Human Language and Our Reptilian Brain

the subcortical bases of speech, syntax, and thought

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For the past 200 years, virtually all attempts to account for the neural bases and the evolution of human language have focused on the neocortex. And in the past 40 years, linguists adhering to Noam Chomsky's theories have essentially equated language with syntax, hypothetically specified by an innate, genetically transmitted "universal grammar." In *Human Language and our Reptilian Brain* (2000), I attempt to shift the focus. My premise is that speech is the central element of human linguistic ability and both speech and syntax are learned skills, based on a neural "functional language system" (FLS). Although neither the anatomy nor the physiology of the FLS can be specified with certainty at the present time, converging behavioral and neurobiological data point to language being regulated by a distributed network that crucially involves subcortical structures, the basal ganglia, often associated with reptilian brains though they derive from amphibians.

Like other distributed neural systems that regulate complex behavior, the architecture of the FLS consists of circuits linking segregated populations of neurons in structures distributed throughout the brain, cortical and subcortical, including the traditional "language" areas (Broca's and Wernicke's areas) as well as other neocortical areas. The FLS rapidly integrates sensory information

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with stored knowledge; it is a dynamic system, enlisting additional neural resources in response to task difficulty. Regions of the frontal lobes of the human neocortex, implicated in abstract reasoning and planning, and other cortical areas are recruited as task difficulty increases. Since natural selection selects for timely responses to environmental challenges, it is not surprising that the FLS also provides direct access to the information coded in a word, i.e., primary auditory, visual, pragmatic, and motoric information. The mental operations carried out in the brain are not compartmentalized in the "modules" proposed by most linguists and many cognitive scientists. The neural bases of human language are intertwined with other aspects of cognition, motor control, and emotion.

The human FLS is unique; no other living species possesses the neural capacity to command spoken language, which serves as a medium for both communication and thought. The FLS appears, however, to have evolved from neural structures and systems that regulate adaptive motor behavior in other animals. In this light, the subcortical basal ganglia structures usually associated with motor control that are key elements of the FLS reflect its evolutionary history: natural selection operated on neural mechanisms that yield adaptive—that is, "cognitive"—motor responses in other species. There is no reason to believe that the basic operations of the human brain differ for motor control and language. Insights gained from the study of the neural bases of motor control apply with equal force to human language; although the neural architecture that regulates motor control and syntax is part of our innate endowment, the details are learned. And the early stages of the evolution of the cortico-striatal neural circuits that regulate human language and thought may have been shaped by natural selection to meet the demands of upright bipedal locomotion, the first defining feature of hominid evolution.

This evolutionary perspective may not be familiar to some cognitive scientists, linguists, and philosophers. I hope, however, that biological linguists working in an evolutionary framework will lead the way to new insights on the nature of language. Paraphrasing Dobzhansky, "Nothing in the biology of language makes sense except in the light of evolution."

FUNCTIONAL NEURAL SYSTEMS

The traditional view of the neural bases of human language derives from 19th-century phrenology. Phrenologists claimed that discrete parts of the brain, which could be discerned by examining a person's cranium, were the "seats" of various aspects of behavior or character. Neo-phrenological theories do not claim that a bump on your skull shows that you are virtuous, but phrenology lives on in the traditional Broca-Wernicke model of the neural bases of language. In 1861, Broca ascribed the word-finding difficulties and speech production deficits of his patient to damage to a frontal region of neocortex,

Broca's area. Shortly after, "receptive" deficits involving comprehension were ascribed to damage to a posterior area of cortex, Wernicke's area (Wernicke 1874). Lichtheim's 1885 model, subsequently reiterated by Geschwind (1970), claimed that the neural basis of human language was a cortical system linking Wernicke's area with Broca's area. According to this model, Wernicke's area processes incoming speech signals; information is transmitted via a cortical pathway to Broca's area, which serves as the "expressive" language output device. The Lichtheim-Geschwind theory is taken by linguists such as Chomsky (1985) and Pinker (1994) to be a valid model of the neural architecture underlying human linguistic ability. According to Pinker, "Genuine language . . . is seated in the cerebral cortex, primarily the left perisylvian region" (p. 334), and the "the human language areas [comprise] Wernicke's and Broca's areas and a band of fibers connecting the two" (p. 350).

Although the Lichtheim-Geschwind model has the virtue of being simple, neurophysiological studies show that it is wrong. Different regions of neocortex are specialized to process particular stimuli, visual or auditory; other regions participate in regulating motor control or emotion or holding information in short-term (working) memory, etc. But complex behaviors, such as looking at and reaching for an object, are regulated by neural circuits that constitute distributed networks that link activity in many different neuroanatomical structures. As Mesulam notes, "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" (1990, p. 598). A given neuroanatomical structure typically supports many segregated neuronal populations that project to different parts of the brain, forming circuits that regulate different aspects of behavior.

In other words, although specific operations are performed in particular parts of the brain, a particular behavior is regulated by a network, a "functional neural system," that integrates activity in structures distributed throughout the brain. Studies that relate brain activity to behavior in humans and other species show that a class of functional neural systems generates timely responses to environmental challenges and opportunities. These functional neural systems integrate incoming sensory information with an animal's knowledge base and modify or generate goal-directed motor activity that enhances biological fitness. The postulated human "functional language system" (FLS) regulates language, the derived feature that sets human beings apart from other living species.

The FLS is a distributed network that includes basal ganglia structures that regulate sequencing in seeming unrelated activities such as moving one's fingers, talking, comprehending distinctions in meaning conveyed by syntax, and solving cognitive problems (Cunnington et al. 1995; Grossman et al. 1991, 1993; Lange et al. 1992; Lieberman et al. 1990, 1992; Natsopoulos et al. 1993; Pickett et al. 1998). Basal ganglia sequence the pattern generators governing

motor activity and cognitive operations, by means of segregated neuronal populations that project to neuronal populations in other subcortical structures and cortical areas throughout the brain.

THE FLS AND BASAL GANGLIA: CORTICO-STRIATAL CIRCUITS

Two sources of evidence are discussed in some detail in *Human Language and our Reptilian Brain*. Since only human beings possess an FLS that regulates spoken language and complex cognitive behavior, it is not possible to employ highly invasive techniques that might reveal the its neural circuitry or the computations that are effected in its component neuroanatomical structures. Since human physiology is manifestly similar to that of other species, however, valid inferences concerning the human brain can be derived from the study of the brains of other species. Comparative neurophysiological studies of other species have revealed many aspects of basal ganglia circuitry and function. In some instances, comparable studies of human brains are feasible. These studies clearly show that neural circuits link basal ganglia structures and cerebellum to prefrontal cortical areas implicated in cognition, as well as to cortical areas associated with motor control.

Experiments in nature involving human subjects constitute the second line of inquiry. Studies of the behavioral effects of brain damage resulting from trauma or disease demonstrate that subcortical structures are essential components of the FLS. While language often recovers after humans suffer cortical damage, perhaps reflecting cortical plasticity (Elman et al. 1996), damage to subcortical circuits results in permanent language deficits (Stuss and Benson 1986). Speech, lexical access, the comprehension of meaning conveyed by sentences, and various aspects of higher cognition are regulated by parallel circuits that involve basal ganglia and other subcortical structures, as well as other neocortical structures.

One of the major findings of clinical studies over the past two decades is that behavioral changes once attributed to frontal-lobe cortical dysfunction can be observed in patients having lesions in subcortical basal ganglia. Cummings (1993) identifies five parallel basal ganglia circuits of the human brain:

> 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. (Cummings, 1993, p. 873)

REPRESENTATIVE EXPERIMENTAL DATA

The Syntax of Rat Grooming

Most linguists believe that the defining characteristic of human linguistic ability is syntax, which binds a finite number of words into "well-formed" sentences that can convey an unbounded set of meanings. Studies of rodents show that they too make use of a "syntax," regulated in basal ganglia, to bind individual movements into "well-formed" grooming programs. The grooming movements of rats do not convey an unbounded set of meanings, or perhaps any meaning to other rats. However, experiments performed on rats show that show that damage to the striatum disrupts the integrity of the *sequences* of gestures that normally occur, but does not disrupt the individual gestures that would make up a grooming sequence. In other words, the "syntax" of grooming is regulated in the basal ganglia. Damage to other neural structures—prefrontal cortex, primary or secondary motor cortical areas, or cerebellum—does not affect the grooming sequence. Electrophysiological data confirm the role of basal ganglia in regulating the sequence in which these individual movements (e.g., forelimb strokes versus body licks) occur (Aldridge et al. 1993).

Learned Behavior

Rats raised completely isolated from other rats execute the same grooming pattern, demonstrating its innate nature. And so it is likely that rodent grooming patterns are coded by a genetically transmitted "universal grooming grammar," analogous to the hypothetical universal grammar that, according to Chomsky, determines the syntax of all human languages. Although most theoretical linguists accept some form of Chomsky's theory, many studies suggest that human beings learn the particular syntax of the languages that they command by means of cognitive processes and neural mechanisms similar in manner and kind to those employed in learning to play a violin, tennis, or even walking (for reviews, see Elman et al. 1996; Lieberman 1991, 2000). Neurobiological studies reveal the role of basal ganglia in the acquisition of learned behavior.

Basal ganglia circuits regulating learned motor tasks appear to be shaped by associative processes. Kimura, et al. (1993), studied the responses of striatal interneurons as monkeys learned a classic Pavlovian conditioned motor task—a sound preceded a task that earned a reward. The data showed that basal ganglia "coded" the learned response. The independent studies of Graybiel and her colleagues (1994) confirm these results. Dopamine sensitive striatal interneurons respond contingent on reward. The striatal architecture noted by Graybiel, et al. (1994), could carry out both associative Hebbian learning and supervised learning in a manner similar to current computer-implemented models of distributed neural networks (Elman et al. 1996). Other independent studies, in which monkeys learned tasks when they were rewarded with fruit juice,

confirm the role of reward-based, "appetitive," activation of midbrain dopamine sensitive neurons.

Human Finger Sequencing

Techniques that indirectly monitor human basal ganglia activity yield data consistent with these invasive electrophysiological studies of motor control. Human basal ganglia circuits regulate sequential, self-paced, manual motor control tasks. Depleted production of the neurotransmitter dopamine degrades basal ganglia activity in Parkinson's disease, largely sparing the cortex (Jellinger 1990). Abnormalities in motor sequencing are one of the signs of Parkinson's (Harrington and Haaland 1991). Cunnington and his colleagues (1995) monitored the activity of the supplementary motor area of the cortex in normal subjects and in patients with Parkinson's disease by means of movement-related potentials, electrical signals that are emitted before a movement. Subjects pushed buttons with their index fingers in various experimental conditions; EEG signals were recorded from the supplementary motor area before and during each button-push. The button-pushing data reveal basal ganglia activity similar to that noted by Aldridge and his colleagues (1993) for rats, as well as studies of spatial sequencing in monkeys that make use of invasive techniques. The basal ganglia

> activate the preparatory phase for the next submovement, thereby switching between components of a motor sequence. Since the basal ganglia and supplementary motor area are more involved in temporal rather than spatial aspects of serial movement, this internal cueing mechanism would coordinate the switch between motor components at the appropriate time, thus controlling the timing of submovement initiation. (Cunnington et al. 1995, p. 948)

Stereotaxic Surgery

Studies of the effects of surgery on humans offer another source of data concerning basal ganglia function. "Stereotaxic" surgical techniques have been perfected that selectively destroy basal ganglia structures or the targets of circuits from these structures in thalamus. Thousands of operations were performed before Levodopa treatment was available to offset the dopamine depletion that is the immediate cause of Parkinson's disease. In many instances, these operations reduced the debilitating rigidity and tremor of patients with Parkinson's. Marsden and Obeso (1994) review the outcomes of these surgical interventions and similar experimental lesions in monkeys. They address the seeming paradox that surgery that destroys subcortical structures, known to regulate various aspects of motor control, reduces tremor and rigidity but has little effect on motor control. As Marsden and Obeso note, the reason appears to be the distributed parallel nature of the basal ganglia system regulating motor control:

> Neurons in supplementary motor area, motor cortex, putamen and pallidum, all exhibit very similar firing characteristics in relation to movement. . . . Within each of these various motor areas, neuronal populations seem to be active more or less simultaneously, rather than sequentially. They appear to cooperate in an overall distributed system controlling the shape of movement (Marsden and Obeso 1994, p. 886)

Marsden and Obeso suggest that the basal ganglia have two different motor control functions in human beings:

> 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. (p. 889)

Many studies show that the basal ganglia circuitry implicated in motor control does not radically differ from that implicated in cognition. Marsden and Obeso conclude:

> 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 allow 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. . . . (p. 893)

Aphasia

Marsden and Obeso's conclusions are supported by the studies of aphasia that have structured theories of mind and brain for more than a century. Although the most apparent linguistic deficit of the syndrome named for Broca, "Broca's aphasia," is labored, slow, slurred speech, other disruptions to normal behavior can occur, such as deficits in fine manual motor control and oral apraxia (Stuss and Benson 1986). Broca's aphasics often have difficulty executing either oral or manual sequential motor sequences (Kimura 1993). Higher-level linguistic and cognitive deficits also occur. The utterances produced by Broca's aphasics were traditionally described as "telegraphic." When telegrams were a means of communication, the sender paid by the word, and "unnecessary" words were eliminated; hence the utterances of English-speaking aphasics who produce messages such as "man eat fish" have a telegraphic quality. Aphasic telegraphic utterances are often thought to be a compensatory behavior: aphasic speakers presumably produce short utterances to minimize

their speech production difficulties. It is evident, however, that Broca's aphasics also have difficulty comprehending distinctions in meaning conveyed by moderately complex syntax. Although agrammatic aphasics are able to judge whether sentences are grammatical, albeit with high error rates, the comprehension deficits of Broca's aphasics have been replicated in many independent studies (cf Blumstein 1995). Nonlinguistic deficits also occur; Kurt Goldstein (1948) noted the loss of the "abstract capacity," deficits in planning and deriving abstract criteria, and "executive capacity generally associated with frontal lobe activity."

Acoustic analyses show that the characteristic speech production deficit of Broca's syndrome is impaired sequencing. It is first useful to note the aspects of speech production that are *not* impaired in Broca's aphasia. The production of the formant frequency patterns that specify vowels and consonants is unimpaired, though there is increased variability (Ryalls 1986). Since formant frequency patterns are determined by the configuration of the supralaryngeal vocal tract (primarily tongue and lip activity) we can conclude that the control of these structures is unimpaired. The "encoding" or "melding" of formant frequency patterns (Liberman et al. 1967) which characterizes the production of human speech is likewise preserved in Broca's aphasia.

Speech Encoding

A short digression on the nature and selective advantage of speech encoding is perhaps germane. Speech encoding is one of the keys to human linguistic ability. It allows us to communicate rapidly, transcending the limits of the human auditory system and the bounds of short-term auditory memory. As the supralaryngeal vocal tract configuration gradually changes, so do the formant frequencies. The result is an acoustic melding of the formant patterns that specify individual sounds into syllable-sized units (Liberman et al. 1967). For example, it is impossible to produce the isolated sound [b] without also producing a vowel or "continuant" such as [ba] or [bs]. The formant frequency transitions specify the initial consonant as well as the vowel—consonant and vowel are fused into a syllable. This process yields the high-information transfer rate of human speech: the encoded syllables are transmitted at a rate that does not exceed the fusion frequency of the auditory system, and listeners then resolve the encoded syllables into the phonetic code. The process yields a transmission rate of 20 to 30 phonetic units per second, exceeding the fusion frequency of the human auditory system. If we were forced to communicate at the slow syllabic rate, we would forget the beginning of a complex sentence before we heard its end.

The encoding process involves two factors. Inertial "coarticulation" effects inherently encode the formant frequency pattern. For example, when producing the sound [t] of the syllable [ta], the tongue blade initially must be in contact with the palate. The tongue can not move instantly away from the palate

to the lower position necessary for [a]. Consequently, the formant frequency pattern gradually changes as the tongue moves from its syllable-initial position to the [a] position. A similar effect holds for the syllable [tu] except that the consonant "transition" flows into the formant frequency pattern of the vowel [u]. However, inertia cannot account for "anticipatory" coarticulation. Human speakers plan ahead as they talk, anticipating sounds that will occur. Speakers, for example, round their lips (move their lips forward and towards each other) at the very start of the syllable [tu], anticipating the vowel [u]. They do not do this when they produce [ti], because the vowel [i] is produced without lip-rounding. (It is easy to see this effect if you look into a mirror and say *tea* and *to*.) The time course for anticipatory planning varies from one language to another; children learn to produce these encoded articulatory gestures in the first few years of life. Acoustic analyses of anticipatory—i.e., planned—coarticulation in aphasics shows that Wernicke's aphasics cannot be differentiated from normal controls (Katz 1988). Broca's aphasics, though they vary in the degree to which anticipatory coarticulation occurs, do not differ markedly from normal controls.

Voice-Onset-Time

The major speech production deficit of Broca's syndrome involves sequencing. Aphasic subjects lose control of the sequencing between larynx and supralaryngeal vocal tract activity. The acoustic cue that differentiates stop consonants such as [b] from [p] in the words *bat* and *pat* is "voice-onset-time" (VOT), the interval between the burst of sound that occurs when a speaker's lips open and the onset of periodic phonation produced by the larynx. The sequence between laryngeal phonation and the burst must be regulated to within 20 msec. Broca's aphasics are unable to maintain motor sequencing control; their intended [b]s may be heard as [p]s, [t]s as [d]s, and so on. Despite this deficit, however, the intrinsic duration of vowels is unimpaired in Broca's aphasia.

Prefrontal Activity and Aphasia

Although aphasia is by definition a language disorder, cognitive deficits were noted in early studies. Kurt Goldstein, a leading figure in aphasia research, stressed loss of the "abstract" attitude (1948). Goldstein described the difficulties that aphasic patients had planning activities and strategies, shifting strategies, formulating abstract categories, and thinking symbolically. Subsequent research has found that these cognitive deficits are associated with impaired frontal lobe—particularly prefrontal—cortical activity (Stuss and Benson 1986). But frontal lobe cognitive deficits do not necessarily result from damage to frontal lobe structures. Studies employing positron emission tomography (PET) and CT scans show that damage to either prefrontal cortex, or to subcortical structures supporting circuits to prefrontal cortex, can yield frontal

lobe cognitive deficits. Metter, et al. (1989), found that all of their Broca's patients had subcortical damage to the internal capsule and parts of the basal ganglia. PET scans showed that these patients had vastly reduced metabolic activity in the left prefrontal cortex and Broca's region.

The Subcortical Locus of Aphasia

Marie (1926) claimed that subcortical lesions were implicated in the deficits of aphasia. This was reasonable, since the middle cerebral artery is the blood vessel most susceptible to thrombotic or embolic occlusion. The central branches of this artery supply the putamen, caudate nucleus, and globus pallidus. Moreover, one of the central branches of this artery is the thin-walled lenticulostriate artery, which is exceedingly vulnerable to rupture. Brain imaging techniques confirm Marie's position: absent subcortical damage, permanent aphasia does not occur. As Stuss and Benson 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" (1986, p. 161). Moreover, subcortical damage that leaves Broca's area intact can result in Broca-like speech production deficits (e.g., Alexander et al. 1987; Mega and Alexander 1994). Damage to the internal capsule (the nerve fibers that connect neocortex to subcortical structures), the putamen, and the caudate nucleus can yield impaired speech production and agrammatism similar to that of the classic aphasias, as well in addition to other cognitive deficits. Alexander and his colleagues (1987) reviewed l9 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. 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 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) 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" (p. 41).

Neuro-Degenerative Diseases

Studies of the behavioral consequences of diseases such as Parkinson's disease and progressive supranuclear palsy provide independent evidence for the

role of basal ganglia in the FLS. These neuro-degenerative diseases result in major damage to basal ganglia, mostly sparing the cortex until the late stages, when cortical receptors may become damaged (Jellinger 1990). The primary deficits of these diseases are motoric: tremors, rigidity, and repeated movement patterns occur. However, subcortical diseases also cause linguistic and cognitive deficits. In extreme form, these subcortical diseases result in a dementia (Albert et al. 1974).

Sentence comprehension deficits linked to syntax have been noted in several Parkinson's studies (Grossman et al. 1991, 1993; Lieberman et al. 1990, 1992; Natsopoulos et al. 1993). Illes, et al. (1988), found that the sentences produced by Parkinson's subjects are often short and have simplified syntax. Illes and her colleagues attributed these effects to the speakers' compensating for speech production difficulties. However, a subsequent study revealed sentence comprehension deficits in Parkinson's disease that could not be attributed to compensatory strategies (Lieberman et al. 1990). In this study, based on a sentence comprehension test designed for hearing-impaired children, 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. Nine of a sample of 40 non-demented Parkinson's subjects showed comprehension deficits. Because the vocabulary of the test is simple and can be comprehended by six-year-old hearing children, the results argue against the subjects having had any difficulties with vocabulary. Cognitive loss was associated with impaired sentence comprehension; the subjects who had sentence comprehension deficits showed no symptoms of Alzheimer's disease, but cognitive decline was apparent to the neurologist who had observed them over a period of time.

Verbal Working Memory

The sentence comprehension deficits of Broca's aphasia and Parkinson's disease appear to reflect impairment of processing in verbal "working memory." The concept of working memory derives from about 100 years of research on short-term memory. Short-term memory is usually thought of as a buffer in which information is briefly stored; working memory includes computation as well as storage. Baddeley and his colleagues (e.g., Gathercole and Baddeley 1993) showed that verbal working memory was implicated in both the storage of verbal material and the comprehension of sentences. Baddeley proposed that verbal working memory involves two components, an "articulatory loop" whereby subjects maintained speech sounds in working memory by subvocally rehearsing them using the brain mechanisms that regulate overt speech, and a "central executive" process.

The central role that speech plays in the human FLS is manifest in the "rehearsal" mechanism, whereby words are subvocally maintained in working memory using the neuroanatomical structures that regulate speech produc-

tion. Many experiments show that subjects have more difficulty recalling a series of longer words than shorter words, as might be predicted if the articulatory buffer had a finite capacity. When the presumed articulatory rehearsal mechanism is disrupted by having subjects vocalize extraneous interfering words (e.g., the numbers *one*, *two*, *three*) during the recall period, recall deteriorates dramatically. Verbal working memory appears to be an integral component—perhaps the key component—of the human functional language system, coupling speech perception, production, semantics, and syntax.

The sentence comprehension test used by Lieberman, et al. (1990, 1992), included sentences with syntactic constructions that are known to place different processing demands on verbal working memory in neurologically intact adult subjects: e.g., "center embedded" sentences, such as "The boy who was fat sat down"; "right-branching" relative clause sentences, such as "I saw the boy who is fat"; conjunctions, like "The boy swam and the girl rowed"; and "simple" declarative sentences, like "I saw the boy." Some of the sentences, such as "The apple was eaten by the boy," were semantically constrained (apples generally do not eat anything); others, such as "The boy was kissed by the girl," were semantically unconstrained. Whereas neurologically intact control subjects make virtually no errors when they take this test battery, the overall error rate was 30 percent for some Parkinson's subjects. The subjects' comprehension errors typically involved repeated errors on particular syntactic constructions. Therefore, their syntax comprehension errors could not be attributed to general cognitive decline or attention deficits. The highest number of errors (40 percent) were made on "left branching" sentences—such as "Because it was raining, the girl played in the house"—that departed from the canonical English form of subject-verb-object. 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." This again points to verbal working memory load being a factor in sentence comprehension; longer sentences place increased demands on verbal working memory.

Similar sentence comprehension error rates for non-demented Parkinson's disease subjects have been found in the independent studies of Grossman, et al. (1991, 1993), Natsopoulos, et al. (1993), and by Pickett, et al. (1998), for a subject having brain damage limited to basal ganglia. Grossman, et al. (1991), also tested Parkinson's subjects' ability to copy unfamiliar sequential manual motor movements, a procedure analogous to that used by Kimura (1993), who found these deficits in Broca's aphasia. The Parkinson's subjects' manual sequencing and sentence comprehension deficits were correlated, and the correlation is consistent with Broca's area playing a role in manual motor control and in verbal working memory through circuits supported by basal ganglia (Lieberman 1984; Marsden and Obeso 1994; Rizzolatti and Arbib 1998).

Sequencing Deficits in Speech, Syntax, and Cognition

Lieberman, et al. (1992), reported striking similarities between the pattern of deficits in Parkinson's disease and Broca's aphasia. Acoustic analysis showed a breakdown in nine subjects' VOT control, similar in nature to Broca's aphasia. The speech of the Parkinson's disease subjects was similar to that of Broca's aphasics in other ways: they produced appropriate formant frequency patterns and preserved the vowel length distinctions that signal voicing for stop consonants when they occur after vowels. The Parkinson's subjects who had VOT overlaps had significantly higher syntax error rates and longer response times on tests of sentence comprehension than did the VOT non-overlap subjects; moreover, the number of VOT timing errors and number of syntax errors were highly correlated.

VOT Deficits: Sequencing or Laryngeal Control?

Impaired laryngeal control, perceptually characterized as hoarse "dysarthric" speech, and low amplitude speech, or "hypophonia," is a sign of Parkinson's disease. Therefore, it would be reasonable to suppose that impaired laryngeal control, in itself, might be the root cause of the VOT deficits of Parkinson's disease. A study of Chinese-speaking Parkinson's subjects resolved this question (Lieberman and Tseng 1994). Chinese makes use of phonemic tones, controlled variations in the fundamental frequency of phonation (F0), to differentiate words. The syllable [ma] produced with a level F0 contour in Mandarin Chinese, for example, signifies *mother*, whereas it signifies *hemp* when produced with a rising F0 contour. Twenty Parkinson's disease subjects read both isolated words and complete sentences in test sessions before or shortly after they took medication that increased dopamine levels. VOT overlap for Parkinson's subjects was significantly greater than that of age-matched speaking normal controls for both the pre-medication and post-medication test sessions. But VOT overlap decreased significantly post-medication for half of the Parkinson's subjects, demonstrating that dopamine-sensitive subcortical circuits were implicated in sequencing the laryngeal and supralaryngeal vocal tract motor commands that yield VOT distinctions. Acoustic analysis showed that the Parkinson's subjects were always able to generate the controlled F0 patterns that specify Chinese phonemic tones. Since the F0 patterns that specify these phonemic tones are generated by precise laryngeal maneuvers, it is apparent that that sequencing deficits are responsible for the observed VOT overlaps.

In short, the VOT overlaps that can occur in Parkinson's disease appear to reflect degraded sequencing of the individual motor commands that constitute the motor "program" that generates speech. This is not surprising, in light of studies of sequential non-speech motor activity in Parkinson's disease (Cunnington et al. 1995; Marsden and Obeso 1994) and the sequencing of sub-

movements of rodent grooming noted by Aldridge, et al. (1993). If the human neural circuitry regulating voluntary laryngeal activity during speech production is similar to that of monkeys, then the locus of VOT sequencing deficits may be the coordination of a laryngeal circuit involving anterior cingulate gyrus and independent circuits involving neocortical areas that regulate supralaryngeal vocal tract maneuvers. Significantly, no neocortical areas appear to be implicated in the regulation of non-human primate vocalizations (Sutton and Jurgens 1988).

Discussions of other experimental data consistent with basal ganglia regulating and shifting sequential motor and cognitive acts await the reader of *Human Language and our Reptilian Brain*. Hypoxia on Mount Everest impairs both VOT and sentence comprehension (Lieberman et al. 1994, 1995). Pickett, et al. (1998), in a study of a subject having brain damage limited to basal ganglia, found speech motor-sequencing deficits as well as deficits involving sequencing in the comprehension of distinctions in meaning conveyed by syntax and in cognitive tasks. Vargha Khadem, et al. (1998), documented speech and language deficits in subjects having a genetically transmitted anomaly that results in bilateral reduction of caudate nucleus volume. Cognitive deficits similar to those occurring with frontal lobe damage can be traced to impaired basal ganglia activity in Parkinson's disease (Lange et al. 1992). Cerebellar damage also yields similar deficits (Pickett 1998). In short, subcortical structures are essential elements of the FLS that regulate human language and some aspects of cognition.

ON THE EVOLUTION OF ADAPTIVE BEHAVIOR

Despite strident claims to the contrary (e.g., Pinker 1994), chimpanzees can produce about 150 words using manual sign language or computer keyboards, roughly equivalent to the abilities of two-year-old children. Chimpanzees also can understand spoken English words (Gardner and Gardner 1984; Gardner et al. 1989; Savage-Rumbaugh and Rumbaugh 1993). Indeed, other species comprehend spoken words. Your dog almost always comprehends a few words. The celebrated circus dog Fellow understood at least 50 words (Warden and Warner 1928). Therefore, lexical ability dissociated from speech production is a primitive feature of language that undoubtedly existed in the ancestral species that was the common ancestor of human beings and apes, and all archaic hominid lineages. Syntactic ability, which was and still is taken by many linguists to be a unique human attribute (Calvin and Bickerton, 2000; Lieberman 1984, 1991), also is present to a limited degree in chimpanzees. Analyses of the American Sign Language communications of the Project Washoe chimpanzees show that they used two-sign combinations and some three-sign combinations. Savage-Rumbaugh, et al. (1986), show that the six-year-old pygmy chimpanzee Kanzi comprehends simple sentences having the canonical

English form in which the subject precedes the object. When he heard sentences like "Put the pine needles on the ball" or "Put the ball on the pine needles," Kanzi responded correctly to the English-language command about 75 percent of the time, demonstrating a sensitivity to basic word order in English.

But chimpanzees cannot talk—speech remains the unique, derived, feature of human language. Despite many attempts over the past 300 years, no one has been able to train a chimpanzee to talk. The supralaryngeal vocal tract anatomy of chimpanzees prevents them from producing the vowels such as [i] and [u] (the vowels of the words *see* and *do*). However, speech communication could take place without the capability of producing the vowel [i], albeit with increased error rates (cf Lieberman 1984, 1991, 2000); analyses of chimpanzee vocalizations and the capabilities of their vocal tracts show that they could speak producing bilabial and alveolar-dental consonants like [b], [p], [m], [t], [d], [s], and so on, and all vowels save [i], [u], and [a] (Lieberman 1968, 1984). But chimpanzees can not even freely permute the sounds that occur in their natural repertoire of calls. The limiting factor is the chimpanzee brain. Chimpanzees can not produce vocalizations that are not "bound" to specific emotional states. As Goodall (1986) notes, "Chimpanzee vocalizations are closely bound to emotion. The production of a sound in the *absence* of the appropriate emotional state seems to be an almost impossible task for a chimpanzee" (p. 125). Human beings are able to freely permute the motor commands, the sub-movements that constitute the sounds of human speech to form words. The utter lack of productive speech and limited cognitive and syntactic abilities of apes and other species may reflect basal ganglia circuitry. *Human Language and our Reptilian Brain* suggests that our ability to "unbind" the articulatory submovements that generate words and to produce a virtually unlimited number of new words that convey referential information derives from the cortico-striatal circuits that are the focus of this inquiry.

THE ANTIQUITY OF HOMINID SPEECH

The probable absence of a modern human vocal tract in Neanderthals, and its almost certain absence in Australopithecines and *Erectus* grad hominids (Lieberman 1984), does not signify the absence of speech. As the initial Lieberman and Crelin (1971) paper on Neanderthal speech capabilities stressed, Neanderthals undoubtedly possessed speech, albeit less efficient speech than modern humans. This conclusion follows from the logic of natural selection. The perceptual process that relates formant frequency patterns to particular speech sounds must take account of the length of a speaker's vocal tract (longer vocal tracts produce lower formant frequencies for the same speech sound than shorter vocal tracts). The vowel [i] (the vowel of *see*) is optimal for this process (Nearey 1979), and it makes the process of speech perception less susceptible to error. However, the human vocal tract increases the risk of choking to death

when we swallow solid food (noted by Charles Darwin) and has other negative consequences (increased risk of death due to impacted teeth and less efficient chewing). But the restructuring of the hominid vocal tract to enhance the speech perception would not have contributed to biological fitness *unless* speech and language were already present in the hominid species ancestral to modern *Homo sapiens*. Otherwise there would have been no reason for the retention of the lower laryngeal position of the human vocal tract. Therefore, speech and some form of language (including syntactic ability, present in rudimentary form in living apes) must already have been present in Neanderthals and in the common ancestors of Neanderthals and modern humans, *Homo erectus* and perhaps Australopithecines.

Indeed, some aspects of human speech are very primitive in an evolutionary sense. The neural processing that allows us to determine the length of the vocal tract of the person to whom we are listening is not a unique human trait; studies of monkeys suggest that they also can judge the length of another monkey's vocal tract, which is a good index of a monkey's size, using a similar process (Fitch 1997). Early hominids must have possessed this ability. Primate calls also can be differentiated through formant frequency patterns generated when monkeys or apes close or open their lips as they phonate (Lieberman 1968). The fundamental frequency of phonation (F0), which is determined by laryngeal muscles and alveolar (lung) air pressure, is one of the principal cues that signals the end of a sentence and major syntactic units. Most human languages make use of controlled variations of F0 to produce tones that differentiate words. Since apes possess laryngeal anatomy that can generate F0 contours, early hominids must have had this ability. In other words, the roots of speech communication may extend back to the earliest phases of hominid evolution.

WALKING AND BASAL GANGLIA

About 5 million years ago, a species lived that was the common ancestor of present-day apes and humans. Early hominid fossils, such as the 4.4-million-year-old *Ardipithecus ramidus*, resemble apes who could have walked upright (White et al. 1994). Upright bipedal locomotion may have been the preadaptive factor that selected for neural mechanisms that enhanced motor ability (Hochstadt 1999). It is apparent that human beings *learn* to walk. The walking reflex that exists in newborn human infants appears to be controlled by a *quadripedal* neural pattern generator that reflects our hominoid ancestry. We crawl and toddle before we are able to walk or run. Heel strike, which marks efficient bipedal locomotion, takes years to develop (Thelen 1984). The subcortical basal ganglia structures of the FLS also regulate upright, bipedal locomotion. Indeed, one of the primary signs of Parkinson's disease, in which basal ganglia circuits are degraded, is impaired locomotion. Thus, upright, bipedal

locomotion may have been the initial selective force for the enhancement of the subcortical-cortical circuits that regulate sequencing of both motor and cognitive acts.

Many topics that are discussed in *Human Language and our Reptilian Brain* can not be treated in this summary. The dopamine depletion that characterizes Parkinson's disease may also directly reduce verbal working memory span. Research in progress suggests that this effect can occur, independent of sequencing deficits. Studies of the neural bases of motor control also show that complex behaviors are generally learned rather than innate. Given the participation of neuroanatomical structures in both motor control and syntactic processing, it is likely that syntax is learned rather than specified by a Chomskian universal grammar. It is also clear that algorithmic descriptions of motor behavior are at best a metaphor: the neural activity that governs motor control is parallel and distributed. In short, the cumbersome and inadequate algorithmic descriptions and innate universal grammar specifying the "rules" of syntax proposed by Chomsky and his disciples are not plausible. Hopefully, the issues discussed in *Human Language and our Reptilian Brain* will bring to linguistics and cognitive science a better appreciation of some of the facts and principles of biology, leading to a better understanding of what makes us human.

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