inputs dissociate. An important example can be found in the establishment of ocular dominance columns, a stripe-like pattern of left-right alternation in primary visual cortex which seems to reflect the brain’s solution to competing (unlike) waves of input from the two eyes that feed into overlapping regions. Because retinal waves produce a hypercorrelation of the activity of spatially adjacent cells in the retina, this information can also be used to fine-tune topographically mapped projections, and it could also produce more detailed spatial structures like orientation sensitivity in visual cortical neurons. This self-organizing process has some very interesting theoretical implications for developmental psychologists: activity-dependent organization occupies a middle ground in the nature-nurture debate, where some of the same mechanisms that will be used later for learning from the outside world (i.e., response to correlations in the input) are used *in utero* to set up the basic functional architecture of the brain. *In utero*, some of this organizing activity may be imposed by the activity of the body itself, or by the intrinsic circuitry of the nervous system.

Third trimester. By the beginning of the seventh month of gestation, a remarkably large number of neural events are complete. The human fetus has matured to the point where the eyes move and remain open for measurable periods of time (though there isn’t much to see—more on this below). Reciprocal connectivity from higher-order cortical areas to primary areas has also begun (Burkhalter, 1993). Pathways exhibit the initial process of myelination (Yakovlev & Lecours, 1967). Large descending pathways from the cortex are also in the process of development. Aside from the more obvious role of descending pathways in motor control, the appearance of descending pathways also means that the brain has started to “talk back” to its input regions, a form of interaction found in all sensory as well as motor systems. The nature and function of this “top-down” connectivity within sensory systems are still poorly understood (the term “top-down” is preferred to “feedback” since it does not prejudge the region of initiation), but it is now clear that simple bottom-up processing sequences are not sufficient to explain many behavioral phenomena. Upon maturity, some top-down projections will actually be many times more robust than their “bottom-up” counterparts (see Churchland, Ramachandran, & Sej-

nowski, 1994). These descending projections are believed to be involved in the dynamic processing strategies that are tied to attention and learning (Cauller, 1995; see collection in Koch & Davis, 1994) and so it is quite interesting in this regard that they continue to develop well after birth.

In the eighth and ninth month, a massive and coordinated birth of synaptic connections begins in the isocortex and related structures, as we will discuss in detail in the next section. In general, however, it is fair to say that the infant arrives in the world with a nervous system whose working components are in place and organized. All cells are generated, all major incoming sensory pathways are in place and have already gone through a period of refinement of their total number of cells, connections, and topographic organization. Intracortical and connectional pathways are well developed, though output pathways lag behind. The microstructure of such features as motion and orientation selectivity in the visual system is already present, though more remains to be elaborated. The “big” cortical regions—primary sensory and motor regions—have their adult input and topography, though we do not know yet if all of the multiple subareas described for the primate cortex have sorted themselves out (Felleman & van Essen, 1991). This brain is up and running at birth, ready to learn. In fact, it has been capable of learning for several weeks, a neurophysiological fact that complements and supports surprising new evidence for prenatal learning of at least some aspects of speech (see Part III above).

2) Postnatal neural events

Now we turn to a consideration of events that extend past birth, with special emphasis on the neural events that surround language learning. As we have already pointed out, the search for punctate and lockstep correlations between neural and behavioral milestones has proven fruitless. However, we can draw some interesting lessons about behavior from the shifting neural landscape that characterizes postnatal brain development in humans.

Myelination. Myelination refers to an increase in the fatty sheath that surrounds neuronal pathways, a process that increases the efficiency of information transmission. In the central nervous system, sensory areas tend to myelinate earlier than motor areas—a fact that has been cited as a possible contributor to the comprehension/production disparity observed in some children. Intracortical association areas are known to myelinate last, and continue to myelinate at least into the second decade of life. Myelination of some callosal and associational cortical regions may continue well into maturity, extending throughout the third and even fourth decade (Yakovlev & Lecours, 1967).

Speculations about the brain basis of behavioral development have often revolved around the process of myelination because it continues for so many years after birth (Parmelee & Sigman, 1983; Volpe, 1987). How-

ever, interest in the causal role of myelination has waned. First, because this is such a protracted process, there are no clear-cut transitions that might provide a basis for major reorganizations in the behavioral domain. Second, we know that "undermyelinated" connections in the young human brain are still capable of transmitting information; additions to the myelin sheath may increase efficiency, but they cannot be the primary causal factor in brain organization of language or any other higher cognitive process. Third, the discovery of other large-scale progressive and regressive events in early brain development that are influenced by interactions of maturation and experience are more appealing candidates as the sculptors of behavioral development—which brings us to a consideration of synaptogenesis.

Synaptogenesis. None of the neural events we have discussed so far span the dramatic events that define early language development. The production and placement of neurons is complete before birth in all structures that do not continue neurogenesis throughout life. Regional connectivity of the isocortex begins in the second trimester and, although completed postnatally, it bears no obvious relationship to changes in language ability except in a permissive sense. A mature reciprocal pattern from secondary to primary visual cortex (although not the mature density) is accomplished somewhere between 4 months and 2 years in developing humans (Burkhalter, 1993). Myelination "brackets" language acquisition only in the most global sense, i.e., it takes place somewhere between gestation and adulthood. Synaptogenesis, however, is an event that occurs in the critical time window for early language development, and seems optimally placed for the rapid statistical learning infants show in both the visual and auditory realms during this time (Saffran et al., 1996).

Synaptogenesis and synapse elimination co-occur over most of early postnatal development, and they co-occur throughout life. However, there are some interesting features to synaptogenesis and elimination within the perinatal period that seem quite closely related to early language acquisition. Because this is such an appealing candidate for a correlational and (perhaps) causal role in language development, we need to provide some important details about the methods used to calculate synaptic growth, and the problems that are encountered in the measurement of this moving target.

The word "synapse" is often used loosely to designate an elemental functional connection between neurons, but anatomists use it more specifically, and look for specific features of synaptic form associated with particular functions. Synapses are chemical junctions between neurons, visible as described here with electron microscopy. Axonal or presynaptic processes contain the metabolic machinery to produce neurotransmitters and package them, often in recog-

nizable packages of vesicles, and a "presynaptic specialization", a thickening of the cellular membrane that can transfer the contents of the synaptic vesicle to the synaptic cleft between neurons in response to activity of the presynaptic neuron. There is also a visible thickening of the membrane of the postsynaptic neuron, on the opposite side of the synaptic cleft, with the machinery to take up, and perhaps degrade, the neurotransmitter released by the postsynaptic cell, and to cause depolarization or hyperpolarization of the postsynaptic cell. Most, but not all, excitatory synapses have "asymmetric" synapses, in which the presynaptic specialization is thicker and denser than the postsynaptic one; most inhibitory synapses are "symmetric", with pre- and postsynaptic thickenings of equal density. The location of the synapse is significant to its function—a synapse can be located on the cell body of the neuron itself, on the shafts of dendrites, or on small spikes appropriately called dendritic spines, which will have consequences for how effectively the presynaptic input can induce changes in the postsynaptic cell. For each neuron, a single synapse usually only contributes a tiny fraction of its input—according to Kandel, Schwartz and Jessell (1991), morphological data indicate an average neuron forms 1000 synaptic contacts (presynaptically) and can receive 10,000->150,000 contacts postsynaptically. A record 200,000 spines on Purkinje cells of the cerebellum have been estimated, but 15,000 is average on layer V cells of the isocortex (Koch & Zador, 1993). It is important to note that chemical synapses are only one part of the total number of ways neurons may communicate—there can be direct electrical coupling between cells (this is particularly prominent in early development), cells can communicate through the release of gases, notably nitric oxide, and by altering—through any means—the extracellular milieu of the neurons surrounding them. Chemical synapses, however, are easily recognizable, countable and are a central component of neuronal signalling, and hence have been much studied.

A primary mode of learning in the nervous system (though not the only mode) takes place when the juncture is formed or modified as a function of experience, a "strengthening" or "weakening" referred to as Hebbian learning. If we ask ourselves where the nervous system stores its "knowledge" (assuming that this term is useful at all), most neuroscientists would agree that synaptic connectivity is the primary means by which knowledge is represented in the brain (Elman et al., 1996), whether that knowledge is innate (set up independent of experience), learned (set up by experience) or somewhere in between (as in the above-mentioned case of prenatal activity dependence). This is why there is so much interest in the role of synaptogenesis and synaptic connectivity in behavioral development.

In cognitive science, the number of synapses is often thought of as an index for the amount and

complexity of information transfer in a structure. Even though synaptic number might be used as such a metric in some comparisons (for example, after certain kinds of experience (Greenough, 1984), it is misleading to understand synaptic numbers in development in this way. “More” in development does not necessarily mean better, more complex, or more mature. To take an extreme case, sudden infant death syndrome (SIDS) is associated with an excess number of persisting synapses in the medulla (O’Kusky & Norman, 1994, 1995). This point is important for understanding a high-profile controversy about synaptogenesis and the peak of synaptic numbers in the isocortex of primates and humans. Briefly, in work with rhesus macaques, Rakic and colleagues described a rapid increase in the number of synapses that seemed to take place almost simultaneously across a number of cortical areas, reaching a peak at around the same time in frontal, cingulate, somatosensory and visual cortical areas (Bourgeois, Goldman-Rakic, & Rakic, 1994; Granger, Teklaia, Lesourd, Rakic, & Bourgeois, 1995; Rakic, Bourgeois, Eckenhoff, Zecevic, & Goldman-Rakic, 1986; Zecevic, Bourgeois, & Rakic, 1989; Zecevic & Rakic, 1991). In contrast, Huttenlocher, working with human material, showed that the peak of synaptic density varies between visual, auditory and somatosensory regions, with the frontal regions not reaching their peak until 3-4 years after birth, while the visual and auditory regions peak more closely to birth (Huttenlocher & Dabholkar, 1997). A closer examination proves that the story these two investigators tell is not very different after all. Part of the confusion lies in mistakenly identifying peak of synaptogenesis with all other aspects of maturational change in different cortical areas.

In order to understand this literature, some of the methodological issues involved in the counting of synapses must be addressed. The most useful information about synaptic growth would be the documentation of a change in absolute number of synapses within a single structure, or perhaps the average number of synapses per neuron within an identified structure. The latter is currently the preferred method of analysis in studies investigating the effects of experience on synaptic changes in adult animals (Jones, Klintsova, Kilman, Sirevaag, & Greenough, 1997). However, this is not the measure used in developmental studies like those of Huttenlocher or Rakic, for a very simple reason: while synapses are growing, the rest of the brain in a young animal is changing as well. It is difficult to calculate a ratio of “synapses per unit” when the nominator and the denominator are both moving targets. In fact, with the exception of primary visual cortex and a few other areas, the borders of most cortical areas are not well defined enough in early development to assess a change in volume of a cortical area, let alone the number of synapses in that area.

An alternative would be to measure the absolute number of synapses per neuron. To obtain the absolute number of synapses per neuron with confidence, a stereological method of relatively recent invention can be used to eliminate the problems involved in counting synapses and neurons of massively different sizes and shapes in very thin sections (discussed in Guillery & Herrup, 1997). This requires a particular method of tissue preparation and analysis that was not used when the initial (and very valuable) samples of macaque and human brain tissue were gathered. Therefore, to analyze the available tissue, “assumption-based” stereology is used, which means that certain assumptions are made about the shape and distribution of the things that we are going to count. Different assumptions (all of them very plausible) produce absolute estimates of synaptic density that can vary by factors of 3-4, as the papers themselves demonstrate. Even so, we can get a reasonable idea of how the number of synapses change in the cortex in a relative sense if not an absolute one (taken with several grains of salt).

Both Rakic and Huttenlocher counted the number of visible synapses in a thin section of cortical tissue, expressed as a fraction of the area of the “neuropil” (which we will define in a moment), and then stereologically or otherwise correct their counts to get the number of synapses in a volume of tissue. The neuropil is defined by exclusion—it is that area of tissue in the brain in cell-dense areas like the cortex that remains after large, “nonsynaptic” chunks of tissue are excluded, such as neural and glial cell bodies, myelinated axons and blood vessels. To show that synaptic density per volume of neuropil rises or falls during development is not particularly informative, because both the numerator and denominator of the fraction can be expected to change significantly and independently of each other in early development. In the numerator, the absolute synapse number can rise or fall. In the denominator (the volume of neuropil), the size and number of spines, varicosities, dendritic inclusions (e.g., vesicles) can also change with age. Moreover, artifactual deviations could be caused by tissue shrinkage (a common event in brain tissue prepared for microscopic analysis). One possible outcome could be that the synapses/neuropil ratio might stay constant, with an increase in synapse number simply mirrored by an increase in its support structure, the neuropil.

Now, back to the question of development: Rakic and Huttenlocher have both shown that the ratio of synapses to neuropil accelerates wildly beginning just before birth, in both the macaque and the human, and across a wide variety of cortical areas. In macaques, the peak of synaptic density across cortical areas is reached two to four months after birth (Figure 4a—replotted from Bourgeois, Goldman-Rakic, & Rakic, 1994; Granger et al., 1995; Rakic et al., 1986; Zecevic et al., 1989; Zecevic & Rakic, 1991). In humans, the curves are very similar, with a marked perinatal increase in

synaptic density that begins around birth and flattens postnatally across all cortical areas (Figure 4b). It should be noted that synapse counts may, or may not, vary across different cortical regions. In the graph, for example, synapse counts in human auditory cortex appear to outnumber those in other human and macaque cortical regions. However, for the technical reasons noted above—and for other methodological considerations, including a possible variability based on the cause of death—absolute values of synapse counts should be considered somewhat conditional, especially in human tissue. Moreover, we have attempted to normalize the data by plotting synapse numbers as a percent of the total at puberty, which we arbitrarily defined as 12 years in human and 3 years in macaque. The "take home" message from the graph lies not in the absolute numbers, but rather in the pattern of *relative* changes. Huttenlocher's claim that synaptogenesis in prefrontal cortex does not reach its peak in humans until approximately 3-4 years of age rests on the accuracy of a single observation of frontal cortex at 3.5 years of age. However, the macaque data show similar variability, with primary visual cortex peaking latest. In other words, there simply are not enough data available at this time to settle the matter. Even if the Huttenlocher pattern does prove true, this is not the most interesting aspect of these events. The most interesting feature in both the macaque and the human data lies in the strikingly similar timing of acceleration and deceleration, not in the peak. To see why, let's look more closely at the broader schedule of synaptogenesis, before and after birth.

Remember that the ratio of synapses to neuropil is a ratio of moving targets. So to understand the development of synaptic connectivity, we need to understand what the denominator of the synaptic ratio (the amount of neuropil) is doing during this same period. Data are not available for humans, but in monkey cortex the relative proportion of neuropil soars from initially insignificant values around PC 50 (50 days post conception) to very high values at PC 100. This prenatal explosion in monkeys corresponds (according to the Finlay/Darlington/Clancy model) to PC 62 to 127 in humans, still well before birth. After that, the amount of neuropil remains constant at about 70 to 75% per section until about one year of life, followed by a long slow decline to a value of about 50% that is reached at some point well past puberty. Meanwhile, the whole brain is getting bigger. In macaques and marmosets, the volume of visual cortex (with comparable increases in both depth and surface area) overshoots its adult size by about 45% at six months of postnatal age, and then regresses to its adult volume. Overall brain volume increases from birth to adulthood by about a factor of two in monkeys, and by a factor of almost four in humans. Because we know that the size of some components like primary visual cortex declines across the same period, the overall

increase in brain size must be due to increases in the size of secondary and tertiary visual areas, nonvisual areas, as well as in the number of supporting elements and myelinated fibers in the brain. We can safely conclude that the generation of synapses in the entire isocortex of humans accelerates around birth, overshoots by a substantial proportion in the first six months or so, and then declines to its adult value. Although less data are available for noncortical regions, a similarly timed burst and decline of synaptogenesis occurs in the striatum (Brand & Rakic, 1984). Where the exact "peak" lies is probably not too important, as it will be influenced by any number of co-occurring additive and subtractive events. The important point is that the brain suddenly starts to generate massive numbers of synapses just before environmental experience, in all of its regions associated with sensory, motor, motivational, and linguistic ability.

What causes the dramatic perinatal acceleration of synaptogenesis? Using visual cortex as a test case, Rakic and colleagues looked into the possibility that this marked increase is actually caused by the barrage of experience that occurs around birth (Bourgeois & Rakic, 1996). However, when monkeys were deprived of visual input, the initial acceleration and peak of synaptogenesis were unchanged, though later events of changing proportions, cortical layering and so forth, did change markedly. O'Kusky (1985) tried a similar experiment with dark-reared cats, and also found no changes in the peak of synaptogenesis. However, one could argue that this kind of deprivation experiment is misleading, because the deprivation might induce a host of compensatory changes in other parts of the system. To control for this possibility, a second, "mirror image" experiment was conducted in which monkeys were delivered three weeks prematurely, so that the hypothetical barrage of experience would begin much sooner than it would normally occur (Bourgeois, Jastreboff, & Rakic, 1989). Again, there was no effect on the timing of synapse acceleration and peak—it occurred precisely when it should occur, based on the monkey's anticipated gestational birthdate, not the prematurely induced one. Secondary effects on types and distributions of synapses were also seen in this study, so experience does matter. However, experience doesn't seem to be responsible for the burst in synaptogenesis.

Humans present an evolutionary experiment that is the opposite of the premature delivery manipulation, because we are born so late with respect to many neural milestones (although we are still quite immature when we finally get around to being born). When we look at the relationship between synaptogenesis and birth in humans, we find a rare and rather exciting exception to the general laws of neural development that create such orderly similarities between humans and other mammals: synaptogenesis seems to occur much later in humans than it occurs in other primates, jumping forward several weeks ahead of the point where it would

occur in “macaque time.” Why should this be? If humans underwent an accelerated period of synaptogenesis at the maturational stage corresponding to the stage when macaques show rapid synaptogenesis, it would occur several weeks prior to birth (Figure 4b). And if this occurred, the human fetus would be in possession of a large reservoir of synaptic plasticity to contemplate the uterine wall! Which would of course be a terrible waste of resources. Timing of peak synaptogenesis to just precede the onset of experience can be seen in other primates (marmoset) (Missler, Eins, Merker, Rothe, & Wolff, 1993; Missler, Wolff, Merker, & Wolff, 1993), and in animals such as rats (Blue & Parnavelas, 1983), where eye opening occurs after birth (which essentially marks a similar transition from a dark, burrow-restricted environment to the external world). The peaking of synaptogenesis is the first instance we have found of a neural maturational event tied explicitly to birth, rather than to the intrinsic developmental timetable of the brain which can be quite dissociated from birth (Darlington et al., in press). Recent work has shown that it is a signal from the fetus that initiates labor, coordinating maturation in the fetus with physiology in the mother. It would be interesting if this same signal might also initiate wholesale neuroanatomical changes in the fetus itself (Nathanielsz, 1998). In any case, the bottom line for present purposes is this: experience does not cause the burst in synaptogenesis, but evolution has coordinated synapse production with birth. Why? Perhaps to guarantee a large reservoir of resources for all the learning that is about to occur. By providing a reservoir of already-formed synapses at the onset of experience in every part of the isocortex, the brain prepares itself for both expected and unexpected learning opportunities.

In fact, the number of synapses present are in excess of the eventual adult number (Zecevic & Rakic, 1991), and we spoke before about how the immediate postnatal phase of development is distinguished by regressive events like axon retraction and synaptic elimination. It has become clear, however, that it would be a mistake to view early development as a solely “regressive” period. In both intermediately aged and mature nervous systems, additive and subtractive events co-occur and overlap (Quartz & Sejnowski, 1997). Perhaps the developing nervous system is simply allowing itself the possibility of both additive and subtractive events, rather than simply additive ones, by the installation of large numbers of synapses just prior to experience. This initial “overproduction” of synapses may be a way of producing continuity in mechanisms of synaptic stabilization from initial development to adulthood.

Why on earth does nature bother to produce so many elements just to throw them away? The massive overproduction and subsequent pruning of synapses is an expensive neural tactic in terms of neural components and energy cost. Between ages 2 and 5, it has

been estimated that 5,000 synapses are disappearing each second in the visual cortex alone (Bourgeois, 1997), and similar recessions are most likely occurring in all cortical areas that participate in language. What purpose could this steady decline serve, especially occurring as it does in a period when details of language (including complex grammar) are mastered? The strategy of excess production followed by pruning has been documented in other neural areas, notably in callosal axonal connectivity, where it has been proposed to permit the neural adjustments that favor evolutionary changes (Innocenti, 1995). Certainly flexibility is a primary outcome of such a system, but refinement, defined in terms of accuracy and speed despite complexity, may be another important consequence of these regressive stages. Empirical studies are limited to observed descriptions of gross synapse counts, but computer simulations have been run that yield interesting information about the computational consequences of this peculiar strategy of overproduction and pruning (Elman et al., 1996). For one thing, in adaptively constructed neural networks that employ overproduction and removal of synapses, input information is more reliably preserved than it is in simple feed-forward networks (Adelsberger-Mangan & Levy, 1993, 1994). Networks constructed using adaptive synaptogenesis also manage to “sculpt” connections that permit quicker transformations of complex data when compared to networks constructed with conventional nonadjustable connective mechanisms. Moving away from machines back to humans, it is true that the net numbers of synapses are decreasing during adolescence; however, new ones are still sprouting, resulting in a constant and co-occurring process of production and trimming that could also serve to adjust and improve on initial connections.

So now let’s take a closer look at the various kinds of synaptogenesis that occur before and after birth, and also consider some of the local and global events that affect the learning potential (and perhaps the learning style) within and across brain regions. A summary of the timetable of synaptic stages can be found in Figure 5, in which milestones of language acquisition and production are mapped alongside sequences of some of the human neural events that are discussed in this section.

Developmental differences in synapse morphology and distribution. The sequence of synaptogenesis can be classified into five stages (reviewed in Bourgeois, 1997). In the initial stage, synapses are present in the preplate (later subplate and marginal zone) which comprises the earliest-generated cortical neurons. This is followed by a secondary stage in which synapses are generated in the cortical plate itself, initially following a gradient corresponding to that of the developing cortical neurons. Phase III of synaptogenesis is the synchronized global perinatal burst phase described above; at its peak in the infant

macaque, it is estimated that 40,000 synapses are formed each second in the visual cortex alone (Bourgeois, 1997). Phase IV is a stabilized high level that lasts from late infancy until puberty, while in the last phase, which extends from puberty to adulthood, synapses steadily decline in density and absolute number.

Variations in morphological characteristics of the third stage of proliferating synapses make it clear that the complexities of the synaptogenic peak extend beyond sheer numbers. There are also interesting development changes in the kinds of synaptic connections that are being made. This includes a change in the ratio of asymmetric to symmetric synapses during the perinatal period—recall that asymmetric synapses are more likely to be excitatory and symmetric inhibitory. During Phase III of synaptogenesis, the asymmetric (putative excitatory) connections decline in number while the numbers of symmetric (putative inhibitory) synapses remain about the same (Bourgeois & Rakic, 1993; Zecevic & Rakic, 1991). Functionally, this means that there may be a developmental shift from a high proportion of excitatory activation toward a more tempered balance between excitation and inhibition, which seems a plausible account of the increasingly better coordination of perception and action.

The sites of synaptic innervation are also altered over development (Zecevic & Rakic, 1991). Early in development (in the more exuberant phase), large numbers of connections are made (or attempted) on the shafts (the trunks and branches) of dendrites. Later there is a shift in contact site, with more connections on dendritic spines. Because spine contact may allow information to be transferred with more specificity than shaft contact (Harris & Stevens, 1989), this shift in site might reflect an increase in connectional efficiency during the early learning process. Theoretical models, as well as imaging experiments which can track ion flow in single cells, also support the role of spine contact in the induction of the plasticity associated with learning (reviewed in Koch & Zador, 1993). Overall, the significance of these changes in the constellations of the types and forms of synaptic interaction is just beginning to be understood. In the future, we may be able to track the changing functions and relative maturity of cortical areas by looking at constellations of varying synaptic morphology alone. Right now, we know that it is these kinds of things that are changing as function matures, without a very direct structure/function link.

So let us leave synaptogenesis and move on to other, larger-scale changes in brain structure and function that start prenatally but extend well into the postnatal period. We will return to the prenatal genesis of some of the patterns that will set up maturational gradients of different features of the cortex, and relate them to postnatal synaptogenesis. If we are looking for the developmental counterpart of “mental organs” somewhere in the brain, this is where we need to look.

Maturational gradients of the isocortex in the early postnatal period. Anyone hoping for fundamental simplicity in patterning of the early maturation of the cortex will be disappointed, but we will argue here that complexity is the message, and that each cortical area can be viewed as a point in a multidimensional space of various maturational gradients which could well provide the reason for later local functional specializations. There is not a single dimension called “maturational state” that any area of the isocortex can be retarded or advanced on (which makes it less likely there could be a moment when a region “turns on”). Rather, each isocortical area is best viewed as an assembly of different features, including neurogenesis, process and axon extension, neurotransmitter inclusion, type and rate of synaptogenesis. The simultaneous peaking of synaptogenesis across all cortical areas, supporting the notion of a global signaling process, is an exception to this general rule. Because different areas of the brain follow maturational gradients that don’t match in order, interesting temporal asynchronies are produced—for example, in some areas, intracortical connections will be relatively more mature than thalamic connections (the frontal cortex), and in others, the reverse will hold (primary visual cortex). Figure 6 contrasts the gradients that are observed in two critically different aspects of cortical development: the timing of peak neurogenesis in different cortical areas compared to peak neurogenesis of their corresponding thalamic nuclei, both superimposed on the cortex of a schematized human brain.

1. Intrinsic cortical gradients. The isocortex has its own gradient of maturation that affects the entire isocortical sheet (because that is what the cortex really is, despite its folds) and is quite conserved across all mammals. Bayer, Altman and colleagues have produced detailed studies of the timing of neurogenesis in rodents (Altman & Bayer, 1979a,b, 1988; Bayer, Altman, Russo, & Zhang, 1993) and we are able to apply the comparative mammalian model of Finlay and Darlington (1995) to predict a similar time sequence for humans. Neurogenesis begins at the front edge of the cortex where frontal cortex abuts inferotemporal cortex and proceeds back to primary visual cortex, framing a period of genesis that can last over 30 days in primates from front to back; in humans, this would lead us to expect a neurogenesis window of about 50 days extending from approximately PC day 42 to PC 92. The limbic cingulate cortices also get an early start, in humans beginning genesis about PC 43. The maturational edge then possessed by frontal and limbic cortex would then be expected to continue in various aspects of the intrinsic development of those parts of isocortex. More mature neurons begin to elaborate neuropil, and extend local and long-range connections, but there is little direct association between the time of a neuron’s genesis and when it makes its connections, as this also depends heavily on the maturational/trophic status of

the regions it must connect to. As depicted in the maturational gradient in Figure 6a paradoxically, the frontal cortex, viewed in hierarchical models as the last maturing cortical area, is in fact one of the first to be produced and thus quite "mature" in some features. This is one more example of the limits of the traditional hierarchical view of brain development, in which the frontal regions are mistakenly viewed as "late" on every dimension.

2. Imposed thalamic gradients. Each area of cortex receives a thalamic input by maturity, but the order of thalamic development in no way resembles the intrinsic cortical gradient (Figure 6b). This is very important, because as we noted earlier, it is the thalamus that carries instructions to the cortex from the sensory and motor periphery, dividing the cortical sheet into specific regions with specific jobs. If intrinsic cortical gradients and imposed thalamic gradients occur in different orders, then we have a dissociation with potentially very interesting consequences. In general, the primary sensory nuclei in the thalamus, including the ventrobasal complex (somatosensory), parts of the medial geniculate body (auditory), and the lateral geniculate body (visual) are generated first, and establish their axonal connections to the cortex first. Various other nuclei, motor and cingulate, are intermediate in their timing, and the last to be produced are the nuclei that innervate the frontal, parietal and part of the inferotemporal cortex. It is the thalamic order of neurogenesis that gives rise to the hierarchical notion of cortical development (e.g., "visual matures early; frontal matures late"), although this gradient is really not a general rule. So what might this mean for frontal cortex, the area that bears so much weight in speculation about human evolution (e.g., Deacon, 1997)? No one actually knows for sure (because the necessary electrophysiological data are not available), but the fact that frontal cortex matures relatively early (intrinsic gradient) but receives its input from the thalamus relatively late (thalamic gradient) might mean that frontal cortex specializes in intracortical communication (through working connections to other cortical regions). In other words, this difference in developmental gradients might mean that frontal cortex is primed for higher-order associative function from the start, not by virtue of being "out of the circuit" early on.

3. General modulatory cortical input. What we have talked about so far has revolved around the construction and interconnectivity of cortical and thalamic regions, i.e., the basic wiring diagram for passage of explicit information. There is another set of subcortical structures that project to the cortex that are deeply implicated in systems of arousal, attention and emotion in adulthood, and in modulation of plasticity and growth in development. Innervation across the different brain areas by the axonal fibers of these systems terminates in patterns specific to each area of the isocortex, but the axons originate from a relatively

small number of neurons in common subcortical structures. Cholinergic fibers arise from the basal forebrain (nucleus basalis), norepinephrine fibers from the locus coeruleus and the lateral tegmentum, serotonin fibers from the raphe nuclei, and dopamine fibers originate in the cells of the substantia nigra and ventral tegmental area, although some connections also originate from neurons intrinsic to the cortex (Parnavelas, Kelly, Franke, & Eckenstein, 1986; Parnavelas, Moises, & Speciale, 1985). During development, the long-range transmitter/modulatory systems are focused throughout the entire isocortex with the exception of the less diffuse dopamine system which focuses more specifically on limbic and prefrontal cortical regions. Unlike the precocious thalamic afferents, cholinergic and aminergic innervation begins relatively late in development, elaborating after birth, although there is some transient embryonic innervation (Dori & Parnavelas, 1989; Kalsbeek, Voorn, Buijs, Pool, & Uylings, 1988) at a time corresponding to the second trimester in humans. We really don't know what these systems are "for" developmentally, except that disturbance of them disturbs normal development. The whole picture of cortical development will have to include these systems in due course; we will briefly review what is known.

In rats, permanent cholinergic innervation begins around eye-opening, reaching adult levels by 1 month postnatal (Dori & Parnavelas, 1989); the serotonergic and noradrenergic timetables are similar (Lidov & Molliver, 1982). Dopamine innervation also begins postnatally but does not achieve mature patterns until full adulthood (2 months postnatal, Kalsbeek et al., 1988). Because we can reasonably conclude that the timing of this innervation, like other neural events, is conserved across species, we can extrapolate these innervation dates to begin in the second or third trimesters of human gestation, likely extending well into the first postnatal year and, for dopamine innervation, into the adolescent years.

What might the protracted and largely postnatal innervation of these neural fibers mean to a human infant or toddler in the process of learning so many behaviors, including language? We know that that these connections transmit substances that are highly implicated in mechanisms of arousal and reward, but it appears that these substances, either alone or acting in combination with other chemicals, can have various and even contradictory effects. For example, acetylcholine, a deficit of which is associated with Alzheimer's disease, can have both excitatory and inhibitory functions in the brain. Norepinephrine is conventionally believed to increase activity and attention, but it too can have inhibitory effects. Serotonin is well known for its role in abating depression, but functions vary; low levels of serotonin have been implicated in aggression and it may also be a factor in the perception of pain. The dopamine system, highly concentrated in the prefrontal

cortex, is believed to participate in aspects of cognition and is also a primary factor in many drug actions, including amphetamines. It is also associated with schizophrenia and Parkinson's disease. It would seem that the progressive innervation of these substances into the developing brain is timed so that they can optimally influence learning behaviors, but much additional research will be needed to tease out a distinct role for each.

Neurochemicals and receptors. The transmitters used in the systems described above are just a small number of the chemicals that can be set in motion from the presynaptic (or launching) side of the synapse. About 20 neurotransmitters (which have rather strict classification criteria) have already been identified (glutamate and GABA are high-profile examples) and many more are under investigation. These include neuromodulatory peptides such as cholecystokinin (CCK) as well as the compound nitric oxide (NO), highly implicated in development. Upon maturity, the neural areas subserving functions associated with language will contain unique combinations of neural transmitters and modulators in distribution patterns distinctive to each area, a form of neural "fingerprint". Some substances arrive from cells located outside the cortex; some are synthesized "on site." Unlike fingerprints, however, synthesis and distributions of these neural substances change over the course of maturity (Goldman-Rakic & Brown, 1982; Hayashi, Yamashita, Shimizu, & Oshima, 1989; Hornung & Fritschy, 1996) making them strong candidates for roles in development. Although much research remains to be done, it is certainly likely—given the timing of the fluctuations and combinations—that these variations in neural substances may play a functional role in maturing behaviors such as language learning.

Neural receptors are the other side of the synapse—the gating (or docking) portion of the postsynaptic complex where neurotransmitters and modulators can exert influence on the cells they contact. One developmental alteration has been consistently documented regardless of the species, the cortical area under investigation, or the related neurosubstance: there is a dramatic overproduction of virtually every type of receptor which occurs around the time of birth (Gremo et al., 1987; Herrmann, 1996; Hornung & Fritschy, 1996; Lidow, Goldman-Rakic, & Rakic, 1991), similar to—and simultaneous with—the perinatal surge of synaptogenesis. The receptor surge greatly supports the notion that escalating phase three synapses described earlier are functional and so likely to participate in developing learning processes. One receptor type in particular, the N-methyl-D-aspartate (NMDA) receptor, routinely implicated in learning and development, may actually influence formation of new synapses (Aoki, 1997; Brooks, Petit, LeBoutillier, & Lo, 1991). The appearance of an NMDA subunit, as well as some other transmitter-related substances, even precedes initial

phases of synaptogenesis (Aoki, 1997; Zecevic & Milosevic, 1997). Interestingly, not all receptors are located on the postsynaptic cell. Neurons also employ an apparent self-monitoring tactic—they contact themselves, so some receptors are located on the cell body of origin. Similar to the many other events we have described in the developing brain, interactions between receptor formation, neurosubstance synthesis, and synaptogenesis are likely to be more complicated than any simple cause-and-effect mechanism.

3) Interactions of neural events and language learning

The picture of human brain development that we have provided here leaves little room for a lockstep table of correlates between language milestones and neural events, but it does provide some useful constraints on how we should conceive of this complex bidirectional relationship, with implications for both normal and abnormal development. In an effort to integrate these ideas about neural development with the behavioral events reviewed earlier in this chapter, we close with four conclusions, or better yet, four working hypotheses to guide future research in this area: (1) readiness for learning, (2) experience-driven change, (3) rethinking two specific postnatal correlates of language, and (4) sensitive periods.

(1) Readiness for learning. There was a period in developmental psychology when the capacities of the newborn infant for perception and learning were vastly underestimated. Much-needed correctives to this misunderstanding have come in two waves: research demonstrating rich perceptual skills in the first few weeks of life (e.g., Bertenthal & Clifton, 1998; Johnson, 1998; Kellman & Banks, 1998), and research demonstrating at least some learning *in utero*, as well as a capacity for rapid learning of arbitrary statistical patterns (including language-specific phonetic details) in the first months of life. With the first wave, there was extensive speculation in the literature on infant development regarding the stock of innate knowledge that infants must possess in order to perform so well in (for example) tasks that require response to complex transformation of objects, including their disappearance and reappearance (Spelke, 1994; Spelke, Breinlinger, Macomber & Jacobson, 1992; Spelke & Newport, 1998). With the second wave, it has become increasingly evident that we have underestimated the power and speed of learning even in very young infants, forcing a reevaluation of the extent to which infant performance is influenced by learning vs. innate perceptual, motor and perhaps even conceptual biases about the nature of the physical and social world (Elman & Bates, 1996; Seidenberg, 1997; Thelen & Smith, 1994, 1998). The material that we have reviewed in this chapter provides support for the idea that the infant brain is up and running at or before birth. The fundamental scaffolding is already in place for learning, and the newborn brain is capable of enough thalamo-

cortical and intracortical communication to permit the acquisition of distributed patterns within and across modalities (e.g., visual-auditory mapping, visual-tactile mapping, auditory-motor mapping, and so forth). Nor do we see any evidence for the hypothesis that whole bounded regions of the brain are “pre-functional”, quiescent, inactive, waiting for some key maturational event before they can “turn on” in the postnatal period.

Of course the newborn still has a lot to learn. It will take weeks or months before she has cracked the speech code for her particular language, and even longer before that code can be mapped in a systematic and productive way to extract and express meaning. Postnatal changes in the brain may make learning more efficient (or less efficient—see sensitive periods, below), and these events will certainly have computational consequences for the kinds of learning and reasoning that the child can carry out. But the capacity for learning is there from the beginning of postnatal life. Future studies of brain and behavioral development may want to focus on postnatal changes in neuroanatomy and neurophysiology as they relate to the nature and efficiency of language learning and language use, including the kinds of planning the child can carry out, and the amount and kinds of memory and attention that a child can deploy in a given task. All the relevant neural systems may be in place and ready to work from the beginning, but that does not mean that they are being used in an adult manner, nor that they are being used in all the tasks for which they will eventually be relevant. An area may “turn on” at some point in postnatal development not because it has finally attained the necessary wiring, or the necessary ratio of neurochemicals, but because the child has finally figured out how that area can and should be employed within a given task. A reorganization in the way that the brain is used (as a result of learning) is not the same thing as a reorganization that is caused by the sudden appearance of a new player. Most classic “insight” experiments make this assumption—if a chimpanzee seems to stack boxes to get bananas, it is not usually assumed a new box-stacking-banana area has suddenly turned on, but, rather, that existing elements have been recombined to solve the task. In language development, we might ask how such a process could occur at a nonconscious level. Recent neural imaging studies of human adults have shown that the configuration of highly active areas changes markedly across a 20-minute period as the subject attains expertise in a new task (Petersen, van Mier, Fiez, & Raichle, 1998). If that is true for mature and sophisticated adults, over a very short period of time, it will undoubtedly prove true for children who are in the process of acquiring language for the first time.

(2) Experience-driven changes. It is now clear that learning itself contributes to the structure of the developing brain, in infants and in adults. Any number of studies have shown that various aspects of the experience presented to an animal will profoundly

alter the time course of synapse elimination, and that experience will also produce increases in synapse numbers (although it does not seem to affect the first fast phase of synaptogenesis, which appears to be locked in to coincide with birth). Particular clear examples of an experience-dependent increase can be found in a series of experiments by Greenough and colleagues examining the effects of enriched housing and/or skill learning on morphological changes in rodent brain. These studies have consistently documented significant increases in dendritic fields and in the ratio of synapses per neurons in rats exposed to complex environments or involved in learning tasks when measured against handled controls (Black, Isaacs, Anderson, Alcantara, & Greenough, 1990; Greenough, Hwang, & Gorman, 1985; Turner & Greenough, 1985). Experience-based synaptogenesis is also accompanied by increases in populations of supporting cells such as astrocytes and oligodendrocytes (Sirevaag & Greenough, 1987), as well as by increases in metabolic activity as measured by mitochondria volume (Sirevaag & Greenough, 1987) and vasculature branching (Sirevaag, Black, Shafron, & Greenough, 1988). We may reasonably conclude that similar reactive neural changes accompany learning in the developing human brain—indeed, although existing human data must be interpreted with caution, post mortem analysis of human adult brains indicates a correlation between high levels of education and increased dendritic branching in Wernicke’s area (Jacobs, Schall, & Scheibel, 1993).

Hence, if we do eventually find evidence for neuroanatomical and neurophysiological events that correlate with milestones in language development, we must be open to the possibility that these correlations are the product rather than the cause of language learning. In the same vein, if we find evidence of neuroanatomical and/or neurophysiological differences between children who are developing normally and children who are substantially delayed in language learning, we should not assume that this neural indicator has caused a language delay. It is equally possible (in the absence of evidence to the contrary) that a particular neural correlate of language impairment reflects the behavioral state of the system. That is, the brain may still be in a relatively immature state because the relevant experience-driven events have not yet taken place. This insight certainly applies to the burgeoning literature on neural correlates of Specific Language Impairment and/or congenital dyslexia, and it may apply to other disorders as well.

(3) Rethinking two postnatal correlates of language. In our earlier review of early language development and its neural correlates, we underscored two potentially interesting postnatal correlates of major language milestones: changes in frontal lobe activity that seemed to coincide with the 8-10-month watershed in comprehension, communication, imitation and reasoning, and changes in synaptic density that seemed

to coincide with bursts in vocabulary and grammar between 16-30 months. We would now like to put these correlates in a different light.

The idea that behavioral events late in the first year of life are correlated with changes in frontal lobe function rested primarily on two sources of evidence. The first (cited earlier) is a positron emission study (PET) of human infants suggesting that there is a marked increase in frontal lobe metabolism starting between 9-12 months postnatal age (Chugani, Phelps, & Mazziotta, 1987). Because these studies were taken in a resting state (when infants were sedated or asleep), and not in response to any particular stimulus or task, Chugani suggested that the sharp increase in glucose metabolism might be caused by a burst in synaptogenesis of the sort postulated by Huttenlocher and Rakic, reflecting a structure-to-function gradient (i.e., more synapses = more glucose metabolism). The second source of evidence comes from lesion studies showing that infant monkeys with bilateral frontal lobe lesions behave very much like age-matched normal controls until a critical point in development (roughly equivalent to 8-12 months in postnatal human life) when normal animals learn to solve short-term memory tasks that are failed by the lesioned animals and by adults with frontal lobe pathology (Diamond & Goldman-Rakic, 1989; Goldman-Rakic, 1987; Pennington, 1994). Findings like these have led to the hypothesis that the frontal lobes “come on line” around 9 months of age, coinciding in humans with dramatic changes in many aspects of language, cognition and social interaction. However, it now seems very clear that the frontal lobes are functional (though still immature) by the end of the second trimester, and may actually be *more* mature than other areas in terms of their intracortical connectivity.

How can we reconcile these apparently contradictory claims? The resolution may lie in the difference between *absolute functionality* (i.e., whether or not an area is working at all) and *task-specific functionality* (i.e., whether the organism has reached a state in which that area is recruited and activated for a given task). Evidence for the latter view comes from another finding by Jacobs, Chugani and colleagues (Jacobs et al., 1995), a positron emission tomography study of infant monkeys that shows high levels of frontal lobe metabolism at birth, well before the point at which monkeys solve the short-term memory tasks that have been associated with frontal lobe function. These authors do find a further increase in metabolism later on, in many regions of the brain including the frontal lobes, compatible with the idea that metabolism and synaptogenesis increase together after birth. However, the amount of activity seen in the frontal lobes of newborn monkeys is not compatible with the standard view that frontal lobes develop especially late. If Goldman-Rakic’s classic findings are not “caused” by the sudden appearance of mature frontal cortex, how can

we explain the sudden relevance of frontal lesions for memory tasks around the human equivalent of 8-10 months of age? We suggest that these results can be reinterpreted with the bidirectional framework that we have recommended here, in which areas are *recruited* into complex tasks across the course of learning. On this argument, normal infants (humans and monkeys) cannot succeed in so-called frontal lobe tasks until they have made enough progress (perceptual, motor, mnemonic) to realize that a new set of strategies is required—strategies that are, in turn, only possible with the involvement of the frontal lobes. We tentatively suggest that the 8-10-month behavioral watershed in human infants may involve a learning-dependent change in social and cognitive systems that have developed in parallel because they began in parallel (at or before birth), are roughly similar in complexity, and may also be in communication with each other. As a result, all of these systems reach a certain critical level of organization around the same time (approximately 8-10 months). At this point, in each of these behavioral domains, frontal lobe regions that are particularly well suited for short-term memory and planning are recruited. Individuals with damage to the relevant frontal regions may be at a significant disadvantage at this point (particularly if the lesions are bilateral), resulting either in developmental arrest or in a significant slowing of progress in language, cognition and communication.

The hypothesized parallel between synaptogenesis and the correlated burst in vocabulary and grammar that are observed from 16-30 months requires more re-characterization still. It is now reasonably clear that the initial burst in synaptogenesis itself is independent of experience, arranged to coincide with the barrage of experience that will arrive at birth. Debates about the timing and location of the peak (or peaks) in synaptogenesis might continue to rage, but they are based on too few samples for us to reach any kind of empirical resolution. Predictions based on cross-species modelling suggest that the human peak is probably reached around 6 months after birth, holding at a plateau for an undetermined period of time and declining slowly into the second decade of life. At this point, that is about all that we can say about this particular set of events. It is intriguing that the peak and plateau of synaptogenesis in humans brackets the primary events in early language development (from word comprehension at 8 months to the mastery of fundamental aspects of grammar, including complex syntax, by 3-4 years). Whether the perinatal burst of synaptogenesis represents an uninstructed, generic set of neural possibilities, such as a neural net set up with random connections, or a richly patterned, prescriptive set of connections, we simply do not know at this point—the reader is asked to project his/her own biases onto this reasonably neutral fact.

Is there any possibility that we should rule out? In our view, it would be wise to rule out the idea that the

“vocabulary burst” and the “grammar burst” depend entirely on synaptogenesis for their shape and size, because such bursts are also observed when learning occurs in a nonlinear dynamical system with a stable architecture (Elman et al., 1996). Such exponential bursts are characteristic of learning, and are observed whether or not they are superimposed on a burgeoning brain. Hence the compelling parallel between the “language burst” and the “synapse burst” may represent a mutually beneficial relationship, but not a crucial and direct relationship of cause and effect.

(4) Sensitive periods. The term “sensitive period” is preferred by neurobiologists over the widely used and widely misunderstood term “critical period”, because the former term implies a softer and more plastic set of developmental constraints and transitions. The term “critical period” is still used in the literature on language development, and it is often used to imply hard boundaries and a crisp dissociation in the mechanisms that are responsible for language learning in children vs. adults (for discussions, see Bates, 1999; Bialystok & Hakuta, 1994; Elman et al., 1996; Johnson & Newport, 1989; Oyama, 1992; Weber-Fox & Neville, 1996). The notion of a critical period for language has been invoked to explain differences between first- and second-language learning, and to account for age-related changes in recovery of language abilities following left-hemisphere injury. It has been shown that adults and children perform at similar levels in the early stages of second-language learning when learning conditions are controlled (Snow & Hoefnagel-Hohle, 1978). The one compelling exception to this general rule is the ability to learn a second language without an accent, which seems to elude all but a very rare subgroup of talented adults. However, studies that focus on the later stages of language learning have shown that adults tend to “fossilize” at a level below native speakers, while children generally go on to acquire full competence (Johnson & Newport, 1989). Results like these provide support for the idea that there is an age-related decrease in plasticity for language learning, but there is no consensus about the shape of this function or its cause. For one thing, the shape of the drop-off in second-language learning seems to vary markedly depending on the aspect of language that is measured (with declines that start in periods varying from 2 years of age to late adulthood). This is true not only across domains (e.g., an earlier decline for accent-free phonetic production than for various measures of grammar), but within domains as well (e.g., a different age-related decline for each and every rule of the grammar—Johnson & Newport, 1989). Results like these have led many investigators to conclude that there is no “single moment” when the window of linguistic opportunity slams shut, but rather, a series of gradients that vary with task difficulty and other poorly understood parameters.

A similar story has emerged for the age-related decline in recovery from unilateral brain injury. For example, Goodman and Yude (1996; see also Bates et al., 1999) have compared performance on verbal and nonverbal IQ tests for adults who suffered from unilateral injuries to the right or left hemisphere at different points from birth to adolescence. To their surprise, both of their outcome measures showed a significant U-shaped relationship to age of lesion onset: the worst outcomes were observed in patients whose lesions occurred between 1-5 years of age; for patients with congenital lesions and for patients whose lesions occurred between approximately 6-12 years, verbal and nonverbal IQ were both within the low-normal range. This kind of nonmonotonic relationship between age and outcome is not predicted by anybody’s theory of critical periods.

Does the literature on brain development shed light on this issue? In the well-studied primate visual system, multiple overlapping sensitive periods have been identified (Harwerth, Smith, Duncan, Crawford, & von Noorden, 1986) and it is likely that human language acquisition is affected by similar complex receptive intervals. Although these learning periods were once thought to be fixed in time, it is now clear that the temporal windows when adequate experience is necessary for proper development are more flexible than previously assumed, and may be retarded or advanced by natural or empirical means. For example, the sensitive vocal learning period for songbirds can be extended or delayed by environmental factors (Kroodsma & Pickert, 1980). Songbirds that hatch in the beginning of the summer normally learn their songs in a restricted 2-3-month period following birth, during which they are exposed to a number of adult song types and long daylight hours, but birds born late in the summer experience shorter daylight periods and greatly reduced song-learning opportunities. Kroodsma found that a reduction of either the length of the photoperiod or the amount of song exposure could delay the critical song learning period—adaptively, the delay lasts until the following spring when the later-born birds have another opportunity to learn adult songs. Sensitive periods in mammals are also affected by activity; dark-rearing will extend visual sensitive periods in cats (Stryker & Harris, 1986).

Links between cortical architecture and plasticity during these sensitive periods have been tested in mammalian cortex by Shatz and colleagues who investigated the distribution patterns of the NMDA R1 subunit, which varies during normal visual cortical development in correlation with the periods of axonal growth that form ocular dominance columns (Catalano, Chang, & Shatz, 1997). Interestingly, visual deprivation through dark-rearing did not alter the developmental time course or normal laminar distribution pattern of the NMDA R1 subunit in visual, auditory or somatosensory cortices. The failure of visual experience to

modify the normal time course of this receptor is similar to the synaptogenesis surge that is also unaffected by lack of visual experience (Bourgeois & Rakic, 1996). However, when all visual activity was blocked with the sodium channel blocker tetrodotoxin (dark-rearing does not block spontaneous firing of retinal ganglion cells), the distribution of the NMDA R1 subunit was drastically altered, implying that activity during sensitive periods is related to underlying physical changes in the developing neural architecture.

We must also be prepared for the possibility that learning itself affects the subsequent ability of the brain to learn something new. We noted that infants have developed a preference for the vowel contrasts in their language by six months of age; around 10 months of age, they start to suppress phonemic contrasts that are not in their native language. In order for the child to “tune in” to the contrasts that she will need, it seems that she must “tune out” contrasts that play no clear predictive role. We know that a similar process is played out on the neural level: like inputs are sorted together, unlike inputs are pushed apart, and unsuccessful connections are eliminated or drafted into some other task. Now, consider the following statistics: assuming a taciturn Calvinist family in which an English-speaking child hears approximately 5 hours of speech input per day (from himself or others), at a mean rate of 225 words per minute, the average 10-year-old child has heard 1,034,775,000 English phonemes (at an average of 25,869,375 trials per phoneme). She has heard just under 250 million words (including 17,246,250 renditions of the most common function word) and 28 million sentences, with more than 6 million past-tense verbs and 12 million plural nouns. In short, she has had many many opportunities to entrench the most frequent elements of her native language. The numbers double by 20 years of age (assuming this Calvinist child does not develop a predilection for the telephone during adolescence). Under these conditions, we should not be surprised that it is hard to reorient the entire system and acquire a similar level of competence in a second language. Brain maturation affects experience, but experience returns the favor, altering the very structure of the brain. Hence the putative critical period for language (which really comprises many overlapping sensitive periods) may be one more example of the bidirectional events that have been the focus of this chapter.

The search for a neuroanatomical basis for language learning has, at this time, no unequivocal conclusion. We have noted here some neural developmental alterations that accompany language milestones. These neural events may drive, or, alternatively, reflect developmental behaviors such as language learning—although the complexity of the interactions remains to be researched. What is clear is that timetables for human neural developmental events cannot be simply mapped onto sequences of language

acquisition and production. As depicted schematically in Figure 5, the human brain develops as an overlapping and inter-connected series of multimodal additive and regressive neural events, many of which are completed prior to birth. Although certain cortical events, especially developmental modifications in the numbers, components, and locations of synapses, may contribute somewhat more directly, all pre- and postnatal events should perhaps be considered essential to the language-learning process.

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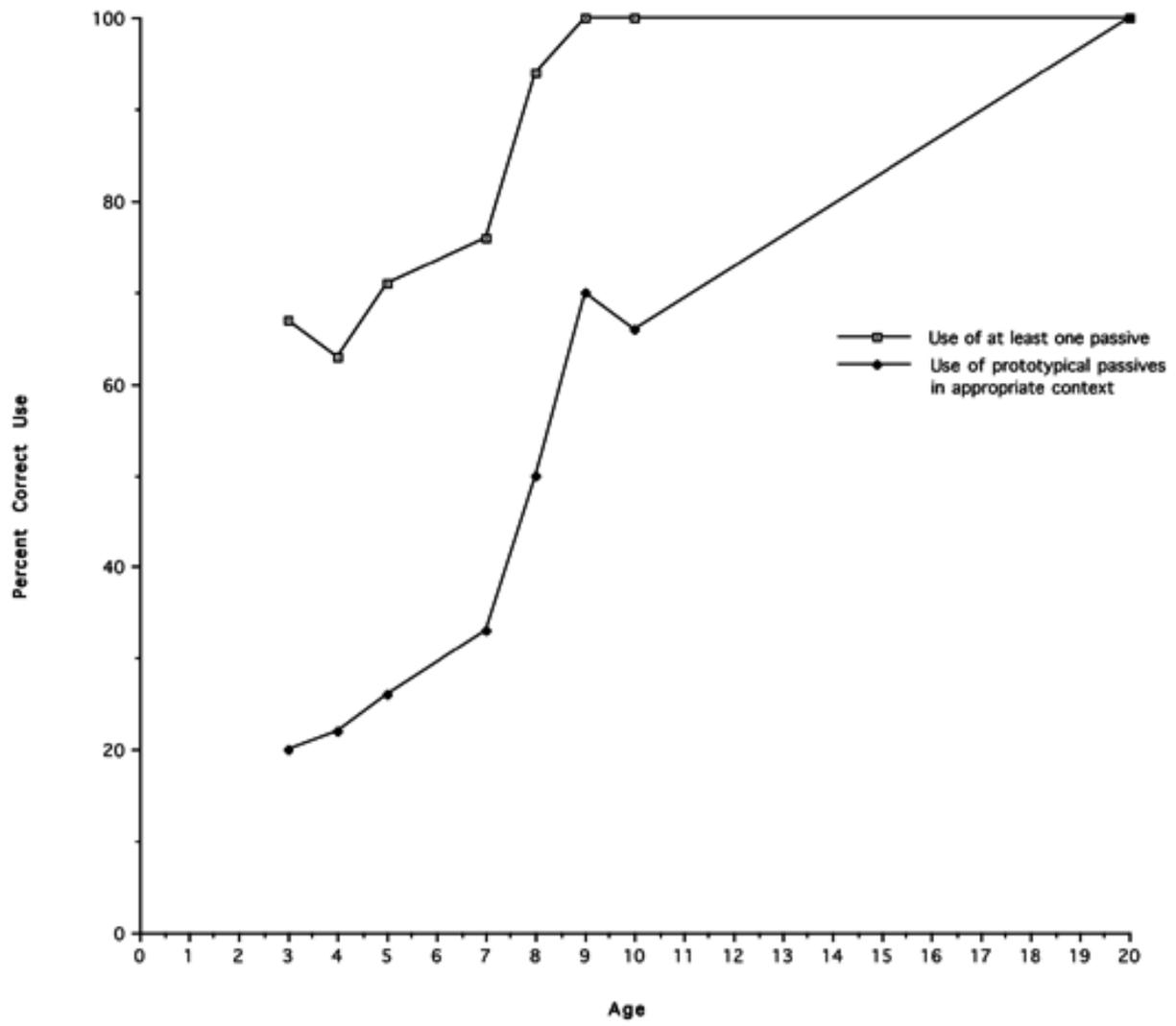
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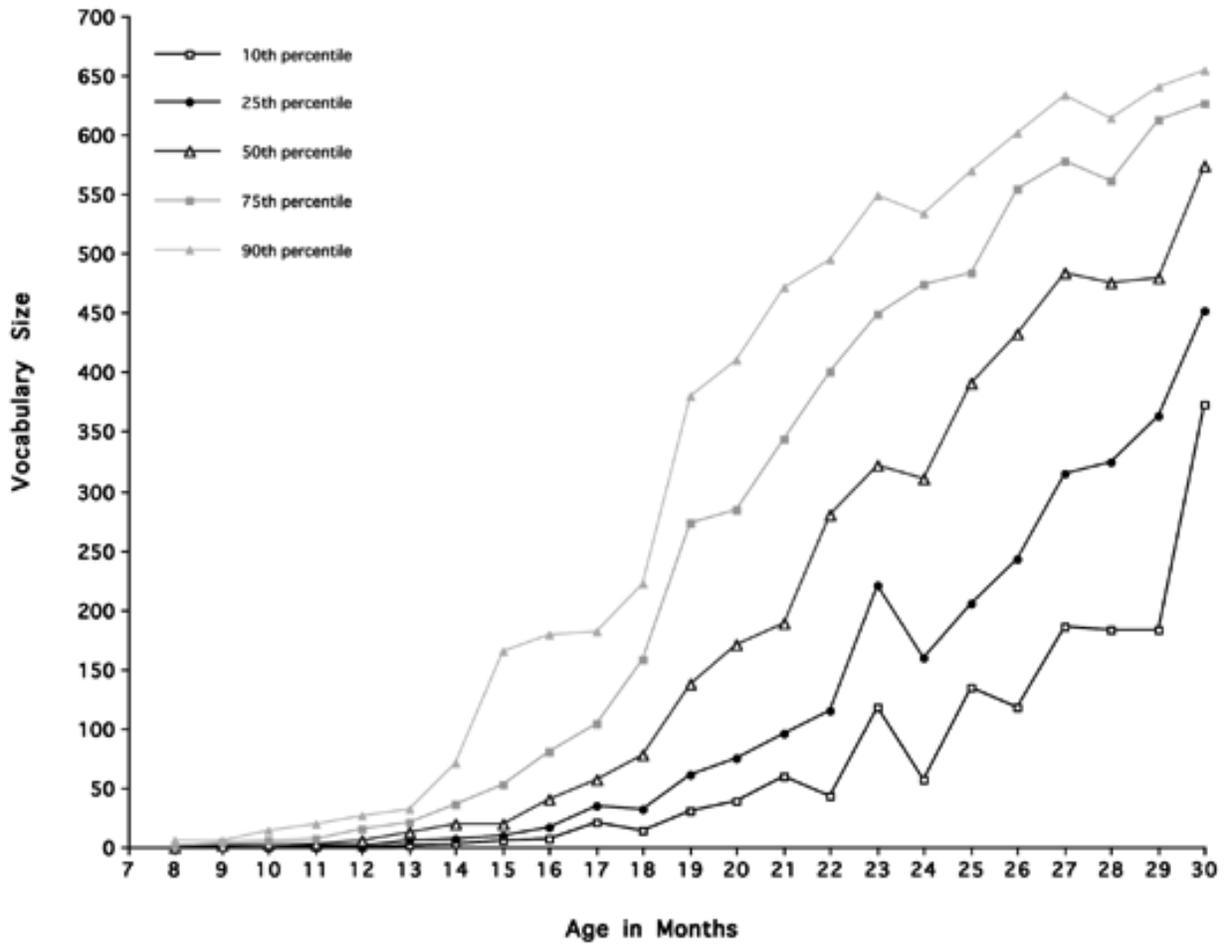
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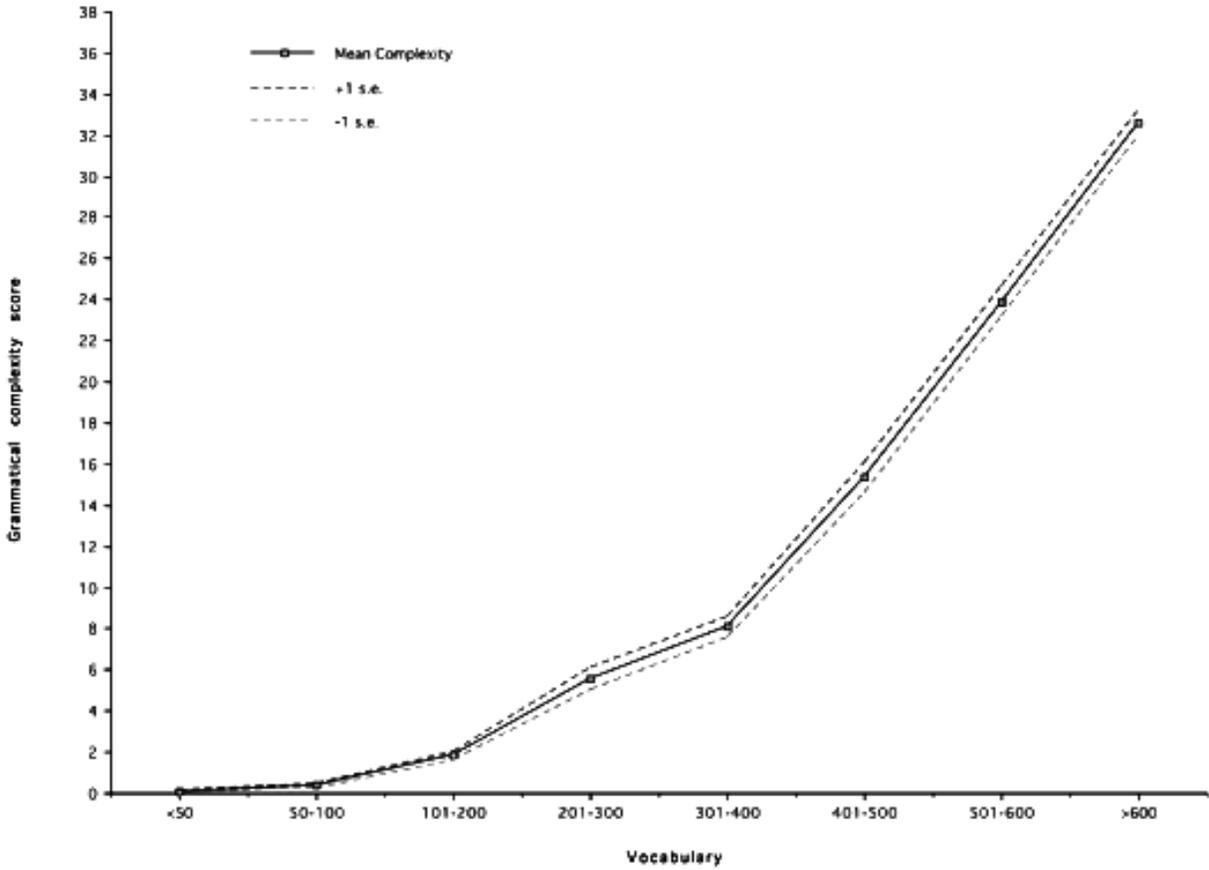
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Knowledge versus Accessibility of Passive Forms

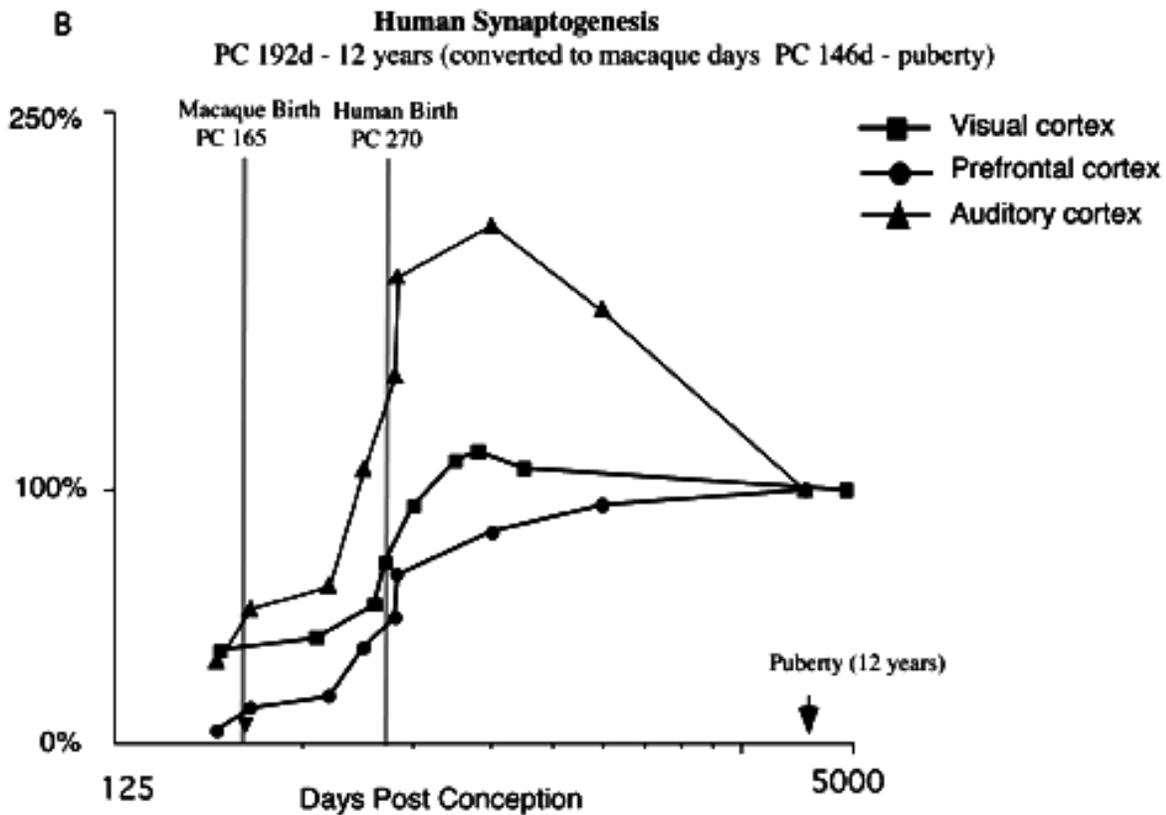
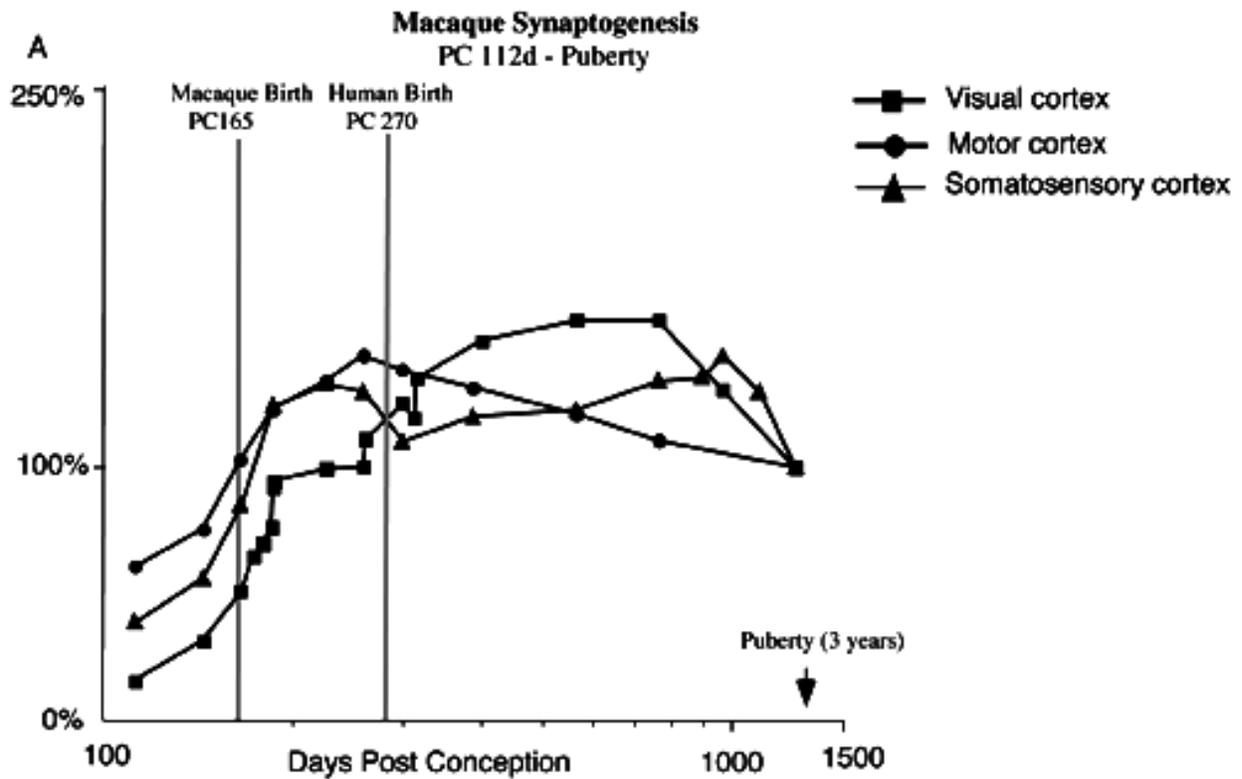






Mean and standard errors for grammatical complexity in children at different vocabulary levels

Figure 4



Neural Events and Language Development

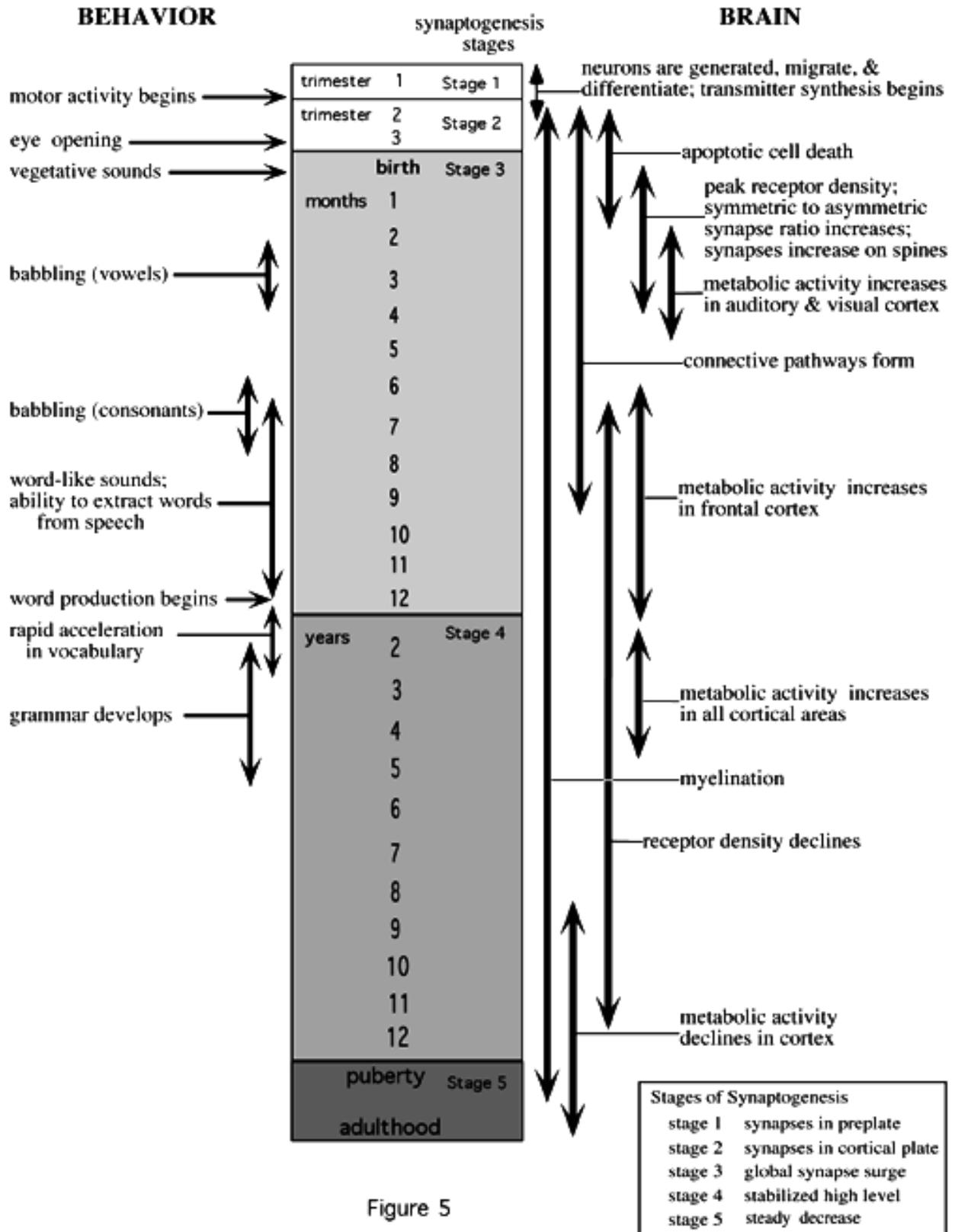


Figure 5

Figure 6

GRADIENTS OF NEUROGENESIS

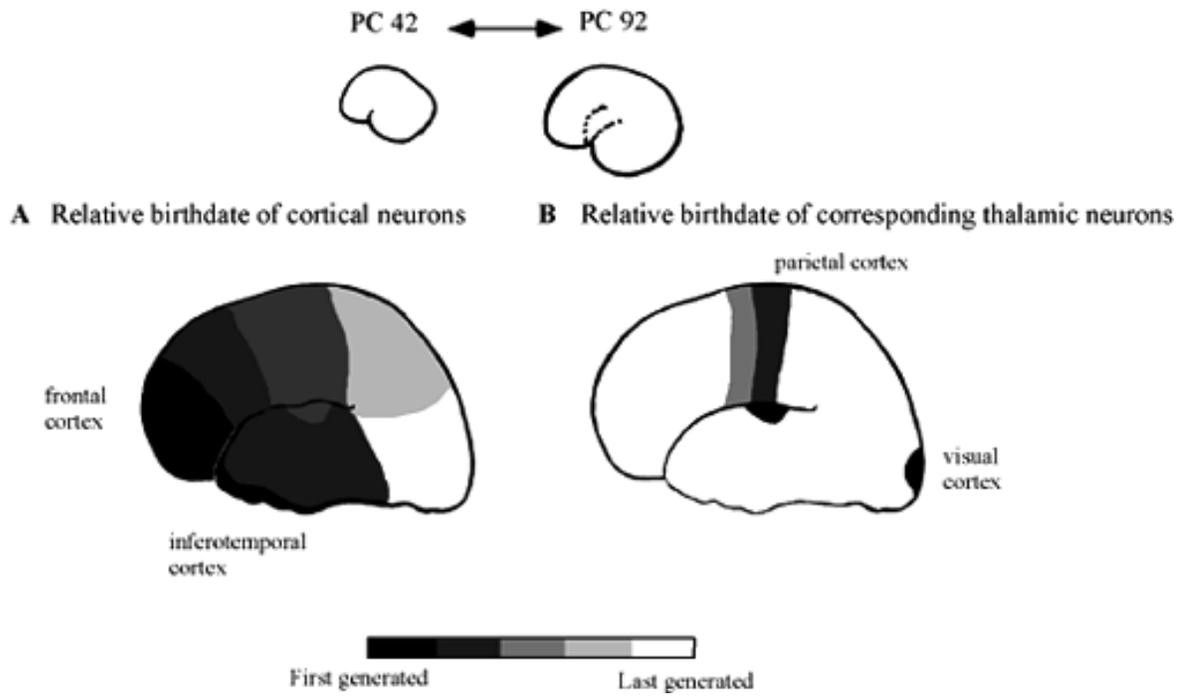


TABLE 1:

PREVERBAL TO VERBAL DEVELOPMENT

| <u>AGE IN MONTHS</u> | <u>SOUND</u> <i>(Perception)</i> | <u>SOUND</u> <i>(Production)</i> | <u>MEANING</u> <i>(Conceptual Content)</i> | <u>INTENTIONALITY</u> <i>(Social & Non-social)</i> | <u>CODING CAPACITY</u> <i>(Imitation and memory)</i> |
|----------------------|--|---|---|--|---|
| 0 | All speech contrasts can be heard | Vegetative sounds. | Object detection. | Innate signals (smiles, cries, etc.). | . |
| 1 | | | | | "Pseudo-imitation" |
| 2 | vocal/facial matching | Cooing, babbling without consonants | Changes in complexity of pattern detection and pattern anticipation | Passive anticipation of actions by others. | Anticipates position of object in a moving visual display. |
| 3 | | | | | |
| 4 | | | | | |
| 5 | language-specific vowel prototypes | Canonical or reduplicative babble with consonants | Changes in complexity of pattern detection and pattern anticipation | Joint attention to objects; objects and people are familiar goals achieved with familiar means. | Ability to retrieve a hidden object at zero delay, if obstacle can be easily removed. |
| 6 | | | | | |
| 7 | | | | | |
| 8 | | | | | |
| 9 | Loss of sensitivity to non-native speech contrasts begins. | Word-like sounds. | Object categorization. | First signs of tool use; novel means to familiar ends; humans as tools to objects; objects as tools to human interaction | True imitation. Ability to retrieve a hidden object after delay of up to 15 sec. |
| 10 | | | | | |

TABLE 2:

LANGUAGE MILESTONES AND NONLINGUISTIC CORRELATES

| <u>LANGUAGE MILESTONE</u> | <u>AGE OF ONSET</u> | <u>NONLINGUISTIC CORRELATES</u> | <u>VARIATIONS IN STYLE</u> |
|---------------------------|---------------------|--|-----------------------------|
| Word Comprehension | 8 to 10 months | Tool use, deictic gestures, gestural routines, causal understanding, shifts in categorization. | Word vs. Intonation |
| Word Production | 11 to 13 months | Recognitory gestures in symbolic play, deferred imitation. | Referential vs. Expressive |
| Word Combinations | 20 to 24 months | Gestural combinations in symbolic play, shifts in categorization, changes in patterns of block building, gestural combinations in motor and social play. | Nominal vs. Pronominal |
| Grammar | 28 months | Active sequencing in spontaneous symbolic play. | Noun Lovers vs Noun Leavers |