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On the Nature and Evolution of the Neural Bases of Human Language

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ABSTRACT The traditional theory equating the brain bases of language with Broca's and Wernicke's neocortical areas is wrong. Neural circuits linking activity in anatomically segregated populations of neurons in subcortical structures and the neocortex throughout the human brain regulate complex behaviors such as walking, talking, and comprehending the meaning of sentences. When we hear or read a word, neural structures involved in the perception or real-world associations of the word are activated as well as posterior cortical regions adjacent to Wernicke's area. Many areas of the neocortex and subcortical structures support the cortical-striatal-cortical circuits that confer complex syntactic ability, speech production, and a large vocabulary. However, many of these structures also form part of the neural circuits regulating other aspects of behavior. For example, the basal ganglia, which regulate motor control, are also crucial elements in the circuits that confer human linguistic ability and abstract reasoning. The cerebellum, traditionally associated with motor control, is active in motor learning. The basal ganglia are also key elements in reward-based learning. Data from studies of Broca's aphasia, Parkinson's disease, hypoxia, focal brain damage, and a genetically transmitted brain anomaly (the putative "language gene," family KE), and from comparative studies of the brains and behavior of other species, demonstrate that the basal ganglia sequence the discrete elements that constitute a complete motor act, syntactic process, or thought process. Imaging studies of intact human subjects and electrophysiologic and tracer studies of the brains and behavior of other species confirm these findings. As Dobzansky put it, "Nothing in biology makes sense except in the light of evolution" (cited in Mayr, 1982). That applies with as much force to the human brain and the neural bases of language as it does to the human foot or jaw. The converse follows: the mark of evolution on the brains of human beings and other species provides insight into the evolution of the brain bases of human language. The neural substrate that regulated motor control in the common ancestor of apes and humans most likely was modified to enhance cognitive and linguistic ability. Speech communication played a central role in this process. However, the process that ultimately resulted in the human brain may have started when our earliest hominid ancestors began to walk. Am J Phys Anthropol 45:36–62, 2002. © 2002 Wiley-Liss, Inc.

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BRAIN-BEHAVIOR MODELS

The data base

The major problem that has vexed studies of the evolution of language is that human beings are presently the single living species to possess complex linguistic ability. The archaic hominids who may have possessed intermediate stages of linguistic ability are extinct. While evidence from genetics and comparative anatomy shows that living apes appear to retain many of the features that characterized the common ancestor that they shared with humans, apes clearly do not have the ability to acquire all aspects of human linguistic ability. The apparent gulf between the communicative abilities of living apes and human language has led to the claim that the neural bases of human language are disjointed from those involved in vocal communications of apes (e.g., Burling, 1993), and to incorrect assertions that apes lack any vestige of linguistic ability (e.g., Terrace et al., 1979; Pinker, 1994). This in turn leads to theories that postulate abrupt discontinuities, or "stages," in the evolution of human language such as "protolanguage" that lacked syntax (Bickerton, 1990), or to claims that language could have not have evolved by means of Darwinian processes (Chomsky, 1976, 1986).

However, these difficulties can be surmounted by applying the principles and techniques of evolutionary biology. Comparative studies, which we will note, clearly show that human language shares many primitive features with the communications systems of other species. By identifying the derived aspects of human linguistic ability that differentiate it from related species, we can focus our attention on identifying and tracing the evolution of the biological substrate that yielded human language. And advances in comparative neurophysiology have generated insights into the biology of language. Therefore, though the absence of the intermediate stages of the evolution of hominid linguistic ability places limits on our ability to ever fully understand how and when human language evolved, the findings of recent neurophysiologic and behavioral studies of human beings and other species place the study of the human brain and language in a different light.

The relevant studies include traditional observations of "experiments-in-nature," deficits resulting from disease or trauma to the brain, and studies employing noninvasive techniques functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) that indirectly track activity in the brain by means of blood flow measurements (Logothetis et al., 2001). Data from studies of neural activity in other species, that make use of invasive techniques employing tracers and direct electrophysiologic recordings of neural activity, also provide essential insights, placing findings from studies of human subjects into a coherent framework. These advances have led to an understanding of the neural bases of human language that transcends the traditional theory equating language with Broca's and Wernicke's areas of the cortex, a theory that dates back to the 19th century. Through these studies, it has become apparent that the evolution of the neural bases of the derived properties of human language, i.e., speech, complex syntax, and an almost limitless vocabulary, derive from Darwinian mechanisms.

The traditional locationist model

History and technology play major roles in structuring human thought. In the early years of the 19th century, phrenologists systematically identified spe-

cific locations in the brain as the locations in which various skills and qualities were regulated. The implicit model underlying the locationist aspect of the theory was the clock. The most common, complex, contemporary machines of the day were clocks and chronometers built out of discrete systems. One mechanical system, i.e., a set of parts, counted out the interval of time, a different set of parts moved the clock's hands, and so on. Therefore, it was reasonable to propose a neural model that sought to find the locations of the specific parts of the brain that regulated various aspects of human behavior. Although phrenology is often portrayed as a quack science, it constituted a locationist scientific theory subject to falsification. Gall (1809) and Spurzheim (1815), in essence, claimed that complex behaviors such as language, mathematical ability, musical ability, and various aspects of human character such as ambition, charity, and veneration were regulated in specific locations of the neocortex. Phrenologists, who obviously lacked any imaging techniques, thought that surface regions of the skull corresponded to the cortical areas in question, and the exterior of the skull was partitioned into regions that were each the "seat" of a "faculty."

According to phrenological theory, the size of these seats, and the areas of the protuberances and bumps of the skull, were innately determined in a given individual. The area of each region was a measure of the complex behavior or particular aspect of character regulated by that region. Phrenological theory was tested through empirical studies that, for example, determined whether people whose skulls had a larger expanse in area 14, the seat of veneration, manifested this attribute in their daily lives more than people whose skulls had a smaller area 14. Gall (1809) measured skulls in such places as prisons and lunatic asylums, correlating behavior with skull measurements. Other studies measured the skulls of clerics, professors, poets, artists, and the like. The skulls of homicidal felons often had larger areas for compassion than clerics, and distinguished mathematicians could have small mathematical areas; thus, phrenology fell into disfavor. However, the underlying premise that guided phrenological research, that all aspects of a complex behavior are regulated in an anatomically discrete, separable, area of the cortex, survives to the present day in the Broca-Wernicke "language area" theory.

When Broca (1861) found that a patient who had lesions in the anterior cortical region of the left hemisphere of his brain was unable to speak anything besides a single monosyllable, he adhered to the phrenological model and concluded that speech production was regulated in this particular region of the cortex. Overlooked was the fact that his patient also had extensive subcortical damage and extensive nonlinguistic motor impairment. Wernicke (1874) found that patients who had suffered damage in the second temporal gyrus of the cortex in the posterior left hemisphere had difficulty comprehending speech. Again, his conclusion was that receptive linguistic ability was localized in a neocortical area. Since making use of language involves both comprehending and producing speech or alternate phonetic systems such as writing or sign language, Lichtheim (1885) proposed a hypothetical cortical pathway linking Broca's and Wernicke's areas. As restated by Geschwind (1970), Lichtheim's revised Broca-Wernicke model persists today in textbooks and (at least implicitly) in some research literature.

The digital computer is the implicit mechanical model underlying current neo-phrenological theories such as those of Fodor (1983), but the basic premises are not very different from those proposed by Gall (1809) and Spurzheim (1815). Complex behaviors are regulated in particular self-contained parts of the brain. However, though the Broca-Wernicke model has the virtue of simplicity, it is wrong. Clinical evidence, which we will review, shows that permanent loss of language does not occur without subcortical damage, even when Broca's or Wernicke's areas have been destroyed. Moreover, damage to subcortical structures, sparing the cortex, can produce aphasic syndromes.

Circuit models

The inherent deficiency of the traditional Broca-Wernicke model is its failure to take account of current knowledge concerning the computational architecture of biological brains. Neurophysiologic activity must be considered at two levels if we are to understand how the brain regulates complex behaviors, such as reaching for a pencil, walking, talking, or comprehending the meaning of this sentence. First, although complex brains contain many distinct neuroanatomical structures, these structures usually do not, in themselves, regulate an observable behavior. An individual neuroanatomical structure instead generally contains many anatomically segregated groups, i.e., "populations" of neurons that carry out a neural process or processes. However, the local process does not constitute a behavior such as walking, talking, or moving a finger. The neuronal population that carries out this process is linked, or "projects," to anatomically segregated neuronal populations in other neuroanatomical structures. Successive links between segregated neuronal populations in different neuroanatomical structures form a neural "circuit;" the linked neural processes carried out in the circuit constitute the brain basis of a complex, observable aspect of behavior that generally has a name, e.g., walking, talking, or striking the key of a piano. And within a given neuroanatomical structure, distinct, anatomically segregated neuronal populations project to neurons in different brain structures to form other circuits. Circuits linking anatomically segregated populations of neurons form neural systems carrying out processes in different parts of the brain. The systems are the neural bases of complex behaviors. As Mesulam (1990, p. 598) notes,

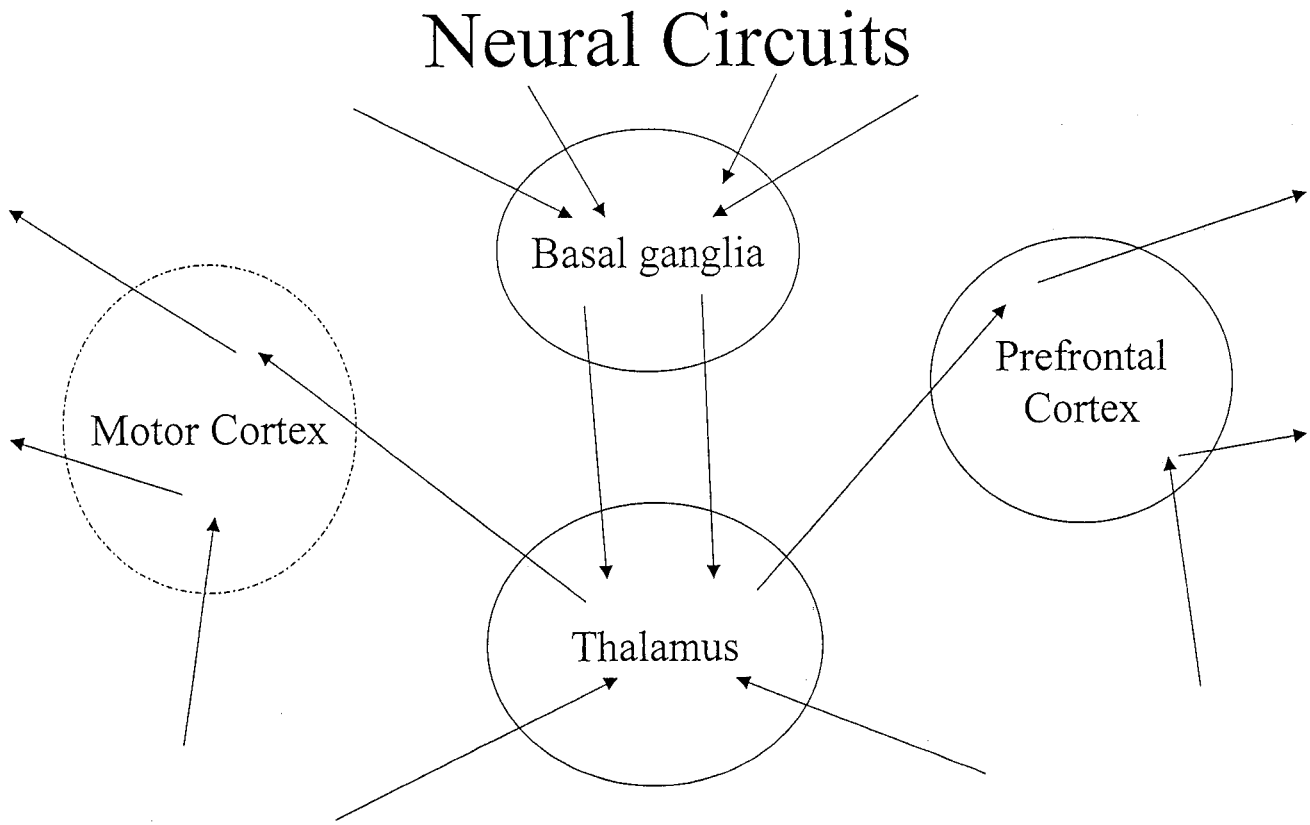

Fig. 1. Anatomically segregated populations of neurons in a particular structure or region of the brain can project to distinct, anatomically segregated populations of neurons in different parts of the brain, forming "circuits" that regulate different aspects of behavior. Thus, damage to a particular part of the brain can result in a "syndrome," an ensemble of seemingly unrelated behavioral deficits. Here, neuronal populations in different cortical areas project into the putamen, and from there indirectly into different regions of the cortex, regulating motor control and different aspects of higher cognition, including the comprehension of syntax.

. . . complex behavior is mapped at the level of multifocal neural systems rather than specific anatomical sites, giving rise to brain-behavior relationships that are both localized and distributed.

In other words, "local" neural operations occur in particular regions of the brain. However, these localized operations in themselves do not constitute an observable behavior such as walking or language for which we have words. Evidence from hundreds of independent studies that span three decades shows that different regions of the neocortex and different subcortical structures are specialized to process particular stimuli (visual or auditory), while other regions perform specific operations that regulate aspects of motor control (such as coding the direction of a movement or its force), or holding information in short-term (working) memory (e.g., Marsden and Obeso, 1994; Mitchell et al., 1987; Mirenowicz and Schultz, 1996; Monchi et al., 2001; Polit and Bizzi, 1978; Sanes et al., 1995). However, these local processes form part of the neural "computations" that, linked together in complex neural circuits, are manifested in behaviors such as walking, pushing a button, speaking, or comprehending the syntax of a sentence (Fig. 1).

For example, within the putamen, a subcortical basal ganglia structure located within the brain, anatomically segregated populations of neurons exist that form part of a system that sequences the submovements that together constitute an overt movement of a monkey's hand, a rat's grooming sequence, and a person's walking or speaking (Aldridge et al., 1993; Cunnington et al., 1995; Lieberman, 2000; Marsden and Obeso, 1994). But the putamen, in itself, is not the "seat" of the motor act. The putamen, like other neuroanatomical structures, supports anatomically segregated neuronal populations that project to different parts of the brain forming a number of circuits that regulate other aspects of behavior. Distinct, anatomically segregated neuronal populations in the putamen project through other subcortical structures to cortical areas implicated in motor control, higher cognition, attention, and reward-based learning (e.g., Aldridge et al., 1993; Alexander et al., 1986; Alexander and Crutcher, 1990; Cummings, 1993; Graybiel, 1995, 1997; Kimura et al., 1993; Lieberman, 2000; Marsden and Obeso, 1994; Middleton and Strick, 1994; Parent, 1986). Complex behaviors are regulated by neural circuits that constitute networks linking activity in many parts of the brain. In short, the neural mechanism that carries out the instruction set manifested in my pecking at my com-

puter's keyboard is a "circuit," linking neuronal populations in different neuroanatomical structures in many parts of the brain.

Some confusion often arises with regard to the precise meaning of the term "module" in neurophysiologic and in linguistic studies. The term "module" is often used in neurophysiologic studies (e.g., Graybiel, 1995, 1997) to refer to complex neural circuits that regulate an observable behavior. In contrast, theories of the mind grounded in linguistics, such as those of Fodor (1983) and Pinker (1998), use the word "module" to refer to localized neuroanatomical structures that they claim regulate specific aspects of language. In these locationist theories, the module that regulates language, or some aspect of language such as syntax, has no anatomical or physiologic relation to other hypothetical neural modules devoted to walking, manual motor control, and so on. In principle, these locationist theories claim that the functional organization of the human brain is similar to that of a conventional digital computer that has a discrete hard disk, a discrete electronic memory, a display, a modem, and so on.

Aphasia

Studies of aphasia, the permanent loss of language, which were the basis for the Broca-Wernicke theory, were among the first to note the deficiencies of this traditional model. Doubts had been expressed in the early years of the 20th century (Jackson, 1915; Marie, 1926). In the past two decades, computer-aided tomography (CT) scans and magnetic resonance imaging (MRI) provided noninvasive information on the nature and extent of brain damage that would result in permanent language loss. The putative basis of Broca's syndrome in the model of Lichtheim (1885) is damage to Broca's neocortical area. However, clinical studies have shown that permanent loss of the linguistic abilities associated with the syndrome does not occur unless subcortical damage is present (Dronkers et al., 1992; D'Esposito and Alexander, 1995; Stuss and Benson, 1986). Patients with extensive damage to Broca's area generally recover linguistic ability, unless subcortical damage also occurs. Patients suffering brain damage that damages subcortical structures but that leaves Broca's area intact also can manifest the signs and symptoms associated with Broca's syndrome. As Stuss and Benson (1986, p. 161) note in their review of studies of aphasia, damage to

> . . . the Broca area alone or to its immediate surroundings . . . is insufficient to produce the full syndrome of Broca's aphasia. . . . . The full, permanent syndrome (big Broca) invariably indicates larger dominant hemisphere destruction . . . deep into the insula and adjacent white matter and possibly including basal ganglia.

Independent studies show that subcortical damage that leaves Broca's area intact can result in Broca-like speech production and language deficits (cf. Alexander et al., 1987; Benson and Geschwind, 1985; Mega and Alexander, 1994; Naeser et al., 1982).

Alexander et al. (1987), for example, reviewed 19 cases of aphasia resulting from lesions in these subcortical structures. Language impairments occurred that ranged from fairly mild disorders in the patient's ability to recall words, to "global aphasia" in which the patient produced very limited nonpropositional speech. In general, the severest language deficits occurred in patients who had suffered the most extensive subcortical brain damage. Damage to the internal capsule (the nerve fibers that connect the neocortex to subcortical structures), basal ganglia structures, the putamen, and the caudate nucleus resulted in impaired speech production and agrammatism similar to that of classic aphasias, in addition to other cognitive deficits. Subsequent studies appear to rule out damage to the internal capsule as the basis for subcortically induced aphasia. Deliberate surgical lesions of the internal capsule aimed at mitigating obsessive-compulsive behavior do not induce aphasia (Greenberg et al., 2000). The studies of neurodegenerative diseases, hypoxia, and focal damage that are discussed below suggest that damage to the subcortical basal ganglia and associated subcortical components of cortical-striatal-cortical circuits yields Broca's syndrome.

The situation for Wernicke's syndrome appears to be similar. The locus for the brain damage traditionally associated with Wernicke's syndrome includes the posterior region of the left temporal gyrus (Wernicke's area), but often extends to the supramarginal and angular gyrus, again with damage to the subcortical white matter below (Damasio, 1991). Indeed, recent data indicate that premorbid linguistic capability can be recovered after complete destruction of Wernicke's area (Lieberman, 2000). As D'Esposito and Alexander (1995, p. 141) conclude in their study of aphasia deriving from subcortical damage, it is apparent

> . . . that a *purely* cortical lesion—even a macroscopic one—can produce Broca's or Wernicke's aphasia has never been demonstrated.

CORTICAL-STRIATAL-CORTICAL CIRCUITS

The basal ganglia are subcortical structures located deep within the brain. They are phylogentically primitive neural structures, which have a functional role that can be traced back to anurans (Marin et al., 1998). The caudate nucleus and the lentiform nucleus constitute the striatum. The lentiform nucleus, which consists of the putamen and globus pallidus (or palladium; the terms refer to the same structure), is cradled in the internal capsule, which forms a bundle snaking through the caudate and lenticular nucleus. Figure 2 shows the general topography. The caudate nucleus, putamen, and globus pallidus are interconnected and form a system with close connections to the substantia nigra, thalamus, other subcortical structures, and the cortex.

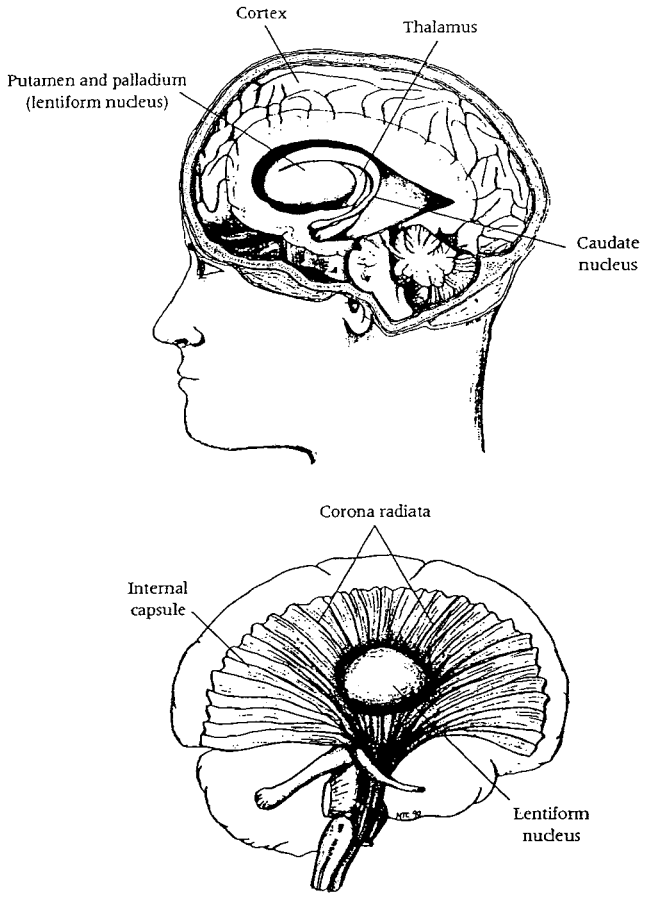

Fig. 2. Basal ganglia are subcortical structures. The putamen and globus pallidus (palladium) constitute the lentiform nucleus, which is cradled in nerves running from the neocortex that converge to form the internal capsule. Caudate nucleus is another primary basal ganglia structure.

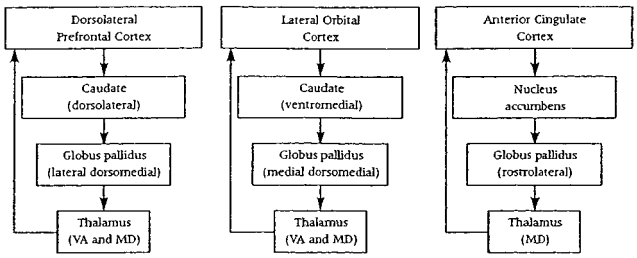

Fig. 3. Organization of three basal ganglia circuits that regulate various aspects of motor control, cognition, and emotion in human beings. Dorsolateral prefrontal circuit, for example, is implicated in speech motor programming, sentence comprehension, and some aspects of cognition. VA, ventral anterior region of thalamus; MD, medial dorsal region of thalamus. Diagrams are simplified and do not show indirect connections of substantia nigra and other details. Damage to any neuroanatomical structures that support neuronal populations of a circuit can result in similar deficits (after Cummings, 1993).

The putamen receives sensory inputs from most parts of the brain. The globus pallidus is an output structure receiving inputs from the putamen and caudate nucleus. Basal ganglia outputs target various regions of the thalamus, which in turn connect to different cortical areas. Connections with the cortex are complex and, as we shall see, not fully understood (Alexander et al., 1986; Alexander and Crutcher, 1990; DeLong, 1993; Marsden and Obeso, 1994). However, the probable subcortical locus of Broca's aphasia is consistent with one of the major findings of contemporary neurophysiological studies.

Disruptions in behavior that are seemingly unrelated, such as obsessive-compulsive disorder (Greenberg et al., 2000), schizophrenia (Graybiel, 1997), and Parkinson's disease (Jellinger, 1990), derive from the disruption of neural circuits that link cortical areas with the basal ganglia structures of the striatum. Anomalous basal ganglia development also appears to implicated in a genetically transmitted deficit affecting speech production and syntax (Lal et al., 2001; Vargha-Khadem et al., 1998; Watkins et al., 2002). Behavioral changes once attributed to frontal lobe cortical dysfunction can be observed in patients with damage to the subcortical basal ganglia (e.g., Alexander et al., 1986; Cummings and Benson, 1984; DeLong, 1983; Flowers and Robertson, 1985; Lange et al., 1992). The review by Cummings (1993), which was based on clinical studies, identified five parallel basal ganglia circuits of the human brain (three of which are shown in Fig. 2). Cumming's (1993, p. 873) notes

> . . . a motor circuit originating in the supplementary motor area, an oculomotor circuit with origins in the frontal eye fields, and three circuits originating in prefrontal cortex (dorsolateral prefrontal cortex, lateral orbital cortex and anterior cingulate cortex). The prototypical structure of all circuits is an origin in the frontal lobes, projection to striatal structures (caudate, putamen, and ventral striatum), connections from striatum to globus pallidus and substantia nigra, projections from these two structures to specific thalamic nuclei, and a final link back to the frontal lobe.

Tracer studies of the brains of other species provide evidence that is consistent with the general model of Cummings (1993). Tracer techniques involve injecting substances (viruses or chemical compounds) into specific locations in the brains of living animals. The tracers attach themselves to the neural circuits formed by the outputs of neurons connecting to other neurons. Postmortem staining techniques then allow neural pathways to be discerned under microscopic analysis. Invasive tracer studies, therefore, are limited to species other than humans. Tracer studies of monkey brains show that the striatum supports circuits that project to cortical areas associated with motor control and cognition (Alexander et al., 1986; Middleton and Strick, 1994; Graybiel, 1995, 1997). The general topography of the striatum and associated structures such as the substantia nigra is quite similar in monkeys and humans (Fig. 3).

In other words, damage to striatal and associated subcortical neuroanatomical structures that support a cortical-striatal-cortical circuit can result in a behavioral deficit that has a seemingly cortical basis. Neurodegenerative diseases such as Parkinson's

disease (PD) and progressive supranuclear palsy (PSP) result in major damage to the subcortical basal ganglia, mostly sparing the cortex (Jellinger, 1990). Independent studies of these neurodegenerative diseases have established the role of the basal ganglia in these circuits. The primary deficits of these neurodegenerative diseases are motoric: tremors, rigidity, and repeated movement patterns occur. However, these subcortical diseases also cause linguistic and cognitive deficits. Speech production, syntax, and cognitive deficits similar in nature to those typical of Broca's aphasia can occur in even mild and moderately impaired PD patients (Cools et al., 2001; Gotham et al., 1988; Harrington and Haaland, 1991; Lange et al., 1992; Lieberman et al., 1992; Morris et al., 1988; Taylor et al., 1990). In particular, deficits in the comprehension of and production of syntax have been noted in independent studies of PD (e.g., Hochstadt et al., unpublished findings; Lieberman, 2000; Lieberman et al., 1990, 1992; Grossman et al., 1991, 1993, 2001; Howard et al., 2001; Illes et al., 1998; Natsopoulos et al., 1993; Pickett, 1998). As is the case for Broca's aphasia (Blumstein, 1995), PD patients have difficulty comprehending sentences that have moderately complex syntax, as well as long sentences that tax the brain's computational resources (Baum, 1989). In extreme form a dementia occurs, different in kind from Alzheimer's dementia (Albert et al., 1974; Cummings and Benson, 1984; Xuerob et al., 1990). The afflicted patients retain semantic and real-world knowledge, but are unable to readily form or change cognitive sets (Flowers and Robertson, 1985; Cools et al., 2001). These seemingly unrelated deficits appear to derive from the "local" operations performed by the basal ganglia in the cortical-striatal-circuits regulating these aspects of behavior.

Probable basal ganglia operations

Sequencing. In the era before medication with levadopa was used to treat Parkinson's disease, thousands of operations were performed. The effects of these surgical interventions on motor control in humans and similar experimental lesions in monkeys were reviewed in a seminal paper by Marsden and Obeso (1994). They noted that the basal ganglia appear to have two different motor control functions (Marsden and Obeso, 1994, p. 889).

> First, their normal routine activity may promote automatic execution of routine movement by facilitating the desired cortically driven movements and suppressing unwanted muscular activity. Secondly, they may be called into play to interrupt or alter such ongoing action in novel circumstances. . . . Most of the time they allow and help cortically determined movements to run smoothly. But on occasions, in special contexts, they respond to unusual circumstances to reorder the cortical control of movement.

Given the fact that the basal ganglia circuitry regulating motor control does not radically differ from that implicated in cognition, Marsden and Obeso (1994, p. 893) concluded that

> . . . the role of the basal ganglia in controlling movement must give insight into their other functions, particularly if thought is mental movement without motion. Perhaps the basal ganglia are an elaborate machine, within the overall frontal lobe distributed system, that allows routine thought and action, but which responds to new circumstances to allow a change in direction of ideas and movement. Loss of basal ganglia contribution, such as in Parkinson's disease, thus would lead to inflexibility of mental and motor response.

Advances in brain imaging and behavioral studies of human subjects support this hypothesis that the basal ganglia perform cognitive sequencing functions. The functional magnetic resonance imaging (fMRI) study of Monchi et al. (2001) monitored brain activity in neurologically intact subjects as they performed a version of the Wisconsin Card Sorting Test (WCST), an instrument that has been used in many studies to assess cognitive dysfunction. The version of the WCST used in this experiment assesses subjects' ability to form and shift abstract categories as they match cards that picture various images to reference cards. The subjects had to match test cards to reference cards based on the color, shape, or number of stimuli pictured on each card. Subjects were informed when they made either correct or incorrect matches, and had to shift the matching criterion as the test progressed. As evidence from many studies of behavioral deficits resulting from brain damage and neurodegenerative diseases predicted, neural circuits involving the prefrontal cortex and basal ganglia were activated during the test. Dorsolateral prefrontal cortical areas (Brodmann areas 9 and 46) were active at points where subjects had to relate the current match with earlier events stored in working memory. In contrast, a cortical to basal ganglia circuit involving the mid-ventrolateral prefrontal cortex (areas 47/12), caudate nucleus, and thalamus was active when subjects had to shift to a different matching criterion. The putamen also showed increased activity during these cognitive shifts. These findings from neurologically intact human subjects matched data from electrophysiologic studies of monkey brains (reviewed in Graybiel, 1995, 1997).

Reward-based learning. Studies of the brains of rodents that selectively destroy basal ganglia structures or use direct electrophysiologic recording are consistent with these human studies. In rodents, the basal ganglia execute innately determined grooming sequences; electrophysiologic studies of basal ganglia neurons show firing patterns that release the sequence of submovements that strung together constitute a grooming sequence (Aldridge et al., 1993). Animal studies also suggest that basal ganglia are implicated in reward-based associative learning. In birds, a basal ganglia to forebrain circuit belongs to a system that regulates vocal learning and production. Lesioning this circuit prevented restructuring of adult zebra finches' songs and young songbirds' acquiring songs (Brainard and Doupe, 2000). In monkeys, neuronal populations form in basal gan-

glia in the course of adaptive reward-based learning (Kimura et al., 1993; Mirenowicz and Schultz, 1996), Graybiel (1995, 1997), reviewing the data of independent studies, noted that neurons in the caudate nucleus, putamen, and globus pallidus control motor acts through response patterns that are built up through learning and memory. In short, converging evidence from independent studies suggests that the basal ganglia and cerebellum (which will be discussed below) are implicated in both learning and executing sequences of motor acts or cognitive processes, forming an internalized repertoire of meaningful, goal-directed acts (e.g., Graybiel, 1995, 1997, 1998; Kimura et al., 1993; Marsden and Obeso, 1994; Mirenowicz and Schultz, 1996).

LINGUISTIC AND COGNITIVE CONSEQUENCES OF SUBCORTICAL BRAIN DAMAGE

As noted earlier, Broca's aphasia does not occur in the absence of subcortical brain damage, suggesting that Broca's syndrome is the result of impairment of subcortical components of cortical-striatal-cortical circuits. A similar pattern of behavioral deficits was documented in neurodegenerative diseases and brain damage affecting the basal ganglia and cerebellum. A brief discussion of the nature of these deficits may be useful, insofar as they may be unfamiliar to nonspecialists.

Speech

One of the primary speech deficits of Broca's syndrome and of compromised basal ganglia function is a breakdown in the sequencing of motor commands necessary to produce stop consonants. The primary acoustic cue that differentiates stop consonants such as [b] from [p] when they occur before a vowel (as in the English words *bat* and *pat*) is an interval of time that reflects the sequence of motor commands that provide these sounds. These speech sounds are produced by closing the lips, obstructing the flow of air from a speaker's mouth, and then abruptly opening the lips, which produces a "burst" of turbulent air that has distinct acoustic properties. At the same time, the speaker must adjust the muscles of the larynx to produce phonation subsequent to the burst. In order to produce a [b], phonation must occur within 20 msec of lip opening; longer delays will yield the sound [p]. Similar temporal contrasts involving the muscles of the tongue and larynx differentiate the sounds [d] from [t] (*do* vs. *to*), and [g] from [k] (*god* vs. *cod*). Lisker and Abramson (1964) coined the term "voice-onset-time" (VOT) to describe this distinction, which appears to hold for all human languages studied to date. In brief, VOT is defined as the time that occurs between the "burst" that results from lip or tongue gestures and the onset of periodic phonation generated by the larynx. Figure 4 shows the waveforms of a [ba] and a [pa] with "cursors" superimposed that mark the onsets of the brusts and phonation.

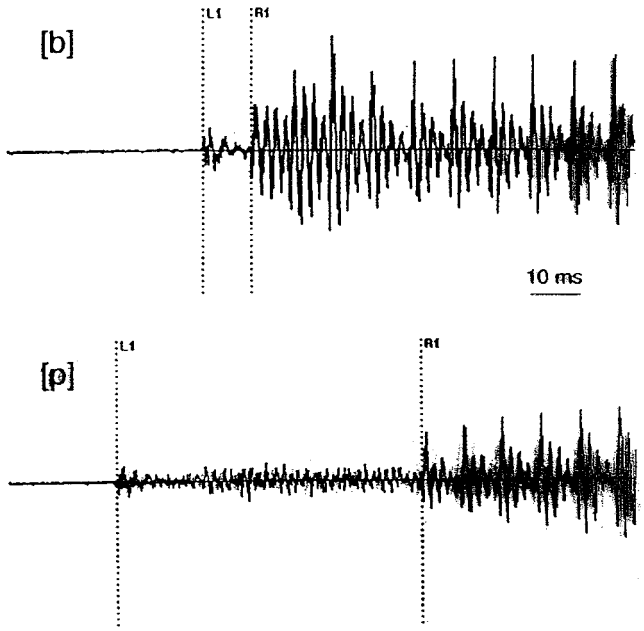

Fig. 4. Speech waveforms of syllables [ba] and [pa]. Amplitudes of speech signal are plotted at ordinate, and elapse of time on abscissa. "Cursors" L1 mark beginnings of "bursts" of "stop consonants" [b] and [p] that occur when the lips open. Cursors. R1 mark onset of phonation that occurs when vocal cords (folds) of speaker's larynx start to produce periodic phonation.

Speakers must precisely control a sequence of independent motor acts to produce these sounds. Broca's aphasics are unable to maintain control of these sequential motor commands: their intended [b] s may be heard as [p] s, [t] s as [d] s, and so on (Blumstein et al., 1980; Baum et al., 1990). The problem was not inherently one of maintaining temporal control, since Broca's aphasics maintain the intrinsic durations that differentiate vowels (Baum et al., 1990), for example, the vowel of the word *bat* is three time longer than that of the word *bit.* Broca's aphasics, moreover, maintain almost normal control of the magnitude and placement of tongue, lip, and laryngeal gestures; no apparent loss of peripheral motor control occurs. The production of the formant frequency patterns that specify vowels is unimpaired in Broca's syndrome, though there is increased variability (Ryalls, 1986; Kent and Rosenbeck, 1983; Baum et al., 1990). Since formant frequency patterns are determined by the configuration of the supralaryngeal vocal tract (tongue, lips, and larynx height), we can conclude that the control of these structures is unimpaired in Broca's aphasic syndrome. The deficit appears to involve sequencing; Broca's aphasics also have difficulty executing either oral, nonspeech, and manual sequential motor sequences (Kimura, 1993). Figure 4 shows the waveforms of [ba] and [pa] marked for VOT.

Voice-onset-time sequencing deficits, similar in nature to those of Broca's syndrome, can occur in the later stages of Parkinson's disease. Computer-implemented analysis revealed overlaps between the

VOTs of stop consonents such as [t] vs. [d], exceeding 19% for some PD subjects; the degree of VOT sequencing deficits depended on the severity of the disease state (Lieberman et al., 1992; Hochstadt et al., unpublished findings). The PD subjects, as was the case for Broca's aphasics, maintained control over vowel duration, other durational speech phenomena, and tongue and lip movements. Similar results occurred for some subjects suffering degeneration of the cerebellum (Pickett, 1998).

The "phonologic" level, i.e., the knowledge and coding of sounds that specify the name of a word, appears to be preserved in Broca's aphasics and in Parkinson's disease for these same sounds. For example, at the phonologic level, the acoustic cues and articulatory gestures that specify a particular stop consonant differ when it occurs in syllable initial position or after a vowel. The speech-sound [t], for example, is signalled by a long VOT when it occurs in syllable-initial position. In contrast, after a vowel, the acoustic cues for [t] are reduced duration of the vowel that precedes it and increased burst amplitude. Broca's aphasics maintain normal control of these cues, although VOT sequencing is disrupted for syllable-initial [t]s. These distinctions are general. The duration of a vowel is always longer, taking into account other factors, before a [b], [d], or [g] than for a [p], [t], or [k]. The fact that Broca's aphasics preserve these durational cues indicates that the phonologic "instruction set" for producing stop consonants is intact. The preservation of these durational cues again indicates that the Broca's VOT deficit derives from the disruption of sequencing rather than impaired ability to control duration. Instrumental analyses of the speech of Broca's aphasics often reveal waveforms showing irregular phonation (Blumstein, 1995). Speech quality is "dysarthric." Noisy and irregular phonation occurs, reflecting impaired regulation of the muscles of the larynx and alveolar air pressure. Similar problems can also occur in advanced stages of Parkinson's disease.

Syntax

"Higher-level" linguistic and cognitive deficits also occur in this aphasic syndrome. The utterances produced by Broca's aphasics often were described as "telegraphic." In the period when telegrams were a means of communication, the sender paid by the word, and words were omitted whenever possible. The utterances of English-speaking aphasics, who omitted prepositions, articles, and tense markers, producing messages such as *man sit tree* in place of *The man sat by the tree,* had the appearance of telegrams. These aphasic telegraphic utterances were generally thought to be the result of the patient's compensating for speech production difficulties by reducing the utterance's length, thereby minimizing difficulties associated with speech production. The presence of language comprehension deficits in Broca's aphasics that appeared to involve syntax was established by studies starting in the 1970s. Broca's aphasics had difficulty comprehending distinctions in meaning conveyed by syntax (Zurif et al., 1972). Although agrammatic aphasics are able to judge whether sentences are grammatical, albeit with high error rates (Linebarger et al., 1983; Shankweiler et al., 1989), the comprehension deficits of Broca's aphasics have been replicated in many independent studies (e.g., Baum, 1989; Blumstein, 1995). For example, higher error rates occur when comprehending distinctions in meaning conveyed by passive sentences such as, "The boy was kissed by the girl" than for the "cannonical" sentence, "The girl kissed the boy." High error rates often occur when comprehending sentences containing embedded relative clauses such as, "The boy who was wearing a red hat fell down." Long sentences generally present additional difficulty. Error rates exceeding 50% can occur using sentences that yield virtually error-free performance by neurologically intact control subjects; cf. Blumstein (1995) for a comprehensive review.

As is the case for Broca's syndrome, Parkinson's disease (PD) can result in sentence comprehension deficits (Grossman et al., 1991, 1993, 2001; Hochstadt et al., unpublished findings; Howard et al., 2001; Lieberman et al., 1990, 1992; Natsopoulos et al., 1993; Pickett, 1998). The first study that associated grammatical deficits with PD was reported by Illes et al. (1988); their data showed deficits similar to those noted in Huntington's disease. The sentences produced by PD subjects were often short and had simplified syntax. However, Illes et al. (1988) attributed these effects to the speakers compensating for their speech motor production difficulties by producing short sentences. A subsequent study of comprehension deficits of PD (Lieberman et al., 1990) showed that syntax comprehension deficits could occur that could not be attributed to compensatory motor strategies. The comprehension deficits noted clearly were not the result of any compensating strategy, since the motoric component of subjects' responses to both sentences that had complex syntax and high rates and sentences with simple syntax and low error rates was identical. The subjects simply had to utter the number (one, two, or three) that identified a line drawing that best represented the meaning of the sentence that they heard. Deficits in the comprehension of distinctions of meaning conveyed by syntax occurred for long conjoined simple sentences as well as for sentences that had moderately complex syntax. Nine of a sample of 40 nondemented PD subjects had these comprehension deficits. The test battery used in this study included sentences with syntactic constructions that are known to place different processing demands on normal adult subjects, e.g., center-embedded sentences, right-branching sentences, conjunctions, "simple" one-clause declarative sentences, or semantically and constrained and semantically unconstrained passives. However, neurologically in-

tact subjects made virtually no errors when they took this test. In contrast, the overall error rate was 30% for some PD subjects. The PD subjects' comprehension errors typically involved repeated errors on particular syntactic constructions. Therefore, the observed syntax comprehension errors could not be attributed to general cognitive decline or attention deficits. The highest number of errors (40%) were made on "left-branching" sentences that departed from the canonical pattern of English having the form subject-verb-object (SVO). An example of a left-branching sentence is, "Because it was raining, the girl played in the house." Thirty percent errors occurred for right-branching sentences with final relative clauses, such as "Mother picked up the baby who is crying." Twenty percent error rates also occurred on long conjoined simple sentences, such as "Mother cooked the food and the girl set the table." Similar sentence comprehension errors reflecting information conveyed by syntax have been found in independent studies of nondemented PD subjects (Grossman et al., 1991, 1993, 2001; Howard et al., 2001; Natsopoulos et al., 1993), using procedures that monitored either sentence comprehension or judgments of sentence grammaticality.

The PD subjects studied by Grossman et al. (1991) were asked to interpret information presented in sentences in active or passive voices when the questions were posed in passive or active voices. Deficits in comprehension were noted when PD subjects had to shift cognitive sets, responding to a question posed in a passive voice concerning information presented in an active voice or the reverse. Higher errors, for example, occurred when the subjects heard the sentence *The hawk ate the sparrow* when asked *Who was the sparrow eaten by?* than when asked *Who ate the sparrow?* Grossman et al. (1991) also tested PD subjects' ability to copy unfamiliar sequential manual motor movements (a procedure analogous to that used by Kimura (1993), who found deficits in this behavior for Broca's aphasics). Deficits in sequencing manual motor movements and linguistic sequencing in the sentence comprehension task were correlated. The correlation between sequencing complex manual motor movements and the cognitive operations implicated in the comprehension of syntax is consistent with Broca's area playing a role in both verbal working memory and manual motor control (Rizzolatti and Arbib, 1998) in circuits supported by basal ganglia (Marsden and Obeso, 1994).

Grossman et al. (2001) interpreted these deficits as an attentional rather than a linguistic deficit. However, this is unlikely, since PD subjects and Broca's aphasics attend to semantic information when they are asked to convey the meaning of a sentence. They consistently perform better when faced with a sentence such as "The banana was eaten by the boy" than the sentence "The clown was poked by the cowboy." They clearly have no attentional deficits regarding the fact that an inanimate banana cannot eat a boy. A study of bilateral damage to the putamen and part of the caudate nucleus (Pickett et al., 1998) revealed similar deficits in sequencing speech motor gestures and the comprehension of distinctions in meaning conveyed by syntax.

Motor and cognitive set-shifting

Motor and cognitive sequencing deficits similar to those noted and predicted by Marsden and Obeso (1994) generally occur in PD. Speech motor sequencing, verbal working memory, syntactic, and cognitive sequencing errors occur in Parkinson's disease. Manual motor sequencing deficits have been noted in many studies of PD (e.g., Cunnington et al., 1995). As noted above, Grossman et al. (1991) found correlated deficits in sequencing manual motor movements and linguistic operations in a sentence comprehension task. Speech production VOT sequencing deficits have also been found to correlate with syntactic comprehension deficits in PD (Lieberman et al., 1992), and with bilateral damage to the putamen (Pickett et al., 1998), as well as for neurologically intact human subjects and subjects having cerebella damage (Pickett, 1998).

Hypoxia (oxygen deficits) commonly occurs in mountain climbers at extreme altitudes, and similar, though generally less extreme, VOT sequencing and syntax comprehension errors again can occur in these subjects (Lieberman et al., 1994, unpublished findings). Histologic studies of the hypoxic brain have identified regions of "selective vulnerability" in the hippocampus, cerebellum, basal ganglia, and layers III, V, and VI of the neocortex (Brierley, 1976). Damage to all of these neural structures may have motor and cognitive consequences. However, the globus pallidus (the principal basal ganglia output structure linking the striatum to the cortex through the thalamus and other subcortical structures) is extremely sensitive to hypoxic damage (Laplane et al., 1984, 1989; Strub, 1989); Moreover, the behavioral deficits at extreme altitude that are discussed below (Lieberman et al., 1994, 1995 submitted; Nelson et al., 1990; Regard et al., 1989) are virtually identical to those occurring with bilateral surgical lesions of the globus pallidus (Scott et al., 2002). Long-term memory remains unchanged, as is performance in a number of psychometric tests that are generally thought to involve the cortex. Cognitive impairment in these subjects appears to be limited to forming and shifting cognitive sets on tests such as the Odd-Man-Out test (Flowers and Robertson, 1985), which involves forming a conceptual category and then shifting to a different category. For example, if the subject starts by sorting pictures by their shapes, s/he must then switch to sorting them by size. Impaired subjects have difficulty shifting to a different sorting criterion; they tend to perseverate, holding or shifting back to a previous sorting criterion. Cognitive perseveration in real-life situations also occurs for hypoxic subjects climbing Mount Everest (Lieberman et al., 1994, 1995). It can

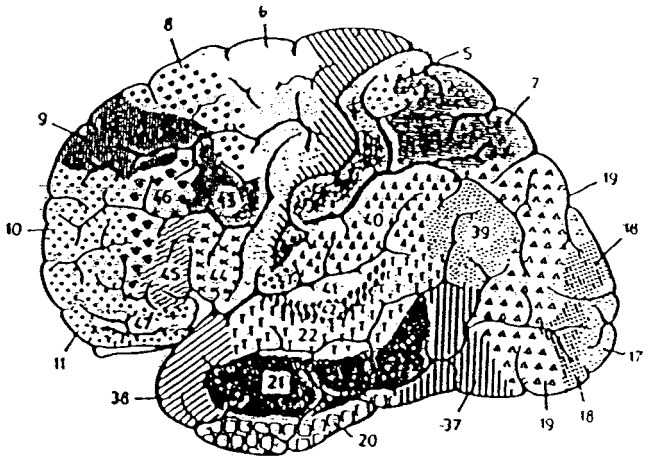

Fig. 5. Brodmann (1912), by means of microscopic examination, partitioned the surface of the cortex into areas whose cells had somewhat different anatomical properties. Different local operations often appear to be performed in these regions. Frontal regions are at left. Areas 44 and 45 are traditional sites of Broca's area.

result in fatal errors of judgment (Lieberman et al., unpublished findings). Cognitive perseveration in the domain of language may account for some of the sentence comprehension deficits discussed above. When comprehending the meaning of a sentence that contains relative clauses, the subject must shift syntactic operations at the clause boundary, for example, as in comprehending the meaning of the sentence, "The boy who was fat fell down."

The fMRI study of Monchi et al. (2001) demonstrated the coordinated activity of the cortex and basal ganglia as human subjects form and shift concepts. Brain activity was monitored in neurologically intact subjects in a task similar to the Odd-Man-Out test, a version of the Wisconsin Card Sorting Test (WCST), an instrument that has been used in many studies to assess cognitive dysfunction. The version of the WCST used assesses subjects' ability to form and shift abstract categories as they match cards that picture various images to reference cards. The subjects had to match test cards to reference cards based on the color, shape, or number of stimuli pictured on each card. Subjects were informed when they made either correct or incorrect matches, and had to shift the matching criterion as the test progressed. Figure 5 shows the cortical areas defined by Brodmann (1912). Neural circuits involving the prefrontal cortex and basal ganglia were activated during the test. Dorsolateral prefrontal cortical areas (Brodmann areas 9 and 46) were active at the points where subjects had to relate the current match with earlier events stored in working memory. In contrast, a cortical to basal ganglia circuit involving the mid-ventrolateral prefrontal cortex (areas 47/12), caudate nucleus, and thalamus was active when subjects had to shift to a different matching criterion. The putamen also showed increased activity during these cognitive shifts. These findings from neurologically intact human subjects match data from electrophysiologic studies of monkey brains (reviewed in Graybiel, 1995, 1997).

Cerebellum

Less is known about the cognitive role of the cerebellum, a subcortical structure that is linked to the prefrontal and motor cortex as well as to the basal ganglia. fMRI and tracer studies show that it is active in motor learning, apparently acting in concert with the prefrontal cortex (Thach, 1996; Deacon, 1997). More general linguistic and cognitive roles for the cerebellum, particularly the neocerebellum, which is disproportionately large in humans, have been proposed (Leiner et al., 1991). However, it is unclear whether the cerebellum plays a role in sentence comprehension and linguistic tasks that do not involve modeling of motor activity (Thach, 1996). A study that tested the ability of persons suffering cerebellar degeneration to comprehend distinctions in meaning conveyed by syntax failed to show deficits attributable to the neocerebellum (Pickett, 1998). The linguistic and cognitive deficits noted by Pickett appeared to derive from damage to the neural pathways linking it to the basal ganglia and cortex (Pickett, 1998). The cerebellum has been linked to the control of timing motor activity (Ivry and Keele, 1989); although VOT sequencing was degraded in some of the subjects studied by Pickett (1998), the intrinsic durations of English vowels were preserved. This might reflect the highly overlearned nature of the neural pattern generators that specify the motor gestures underlying human speech.

THE "LANGUAGE GENE" AND UNIVERSAL GRAMMAR

Some attention has been drawn to the identification of a putative "language gene" that has been interpreted as evidence for the claim by Chomsky (1986) that the syntactic "rules" of all human languages are determined by an innate neural mechanism (Gopnik, 1990; Gopnik and Crago, 1991; Pinker, 1994). Over the course of many years, Chomsky (1966, 1972, 1986) developed the theory of "universal grammar" (UG). No person would dispute that human beings have an innate capacity to acquire language. It is clear that neurologically intact infants and children raised under "normal" circumstances have the biological capacity to learn any language. However, Chomsky (1966, 1972, 1986) goes further, claiming that the detailed syntax of all human languages is an innate attribute of the human brain. For example, English syntax has a "regular plural rule" which predicts the plural form of most nouns: dog-dogs, car-cars, etc. Children raised in an English-speaking environment acquire this knowledge without explicit tutoring. While many specialists would argue that the processes that allow

children to master other aspects of cognitive behavior can account for this and other aspects of the acquisition of language (e.g., Bates et al., 1992; Elman et al., 1997; Greenfield, 1991; Lieberman, 1984, 1998, 2000), Chomsky (1966, 1972, 1976, 1986) claims that the UG instantiates innate knowledge of this "rule," in effect "triggering" the genetic program if a child is exposed to regular English plural nouns early in life. In short, the UG constitutes an innate store of detailed knowledge of syntax. Other genetically transmitted components of the UG would specify the rules governing the formation of yes-no questions, while other UG genes would confer the ability to form passive sentences, and so on. Nowak et al. (2000) make two claims based on a computer-modelling study: 1) that syntax becomes necessary as a language acquires many words, and 2) that the rules of syntax must be innately determined. The first claim is consistent with studies of the development of lexical ability and syntax in young children. Bates and Goodman (1997) showed that syntax develops as vocabulary size increases as children mature. The second claim, that the rules of syntax are innately specified, reiterates the claim of Chomsky (1966, 1972, 1986) that children could not possibly learn the rules of syntax. Nowak et al. (2000) disregard the body of studies that suggest that children acquire words and syntax by means of associative learning, imitation, and subtle social cues that indicate their errors to them (e.g., Bates and Goodman, 1997; Elman et al., 1997; Greenfield, 1991; Lieberman, 1984).

It is clear that the vocal and gestural signals of many species are genetically specified and need only triggering stimuli. For example, ducklings require very limited exposure to duck calls as they hatch to develop normal duck signaling behavior months later (Gottlieb, 1975). In effect, the claim inherent in UG is that human beings possess a vastly more elaborate set of genetically transmitted linguistic information than ducks, allowing children who receive limited exposure to the utterances of a language to master syntax. The hypothetical UG must, of course, encode the different syntactic schemes that occur in the world's languages. Therefore, UG must contain many detailed syntactic rules. Since diseases such as diabetes which have a strong genetic component result in specific deficits, one source of evidence for UG would be a genetic anomaly that prevented afflicted individuals from mastering a specific aspect of English syntax, while retaining other aspects of normal linguistic ability. This was reported to be the case for the afflicted members of a large extended family (KE) who suffer from a genetically transmitted anomaly. Gopnik (1990) and Gopnik and Crago (1991) claimed that these individuals were unable to master the regular past tense of English verbs and regular plural nouns. Other aspects of English syntax, and cognitive and motor behavior, supposedly were similar to the normal members of family KE. However, this is not the case. Intensive study of family KE reveals the occurrence of a suite of severe speech and orofacial movement disorders, cognitive deficits, and linguistic deficits that are not limited to specific aspects of the syntax of English (Lal et al., 2001; Vargha-Khadem et al., 1998; Watkins et al., 2002). Major orofacial sequencing errors (they are not able to stick out their tongues while closing their lips) occur in these individuals; they have difficulty repeating two words in sequence. In a filmed interview of afflicted family KE children (BBC broadcast, 1994), subtitles were provided because their speech was scarcely intelligible. On standardized intelligence tests, afflicted members of family KE, raised in the same immediate family, have significantly lower scores than their nonafflicted siblings, which rules out environmental factors that might affect intelligence.

Watkins et al. (2002) concluded that these "verbal and non-verbal deficits arise from a common impairment in the ability to sequence movement or in procedural learning." MRI and PET data on a limited sample of family KE members indicate that these cognitive and motor impairments appear to derive from a basal ganglia anomaly. The afflicted members of this large extended family have bilaterally small caudate nuclei. Although other as yet undetermined neural structures may be at risk in family KE, their conclusion is consistent with the pattern of motor and cognitive sequencing deficits associated with the basal ganglia dysfunction noted above.

Other studies point to damage to components of cortical-striatal-cortical circuits yielding motor, cognitive, and linguistic deficits. Kimura and Watson (1989), in a study of aphasic patients with focal brain damage, found that their patients, as is the case for the afflicted members of family KE, had coordinate oral sequencing and speech production deficits. Developmental verbal apraxia in children, which is defined as an impairment in the programming of the sequences of movement, also results in verbal speech production and cognitive deficits deriving from a breakdown in sequencing (Dewey at al., 1988).

CORTEX

As noted above, subcortical neural structures work in concert with regions of the cortex in linguistic and cognitive tasks as well in motor control. Although the specificity of the traditional Broca-Wernicke theory, localizing language to these cortical areas, is incorrect, these and other cortical areas play critical roles in the neural networks that confer human linguistic ability.

Verbal working memory: Broca's and Wernicke's areas

Imaging studies that monitor brain activity during different linguistic tasks consistently show activation of Broca's and Wernicke's areas of the cortex,

as well as many other cortical areas. In neurologically intact subjects, Broca's area clearly is implicated in sentence comprehension. Stromswold et al. (1996), using PET, studied neurologically intact subjects whose task was to decide whether sentences were grammatical. The sentences varied in grammatical complexity; the greatest activation of Broca's area occurred in the sentences that were most complex, leading to the conclusion that Broca's area was implicated in analyzing the syntax of a sentence. Indeed, Stromswald et al. (1996) concluded that Broca's area is the brain's "syntax" organ.

But this is not strictly the case; a body of evidence that extends back 30 years shows that the meaning of a sentence involves recourse to the brain's neural dictionary as well as short-term storage and operations in "verbal working memory," a short-term neural memory buffer (Baddeley, 1986; Gathercole and Baddeley, 1993). Scores of independent studies show that the words of a sentence are held in verbal working memory by means of a process of phonetic "rehearsal," silent speech that makes use of the neural mechanisms that also control overt speech. The data of Awh et al. (1996), for example, show that neurologically intact subjects use neural structures implicated in speech production to subvocally "rehearse" letters of the alphabet, maintaining them in working memory. Subtractions of PET activity showed increased metabolic activity (rCBF values) in Broca's area (Broadmann area 44) as well as the premotor cortex (area 6), supplementary motor area, cerebellum, and anterior cingulate gyrus when PET data from a task involving verbal working-memory were compared with a task that had a substantially lower working memory load. These brain regions are all implicated in speech motor control. Electrophysiologic data from nonhuman primates, for example, show that the anterior cingulate gyrus is implicated in regulating phonation (Newman and Maclean, 1982) as well as in attention (Peterson et al., 1988). Left hemisphere posterior (Wernicke's area) and superior parietal regions also showed greater activity as working memory load increased. These PET data are consistent with the results of studies of patients having lesions in these cortical areas: they show deficits in verbal working memory that appear to reflect impairment to phonological knowledge, i.e., the sound pattern of words (Warrington et al., 1971; Vallar et al., 1997).

Imaging studies confirm that Broca's area and these cortical areas are involved in overt speech as well as in silent reading. The PET study of Peterson et al. (1988), in which neurologically intact subjects were asked to either read or repeat spoken isolated words, showed activation of the primary motor cortex, premotor cortex, and supplementary motor cortex in the subjects' left hemispheres, and bilateral activation of areas near Broca's area and its right-hemisphere homologue. Bilateral activation of areas near Broca's region also occurred when subjects were asked to simply move their mouths and tongues. This finding is consistent, to a degree, with the data of many studies of patients having cortical lesions, since lesions confined to Broca's area often result in oral apraxia, i.e., deficits in motor control instead of the deficits in motor planning associated with aphasia (Stuss and Benson, 1986; Kimura, 1993).

Frontal and posterior regions of the cortex also activate when people listen to speech and talk. A series of PET studies performed at the Montreal Neurological Institute consistently showed increased activity in Brodmann's areas 47, 46, 45, and 8 in the left frontal region, as well as activity in the subcortical left putamen and posterior secondary "auditory" cortex (Klein et al., 1995; Paus et al., 1996). These studies demonstrate the presence of pathways from the "motor" to "auditory" cortex. Signals transmitted from neural structures regulating speech motor control result in increased activity in regions of the posterior temporal cortex associated with speech perception when a person talks.

Broca's area thus does not constitute a localized "speech production," "syntax comprehension," or "sentence-comprehension" organ. The posterior parietal regions, anterior cingulate gyrus, premotor cortex, and supplementary motor area are all implicated in these processes. It is also evident that Broca's area is also implicated in manual motor control (Kimura, 1973). Recent data show that Broca's area and its homologue in monkeys support a functional neural system that generates and monitors grasping and manual gestures (Rizzolatti and Arbib, 1998).

Dynamic neural systems

Moreover, the neural system that carries out sentence comprehension is dynamic, recruiting additional resources as task demand increases. The fMRI study of Just et al. (1996) made use of the same "subtraction" technique as Stromswold et al. (1996). Neural metabolic activity was monitored as subjects read sentences that expressed the same concepts and had the same number of words, but differed with respect to syntactic complexity. The sentences all had two clauses. The sentences with the simplest syntactic structure were active conjoined sentences (type 1) such as, *The reporter attacked the senator and admitted the error.* The same information was conveyed by the subject relative clause sentence (type 2), *The reporter that attacked the senator admitted the error,* and the object relative clause sentence (type 3), *The reporter that the senator attacked admitted the error.* These three sentence types differ with respect to syntactic complexity by several generally accepted measures. Progressively longer reading times and higher comprehension error rates occur in these sentence types. Neurologically intact subjects read sets of exemplars of each sentence type while activity in their brains was monitored by means of fMRI. Measures of comprehension were also obtained, as well as mean processing time and error rates. Activity in the left

temporal cortex, superior temporal gyrus, superior temporal sulcus, and sometimes the middle temporal gyrus, Wernicke's area (Brodmann's areas 22, 42, and sometimes 21), increased as subjects read the sentences with increasing syntactic complexity. Similar increases in activity occurred in the left inferior frontal gyrus, i.e., Broca's area (Broadmann's areas 44 and 45). The novel finding was that the three sentence types resulted in increased activity in areas that were spatially contiguous or proximal to the areas activated while reading simpler sentences. Furthermore, the right hemisphere homologies of Broca's and Wernickes's areas became activated, though to a lesser degree, as syntactic complexity increased. Moreover, the dorsolateral prefrontal cortex (generally not associated with language) showed bilateral activation for 3 of the 5 subjects who were scanned in an appropriate plane (coronal scans). Activation levels in the dorsolateral prefrontal cortex also increased with sentence complexity for these subjects. The dorsolateral prefrontal cortex is implicated in executive control, visual working memory, tasks requiring planning, deriving abstract criteria, and changing criteria in cognitive tasks (Grafman, 1989; Paulesu et al., 1993; D'Esposito et al., 1995). In a PET study of bilingual neurologically intact subjects, increased activity in the left putamen was observed when subjects were speaking their "second," less established language (Klein et al., 1994).

It is clear that the neural bases of language are complex and appear to involve many different neural circuits (Mesulam, 1990). Moreover, our knowledge is imperfect. For example, cortical-striatal-cortical circuits linking the prefrontal cortex to other neural structures do not appear to be implicated in the linguistic deficits associated with Wernicke's syndrome, i.e., fluent, often meaningless speech containing neologisms (cf. Blumstein, 1995). While PET studies show prefrontal hypometabolism in patients with Broca's syndrome, this is not the case for Wernicke's syndrome (Metter et al., 1987). The neural bases of Wernicke's syndrome are still unclear.

The brain's dictionary

It is clear that comprehending the meaning of a sentence cannot proceed without first identifying its words, their meanings, and syntactic constraints, for example, the argument structures of verbs that determine, among other things, whether they can refer to animate subjects or not (Croft, 1991). And, in fact, a growing body of psycholinguistic research based on interactive-activation models of linguistic representation and processing indicates that sentence processing is lexically driven and takes into account probabilistic, semantic, and syntactic knowledge coded in the lexicon (Bates and Goodman, 1997; MacDonald, 1994). Moreover, the neural structures that "define" the meaning of a word appear to be the ones that are relevant in real life. Neuroimaging studies show that when we think of a word, the concepts that are coded by a word result in the activation of the brain mechanisms that concern the real-world attributes of the word in question. For example, the PET data of Martin et al. (1995b) show that the primary motor cortex implicated in manual motor control is activated when we think of the name of a hand tool. Primary visual cortical areas associated with the perception of shape or color are activated when we think of the name of an animal. Neurologically intact subjects who were asked to name pictures of tools and animals activated the ventral temporal lobes (areas associated with visual perception) and Broca's area.

A second PET study of neurologically intact subjects, who were asked to retrieve information about specific objects and words, reinforces the premise that the knowledge "coded" in words is stored and accessed by activating the neuroanatomical structures and circuits that constitute the means by which we attain and/or make use of the knowledge coded by words. Subjects were asked to either name the color associated with an object or word (e.g., *yellow* for a pencil), or state the action associated with the word or object (e.g., *write* for a pencil). As Martin et al. (1995a) noted.

> Generation of color words selectively activated a region in the ventral temporal lobe just anterior to the area involved in the perception of color, whereas generation of action words activated a region in the left temporal gyrus just anterior to the area involved in the perception of motion.

It is significant that the areas of cortex involved in these aspects of visual perception are multisensory. Other neural circuits supported in these regions of the cortex are implicated in tactile sensation and audition (Ungerleider, 1995). There may be no clear distinction between the neural mechanisms involved in storing "nonlinguistic" concepts in our mind-brain and those implicated in perception. Neurophysiologic data, for example, show that Brodmann's area 17, an area of the cortex associated with early stages of visual perception, is activated when subjects are asked to image simple patterns (Kosslyn et al., 1999).

As is the case for the dictionaries that we are accustomed to using, the sound pattern, i.e., the word's spelling, seems to be its "address" in the brain's dictionary. Damasio et al. (1996), by means of behavioral studies of brain-damaged patients and imaging studies of neurologically intact subjects, again showed that the neural substrate that constitutes the brain's dictionary extends far beyond Wernicke's area. Their data suggest that the brain's lexicon is instantiated in circuits that link conceptual knowledge to the words' spellings, i.e., the sounds of speech. Deficits in naming were studied in patients who had focal brain damage. The subjects were shown photographs that fell into three general categories: 1) the faces of well-known people, 2) animals, and 3) tools. Subjects were asked to provide the most specific word for each item and were com-

pared with the responses of normal controls matched for age and education. Subjects were also asked to describe the photograph as best they could.

Twenty-nine subjects were found who could not name the photographs, though they knew what they represented. Seven patients were impaired solely on persons, 2 on persons and animals, 5 only on animals, 5 on animals and tools, 7 only on tools, and 4 on persons, animals, and tools. All of these subjects had cortical and underlying subcortical lesions localized along the temporal pole and inferotemporal regions inferior to Wernicke's area (all but one had left hemisphere damage). The naming deficits roughly correlated with damage to three adjoining cortical areas and the underlying subcortical structures in this region. fMRI activation of these same regions occurred when these photographs were shown to nine neurologically intact subjects. Variations occurred from subject to subject, which Damasio et al. (1996) considered to have resulted from different life histories; the detailed circuitry in their view was acquired rather than genetically specified.

Cortical plasticity

One of the surprising findings of current research on the brain is the presence of cortical plasticity. The supposition of Damasio et al. (1996) regarding the phenotypic acquisition of the circuits that neurally instantiate words is well-founded. Neurophysiological studies indicate, beyond reasonable doubt, that the particular neural circuits that regulate complex aspects of human and animal behavior are shaped by exposure to an individual's environment. For example, they show that inputs to the visual cortex develop in early life in accordance with visual input; different visual inputs yield different input connections (Edelman, 1987; Hata and Stryker, 1994). Cortical regions that normally respond to visual stimuli in cats respond to auditory and tactile stimuli in visually deprived cats. Rauschecker and Korte (1993) monitored single-neuron activity in the anterior ectosylvian visual cortical area of normal cats and cats that had been vision-deprived. Neurons in this area in normal cats had purely visual responses. In young cats who had been deprived of vision from birth, only a minority of cells in this area responded to visual stimuli: most responded vigorously to auditory and to some extent somasensory stimuli. Imaging and behavioral data indicate that similar processes account for the formation of such basic aspects of vision as depth perception in children.

PET data show that both the primary and secondary visual cortex in persons blinded early in life are activated by tactile sensations when they read Braille text (Sadato et al., 1996). Children who have suffered large lesions to the classic "language areas" of the cortex usually recover language abilities that cannot be differentiated from those of other normal children (Bates et al., 1992). Sign language is "heard" in the auditory cortex of deaf people (Nisimura et al., 1999). The cortical representation of the tactile finger receptors involved in playing stringed instruments is a function of the age at which musicians started musical lessons (Pantev et al., 1998).

Acquisition of plans for motor control, cognition, and language

The details of the motor programs instantiated in the motor cortex likewise are phenotypically acquired (Edelman, 1987; Nudo et al., 1996; Sanes and Donoghue, 1994, 1997). Given the architectural similarity of the cortical-striatal-cortical circuits implicated in motor control and cognition noted earlier (Cummings, 1993; Graybiel et al., 1994; Kimura et al., 1993; Marsden and Obeso, 1994; Middleton and Strick, 1994; Mirenowicz and Schultz, 1996), it is most unlikely that the cognitive "pattern generators" (Graybiel, 1997, 1998) that specify syntactic operations (Lieberman, 2000) are innately specified. The neural mechanisms implicated in motor control are massively parallel (Alexander et al., 1986, 1992; Alexander and Crutcher, 1990; Marsden and Obeso, 1994; Sanes and Donoghue, 1996, 1997), and cannot usefully be described by means of sequential algorithms similar to those commonly used by linguists to describe the "rules" of syntax (Alexander et al., 1992). As Croft (1991) noted, the inability of formal linguistics to describe the sentences of English, arguably the most intensively studied language on earth, may derive from an overreliance on algorithmic procedures that do not take account of graded semantic and "real-world" knowledge (MacDonald, 1994). Even formal linguists committed to the Chomskian school, such as Jackendoff (1994), note that it has not been possible to describe the syntax of English by these procedures. The problem most likely rests in the fact that language is the product of a biological brain that does not resemble the digital computers programmed by means of sequential algorithms that linguists implicitly use as a model of the mind (e.g., Pinker, 1994).

Brain lateralization and language

The human brain is lateralized, and the left hemisphere in about 90% of the present human population has a dominant role in regulating both motor control and language. Lenneberg (1967), in one of the first modern studies of the biology of language, believed that brain lateraliziation was the key to the presence of language; many subsequent studies have sought to establish brain lateralization in extinct hominids. However, lateralization is a primitive feature (Bradshaw and Nettleton, 1981). The brains of some species of frogs are lateralized; the left hemisphere of their brains regulates their vocalizations (Bauer, 1993). Moreover, studies of many mammalian species show that paw movements are under lateralized neural control (Denneberg, 1981; MacNeilage, 1991). And as noted earlier, current neural imaging studies show that although one

hemisphere generally is more active during linguistic tasks, both hemispheres of the brain are activated (Just et al., 1996). Theories that identified asymmetric development of the traditional "language areas" of the neocortex with linguistic ability have not stood the test of time. The planum temporale of Wernicke's area was thought to be symmetric in apes, in contrast to the asymmetrically larger planum temporale in the human dominant hemisphere. However, further study shows that apes and humans both have a similar asymmetric planum temporale (Gannon et al., 1998). Broca's area likewise has been found to be asymmetric in apes (Cantalupo and Hopkins, 2001). Since apes who have asymmetric neocortical "language areas" lack human language, it is apparent that the asymmetric characteristics of the human brain by themselves do not confer linguistic ability.

SUMMARY: NEURAL BASES OF LANGUAGE

Summarizing the discussion above, the neural bases of human language are not localized in Broca's and Wernicke's areas of the cortex. The brain's dictionary appears to be instantiated by means of a distributed network in which neuroanatomical structures that play a part in the immediate perception of objects and animals as we view them, or the gestures associated with tools as we use them, are activated. The lexicon appears to connect real-world knowledge with the sound-patterns by which we communicate the concepts coded by words. It, like other neural structures implicated in language, is plastic and is shaped by life's experiences.

Human beings possess a verbal working memory system that allows us to comprehend the meaning of a sentence, taking into account the syntactic, semantic information coded in words as well as pragmatic factors. Verbal working memory appears to be instantiated in the human brain by a dynamic distributed network that recruits neural "computational" resources in response to task demands such as syntactic complexity and sentence length. The neural network that is the basis of verbal working memory links activity in posterior, temporal regions of the neocortex, including Wernicke's area, with frontal regions such as Broca's area (Brodmann areas 44 and 45), frontal regions adjacent to Broca's area, the premotor cortex (area 6), motor cortex, supplementary motor area, right hemisphere homologies of Wernicke's and Broca's areas, and prefrontal cortex. Frontal regions of the cortex generally associated with "nonlinguistic" cognition are activated as task difficulty increases. The anterior cingulate cortex, basal ganglia, and other subcortical structures such as the thalamus and cerebellum are also implicated.

Cortical-striatal-cortical neural circuits are implicated in sentence comprehension, cognitive sequencing, and speech and other aspects of motor control. The subcortical neuroanatomical structures that support the neuronal populations that constitute these circuits also play a part in regulating emotion. The basal ganglia play a critical role in these circuits and carry out at least three motor and cognitive control functions:

1. They are involved in learning activities that yield a reward.
2. They play a part in sequencing the individual elements that constitute a motor or cognitive "pattern generator."
3. They interrupt an ongoing sequence, contingent on external events and prior knowledge.

The motor patterns that generate the articulatory gestures that produce human speech appear to be learned (Lieberman, 1984), as is the case for other acquired motor patterns. Cognitive, procedural knowledge, including the syntactic operations specific to a particular language, also appear to be learned by children (Bates et al., 1992; Bates and Goodman, 1997; Deacon, 1997). The cerebellum is implicated in motor learning and may play a part in cognitive and linguistic tasks involving motor imagery. Our knowledge of the brain is imperfect. However, localized areas of the brain do not appear to constitute "language organs" devoted to language and language alone.

ON THE EVOLUTION OF THE BRAIN BASES OF LANGUAGE

Here, given the imperfection of human knowledge, we go with some trepidation. However, some hypotheses concerning the evolution of language can be rejected, while other hypotheses may lead to useful insights on both the nature and evolution of the human brain and human nature. It is most improbable that one single factor could account for the evolution of language, given the complexity of the neural bases of human linguistic ability. Indeed, linguistic theories since the time of the Sanskrit grammarians generally propose that the ability to communicate and think by means of language involves different elements. Syntax, the store of words in the brain's dictionary, and the ability to talk (or alternate manual systems) all make human language possible, and different evolutionary processes and timetables may account for their evolution.

The principles and techniques of evolutionary biology noted at the start of this discussion can clarify the issues that must be addressed. Biologists differentiate a species from its ancestral species and "cousins" (related species that can be traced to a common ancestor) by means of its "derived" characteristics that differentiate it from its ancestors and cousins. But in order to track derived features, we first must identify the "primitive" characteristics (aspects of morphology, physiology, and behavior) shared with the ancestral species and the particular species' "cousins." Comparative studies clearly show that human language shares many primitive features with the communication systems of other species.

Lexical ability

Lexical ability, the ability to name objects, actions, and states of being, clearly is a primitive feature of human language. Although Chomsky (1976) claimed that no other living species can understand or communicate by means of words, dogs can understand a limited number of spoken words (Warden and Warner, 1928). Present-day chimpanzees exposed to nonverbal forms of human language can acquire about 150 words and devise new words and can modify the meaning of words that they already have (Gardner and Gardner, 1969, 1984; Savage-Rumbaugh et al., 1985; Savage-Rumbaugh and Rumbaugh, 1993). Moreover, monkeys develop predator-specific calls (Zuberbuhler, 2001). Baboons learn to respond to conspecific barks that have different referents, but have subtle acoustic distinctions (Fischer et al., 2000). It is quite probable that some aspects of lexical ability were present at the dawn of hominid evolution.

Syntax

Virtually all theoretical linguists have focused on syntax as the defining aspect of human language. Many linguists have claimed that syntax is totally absent in the communication of other species and propose that "protolanguage," that lacked any aspect of syntax, constituted the early stages of hominid language (e.g., Bickerton, 1990; Calvin and Bickerton, 2000; Pinker, 1994). This position is perhaps based on the erroneous claim of Terrace et al. (1979), who stated that chimpanzees using American Sign Language (ASL) were simply imitating the signs used by their human attendants. This clearly was not the case, since the chimpanzees in the study by Gardner and Gardner (1969) were observed signing the names of objects pictured in magazines that they were "reading" by themselves in much the same manner as young children. Moreover, those chimpanzees correctly answered questions such as, "What are you holding?" No chimpanzee has demonstrated syntactic ability equivalent to a neurologically intact human, raised in a "normal" environment. However, rudimentary syntactic ability limited to short sentences lacking embedded clauses is present in chimpanzees exposed to human language for prolonged periods starting in infancy. Chimpanzees can also understand simple spoken sentences that lack embedded clauses, deriving meaning from the sentence's syntax (Savage-Rumbaugh and Rumbaugh, 1993). Chimpanzees exposed to ASL use simple phrases (Gardner and Gardner, 1994). Moreover, a simple syntactic "rule" has been observed in the responses of monkeys to alarm calls: Diana monkeys respond differentially to the alarm calls of Cambell's monkeys (a different species), depending on the particular sequence of calls emitted by the Cambell's monkeys (Zuberbuhler, 2002). Therefore, it is most unlikely that the earliest hominids' command of syntax was less than that of language-trained chimpanzees. We can conclude that "protolanguage" most likely never existed in any hominid. The ability to generate and comprehend complex syntax, which involves switching sequences at clause boundaries, may be tied to the evolution of the cortical-striatal-circuits that regulate the production of human speech.

Brain size

In short, lexical ability and simple syntax are primitive characteristics. Though reduced in extent in living apes, these aspects of language are present in them. Evolution by means of natural selection, gradually increasing the neural memory base that constitutes the brain's dictionary, could in part account for our enhanced lexical ability. The gradual increase in brain size noted by Jerison (1973) and Deacon (1997) could, in part, account for increases in the size of the human neural dictionary. The focus by Deacon (1997) on the role of the prefrontal cortex and cerebellum in acquiring conceptual knowledge is consistent with research showing their increased activation during motor learning (Thach, 1996). Although neither the prefrontal cortex nor cerebellum appears to be disproportionately larger in the human brain compared to apes and other primates (Semendeferi et al., 1997; Stephan et al., 1981), the almost threefold increase in the volume of these structures and the basal ganglia, compared to chimpanzees, could have yielded the computational base and memory size necessary to rapidly learn and store the meanings of new words. Recent findings (Semendeferi et al., 2002) suggest that the posterior human brain, which current studies suggest is critical for accessing words from the lexicon (Damasio et al., 1996), is disproportionately large in humans compared to apes. However, memory size, in itself, is useless without the capacity to learn words. As noted above (Graybiel, 1995, 1997, 1998; Kimura et al., 1993; Lieberman, 2000; Marsden and Obeso, 1994; Mirenowicz and Schultz, 1996), the basal ganglia and prefrontal cortex play a critical role in cortical-striatal-cortical circuits implicated in learning motor control programs, cognitive sets, and most likely new words. The cerebellum clearly is implicated in motor learning, and may also take part in learning other aspects of behavior (Thach, 1996).

Fossil evidence for lateralization: Broca's and Wernicke's areas

A number of claims (e.g., Wilkins and Wakefield, 1995) concerning the presence of fully developed language in extinct hominids have been made that are based on being able to discern brain lateralization in the form of asymmetric Broca's and Wernicke's areas in the endocasts of fossil skulls. Wilkins and Wakefield (1995) claimed to discern evidence in fossil endocasts for a Chomskian universal grammar. Apart from the difficulties of discerning the presence of these cortical areas from endo-

casts, a process likened by Holloway (1995) to reading tea leaves, the presence of Broca's area or its homologue in an archaic hominid would not be an indisputable "proof" of fully developed human linguistic ability, since Broca's area is also implicated in manual motor control. Studies of aphasia have linked lesions in or near Broca's area to deficits in programming manual motor control (Kimura, 1993). Moreover, the homologue of Broca's area in monkeys contains mirror neurons implicated in manual motor control (Rizzolati and Arbib, 1998). Therefore, even if an extinct hominid's brain had a cortical area resembling Broca's area, that fact might reflect the result of adaptation for manual motor control.

Gestural language

Rizzolati and Arbib (1998) proposed that natural selection that initially enhanced manual motor control played a part in the evolution of Broca's area and human language, a view previously presented by Greenfield (1991), Hewes (1973), Kimura (1993), Lashley (1951), Lieberman (1975, 1984), and others. A stage at which manual gestures rather than speech was the phonetic medium for language has been proposed many times (e.g., Burling, 1993). The basis for this claim is the apparent discontinuity between the stereotyped vocalizations of nonhuman primates and human speech. However, studies that claim a total discontinuity between human speech and primate vocalizations fail to take account of the repertoire of phonetic contrasts shared by humans and other primates, which is not evident in the absence of quantitative acoustic analysis (Hauser, 1996; Lieberman, 1968, 1975; Fitch, 1997; Fischer et al., 2002).

Manual gestures continue to play a part in linguistic communication, even in hearing individuals (MacNeill, 1985), and adaptations that enhanced manual gestures may have played a part in the evolution of language. These adaptations may also have played a role in the evolution of the neural bases of vocal control, but speech is the default medium for human language, and the human brain is singularly adapted to regulate speech. Any theory for the evolution of human language must account for that fact. Hewes (1973), to whom contemporary gestural language theories are indebted, traced the development of gestural theories for the evolution of human language back to the 19th century.

Social factors

Many social factors have been cited as stimuli driving the natural selection that increased neocortical size, thereby enhancing lexical ability. In this context, Dunbar (1993) noted group size, cohesion, and grooming (though the brain size of solitary orangutans does not fit his model). Lieberman (1984) cited "collective insight," i.e., sharing of information among the members of a group so as to enhance problem-solving. However, it is the case that language enhances the conduct of virtually every aspect of human behavior that could enhance biological fitness. Paraphrasing Darwin (1859), in the infinitely complex relations of hominids to each other and external nature, language would have been an asset.

SPEECH AND COMPLEX SYNTAX

The derived features of human language clearly are the ability to talk and to regulate complex syntax. Although apes can, to a degree, comprehend human speech, they cannot vocally reply. The ability to produce human speech is a derived feature of human language, which as we shall see, enhances biological fitness—the edge in the Darwinian struggle for existence. The mechanism first proposed by Charles Darwin in *On the Origin of Species* (1859, p. 190), that "an organ might be modified for some other and quite distinct purpose," may provide insights into the evolution of this derived feature of human language and the nature of the "rules" of syntax. The role of the striatal sequencing engine in both speech and syntax (as well as in other aspects of cognition) suggests that the neural substrate that regulated motor control in the common ancestor of apes and humans apparently was modified to enhance cognitive and linguistic ability. Similar neural computational processes appear to govern motor activity and syntax; adaptations for speech communication may have played a central role in this process.

Speech

Despite many attempts, spanning several centuries, to teach apes to talk, they cannot speak. Human speech achieves its productivity by altering the sequence in which a limited number of speech sounds occur. The words "see" and "me" contain the same vowel; the initial consonants signify different concepts. Changing vowels also signifies different words: "sue, ma, sit, mat." Human speakers are able to alter the sequence of articulatory gestures that generate the meaningful speech sounds, the "phonemes," that convey the words of their language. Different languages have particular constraints, but any neurologically intact child raised in a "normal" environment learns to speak his or her native language or languages. But speech production arguably is the most complex motor activity "normally" attained in the course of human development. Normal children do not even attain the ability to talk with the articulatory precision or speed of adults until age 10 years (Smith, 1978).

In contrast, apes appear to lack the neural capacity to even freely alter the sequence of muscle commands that generate phonemes. Anatomical limitations, which will be discussed below, limit the range of phonetic forms that apes could produce. However, acoustic analysis of the vocal signals that they produce in a state of nature reveals many of the segmental phonetic elements that could be used to form

spoken words. Chimpanzee vocalizations, for example, include segments that could convey the consonants [m], [b], and [p] and the vowel of the word "but" (Lieberman, 1968; Hauser, 1996). Computer modelling studies (Lieberman et al., 1972) show that chimpanzee speech apparatus is capable of producing the sounds [n], [d], and [t] and most vowels other than [i], [u], and [a] (the vowels of the words "tea," "too," and "ma"). However, chimpanzees are unable to voluntarily speak any of the words of English that could be formed from the range of speech sounds that their vocal apparatus could generate. Exhaustive field observations of chimpanzee communication reveal that their vocal signals are bound to particular emotions or situations. They cannot alter these stereotyped vocal calls, and even have great difficulty suppressing their calls in situations where that would appear to be warranted (Goodall, 1986). Similar constraints limit the vocal communications of other primate species. Male baboon cries signaling different "semantic" concepts (predator alarm calls vs. contact with other baboons) are limited to a narrow acoustic range (Fischer et al., 2002). Thus, if we follow the logic of evolutionary biology, we must conclude that speech production is perhaps the primary derived characteristic of human language. And, although virtually all theoretical linguists have focused on syntax for the past 40 years, we must account for the evolution of the anatomy and neural apparatus that makes human speech possible.

The selective advantages of human speech

In most discussions of human language, little attention is paid to the rate of information transfer. At normal speaking rates, 20–30 phonemes per second are transmitted from a speaker to listeners. This rate exceeds the temporal resolving power of the human auditory system; individual nonspeech sounds merge into a buzz at rates in excess of 15 per second (Liberman et al., 1967). Indeed, it is difficult to even count more than 7 sounds in one second. Studies of the evolution of the physiology of speech production indicate that the anatomical structures involved have been modified from the common ancestor of apes and humans to facilitate this process (Bosma, 1975; Lieberman and Crelin, 1971; Lieberman et al., 1972; Lieberman, 1984; Negus, 1949). The relevant point is that the pongid airway inherently lacks the capacity to produce speech sounds that facilitate the process of speech perception.

A short discussion of the anatomy and physiology of speech production may be useful in understanding the contribution of human speech to human linguistic ability. The physiology of speech production has been studied since the time of Müller (1826). The lungs power speech production. The outward flow of air from the lungs is converted to audible sound by the action of the larynx, which during "phonation" generates a series of quasiperiodic puffs of air as the vocal cords rapidly open and close. The larynx operates in much the same manner as the reed of a woodwind instrument, producing acoustic energy which is then shaped into the notes of a musical composition by the air passages above it. Acoustic energy ("noise") can also be generated by air turbulence at a constriction, in much the same manner as the source of acoustic energy in a flute or organ. The length of the air passage above the reed or turbulent energy source acts as a "filter," allowing maximum acoustic energy through at a particular frequency or "note." The differing tube lengths of a pipe organ act in a similar manner: long pipes produce "low" notes, short pipes "high" notes. The airway above the human larynx filters the acoustic energy produced by the larynx or the noise sources of consonants, except that the perceptual bases of the sounds of speech are the particular frequencies at which maximum acoustic energy occurs.

The supralaryngeal vocal tract

The shape of the airway above the larynx, termed the "supralaryngeal vocal tract" (SVT), is continually modified as a person talks, in effect yielding a plastic air passage that can dramatically change its shape. Major changes in SVT shape are produced by the tongue. The human tongue, which extends down into the pharynx, can be depressed or elevated backwards or forward to dramatically change the shape of the SVT (Nearey, 1979). The position and degree of constriction of the lips and vertical position of the larynx, which can move up or down about 25 mm, also can change the SVT configuration. The filtering properties of the SVT are determined by its shape and overall length (Chiba and Kajiyama, 1941; Fant, 1960; Perkell, 1969; Stevens, 1972). Peak energy can potentially be transmitted through the SVT at particular "formant frequencies." Systematic research since the end of the 18th century (Hellwag, 1781) shows that the phonetic properties of many speech sounds are determined by these formant frequencies. The vowel [i] of the word "see," for example, differs from the vowel of [a] of the word "ma" solely because of its different formant frequencies. The pitch of a person's voice, which is determined by the rate at which the vocal cords of the larynx open and close, has no effect on formant frequencies.

The rapid transmission rate of human speech follows from the "encoding" of these formant frequencies. As a person talks, the SVT continually changes its shape, thereby changing the formant frequencies that specify the "phonemes" that make up words. However, the shape of the SVT changes gradually; for example, it is impossible to move the lips open instantly from the position that is necessary to produce the consonant [b] of the word "bat" into the position necessary to produce the word's vowel. Nor can a person's tongue instantly move from the position that produces the formant frequencies that convey the vowel of "bat" to produce the [t] sound. Therefore, the articulatory gestures that make up the initial and final consonants and vowel of "bat"

gradually flow into a unit, melded together to form a syllable in which the formant frequencies are also melded together. It is impossible to isolate the formant frequencies of the individual sounds that constitute the syllable. In the case of words like "bat," all three phonemes are transmitted together, or "encoded," as a syllabic unit. In perceiving speech, human listeners "decode" the merged formant frequency pattern, recovering the three phonemes that were transmitted as a package at the slower syllabic rate.

The rapid information transfer rate and complex conceptual and syntactic properties of human language derive from the encoded nature of human speech. It is impossible to comprehend the meaning of a complex sentence when individual phonemes are transmitted at the slow nonspeech rate. Nonspeech sounds fare no better. Research at Haskin's Laboratories in the 1960s showed that listeners forgot the beginning of a sentence before reaching its end when arbitrary acoustic signals were used in place of speech (Liberman et al., 1967). Moreover, the listeners' full attention was occupied with transcribing these nonspeech signals. In contrast, human listeners effortlessly derive the sounds that make up the "encoded" syllables of speech by means of a process that involves an implicit neural representation or knowledge of the constraints of speech production (Liberman et al., 1967). We perceptually "decode" the syllables to recover the sounds of speech using some knowledge of speech production. The decoding process must take into account the length of the SVT that produced a speech sound (Nearey, 1979), since the absolute values of the formant frequencies produced by a long SVT are lower than those of a shorter SVT.

Studies of speech-decoding shed light on the evolution of human language. The perceptual mechanism that allows humans to perform this feat has a long evolutionary history; it is a primitive attribute of human linguistic ability used by other species to gauge the size of a conspecific by listening to its vocalizations (Fitch, 1997). The length of a monkey's upper airway, its SVT, is highly correlated with its height and weight. The vocal calls of a large monkey have lower formant frequencies than a smaller monkey's, and other monkeys can gauge its size by listening to it. Many species make use of this mechanism. Human beings can make similar estimates of body size and height (Fitch, 1994). This innate, primitive perceptual mechanism, which is apparent at age 3 months in infants (Lieberman, 1984), is used by human listeners as they perceive speech to decode the formant frequency patterns. Talkers with different SVT lengths can produce different formant frequencies when they say the same words. For example, an adult woman's [a] can have the same formant frequencies as an adolescent girl's [ae] vowel. Listeners, therefore, must have some knowledge of the length of a speaker's SVT in order to relate specific formant frequency patterns to the phonemes that the speaker intended to communicate. In this regard, experimental studies show that listeners often confuse one word for another when faced with the problem of rapidly shifting from one speaker's voice to another's (Peterson and Barney, 1952; Ladefoged and Broadbent, 1957; Hillenbrand et al., 1995).

However, under most conditions, human listeners rapidly adjust to the speech produced by speakers having different SVT lengths; the most effective vowel sound for this purpose is that of the word "see" -- [i] in phonetic notation. Many studies have shown that [i] is the "supervowel" of human speech. It is less often confused with other sounds (Peterson and Barney, 1952; Hillenbrand et al., 1995). Words formed with the vowel [i] are correctly pronounced more often by young children as they acquire their native languages (Olmsted, 1971). This follows because the vowel [i] is less confused, because it yields an optimal reference signal from which a human listener can determine the length of the supralaryngeal vocal tract of a speaker's voice (Nearey, 1979; Fitch, 1994, 1997).

Anatomically modern human beings are the only living species who can produce the vowel [i] (Lieberman and Crelin, 1971; Lieberman et al., 1972; Lieberman, 1968, 1975, 1984; Carre et al., 1995). The adult human tongue and SVT differ from those of all other living animals (Negus, 1949). In apes, the body of the tongue is long and relatively flat, and fills the oral cavity. During swallowing, the tongue propels food through the oral cavity. The nonhuman larynx is positioned high and can lock into the nasopharynx, forming an air pathway sealed from the oral cavity, thereby enabling an animal to simultaneously breath and drink or swallow small food particles. In contrast, the posterior portion of human tongue in a midsaggital view is round, and the larynx is positioned low. The resulting human supralaryngeal airway has an almost right-angle bend at its midpoint. As we talk, extrinsic tongue muscles can move the tongue upwards, downwards, forwards, or backwards, yielding abrupt and extreme changes in the cross-sectional area of the human supralaryngeal airway at its midpoint (Chiba and Kajiyama, 1941; Fant, 1960; Nearey, 1979). The vowel sounds that occur most often in the languages of the world (Jakobson, 1990; Greenberg, 1963; Maddieson, 1984), [i], [u], and [a] (the vowels of the words *see, do,* and *ma*), can only be formed by extreme area function discontinuities at the midpoint of the human supralaryngeal vocal tract (Lieberman et al., 1972; Stevens, 1972; Carre et al., 1995).

Examination of the skull and airways of newborn human infants and apes shows that the base of the skull and hard palate (the roof of the mouth) support SVTs in which the tongue is positioned almost entirely within the oral cavity. Acoustic analyses of the speech of human infants and living apes, and computer modelling of the possible range of speech sounds that they and the reconstructed SVTs of

early australopithecine hominids, whose skull bases resemble those of apes, could have made, show that they could not have produced the vowel [i], as well as the vowels [u] and [a] (Lieberman and Crelin, 1971; Lieberman et al., 1972; Carre et al., 1995). Between birth and age 6 years, the skeletal structure and soft tissue of the human skull, tongue, and other aspects of human anatomy that define the SVT restructure. The human face moves backward from its birth position; the human face is almost in line with the forehead (Lieberman DE, 1998; Lieberman and McCarthy, 1999). In contrast, the faces of apes follow a diametrically opposite growth pattern. Their faces instead gradually project forwards, yielding long, thin tongues positioned almost entirely in long mouths. Neanderthal hominids, who survived until about 35,000 years ago, appear to have followed the nonhuman growth trajectory (Vleck, 1970; Lieberman DE, 1998). The claims of Boe et al. (1999) to the effect that newborn human infants can, and Neanderthals could, produce the vowel [i] are based on their modelling a supposed "newborn" vocal tract that in actuality is similar to that of a 5-year-old human child who has already attained an adult-proportioned SVT in which the pharynx and oral cavities have almost equal lengths: a criterion necessary to produce the vowel [i]. The SVTs of newborns and children younger than age 5 years do not have this pharynx-to-oral-cavity proportion (Lieberman et al., 2001), which is necessary to generate the vowel [i]. The computer modelling technique employed by Boe et al. (1999) also inherently forces the parameters modelled into the right-angle-bend SVT configuration characteristic of human adults (Lieberman, 1984). In short, the putative "infant" SVT modelled by Boe et al. (1999) bears little relation to newborn SVTs documented in independent anatomical studies (Negus, 1949; Bosma, 1975; Lieberman and Crelin, 1971) and in studies based on cephalometric radiographs or MRI images of living subjects (Lieberman et al., 2001; Fitch and Giedd, 1999). Indeed, the "infant" SVT modelled by Boe et al. (1999) is virtually identical to that of the 5-year-old pictured in the MRI study by Fitch and Giedd (1999).

WHEN DID HUMAN SPEECH, COMPLEX SYNTAX, AND THE LEXICON EVOLVE?

Paradoxically, the phonetic deficits of living apes and probable speech deficits of archaic hominids suggest that the neural substrate that regulates speech evolved gradually, long before the appearance of anatomically modern human beings. The role of the basal ganglia in the cortical-striatal-cortical circuits that regulate speech and syntax also suggests that syntactic capabilities gradually evolved. The primary life-supporting functions of the mouth, pharynx, throat, and anatomical components of the SVT are eating, swallowing, and breathing. These functions are, as Darwin (1859) and anatomists such as Negus (1949) noted, impeded by the human SVT. In apes and newborn human infants, the larynx is positioned close to the base of the skull. When swallowing, the infant larynx moves upwards, forming a tight seal with the opening to the nose. Infants, apes, and virtually all other mammals can simultaneously drink or swallow small pieces of solid food while they breathe. The low position of the adultlike human larynx, and the shape and position of the human tongue, necessitate solids and liquids being propelled past the opening to the larynx. Foreign matter lodged in the larynx results in death. Chewing is also less efficient in the shorter human mouth; our teeth are crowded, and molars can become impacted and infected, resulting in death in the absence of dental intervention.

The only apparent selective advantage that the human SVT yields is to enhance the robustness of speech reception; a human face, tongue, and pharynx are necessary to produce the quantal vowel [i] which is an optimal signal for the preexisting, phylogenetically primitive neural process for estimating SVT length. This yields the conclusion that speech communication has a long evolutionary history, probably extending back to the earliest phases of hominid evolution. There would have been no selective advantage for the retention of variations that yielded the modern human SVT in the absence of some form of speech. Speech that lacked the optimal vowels [a], [i], and [u] of human speech still would have sufficed as a means of rapid vocal communication. It would not have been as error-free as modern human speech, but it nonetheless would have been a medium for rapid vocal information transfer. But as comparative studies of living apes show, any form of voluntary speech necessitates a neural substrate that can learn, sequence, and execute a series of complex motor acts. Thus, before the evolution of the modern human SVT, the neural substrate that sequences the motor pattern generators that generate speech must have been in place. In short, speech lacking the full phonetic range of modern humans must have been the mode of linguistic communication. In the absence of some form of speech, there would have been no selective advantage for the anatomical development of the face, skull, and vocal tract that marks anatomically modern human beings.

Complex syntax

It also is probable that complex syntactic and cognitive ability existed well before the appearance of modern human beings. The studies noted here demonstrate the interwoven nature of the neural bases of speech and syntax. The mark of evolution is apparent in the brain bases of human speech and syntax. The studies noted above show that human basal ganglia carry out similar sequencing operations in the cortical-striatal-cortical circuits implicated in speech, abstract cognition, and syntax. In comprehending a sentence with simple "canonical" syntax, such as "The boy is fat," only one syntactic "pattern

generator" (syntactic scheme or set of "rules") need be invoked. The words "the boy" constitute a noun-phrase (NP) that is the sentence's subject. The words "is fat" constitute a verb phrase (VP) that is the sentence's predicate. The syntactic "formula" Sentence = NP + VP describes the basic syntax of the sentence. In contrast, comprehending a complex sentence, such as "The boy who is fat fell down," entails being able to interrupt the sequence of cognitive acts involved in interpreting syntax at the relative clause's boundary (the word "who"), where an "embedded" sentence has been inserted into the canonical form. As syntactic complexity increases, different "rules" of syntax must be continually suppressed and invoked. As Marsden and Obeso (1994) and Graybiel (1997, 1998) noted, the basal ganglia appear to perform similar cognitive and motor processing sequencing functions, i.e., interrupting an ongoing activity and starting a different act when appropriate. The basal ganglia most likely are active in acquiring the cognitive pattern generators that are relevant to the syntax of language: the "rules of grammar," and perhaps the acquisition of the increased store of words that marks human lexical ability. Further study obviously is necessary.

Walking

As noted above, a half century ago, Lashley (1951) explicitly pointed out the similarities between the syntax of motor control and language. Data-linking deficits in serial manual motor control, speech, and syntax in aphasia suggest that neural mechanisms initially adapted for motor control were modified in the course of human evolution to yield syntactic ability (Lieberman, 1975, 1984, 1985), a view independently shared by Kimura (1993). Studies of the acquisition of gesture and speech by children support this view (Greenfield, 1991); a seamless transition between gesture and speech occurs in the course of maturation. Neurophysiologic studies of motor control and cognition (Graybiel, 1997, 1998), and the studies of neurodegenerative diseases noted herein, show that the basal ganglia support neuronal populations that carry out similar operations in the domains of motor control, cognition, and syntax. An insight regarding the start of the evolutionary process that yielded the neural bases that allow human languages to sequence the sounds of speech (syntactic rules), and that confer flexability to human thought, may follow if we take account of the role of basal ganglia in walking (Hochstadt, personal communication). Although the striatal components of human cortical-striatal-cortical neural circuits play a role in language and cognition, they continue to regulate motor control. In Parkinson's disease, one of the most apparent behavioral deficits of degraded basal ganglia function is the marked deterioration of walking and upright balance. The PD rating scale of Hoehn and Yahr (1967) is essentially a measure of a patient's ability to recover a stable upright position after being gently perturbed. Moderate to severe PD generally results in deficits in a person's ability to sequence internally generated motor sequences (Cunnington et al., 1995; Harrington and Haaland, 1991; Jellinger, 1990), and one of the most symptomatic signs is impaired ability to execute the complex sequence of motor acts involved in upright bipedal locomotion. Moreover, upright human bipedal locomotion appears to be learned rather than innate (Thelen and Cooke, 1987), and the basal ganglia (Mirenowicz and Schultz, 1996; Graybiel, 1995) as well as cerebellum and prefrontal cortex (Thach, 1996) are all implicated in motor learning. Given the role of the basal ganglia in the motor control necessary for upright locomotion, natural selection directed at enhancing upright bipedal locomotion and subsequent Darwinian "preadatation" (the direction of the basal ganglia "sequencing engine" to speech production, syntax, and thinking) may have been triggered in the earliest phases of hominid evolution.

The archaelogical record

The archaeological record has often been used to date the appearance of the modern human mind (Klein, 1999). Until recently, the artifacts associated with modern human beings appeared to occur after 40,000 years ago. However, it is apparent that artifacts associated with anatomically modern humans appeared in Africa well before that time (McBrearty and Brooks, 2000). Moreover, the modern human mind and full linguistic capabilities must have evolved before the probable dispersal of modern humans from Africa, since any neurologically intact child from any location on earth can effortlessly acquire any language when exposed to it in a normal environment before age 7 years. Since modern humans reached Australia about 60,000 years ago, the human brain must have reached its present state of development long before that date.

CONCLUDING COMMENTS

The neural bases of human linguistic ability are complex, involving structures other than Broca's and Wernicke's areas. Although our knowledge is at best incomplete, it is clear that many other cortical areas and subcortical structures form part of the neural circuits implicated in the lexicon, speech production and perception, and syntax. The subcortical basal ganglia support the cortical-striatal-cortical circuits that regulate speech production, complex syntax, and the acquisition of the motor and cognitive pattern generators that underlie speech production and syntax. They most likely are involved in learning the semantic referents and sound patterns that are instantiated as words in the brain's dictionary. The cerebellum and prefrontal cortex are also involved in learning motor acts. Frontal regions of the cortex are implicated in virtually all cognitive acts and the acquisition of cognitive criteria; posterior cortical regions are clearly active elements of

the brain's dictionary. The anterior cingulate cortex plays a part in virtually all aspects of language and speech. Real-word knowledge appears to reflect stored conceptual knowledge in regions of the brain traditionally associated with visual perception and motor control. Some aspects of human linguistic ability, such as the basic conceptual structure of words and simple syntax, are phylogenetically primitive and most likely were present in the earliest hominids. Speech production, complex syntax, and a large vocabulary developed in the course of hominid evolution, and *Homo erectus* most likely talked, had large vocabularies, and commanded fairly complex syntax. Full human speech capability, enhancing the robustness of vocal communication, most likely is a characteristic of anatomically modern humans.

The computational architecture and neurophysiology of the human brain and comparative evidence suggest that neural systems that enhanced adaptive motor control may have been the starting point for the evolution of human speech and complex syntax. Given the involvement of the basal ganglia in the cortical-striatal-cortical circuits regulating upright bipedal locomotion, one of the first derived hominid features, adaptations aimed at enchancing walking may have initiated the process that yielded the neural bases of human linguistic ability.

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