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What dysarthrias can tell us about the neural control of speech

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Dysarthrias, part of the class of neurogenic speech disorders, provide several sources of evidence concerning the neural control of speech. Although the dysarthrias have been studied primarily from a clinical perspective directed to issues of assessment and management, they have much to tell us about how the brain regulates the act of speaking. This paper considers five major areas in which disordered and normal speech can be integrated into an improved understanding of speech motor control: sensory function in the regulation of speech; rhythm as a temporal substrate for the organization of speech movements; kinematics of individual movements and motor systems; neural bases of multi-articulator coordination; and strategies for compensation, adaptation, and re-organization. A theme that runs through these five areas is consideration of the overarching hypothesis that speech motor regulation is based on a modular organization that can be defined partly by consideration of results from neurogenic speech disorders.

1. Introduction

The dysarthrias (neurogenic speech disorders) have been studied largely from a clinical perspective that addresses issues in assessment and treatment, but these disorders ultimately are a proving ground for the understanding of how the brain controls spoken language. Speech is a remarkable motor accomplishment in which sound segments are produced at rates of up to 30 per second in a precisely coordinated action that requires more muscle fibers than any other human mechanical performance (Fink, 1986). Therefore, speech is interesting and important not only as a primary means of human communication, but also as an exemplar of human motor coordination. A synthesis of research on normal and neurologically disordered speech should lead to an improved understanding of the neural regulation of speech. Consideration of neurogenic speech

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disorders can complement the knowledge gained through studies of normal speech production, so that both areas of study ultimately can be placed in a common theoretical framework. This paper reviews the literature on dysarthria with the objective of interpreting the accumulated knowledge in terms of its implications for understanding the neural control of speech. This review is timely insofar as dysarthria has been studied by a number of methods that have generated a large amount of data, particularly over the last two decades. Of particular interest is the possibility of linking acoustic and physiologic studies of speech with clinical and neuroimaging methods of determining site of lesion. Highly selected information from other speech disorders (apraxia of speech, structural disorders) also is mentioned in relation to some general issues, but the focus of this review is on the dysarthrias.

Dysarthrias, apraxia of speech, and the aphasias are sources of information on neurologically impaired speech production. This paper focuses on the dysarthrias, which can be defined as "a group of speech disorders resulting from disturbances in muscular control over the speech mechanism due to damage to the central or peripheral nervous system" (Darley, Aronson & Brown, 1969*a*, p. 246). Darley *et al.* went on to note that the dysarthrias involve "problems in oral communication due to paralysis, weakness, or incoordination of the speech musculature". Darley *et al.* (1969*a*, *b*) identified seven types of dysarthria: *spastic*, *flaccid*, *mixed spastic-flaccid* (in amyotrophic lateral sclerosis), *ataxic*, *hypokinetic*, *hyperkinetic in chorea*, and *hyperkinetic in dystonia*. This nosology has been highly influential and continues to be used for both clinical and research purposes (Edwards, 1984; Gerratt, Till, Rosenbek, Wertz & Boysen, 1991; Duffy, 1995; Love & Webb, 1996; Simmons & Mayo, 1997; Weismer, Laures, Jeng, Kent & Kent, 2000). The different forms of dysarthria were hypothesized by Darley *et al.* to be associated with damage to distinctive parts of the neural circuitry that regulate speech production. That is, Darley *et al.* not only delineated major types of dysarthria, but they also developed clinicoanatomic hypotheses for each type. These hypotheses are summarized in Table I, which shows the types of dysarthria thought to result from lesions to specific parts of the central or peripheral nervous system. To our knowledge, there has never been a systematic reevaluation of these clinico-anatomic correlations, even with the availability of neuroimaging techniques. Table II lists the major perceptual clusters for the dysarthria types. These clusters are useful in summarizing the overall perceptual features of the various types of dysarthria, but they have peculiarities and shortcomings that may limit their application (see review in Kent, Kent, Duffy & Weismer, 1998*b*). No alternative system of comparable scope has been as widely adopted as the system of Darley *et al.* (1969*a*, *b*).

TABLE I. Clinicoanatomic relationships hypothesized by Darley *et al.* (1969*a*, *b*). Shown for each perceptual type of dysarthria is the primary lesion site

Dysarthria type	Primary lesion site
Flaccid	Lower motor neuron (one or more cranial nerves)
Spastic	Upper motor neuron
Spastic–flaccid	Both upper and lower motor neurons
Ataxic	Cerebellum or its outflow pathways
Hypokinetic	Basal ganglia, especially substantia nigra
Hyperkinetic	Basal ganglia, especially putamen or caudate

TABLE II. Major clusters of deviant perceptual dimensions for dysarthria, as reported by Darley *et al.* (1999*a*, *b*)

Type of dysarthria	Clusters of deviant dimensions
Ataxic dysarthria	Articulatory inaccuracy, prosodic excess, phonatory–prosodic insufficiency
Spastic dysarthria	Prosodic excess, prosodic insufficiency, articulatory–resonatory incompetence
Flaccid dysarthria	Phonatory incompetence, resonatory incompetence, phonatory–prosodic insufficiency
Spastic-flaccid dysarthria	Prosodic excess, prosodic insufficiency, articulatory–resonatory incompetence, phonatory stenosis, phonatory incompetence, resonatory incompetence
Hypokinetic dysarthria	Prosodic insufficiency, phonatory incompetence
Hyperkinetic dysarthria (Chorea)	Articulatory inaccuracy, prosodic excess, prosodic insufficiency, articulatory–resonatory incompetence, phonatory stenosis
Hyperkinetic dysarthria (Dystonia)	Articulatory inaccuracy, prosodic excess, prosodic insufficiency, phonatory stenosis

With the important exception of neural lesions to individual cranial nerves, dysarthrias often involve global, rather than restricted, impairments of the speech production system (Auzou, Ozsancak, Jan, Leonardon, Menard, Gaillard, Eustache & Hannequin, 1998; Kent *et al*., 1998*b*). That is, they often affect the regulation of the respiratory, laryngeal, and upper airway (articulatory) systems. This multisystem dysregulation means that the dysarthrias are characterized by impairments of articulation, voice, and prosody, but the nature of the impairment may vary with the type and severity of the dysarthria.

Apraxia of speech has been defined as "a neurogenic speech disorder resulting from impairment of the capacity to program sensorimotor commands for the positioning and movement of muscles for the volitional production of speech. It can occur without significant weakness or neuromuscular slowness, and in the absence of disturbances of conscious thought or language" (Duffy, 1995, p. 5). Although apraxia of speech is controversial, most definitions of the disorder emphasize an impairment in programming, planning, or sequencing the movements of speech. The responsible lesion is frequently, but not invariably, in the language-dominant cerebral hemisphere. Apraxia of speech is potentially complementary to dysarthria in describing the neural control axis of speech production. Presumably, apraxia of speech reflects damage to relatively high-level mechanisms of planning or programming, whereas the dysarthrias reflect disorders of motor execution, with disruptions at different levels depending on the type of dysarthria. However, this duality may not be quite as simple as it appears at first look, especially because a variety of explanations have been offered to account for apraxia of speech (Code, 1998; Dogil & Mayer, 1998; Whiteside & Varley, 1998).

Progress toward the goal of integrating normal and neurologically disordered speech in a comprehensive model of neural control is particularly important in five major areas reviewed here. The major goal of this paper is to review the information in each area with respect to its implications for understanding the motor control of speech. A secondary goal is to evaluate each of these areas with respect to the overarching hypothesis that

speech motor regulation is based on a system of neural specializations in which particular functions are carried out in individual structures or circuits. A weak form of this hypothesis focuses on the complementarity of motor control operations that are organized into parallel processing streams (Grossberg, 2000). A stronger form of the hypothesis is a modular organization. This modular hypothesis requires an eventual statement in terms of (a) the autonomous functions of individual modules, (b) informational encapsulation in the modular architecture, and (c) the neuroanatomic sites (structures or pathways) of the modules. Although modularity also has been defined in terms of genetic prespecification (Fodor, 1983), more recent thinking allows the possibility of a gradual, experience-based specialization (Karmiloff-Smith, 1999).

Much remains to be done to describe putative modules for speech motor control, but some preliminary evidence is available to chart the path to a more complete examination of this hypothesis. Modularity has been discussed largely in relation to the brain's representation of language and cognition (Frazier, 1999; Karmiloff-Smith, 1999), but recent evidence has been reported for a modular organization of motor behavior (Gentilucci, Negrotti & Gangitano, 1997; Wolpert & Kawato, 1998). The perspective taken here is that modularity is conceptually consistent with the clinicoanatomic approach of Darley *at al.* (1969*a*, *b*) and can be evaluated as a theoretical extension of their proposals.

2. Sensory function in the motor regulation of speech

The role of sensory information in regulating movement is conceived quite differently in the various published models of speech production (Kent, Martin & Sufit, 1990; Kent, Adams & Turner, 1996; Kent, 1997). Some models give only passing mention to the role of afference, but others place a premium on sensory information as a reference for the planning and amendment of movements. The ambivalence is perhaps why the word *sensation* does not appear in the index of the Hardcastle & Laver (1997) *The Handbook of Phonetic Sciences*, a generally comprehensive account of topics and issues in speech production (consisting of 26 contributed chapters). It may be concluded that sensory function in speech production is poorly understood and often neglected in theories and models of speech production.

Feedback (and feedforward) information for speech production is potentially plurimodal, given that the available modalities include auditory, tactile, kinesthetic, and barometric information (Kent *et al.*, 1990). Some combination of these signals is generated with every speech event, but speech production models give little attention to the diverse afference that could be used to monitor and adjust motor activities in speech. One possibility is that talkers select the type(s) of information most pertinent to a particular motor objective. Attempts to disrupt afference have generally led to the conclusion that speech production is relatively little affected by short-term corruptions in sensory information (Kent *et al.*, 1990; Smith, 1992). One limitation in this line of research is the great difficulty of eliminating afference, especially because so many types of sensory information may be available and a speaker conceivably could rely on whatever afferent channels are available. Another limitation is that sensory information may be utilized either on a sampled or long-term basis, but these conditions are not easily investigated. Suffice it to say that the understanding of sensory function in normal speech production is clouded by limited data and incomplete theory.

Although empirical data on sensory function in speech continue to be disappointingly meagre, some important observations point to specific roles of sensory function in the regulation of skilled movements, including those in speech. Furthermore, these observations fit into recent theories that emphasize internal models as a means of controlling the skilled motor behavior in speech (Guenther, Hampson & Johnson, 1998; Perkell, Matthies, Lane, Guenther, Wilhelms-Tricarico, Wozniak & Guiod, 1997; Guenther, Hampson & Johnson, 1998; Callan, Kent, Guenther & Vorperian, in press; and see Perkell, Guenther, Lane, Matthies, Perrier, Vick, Wilhelms-Tricarico & Zandipour, 2000). The basal ganglia and cerebellum are likely neural sites for the maintenance of these internal models, and an examination of the dysarthrias associated with damage to these neural structures provides some ideas about the role of sensory information in speech motor control. Relevant information comes especially from Parkinson disease for the basal ganglia and from ataxic dysarthria for the cerebellum.

2.1. *Parkinson disease*

Growing evidence points to impaired sensory function in Parkinson disease (Koller, 1984; Schneider, Diamond & Markham, 1986; Klockgether, Borutta, Rapp, Spieker & Dichgans, 1995; Demirci, Grill, McShane & Hallet, 1997; Jobst, Melnick, Byl, Dowling & Aminoff, 1997; Forrest, Nygaard, Pisoni & Siemers, 1998). Schneider *et al.* (1986) reported on a deficit in orofacial sensory function that could be significant in understanding dysarthria and dysphagia in individuals with Parkinson disease. They pointed specifically to limitations in processing kinetic (moving) stimuli. A fundamental deficit in the sensory processing of time-varying information in subjects with Parkinson disease is indicated by a poorer temporal discrimination than in age-matched controls for tactile, auditory, and visual stimuli (Artieda, Pastor, LaCruz & Obeso, 1992), poorer accuracy than neurologically healthy subjects in a task of finger tapping in synchrony with an auditory cue (Freeman, Cody & Schady, 1993), and reduced orofacial kinetic sensitivity compared to control subjects (Schneider *et al.*, 1986). Demirci *et al.* (1997) concluded that subjects with Parkinson disease underestimate movement displacements when provided with kinesthesia. The authors suggested that the reduced kinesthesia, combined with reduced motor output and the likelihood of reduced corollary discharges, could mean that the sensorimotor apparatus is "set" smaller in Parkinson disease. A reduced envelope of movement is characteristic of this disorder, and can be manifest as a festinating gait, micrographia, and accelerated and attenuated speech ("short rushes of speech"; Darley *et al.*, 1969*a*). A similar conclusion was expressed in a study of volume control in speech, in which the authors noted that subjects with Parkinson disease appear to use a preset amplitude that is abnormally low (Ho, Iansek & Bradshaw, 1999).

One explanation for motor deficits in Parkinson disease is that the basal ganglia are involved in the formation of sensory templates that are used to guide movements (Schneider *et al.*,1986; Kent *et al.*, 1990; Leiner, Leiner & Dow, 1991). Damage to the basal ganglia may impair the preparation of templates and therefore contribute to disordered movements. In this view, the basal ganglia do not actually prepare motor programs but rather contribute to the specification of individual movements and their fluent execution. This conception agrees with research showing that Parkinson disease does not impair motor programming but does affect the performance of movement (Jennings, 1995; Weiss, Stelmach & Hefter, 1997).

Significantly, sensory training is emphasized as one step in the Lee Silverman Voice Treatment, a behavioral treatment for hypokinetic dysarthria (Ramig, Bonitati, Lemke & Horii, 1994; Ramig, Pawlas & Countryman 1995). This aspect of treatment is based on reports of laryngeal, respiratory, articulatory, velopharyngeal, and sensory kinesthesia disorders in this population (Ramig, 1998). Additional studies of this treatment should enhance the understanding of sensory deficits related to speech motor control.

2.2. *Cerebellar disease*

Ataxic dysarthria is another source of information on sensory function, especially because the cerebellum has privileged access to a wide range of sensory information that apparently is used in the preparation and revision of movements. The classic ataxic triad of dysmetria, dysynergia, and dysdiadochokinesis may be rooted in part in an inefficient processing of sensory information needed to produce accurate and well-timed movements. Some support for this possibility comes from studies that indicate a sensory impairment in individuals with cerebellar lesions (Keele & Ivry, 1990; Ackermann, Graber, Hertrich & Daum, 1997; Shimansky, Saling, Wunderlich, Bracha, Stelmach & Bloedel, 1997). Cerebellar processing of sensory information may be a primary reason for cerebellar activation in a variety of cognitive–linguistic tasks (Kent, 1998). Bower (1997) considers the cerebellum to be responsible for monitoring and adjusting the acquisition of various types of sensory data used by the rest of the nervous system. In his view, the cerebellum contributes to the efficiency of overall neural processing but is not itself required for all the behaviors to which it gives precision and fluidity. This suggestion accords with the observation that cerebellar lesions in the adult typically are associated with discoordinated movements in an essentially preserved motor plan (Kent *et al*., 1979). Additional support for a cerebellar involvement in sensory processing comes from Jueptner & Weiller (1998), who concluded that the neocerebellum is responsible for monitoring and optimizing movement outcomes. Maill (1998) hypothesized that the cerebellum has two roles: first, to provide an internal state estimate or sensory prediction that is used for on-line regulation of movements, and second to use the predictive state estimates to coordinate actions by different effectors.

Of the many different theories of cerebellar function that have been proposed, one influential proposal pertaining to skilled movement is that the cerebellum constructs a model of the skeletomuscular system (Ito, 1984, 1999). The motor cortex can then use the cerebellar model, rather than the skeletomuscular system, to prepare a precise movement. According to this view, dysmetria results because the internal model is dysfunctional. It also is possible that, through motor learning, the cerebellum replaces the motor cortex as the controller of skilled movements (Kawato, Furukawa & Suzuki, 1987; Shidara, Kawano, Gomi & Kawato, 1995). These issues are considered in more detail in a later section on the cerebellar role in the neural circuitry for speech and in the section on compensation or reorganization of speech movements.

2.3. *Conclusion*

Studies of individuals with Parkinson disease and cerebellar disease help to define the role of sensory information in the regulation of speech. Although the picture is incomplete, one unifying hypothesis is that the basal ganglia and the cerebellum contribute to the execution of precise and fluent speech by planning and amending movements based

on sensory information. Furthermore, these neural structures may contribute to the development and maintenance of internal models that are used to prepare and guide movements. Internal models are a component of several contemporary models of speech production, and the study of the dysarthrias may help to identify the neural mechanisms for the construction and maintenance of these models. Possibly, the emerging conclusions from studies of neurogenic speech disorders will be confirmed from functional neuroimaging studies of both normal and disordered speech. A major hypothesis is that sensory planning information for movements is vested especially in the basal ganglia and cerebellum.

3. Rhythm as a temporal substrate for the organization of speech movements

Rhythm has been one of the most recalcitrant concepts in speech research (Handel, 1989; Kent *et al*., 1996; Nooteboom, 1997). Although many writers allude to rhythm, empirical evidence for rhythm has been limited and controversial. Because many dysarthrias are described as having rhythmic disturbances (Duffy, 1995), these disorders may be useful to (a) define what rhythmic organization is (or is not), (b) identify the neural structures or loops that participate in rhythmic patterning, and (c) describe the contribution of rhythm to the organization of respiratory, laryngeal, and articulatory processes.

3.1. *Definitions*

Because rhythm is understood in different ways, it is necessary to define this concept as it pertains to speech. A simple definition is that rhythm is the distribution of various levels of stress across a series of syllables (Kent, Adams & Turner, 1996). This definition is specific to speech but accords with a more general definition in which the "experience of rhythm involves movement, regularity, grouping, and yet accentuation and differentiation" (Handel, 1989, p. 384). Guaitella (1999) notes that the rhythm of speech can be defined in two general ways that carry quite different implications for empirical study. The first way is a metric one, expressed, for example, as "an assimilation tendency involving the regulation of intervals" (Guaitella, 1999, p. 509). The concept of isochrony (equalized time intervals) is an example of this metric approach. The other general way is a rhythmic concept that emphasizes a dissimilarity tendency over the events of speech. Guaitella explains the difference in approach as follows: "Metric analysis is based on the premise that a temporal continuum can be analyzed by quantification, while rhythmic analysis approaches temporal organization through the mechanisms of perception (p. 509). Somewhat the same distinction was expressed by Duffy (1995) in his discussion of rhythmic cuing in the treatment of neurogenic speech disorders: "It may be that external pacing of rate is more effective when it is 'metered' and each word is given equal time, as opposed to 'rhythmic', in which timing patterns more closely simulate natural speech" (pp. 402–403). It is important to note the tension between the metric and rhythmic approaches, because they are not entirely compatible in their implications for method of study and interpretation of data. It should be emphasized that rhythm is relational, in the sense that the same rhythm can apply to different rates of production. Therefore, rhythm and tempo are nearly orthogonal, at least within the typical parameters of speech production. At extremely fast or slow speaking rates, rhythm may have to be adapted to tempo (as discussed later).

3.2. *Concepts of rhythm*

In psychology, rhythm has been studied partly because it is a means to understand the control mechanism for timed responses. Although some have proposed that a central clock coordinates behavior in all sensory modalities and response modes (Eijkman & Vendrik, 1965), evidence has been presented to support the alternative view that timing depends on the stimulus modality used to mark the presented intervals and on the task performed by the subject (Kolers & Brewster, 1985). Interestingly, in a task of rhythmic tapping synchronized to auditory, tactile, or visual stimuli, performance was least variable for auditory stimuli (Kolers & Brewster, 1985). The potency of audition for timing control has implications for speech development in children and it holds clinical relevance in that an auditory metronome appears to be better than visual stimuli in accomplishing rate control in dysarthric speakers (Pilon, McIntosh & Thaut, 1998). Because speech normally generates its own auditory pattern, it is possible that self-produced rhythms perceived through audition reinforce timing patterns in speech. A major conclusion from this line of research is that studies of the timing of motor responses should consider the stimulus modality and response task.

A possible role of rhythm is that it establishes a temporal framework for the coordination of sensory and motor information for an evolving movement. For example, Edelman (1989) noted that rhythm could be a means for reliable timing of reentrant signaling across neural maps. In a motor skill with plurimodal afference (such as speech), rhythmically gated sensory information may be a key factor in controlling movement. In particular, rhythm may be part of a predictive neural strategy that uses selective sensory information on a time-sampled basis to confirm movement execution. In this view, afference need not be sampled continuously but at times most pertinent to motor control in a specified task.

Rhythm, as applied to speech, has been notoriously difficult to define and to measure. Perhaps some progress can be made by characterizing disordered rhythm as it is manifest in various dysarthrias. Examples are discussed with respect to tremor, ataxic dysarthria, hypokinetic dysarthria, and apraxia of speech.

3.3. *Tremor*

Simple periodicity is the most basic rhythm. Periodic movement occurs both normally and clinically as a *tremor*, defined as involuntary rhythmic oscillations occurring about an equilibrium position of either the whole body or some part of the body (Rondot, Jedynak & Ferrey, 1978). Essential tremor is the most common movement disorder (Britton, 1995) and, in its severe forms, can be very disturbing to motor performance. The typical tremor frequencies observed in normal and pathologic tremor vary from 1 or 2 Hz up to about 16 Hz. The variation in tremor frequency may explain some aspects of the timing of voluntary movement in individuals with movement disorders. One possibility is that tremor is an attractor for phasic voluntary movements in Parkinson disease (Hertrich, Ackermann, Ziegler & Kaschel, 1993; Staude, Wolf, Ott, Oertel & Dengler, 1995), essential vocal-oromandibular tremor (Kent, Duffy, Vorperian & Thomas, 1998*a*), and cerebellar ataxia (Kent, Kent, Duffy, Weismer & Stuntebeck, 2000). It appears that one means by which an individual with a tremor can contend with the movement oscillations is to coordinate voluntary movements with the tremor, which then acts as an internal pacemaker. Tremor is therefore a means of investigating the relationship

between phasic voluntary movements and the rhythmic substrate for a movement sequence.

3.4. *Ataxic dysarthria*

Dysrhythmia is a hallmark of ataxic dysarthria, and investigations into the temporal pattern of syllable production in this disorder help to provide both a quantitative index of rhythm and suggestions on the neural origins of rhythm in speech. Ataxic dysarthria is classically associated with a speech pattern described with terms such as *staccato*, *explosive*, *scanning*, and *equal and excess stress*. Ackermann & Hertrich (1994) proposed an index of temporal structure that was used to study the "scanning" pattern of speech typical of ataxic dysarthria. The proposed *scanning index* (SI) is defined as

$$\mathrm{SI} = (S_1 \times S_2 \times \cdots \times S_n)/[S_1 + S_2 + \cdots + S_n)/n]^n$$

where S_n is the duration of a given syllable in a sequence of syllables, and n is the total number of syllables in the sequence.

Ackerman & Hertrich explain SI as follows: "Provided that all of the [n] syllables have equal lengths the index amounts to unity. In any other case, especially if one syllable is considerably shorter than the other ones, this measure will be < 1" (p. 80). The SI is an example of a metric approach to the problem of speech rhythm.

Because rhythm is manifest partly as temporal structure, the potential exists to alter the rhythm of speech patterns by artificially modifying segment durations. Hertrich & Ackermann (1998) applied this method to sentences produced by two individuals with ataxic dysarthria and two neurologically normal speakers. As expected, the modifications affected judgments of slowness, dysfluency, and rhythmic adequacy. However, the synthetic changes made to correct the dysarthric tempo in the ataxic speech samples did not lead to improvements in intelligibility and naturalness, although changes made to simulate ataxic dysarthric tempo in the normal speech samples did result in some loss of intelligibility and naturalness. It has also been reported that ataxic dysarthria hinders the ability of listeners to determine lexical boundaries in samples of the dysarthric speech, partly because of the abnormal speech rhythm (Liss, Spitzer, Caviness, Adler & Edwards, 2000).

3.5. *Hypokinetic dysarthria*

Dysarthrias provide other opportunities to examine the effect of rhythmic disturbances on speech intelligibility. It has been concluded from studies of normal speech that prosody contributes to spoken word recognition (Grosjean & Gee, 1987; Cutler & Butterfield, 1992; Cutler, Dahan & Van Donselaar, 1997). The hypokinetic dysarthria in Parkinson disease often is associated with reduced syllabic contrastivity (a form of dysprosody), and it appears that this speech pattern contributes to reduced intelligibility, especially when articulatory precision is compromised (Liss, Spitzer, Caviness, Adler & Edwards, 1998). Liss *et al*. concluded that stressed syllables and the rhythmic pattern of speech are important in the listener's segmentation of speech into words.

3.6. *Apraxia of speech*

Apraxia of speech is similar to many dysarthrias in that it typically has a disturbed rhythm defined by a slow speaking rate and deviant stress patterns. But, unlike the

dysarthrias, apraxia of speech presumably is not associated with muscle weakness or slowness. The slow speaking rate in apraxia of speech may be the result of impairments in the selection or programming of movements. It appears that a slow speaking rate in itself, can affect speech rhythm in both neurologically normal and neurologically impaired speakers (Deger & Ziegler, 1998). That is, rhythm and tempo are not independent dimensions of speech production, and, at very slow speaking rates, rhythm may deteriorate. It has been suggested that at slow rates, movements are no longer automatized but come under a form of closed-loop control (Adams, 1971; Deger & Ziegler, 1998). The dysrhythmia in dysarthria and apraxia of speech may therefore be, at least in part, a consequence of the slow speaking rate that typifies these disorders.

3.7. *Aphasia*

Although aphasia is not within the purview of this paper, it should be mentioned that this disorder is also a source of information on prosody and its neural control. Grela & Gandour (1998) studied the *rhythm rule* (a phonological phenomenon in which adjacent stresses are adjusted to avoid "stress clash") in two individuals with aphasia. Both subjects demonstrated rhythmic disturbances associated with the rhythm rule, and the authors concluded that (1) the impairment was associated with phonetic implementation (and not loss of word-level stress or loss of the rule itself) and (2) the neural substrates of prosody "are broadly distributed in the left and right cerebral hemispheres" (p. 361).

3.8. *Conclusion*

Several neurogenic speech disorders involve an apparent dysrhythmia, and the study of these rhythmic disorders should supplement studies of rhythm in normal speech. To some degree, impairments of rhythm can occur separately from impairments of other aspects of speech. But, rhythmic disturbances also can come about as the compensation for a neurogenic speech disorder, or from a slow speaking rate. Whatever the cause of dysrhythmia, its appearance in the dysarthrias provides a counterpart to the description of rhythm in normal speech. The various types of dysrhythmia that occur in neurologic diseases provide insight into both the nature of rhythm and the neural systems that control temporal patterns in speech. A particularly promising direction for future research is the computer modification of natural speech to derive normal rhythm from dysrhythmic patterns or vice versa.

4. Kinematics of individual movements and motor systems

Stevens (1998) divides the control processes for kinematic adjustments in speech into three groups: (1) control of subglottal structures that generate pulmonic airflow, (2) regulation of vocal fold activity, and (3) movements of structures that shape the supratracheal airway. Drawing from various speech movement studies, he summarizes kinematics in terms of unidirectional movement from one configuration to another (e.g., abducted to adducted vocal folds) and cyclic movement from one configuration to another and return to the original configuration (e.g., abducted to adducted to abducted vocal folds). The fastest adjustment times for either unidirectional or cyclic movements

occur for (a) lip or tongue movements for stop consonants, (b) jaw raising–lowering, and (c) vocal fold abduction–adduction. Slower adjustment times characterize tongue body movements for vowels, velar raising–lowering, and subglottal pressure changes. These kinematic values place limits on the rate of speech articulation, and Stevens remarks that the timing limitation is due mainly to neuromuscular processes. Similarly, Tsao & Weismer (1997) concluded that neuromuscular factors contribute to individual differences in speaking rates among neurologically normal individuals. The question arises if timing limitations on individual articulators, especially the slowest ones, are a determining factor in speaking rate.

4.1. *Kinematics in dysarthria*

Dysarthria generally is characterized by slow and weak movements of the articulators. Articulatory movements are slow not only for slow speaking rates but even for normal or faster-than-normal rates (Weismer, 1997). This slowness is observed in both kinematic and acoustic studies and defines a temporal substrate for speech patterns. It is possible that the slow rate of speech typically seen in dysarthria is determined by lengthened motor response times of individual structures of speech production. That is, slow speaking rate may be an inevitable consequence of slow motor responses in one or more articulators. This possibility raises the question: Can speakers with dysarthria increase their speaking rates when asked to do so? The answer probably depends on the type of dysarthria. At least some individuals with ataxic dysarthria do not seem to increase speaking rates appreciably (Kent *et al*., 2000), but subjects with dysarthria related to amyotrophic lateral sclerosis can increase their rates while maintaining speech intelligibility, even in the presence of a reduced acoustic vowel space (Weismer *et al*., in press).

Table III summarizes published data that can be used directly or indirectly to estimate unidirectional or cyclic movements in dysarthria. Specifically, the table entries are: (1) stop gap duration, or an index of constriction duration; (2) CV period duration extracted from a CvCVCv utterance (where v is the unstressed vowel and V the stressed vowel); (3) duration of a target unstressed vowel (in normal speech, the minimal duration for a vowel, and therefore a possible index of minimum duration for a vocalic nucleus); (4) syllable nuclei containing a vowel nucleus and its associated CV or VC transitions; (5)–(7) alternating motion rate (AMR) period for repeated CV syllables. The AMR data are particularly relevant to estimating cyclic movement times. Because the syllables typically used in this task involve a reciprocal (opening–closing) consonant articulation involving the lips ([p] or [b]), tongue tip ([t] or [d]), or tongue dorsum ([k] or [g]), the period in the AMR task is an index of cyclic movement. The data in Table III show that this cycle is longer in some dysarthrias than in normal speech, and often appreciably so. The data in Table III pertain to an interval that spans transitional and steady-state segments in speech. Possibly, these two types of segments are differentially affected by neuropathologies. Vollmer (1997) made acoustic measures to determine the relative contribution of steady states and transitions to the durations of words in four groups—subjects with (a) neurologically normal speech, (b) aphasia, (c) dysarthria, and (d) apraxia of speech. The subjects with dysarthria differed from the other three groups in having a nearly equal contribution from steady states (49.95%) and transitions (50.05%). In contrast, the normal speakers had a larger contribution from steady states (82.14%) than from transitions (17.86%). The subjects with apraxia of speech had a remarkably large

TABLE III. Data pertaining to unidirectional or cyclic movements in dysarthric *vs.* normal speech. Listed for each feature is the value in ms obtained for dysarthric subjects, normal subjects, and the ratio between the two

	Dysarthria	Normal	Ratio
Feature			
(1) Stop gap—ALS *vs.* normal (Caruso & Burton, 1987)	291	116	2.5
(2) CV period duration in /gvCVCv/tokens—spastic dysarthria *vs.* normal (Ziegler & von Cramon, 1986)	422	220	1.9
(3) Unstressed vowel (schwa)—ataxia *vs.* normal (Kent, Netsell & Abbs, 1979)	119	38	3.1
(4). Syllable nuclei—severe ALS *vs.* normal (Weismer, Martin, Kent & Kent, 1992)	507	236	2.1
(5) AMR period (mean for [pʌ], [tʌ], [kʌ]—ataxia *vs.* normal			
(a) Portnoy & Aronson (1982)	270	164	1.6
(b) Kent *et al.* (submitted)	256	154	1.7
(6) AMR period (mean for [pʌ], [tʌ], [kʌ]—spastic *vs.* normal speech (Portnoy & Aronson, 1982)	244	164	1.5
(7) AMR period (mean for [bV], [dV], [gV]—Parkinson disease *vs.* normal speech (Canter, 1965)	217	152	1.4

contribution from steady states (98.16%). One interpretation of these data is that the subjects with dysarthria had a general slowness that affected the steady states and transitions equally.

Table III pertains to durations for steady-state or dynamic intervals in speech, but they do not necessarily indicate actual velocity differences between dysarthric and neurologically normal speech. Limited data have been published on articulatory velocities, as summarized in Table IV. These results make it clear that in some individuals with dysarthria, articulatory velocities are reduced compared to neurologically normal speech. Although reduced velocities may accompany (and perhaps cause) a slow rate of speech, this is not necessarily the case. Connor, Ludlow & Schulz (1989) observed reduced $F1$ and $F2$ transition rates (and presumably reduced articulatory velocities) in syllable repetitions by speakers with Parkinson disease. Connor *et al.* concluded that these speakers used reduced articulatory displacements, even while achieving normal repetition rates.

In some dysarthrias, the movement disorder is confined to an individual articulator or individual system so that the effects of articulator- or system-specific dysregulation can be assessed. The dysarthrias most pertinent to this objective are: (a) flaccid dysarthria with specific cranial nerve involvement, (b) focal dystonias affecting speech (e.g., spasmodic dysphonia, lingual dystonia, jaw-opening dystonia, oromandibular dystonia); (c) spastic or unilateral upper motor neuron dysarthria with effects on the corticolingual or corticofacial tracts; and (d) flaccid–spastic dysarthria associated with some periods in the natural history of amyotrophic lateral sclerosis. Each of these is discussed further, with an emphasis on the potential knowledge that could be gained.

Although the data are limited, studies of flaccid dysarthria should help to construct a kinematic portrait of weak (flaccid) muscle systems in speech. An example is the mild dysarthria resulting from isolated hypoglossal nerve palsy (Omura, Nakajima, Kobayashi, Ono & Fujita, 1997). These disorders offer an opportunity to determine the way in

TABLE IV. Velocities of articulatory movements in dysarthric *vs.* normal speech. Shown for each movement is the value (mm/s) for dysarthric subjects, normal subjects, and the ratio between the two

	Dysarthria	Normal	Ratio
Movement			
(1) Ackermann, Hertrich & Scharf (1995)*			
Opening movement of lower lip	93–118	123	0.8–1.5
Closing movement of lower lip	120–212	151	0.8–1.4
(2) Caligiuri (1989)†			
Closing movement of lower lip	17	140	8.2
(3) Forrest, Weismer & Turner (1989)†			
Opening movement of lower lip	134	197	1.5
Opening movement of jaw	51	108	2.1
(4) Hirose, Kiritani & Sawashima (1982)‡			
Lower lip closing movement	104	222	2.1
Lower lip opening movement	99	165	1.7
Velar elevation	88	163	1.8
Closing movement of tongue dorsum	124	182	1.5
Opening movement of tongue dorsum	115	168	1.5

*Ataxia *vs.* normal comparison; dysarthric data shown are lowest and highest values for four subjects with ataxia.
†Parkinson disease *vs.* normal comparison.
‡Amyotrophic lateral sclerosis *vs.* normal comparison.

which impaired movement of one structure affects the sequence of movements in speech. The kind of question that can be asked is: How does the neural control system accommodate one weak articulator? Is the entire temporal plan of speech movements altered, or are there islands of adjustment that occur only when the affected articulator is part of the ongoing movement sequence?

Disorders such as spasmodic dysphonia, jaw-opening dystonia, and lingual dystonia may provide some effective contrasts to flaccid dysarthrias affecting the same muscle groups. Such contrasts present an opportunity to observe the similarities or differences that occur when a given structure is affected by different underlying pathophysiology (weakness *vs.* dystonia). Also, because dystonias are presumably focal, they allow investigation of the same question posed earlier with respect to flaccid dysarthrias: How does the movement plan accommodate a movement disorder affecting one articulator?

Concerning spastic or unilateral upper motor neuron dysarthria (the typical types of dysarthria associated with supratentorial strokes), studies could yield a better understanding of the higher levels of speech neural control. Urban and associates (Urban, Hopf, Zorowka, Fleischer & Andreas, 1996; Urban, Hopf, Fleischer, Zorowka & Mullerforell, 1997) concluded that disruption of the corticobulbar (corticolingual and corticofacial) tracts is central to the pathogenesis of dysarthria from supratentorial ischemic stroke. Perceptual studies of upper motor neuron dysarthrias evince several types of articulatory errors involving especially the structures innervated by the facial and hypoglossal nerves, which accords with the suggestion by Urban and associates. Data from a study in progress support this conclusion but also point to a variety of dyscoordinations. The neurophysiologic study of unilateral lingual paralysis following monohemispheric ischemic stroke is an example of how clinical studies can provide information on the neural innervation of speech structures. Muellbacher, Artner

& Mamoli (1998) examined compound muscle action potentials in lingual muscles following transcranial magnetic stimulation of motor cortex and peripheral electrical stimulation of the hypoglossal nerve. Comparison of data from neurologically normal controls and patients with monohemispheric ischemic stroke showed considerable inter-subject variability in the clinical group.

Acquired motor neuron disorders, such as amyotrophic lateral sclerosis (ALS), are identified clinically especially by weakness unaccompanied by a sensory defect (Ross, 1997). Both acoustic and physiologic data point to specific speech correlates of the neural degeneration, for example, reduction of the *F*2 slope in acoustics and slow force generation in physiology (Weismer & Martin, 1992; Weismer *et al*., 1992; Weismer, 1997; Kent *et al*., 1998*b*). Because ALS is progressive, the dysarthria increases in severity until most patients are nonspeaking in the final stage of the disease. The accrual of symptoms and the selectivity of the pathology (e.g., affecting lingual more than mandibular muscles) mean that quantitative studies can demonstrate the effects of specific impairments on speech. Interestingly, clinical symptoms are not evident until about 80% of motoneurons are lost (reviewed in Kent *et al*., 1998*a*, *b*).

It is often assumed that apraxia of speech results from impairment of sequencing or temporal coordination in the face of essentially normal individual gestures. Some evidence of articulatory slowing has been reported (Ackermann, Scharf, Hertrich & Daum, 1997), but given the mixed results on this issue (McNeil & Kent, 1990), it is better to await further study before asserting articulatory slowing as a common feature of apraxia of speech.

4.2. *Conclusion*

Generally, the dysarthrias and apraxia of speech result in a slowing of speech that is manifest as lengthened movement times and reduced articulatory velocities. Sometimes, only one articulator or speech subsystem is directly affected by the slowing, which opens the possibility of determining how an overall movement pattern is adjusted to contend with slowing in one part of the effector apparatus. Dysarthria can be studied to determine the neuromuscular determinants of speaking rate, coarticulation, and other aspects of speech production.

5. Neural bases of multi-articulator coordination

Because speech is one of the most precisely controlled human motor skills, its regulation should carry important lessons for coordinated motor behavior in general. The term "dyscoordination" is frequently applied to the dysarthrias and to apraxia, although exactly what is meant by this term is not always clear (Kent & Adams, 1989). Multi-articulate movement data are not abundant, except for observations of lip and jaw articulation. However, data on dysarthria associated with stroke, cerebellar lesion, Parkinson disease, and essential tremor are beginning to show aspects of dyscoordination, which may lead to new formulations on the preparation of coordinated movement. The terms programming, sequencing, and coordination are frequently used in referring to the control of skilled movements, but unfortunately, they are rarely defined. They do not necessarily have the same meaning. In this discussion, the following definitions apply. Programming is a plan for a motor action (generally a learned, skilled response).

TABLE V. Hypothesized roles of select neural structures in the regulation of speech

Structure	Function
Insula	Prepares sequential representations of speech segments and/or movements, given a phonological input; the posterior and anterior regions may play different roles in speech regulation
Broca's area	Maintains the syllable string for an utterance while keeping track of the grammatical influences that shape phonological boundaries and other prosodic effects
Supplementary motor area	Initiates and controls sequential movement plans that activate muscle-specific regions of primary motor cotrex; possibly includes specific functions of retrieval and execution of a motor plan
Basal ganglia	Prepares and select movements, perhaps in accord with a sensory plan or trajectory that is formulated within this structure
Cerebellum	Creates a model that mimics the dynamics of the skeletomuscular system of speech, so that the motor cortex, can use these models, rather than the skeletomuscular system itself, to regulate precise movements
Primary motor cortex	Selects and regulates specific muscles in accord with movement plans specified by other structures

Although some notions of programming deny any role of feedback, it seems more appropriate to include both motor and sensory components in the construction of programs. Sequencing refers to the order of succession, as in the case of phonetic segments, movements, or muscle contractions. For present purposes, sequencing refers especially to the latter two of these. Coordination refers to the processes of adjustment by which separate components of action are unified in a common motor objective. An implication of these definitions is that programming does not necessarily specify all aspects of sequencing or coordination. For example, programming may pertain to higher levels of motor organization, and some details of sequencing or coordination may be accomplished separately by lower levels of control.

Some new hypotheses about movement scaling and coordination in speech are being formed from gestural theory (Browman & Goldstein, 1986, 1988, 1990, 1992; Salzman & Munhall, 1989; Kröger, 1993; Kröger, Shröder & Opgen-Rhein 1995). As applied to dysarthria, these hypotheses may account for certain dyscoordinations in terms of phasing errors that occur between concurrent movements in a motor score (Weismer, Tjaden & Kent, 1995*a*). To date, gestural theory has been considered almost exclusively within the domain of neurologically normal speech, but it may pertain in interesting ways to dysarthria. In particular, the altered kinematics in many dysarthrias provide an opportunity to determine how the hypothesized gestures are affected by neuromuscular abnormalities.

Coordination is predicated on either a prescribed pattern of motor activity (e.g., a motor score that stipulates the events in a motor sequence) or an emergent function in system dynamics. The former view usually is taken to mean that some part or system of the brain formulates and plans the essential pattern of speech. Although this role classically was assigned to Broca's area (Brodmann Area 44 and 45), more recent studies demonstrate that the insula may be responsible, perhaps along with other cortical regions, as discussed next. The discussion also includes hypotheses on modularity of speech motor control (summarized in Table V).

5.1. *Insula*

Dronkers (1996) determined that patients with strokes and "articulatory planning" deficits (apraxia of speech) had lesions that included a region of the left precentral gyrus of the insula. Patients who did not have lesions in this structure did not have deficits in articulatory planning. A recent case report also notes the occurrence of apraxia of speech following an acute infarct limited to the precentral gyrus of the left insula (Nagao, Takeda, Komori, Isozaki & Hirai, 1999). Confirmation of the insula's role in speech planning comes from recent studies of normal subjects using the functional imaging methods of MEG (Kuriki, Mori & Hirata, 1999) and PET (Wise, Greene, Buchel & Scott, 1999). Kuriki *et al.* (1999) reported that a broad MEG response that occurred 120–320 ms before speech onset had current dipole sources around the superior end of the left insula. The MEG response was localized to a region extending from the superior end of the insula to the lower deep part of the precentral gyrus. Wise *et al.* (1999) concluded that the articulatory plan is formulated in the left anterior insula and lateral premotor cortex. They also noted that the left basal ganglia are dominant for speech. Apparently, the classic picture of the neural regulation of speech in which Broca's area was the primary motor center should be revised to make the insula at least one center, if not a major center, for articulatory planning. Consistent with this role is the suggestion by Shi & Cassell (1998) that the anterior insula is an interface between the posterior insular cortex and the motor cortex. If the posterior insular cortex is concerned more with word morphology and phonology, then the anterior insula could be responsible for the motoric interpretation of phonologic sequences, with the products of this interpretation being sent to the premotor cortex where instructions are prepared for sequences of movements by the articulators. Specifically, the insula may be involved in sequential representations of speech segments and/or movements, which are needed before effectors are selected for movement execution (Keele, Jennings, Jones, Caulton & Cohen, 1995). In a modular view of the neural control of speech production, the insula is a candidate for the sequential representation of either the phones or the movements of speech. The insula also may be one of the neural structures that insures compliance with phonotactic constraints, even when greatly disturbed sequences are generated, as in the case of paraphasias (Wheeler & Touretzky, 1997).

The insula has connections with a number of brain structures and regions, including the orbital cortex, frontal operculum, lateral premotor cortex, ventral granular cortex, medial area 6 in the frontal lobe, the second somatosensory area, and the superior temporal sulcus of the temporal lobe (Augustine, 1996). These connections include structures serving both motor (lateral premotor cortex, area 6) and auditory (superior temporal sulcus) functions in speech. Augustine (1996) suggests that the insula is a limbic integration cortex, a role that could be highly suited to the motivational and affective foundations of speech production. It also has been suggested that the insula is part of the network of verbal memory (Manes, Springer, Jorge & Robinson, 1999).

5.2. *Broca's area* (*Brodmann areas* 44 *and* 45)

The insula is an example of a brain structure that is increasingly implicated in speech and language functions, even as certain classic language areas (e.g., Broca's area) and classic language pathways (e.g., arcuate fasciculus) are being reexamined, and sometimes discounted, as to their role in language processing (Aboitiz & Garcia, 1997*a*, *b*). Contrary to

the historic understanding of Broca's area, this cortical region is not necessarily active in the production of single words for nouns (Raichle, 1996), although it is more likely to be activated in verb generation (Raichle, 1996), sentence reading (Muller, Rothermel, Behen, Muzik, Mangner & Chugani, 1997), and verbal fluency tasks (Phelps, Hyder, Blamire & Schulman, 1997; Schlosser, Hutchinson, Joseffer, Rusinkek, Saarimaki, Stevenson, Dewey & Brodie, 1998).

But Broca's area cannot be dismissed from a role in the neural circuitry of speech production. Damage to this area and surrounding cortical regions is associated with a progressive loss of speech that has been termed *primary progressive aphasia*, *progressive anarthria*, or *progressive dysarthria*. Patients with this disorder present with impaired articulation (or apraxia of speech), telegraphic style, and a difficulty of performing complex orofacial and hand movements. In one of the larger studies, eight patients were examined, with long-term follow-up (6–10 years) for four cases (Broussolle, Bakchine, Tommasi, Laurent, Bazin, Cinotti, Cohen & Chazot, 1996). CT and MRT findings showed an asymmetric (left more than right) progressive cortical atrophy of the frontal lobes "predominating in the posterior inferior frontal region, notably the operculum" (Broussole *et al*., 1996, p. 44). The patients studied in follow-up progressed to muteness and bilateral suprabulbar paresis. In five patients studied by Didic, Ceccaldi & Poncet (1998), the neural damage associated with the early stage of the disease was thought to be in the ventral compartment of the premotor cortex. However, exact localization of the lesion was difficult with CT or MRI. Progression of the disorder apparently was associated with more extensive frontal lobe damage, perhaps including dorsolateral premotor cortex. In a group of three patients with "slowly progressive loss of speech and dysarthria associated with orofacial dyspraxia", PET revealed bifrontal hypometabolism which was especially marked in the inferior and lateral portions of both frontal lobes (Tyrrell, Karsounis, Frackowiak, Findley & Rossor, 1995). In two patients with progressive dysarthria that was the sole initial sign of a neurodegenerative condition, neuroimaging revealed bilateral involvement of the posterior inferior frontal lobe structures (Santens, Van Borsel, Foncke, Meire, Merkx, De Bleecker & De Reuck, 1999). Selnes, Holcomb & Gordon (1997) studied one patient with progressive dysarthria. MRI revealed that he had a localized left-sided perisylvian lesion and PET showed a left and possibly right perisylvian hypometabolism localized to the area of tissue loss. A common feature to these patients was the initial impairment of speech (often the earliest sign of disorder), the progressive deterioration usually leading to mutism, and involvement of the posterior inferior frontal lobe, especially the operculum.

How, then, does Broca's area participate in speech production? One possibility is that this region is activated in tasks that require analysis of hierarchical structure (e.g., sentences) or complex sequences (e.g., extracting and manipulating phonetic segments; Zatorre, Meyer, Gjedde & Evans, 1996). The minimal activation of Broca's area in the production of single words may mean that individual words can be executed through automatized motor plans that do not require operations in Broca's area. But when more complex hierarchical or sequential structure must be analyzed, Broca's area cooperates with other regions, especially the insula and premotor cortex, to effect precise motor programs that accord with linguistic structure. Broca's area could play a critical role in the neural implementation of Fujimura's (1992, 1994*a*, *b*) Converter–Distributor (C/D) Model, in which speech is governed by a string of sequentially ordered syllables that also includes syntactically motivated phonological boundaries of varying strengths. Prosodic organization is accomplished by a metrical tree that attaches to the linear string of

syllables. Broca's area, with its presumed capability for hierarchical and sequential ordering, could maintain the syllable string while keeping track of the grammatical influences that shape phonological boundaries. Simple speech production tasks, such as single word production, may not require participation of this area.

It also has been proposed that Broca's area is the location of a "mirror system" that matches observation and execution of gestures (Rizzolatti & Arbib, 1998). A similar idea is expressed by Skoyles (1998), who theorizes that phones are a "replication code" between auditory stimuli and speech motor patterns. Broca's area is perhaps the neural means to the execution of gestures that match or replicate gestures that are seen and/or heard. This role presumably would be very important in the evolution and development of language, but it also could apply to the McGurk effect in which visual and auditory information about speech is integrated into a single phonetic decision even when the visual and auditory cues are noncompatible. It is noteworthy that about 80% of the variance in vocal tract activity can be estimated from facial movements (Yehia, Rubin & Vatikiotis-Bateson, 1998). Therefore, an observer can use facial information to predict the general pattern of vocal tract behavior. The "mirror system" hypothesis is consistent with the idea that Broca's area is specialized to perform various analyses that enable the execution of complex motor acts.

5.3. *Supplementary motor area* (*SMA*) (*Brodmann area* 6)

The SMA is frequently activated in speech production (and other skilled movements) and it has been implicated especially in the control of sequential movements. Damage to the SMA results in various disruptions of speech, including mutism, initiation difficulties, short phrases, and dysfluencies (Jonas, 1981; Gelmers, 1983; Caplan, 1987; Ziegler, Kilian & Deger, 1997). The SMA also may be involved in the transient mutism that occasionally occurs following posterior fossa surgery. The mutism is thought to result from interruption of the pathway that connects the cerebellar dentate nucleus, ventrolateral nucleus of the contralateral thalamus and the SMA (Germano, Baldari, Caruso, Caffo, Montemagno, Cardia & Tomasello, 1998). Both functional and anatomic studies indicate that the SMA is not a single region but 2 or 3 regions. A two-fold division recognizes the pre-SMA and the SMA. The pre-SMA is an anterior region that is activated early in the period of movement preparation, while the SMA is activated with movement execution (Lee, Chang & Roh, 1999). Cytoarchitectonic data indicate that the SMA consists of three separate regions (Vorobiev, Govoni, Rizzolatti, Matelli & Luppino, 1998). Given the connection between the insula (a possible articulatory planning center) and the SMA, it is conceivable that the SMA initiates and controls sequential movement plans that activate muscle-specific regions of primary motor cortex. One possible role of the SMA is the retrieval of information from an articulatory buffer (Ziegler *et al*., 1997). In addition, the SMA may have specific responsibilities for activating the selected and retrieved movements. Connections between the SMA and the cerebellum insure that the movements are performed with requisite precision, using various forms of sensory information generated during the nascent movement.

5.4. *Motor cortex*

The motor cortex selects the activation of individual muscles in keeping with a motor plan that is constructed by neural circuits involving the insula, SMA, and, for some

purposes, Broca's area. The analogy sometimes used is that the motor cortex is a kind of motoric keyboard in which the individual keys correspond to instructions to individual muscles. This idea is compatible with the effects of unilateral lesions in primary motor cortex. Typically, the motor impairment is transient, perhaps because individual muscles (or movements) are redundantly represented. Compensation for focal injury is accomplished by using spared neural tissue that can activate the same muscle. Bilateral injury, or more extensive unilateral damage, may result in a more lasting behavioral deficit. Although it is somewhat controversial as to how spasticity is related to damage to the motor cortex and its pathways, the classic understanding is that spasticity is associated especially with lesions of the upper motor neuron. Studies of pointing movements in spastic hemiparesis show that trajectory planning in extrapersonal space is preserved but interjoint coordination is disturbed (Levin, 1996). If this result can be extended to movements in general, then spasticity can be understood primarily as a difficulty in specifying interjoint coordination in accord with a planned trajectory that is essentially intact. As noted earlier, the cerebral cortex and basal ganglia may cooperate in the determination of the trajectory and selection of movements to accomplish it.

5.5. *Basal ganglia*

The basal ganglia are comprised of the striatum (caudate, putamen, and ventral striatum), globus pallidus (GP), subthalamic nucleus (STN), and substantia nigra (SN). The striatal structures are the input system for the basal ganglia, receiving information directly from various regions of the cerebral cortex. The GP consists of an external segment (GPe) and an internal segment (GPi). The GPe and STN sometimes are thought of as intermediate structures of the basal ganglia, but the STN does receive some direct cortical input. The SN is divided into two cell groups, the pars compacta (SNpc) and the pars reticulata (SNpr). The SNpr and the GPi are the primary output structures that reach the cortex through thalamo-cortical pathways.

Studies of Parkinson disease have been particularly important in understanding the role of the basal ganglia. As noted previously, it appears that the kind of neural damage that occurs in Parkinson disease does not disrupt motor programs so much as it interferes with the effective execution of complex movement sequences. Therefore, the basal ganglia may be responsible for sensorimotor integration that insures the smooth performance of movement. One way of explaining this contribution to movement is that the basal ganglia prepare sensory templates that guide movement execution and perhaps also use afference to update and revise internal models of the muscle system. However, Jueptner & Weiller (1998) concluded from a functional imaging study of learned finger movements that the basal ganglia are involved more with movement/muscle selection than in sensory processing. In particular, they reported that (a) initial learning activated the dorsolateral prefrontal cortex and the striatum (caudate nucleus and anterior putamen), (b) movement selection activated the premotor cortex and mid-putamen, and (c) automatic (overlearned) movements activated the sensorimotor cortex and posterior putamen. If the basal ganglia contribute in the same way to speech, then their primary function would be to select movements, perhaps in accord with a sensory plan or trajectory.

It is relevant here that both neuroimaging and genetic data have been reported for members of a large three-generation pedigree (the KE family), a large proportion of who

have verbal dyspraxia, with particular difficulties in sequential articulation and orofacial praxis. Vargha-Khadem, Watkins, Price, Ashburner, Alcock, Connelly, Frackowiak, Friston, Pembrey, Mishkin, Gadian & Passingham (1998) concluded from PET and MRI studies that abnormalities in the affected members were found in both cortical and subcortical motor areas of the frontal lobe. MRI results showed that the caudate nucleus was abnormally small bilaterally. A linkage study of the family localized the abnormal gene to a 5.6-centiMorgan interval in the chromosomal band 7q31 (Fisher, Vargha-Khadem, Watkins, Monaco & Pembrey, 1998).

5.6. *Cerebellum*

The cerebellum, amounting to about 10–15% of the entire weight of the brain, comprises about 50% of all CNS neurons. These are arranged in a complex circuitry involving five types of cells: Purkinje, basket, strellate, Golgi, and granule cells. All of these except for the granule cells are inhibitory in their synaptic action. The cerebellum is highly compartmentalized, and it has been proposed that the existence of several hundreds of reproducible structural/functional modules contributes to an efficient parallel processing of information for motor control (Ozol & Hawkes, 1997). Many theories have been developed to account for cerebellar control of behavior. As mentioned earlier, one influential theory is that the cerebellum creates models that mimic the dynamics of the skeletomuscular system (Ito, 1984, 1999). The advantage of these internal models is that the motor cortex can use them, rather than the skeletomuscular system itself, to regulate precise movements. Another proposal is that the cerebellar model can act as a controller to replace the motor cortex, so that learned movements can be executed precisely without conscious effort (Kawato *et al*., 1987; Shidara *et al*., 1995).

Because the cerebellum has an elaborate neural circuitry, a goal in understanding its functions is to identify regions of the cerebellum that are involved in particular actions or behaviors. The cerebellum can be divided anatomically into the flocculonodular lobe and the corpus cerebelli. The latter is subdivided into vermis (a midline structure), parvermis (intermediate region), and hemisphere (lateral structure). Cerebellar damage leading to ataxic dysarthria is one way of determining which parts of the cerebellum control speech. In fact, ataxic dysarthria has been linked to several cerebellar lesions, including: the superior cerebellar vermis, both cerebellar hemispheres, paravermal and lateral aspects of the hemispheres, and left paravermal area (Lechtenberg & Gilman, 1978; Amerenco, Chevrie-Muller, Roullet & Bousser, 1991; Amerenco, Roullet, Goujon, Cheron, Hauw & Bousser, 1991; Ackermann, Vogel, Peterson & Poremba, 1992; Gilman & Kluin, 1992); the paramedian regions of the superior cerebellar hemispheres (Ackerman & Ziegler, 1992); the midline structures of vermis and fastigial nucleus (Chiu, Chen & Tseng, 1996), and even noncerebellar regions such as frontal cerebral cortex (Terry & Rosenberg, 1995; Marie, Rossa, Lambert, Verard, Marchal & Viader, 1998). An impairment in sensory processing may result especially from bilateral damage to the cerebellum (Ackermann *et al*., 1997).

Finally, the cerebellum also has been proposed as the neural site for a time computation that is used by different motor and sensory systems (Ivry, Keele & Diener, 1988; Keele & Ivry, 1990; Keele *et al*., 1995). One interpretation is that the cerebellum's time computation is an example of a modularity in which a single representation in the nervous system supports a number of sensory, motor, or cognitive activities.

TABLE VI. Lesions resulting in pure or isolated dysarthria. Shown for each lesion location is the number of patients and source (? indicates uncertainty about one or more patients)

Lesion location	No. of subjects and source
Cerebral cortex	1 of 9 patients in Ichikawa & Kageyama (1991), 2 of 13 patients in Kim (1994), single patient in Lampl, Steinmetz, Gilad, Eshel, Chamovitz & Sarova-Pinhas (1997), 8 of 12 patients in Okuda *et al.* (1999)
Corona radiata (unilateral)	4 (?) of 7 patients in Urban, Wicht, Hopf, Fleischer & Nickel (1999); 4 of 13 patients in Kim (1994)
Internal capsule (unilateral)	2 (?) of 7 patients in Urban *et al.* (1999)
Corona radiata and/or internal capsule (Bilateral)	11 of 12 patients in Okuda *et al.* (1999)
Corona radiata and internal capsule (Unilateral)	9 of 10 patients in Ichikawa & Kageyama (1991), 1 of 12 patients in Okuda *et al.* (1999), 5 patients in Ozaki *et al.* (1986), 3 patients in Tohgi *et al.* (1996)
Basal ganglia	3 patients in Kim (1994)
Pons	3 patients in Kim (1994), 2 patients in Orefice *et al.* (1999); 9 patients in Tohgi *et al.* (1996)

5.7. *The essential motor pathway for speech*

The vast majority of dysarthrias occur in relation to other motor and sensory abnormalities, but infrequently, dysarthria is the sole or predominant sign of stroke (Fisher, 1982; Ozaki, Baba, Narita, Matsunaga & Takebe, 1986; Arboix, Massons, Oliveres & Titus, 1991; Ichikawa & Kageyama, 1991; Kim, 1994; Tohgi, Takahashi, Takahashi, Tamura & Yonezawa, 1996; Orefice, Fragassi, Lanzillo, Castellano & Grossi, 1999). These instances of *isolated* or *pure dysarthria* offer an opportunity to identify neural lesions that apparently are restricted to the regulatory pathways for speech. However, some caveats should be noted. First, at least some of the so-called pure or isolated dysarthrias described in the literature were accompanied by mild concomitant deficits (particularly orofacial paresis), so that the speech disorder may have been the most noticeable, but not singular, consequence of the lesion. Secondly, synthesis and interpretation of the published studies is difficult because the lesions are widely distributed (cortical and subcortical, unilateral and bilateral) and it is possible that remote effects (diaschisis) and compensations occurred (Okuda, Kawabata, Tachibana & Sugita, 1999). These problems notwithstanding, it may be instructive to survey the lesion sites associated with isolated dysarthria (Table VI). The lesions occur along a pathway that includes the cerebral cortex, corona radiata, internal capsule, basal ganglia, and pons. This pathway is essential to the motor regulation of speech. In addition to isolated dysarthrias related to stroke, it has been reported that an isolated, reversible dysarthria can result from a basilar artery balloon occlusion (Hartmann, Conolly, Duong, Prestigiacomo, Joshi, Mohr & Mast, 1999).

5.8. *Conclusion*

It is not likely that the task of motor coordination could be assigned to any single neural structure. It is more likely that several different structures participate, depending on the

nature of the speech production task. There may be some critical structures or pathways, without which any complex motor coordination would be difficult or impossible. It also may be helpful to recognize different aspects of the organization of complex motor acts such as speech (e.g., sequencing, selection, coordination). There is some evidence for a modular organization of these aspects, and the foregoing review identifies certain neural structures that are candidates for a modular architecture.

6. Compensation, adaptation, and reorganization

A remarkable feature of speech is the capacity of speakers to compensate for a variety of perturbations and disruptions (oral anesthetization, mandibular fixation, dental appliances, prosthetics, and even food in the mouth). In this sense, speech exemplifies functional equivalence, in which a variety of movement patterns can be used to accomplish a specific goal or task. It is often remarked that individuals with speech disorders, especially structural disorders, can learn to use compensatory adjustments to overcome some of the negative effects of the disorder. It is an extremely interesting question to know how (and how well) individuals with neurologic disorders or structural abnormalities compensate for neurogenic disorders.

6.1. *Evidence for compensation*

The desiderata for compensation in speech are: (1) *acoustic equivalence* (*or near equivalence*) (the compensation should yield an acoustic product similar to that generated by the motor action that it is designed to replace); (2) *ease of motor execution* (the compensation should be executable as a readily performed articulatory movement); and (3) *compatibility with other movements in a sequence* (the compensation should not interfere with preceding and ensuing motor events in the phonetic sequences of speech). Neurologically intact individuals demonstrate considerable facility in compensating for dental appliances (Haydar, Karabulut, Ozkan, Aksoy & Ciger, 1996), jaw fixation by a bite block (Lindblom, Lubker & Gay, 1979), transient perturbations to an articulatory movement (Löfqvist, 1997), modification of oral structure by an artificial palate (McFarland, Baum & Chabot, 1996), or alterations of sensory feedback (Houde & Jordan, 1998). Studies of individuals who have undergone laryngectomy, glossectomy or osteotomy often show fairly successful compensation for structural defects, including some that result in nearly total loss of an articulator (Wakumoto, Isaacson, Friel, Suzuki, Gibbon, Nixon, Hardcastle & Michi, 1996; Laccourreye, Crevier-Buchman, Muscatello, Hans, Menard & Brasnu, 1998; Mahanna, Beukelman, Marshall, Gaebler & Sullivan, 1998; Sorokin, Olshansky & Kozhanov, 1998; Cotert & Aras, 1999). Sorokin *et al.* (1998) reported that some laryngectomized individuals recover speech function well enough not only to produce normal formant patterns but also to make voiced–voiceless distinctions. They interpreted this ability as being consistent with an internal model that can be used to re-assign muscles to accomplish phonetic distinctions. The study of speech production in both normal and disordered speech is replete with examples of compensation or adjustment. What is needed is a theory that accounts for these capacities.

A difficulty in this line of research in dysarthria is that compensation for a neurogenic disorder is rarely easily distinguishable from the disorder itself. For example, decomposition of movement (moving one joint at a time) is regarded as a pathological sign of

cerebellar ataxia (Holmes, 1939), and it might be supposed that decomposition is a direct consequence of failed mechanisms of the cerebellar coordination of movement. But it has been suggested that decomposition is an adopted, voluntary strategy that enables more accurate movements (Goodkin, Keating, Martin & Thach, 1993; Bastian & Thach, 1995). Similarly, some of the articulatory features in ataxic dysarthria may be the consequence of compensatory strategies employed to overcome faulty coordination of multi-articulator movement sequences. Bastian (1997) recommended that physical therapy for ataxia should emphasize avoidance of rapid multi-joint movements and a preference for slower movements of single joints.

A general approach to modeling control systems to ensure that they have the ability to contend with environmental variations is to equip them with both inverse (controller) and forward (predictor) models. Wolpert & Kawato (1998) proposed a new modular architecture that is based on multiple pairs of inverse and predictor models. This highly flexible system relies on a tight coupling of the inverse and forward models during acquisition of a skill, so that a given inverse model can be selected to meet a particular environmental need. The complementarity of inverse and forward models would offer distinct advantages in providing for compensatory or reorganizational capabilities in speech production.

6.2. *Conclusion*

One of the most remarkable aspects of speech production is its robustness, which is manifested in part by the ability of speakers to compensate for a variety of intrinsic and extrinsic disturbances. Although this issue is often mentioned in the study of both normal and disordered speech, a comprehensive model is still wanting. A model that holds great potential is one that combines inverse and forward models in a modular architecture.

7. General discussion

This review adds to recent efforts to bridge between studies of normal speech and studies of disordered speech (Weismer *et al*., 1995*a*, *b*). It appears that both normal and neurologically disordered speech can be explained in large part by a theory of speech motor control that is based on (a) internal models of the articulators, (b) rhythm-based sensory-motor integration, and (c) specification of articulatory dynamics within a motor program or motor score. Studies of dysarthric speech can provide information that complements data from normal speech to develop theories of speech motor control that account for speech development, normal speech regulation, and speech disorders resulting from neurologic disease.

There is fertile ground for the development of models for the neural control of speech. Much new information has been obtained through the study of individuals with neurogenic speech disorders and through the application of neuroimaging (Lauter, 1995) and stimulation methods (Urban *et al*., 1997; Epstein, 1998) to investigate neural mechanisms that control speech production and perception. The confluence of information from clinicoanatomic (lesion), neural activation, and stimulation studies should accelerate progress in understanding the neural control of speech. Even now, the classical description of the neural circuitry for spoken language is under revision (Aboitiz & Garcia, 1997*a*, *b*), and new ideas are emerging on the contribution of individual structures such

as the insula, SMA, basal ganglia, and cerebellum and the regulatory loops by which these structures govern complex behavior. This paper reviewed some examples of modularity in neural control to define hypotheses for research on normal and disordered speech. Eventually, it should be possible to relate information on neural circuitry to proposals for inverse and forward models for the regulation of movements.

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