This paper reviews issues in speech motor control and a class of communication disorders known as motor speech disorders. Speech motor control refers to the systems and strategies that regulate the production of speech, including the planning and preparation of movements (sometimes called motor programming) and the execution of movement plans to result in muscle contractions and structural displacements. Traditionally, speech motor control is distinguished from phonologic operations, but in some recent phonologic theories, there is a deliberate blurring of the boundaries between phonologic representation and motor functions. Moreover, there is continuing discussion in the literature as to whether a given motor speech disorder (especially apraxia of speech and stuttering) should be understood at the phonologic level, the motoric level, or both of these. The motor speech disorders considered here include: the dysarthrias, apraxia of speech, developmental apraxia of speech, developmental stuttering, acquired (neurogenic and psychogenic) stuttering, and cluttering. © 2000 by Elsevier Science Inc.

Educational Objectives: On the basis of this article, the reader will (1) be acquainted with recent directions in the study of normal speech motor control; (2) be able to discuss ways to separate motoric and linguistic factors potentially involved in dysarthria, apraxia of speech, and stuttering; (3) appreciate major current directions for research on speech motor disorders; and (4) know the relationship between research and clinical practice pertaining to motor speech disorders.

KEY WORDS: Dysarthria; Apraxia of speech; Stuttering; Speech motor control; Speech disorders

INTRODUCTION

Speech motor control refers to the systems and strategies that control the production of speech. Typically, it is assumed that the input to the system of speech motor control is a phonologic representation of language, especially a sequence of abstract units such as phonemes. The output of speech motor control is a series of articulatory movements that convey the intended linguistic message.
through an acoustic signal that can be interpreted by a listener. Therefore, the processes of speech motor control intervene between those of language formulation and those of the acoustic signal by which the speaker’s message is usually received. This paper focuses on speech motor control and largely, but not completely, excludes related processes in audition and language formulation. In fact, speech motor control ultimately must be understood in relation to the overall process of human speech communication, which is summarized diagrammatically in Figure 1. The investigation of how we speak draws on the ingenuity of several disciplines, including linguistics, psychology, engineering, physics, anatomy, biology, speech and hearing science, and computer science. Each of these, and more to be sure, has contributed to knowledge that is reflected in a skeletal form in Figure 1, which is based largely on Cleary and Pisoni (1998), Cutler, Dahan, and Van Donselaar (1997), Kent, Adams, and Turner (1996), Levelt (1989), Levelt, Roelofs and Meyer (1999), Meyer (1997), and Tyler and Frauenfelder (1987). In Figure 1, processes of language formulation and utterance production are represented on the left, and processes of speech perception and language comprehension are represented on the right. It is assumed that both speaker and listener maintain a discourse record, which is a mental log of primary points of information exchanged. In much of current theorizing, segmental structure and prosodic-affective patterns are processed somewhat independently, but merge into a unified representation of an utterance.

For all its ordinariness, speech is a remarkable and unique motor accomplishment. It is not unusual for speech to be produced at rates of up to six to nine syllables (or 20 to 30 phonetic segments) per second, which is faster that any other discrete human motor performance. Furthermore, speech production involves more motor fibers than any other human mechanical activity (Fink, 1986). In the face of these complications, speech must generate an acoustic signal that is understood by other listeners both as a linguistic (propositional) message and an affective signal. Spoken language is one of the greatest achievements of childhood, for it opens the door to a variety of educational and social experiences. Fortunately, speech is a robust faculty that serves most people in the face of various challenges to well-being and health. However, it can be impaired by a number of genetic and acquired conditions. A major group of these impairments are the motor speech disorders. This paper briefly reviews current research issues in normal speech motor control and selected motor speech disorders.

NORMAL SPEECH MOTOR CONTROL THROUGH THE LIFESPAN

The precursor to speech is the birth cry, which is the threshold to vocal communication. Infants typically acquire speech readily and naturally, and it appears that babbling anticipates important aspects of spoken language (de Boysson-
Figure 1. A diagram of the conceptual structures and processes involved in the production (left side of diagram) and perception (right side of diagram) of spoken language (based on sources listed in text). A variety of linguistic, cognitive, and sensorimotor processes are involved in spoken language, and the intent of this diagram is to portray some of the major sources of information that cohere in speech communication. The production of spoken language includes: prelinguistic aspects (intentions, preverbal message), discourse regulation (such as a discourse record mutually maintained by a speaker and listener), language formulation (lexical selection and syntactic construction), phonologic operations, phonetic specifications, and the motor control of the speech production system to generate acoustic patterns. To some degree, the comprehension of the spoken message involves operations inverse to those used in its formulation and production.
Bardies, 1999; Vihman, 1996). As soon as speech is acquired, children and adults use it with remarkable flexibility. Because speech production reflects the maturational status of sensory, motor, and cognitive systems, it is helpful to take a lifespan perspective, beginning with infancy.

Infancy

The developmental period of infancy takes its name from the child’s lack of articulate speech (infant means “incapable of speech”). However, it is clear from the last two decades of research that infants make rapid and substantial progress in both the perception of speech and the production of vocalizations that prefigure adult speech. Recent research has applied acoustic and physiologic methods to the study of infant vocalizations and related oral motor behaviors such as sucking, chewing, and swallowing (Finan & Barlow, 1998; Green, Moore, Ruark, Rodda, Movee, & van Wittenburg, 1997). Modern physiologic methods enable the study of important questions in the genesis of speech, such as the hoary issue of whether speech emerges from earlier appearing movements such as those used in mastication and deglutition. Recent results indicate that it does not, which means that in the early developmental period, the motor control for speech is distinct from that for nonspeech oral functions (Moore & Ruark, 1996).

An additional promise of this research is the possibility of using prespeech vocalizations to identify infants at risk for communication disorders or developmental disabilities. The vocalizations of interest include both cries (Moller & Schonweiler, 1999) and babble (Oller, Eilers, Neal, & Schwartz, 1999). Some particular points of progress in recent research include: evidence that the onset of canonical babbling marks a period in which infants use auditory awareness to control motor activities (Ejiri, 1998); physiologic studies of the coordination of lip and jaw movements in infants (Green, Moore, Higashikawa, & Steeve, 2000); description of patterns of breathing associated with different vocalization types (Boliek, Hixon, Watson, & Morgan, 1996, 1997); development of computer programs for automatic analysis of cry or babble (Fell, Macauslan, Ferrier, & Chenausky, 1999; Moller & Schonweiler, 1999); incorporation of babbling and auditory feedback in computer models of speech development in children (Bailly, 1997; Callan, Kent, Guenther, & Vorperian, 2000); and reports that babbling is sensitive to early-onset otitis media (Petinou, Schwartz, Mody, & Gravel, 1999; Rvachew, Slawinski, Williams, & Green, 1999), cerebral palsy (Levin, 1999), and maternal smoking (Obel, Henriksen, Hedegaard, Secher, & Ostergaard, 1998). The general value of babbling in the prediction of communication disorders in children has been described by Oller et al. (1999), among others.

Taken together, these studies demonstrate the value of babbling in under-
standing communicative development and the possibility of early identification based on babbling patterns. Babbling, one of the infant’s earliest ventures into speech motor control, reflects the child’s neurologic integrity as well as specific sensory and motor functions. The apparent early divergence between speech and nonspeech motor activities indicates that separate neural control systems are established in infancy. Further evidence of this separation is reviewed later in this paper. The continuity of babbling and other early vocalizations with phonetic patterns in early speech development points to the use of early vocalizations as a tool to identify infants at risk for communication disorders. This possibility speaks to an important health issue, given that “speech delays are the most common developmental concern seen by the general pediatrician, yet they often are not well understood or diagnosed expediently” (Johnson & Blasco, 1997, p. 224).

**Childhood and Adolescence**

The traditional studies of speech development were based on perceptual phonetic methods in which one or more examiners transcribed the speech of child participants (Vihman, 1996). Although these data were important in charting the developmental progress toward a mature phonetic system, the studies had limitations that may have affected the validity and reliability of the observations. Several advances have been made toward quantitative assessment of speech, including: structural magnetic resonance imaging (MRI) studies of the developmental anatomy of the vocal tract (Fitch & Giedd, 1999; Vorperian, Kent, Gentry, & Yandell, 1999), structural MRI studies of human brain development related to speech (Paus, Zijdenbos, Worsley, Collins, Blumenthal, Giedd, Rapoport, & Evans, 1999), acoustic profiles of speech development spanning childhood and adolescence (Lee, Potamianos & Narayanan, 1999), and improved indices of the maturation of speech movements in the articulatory and respiratory systems (Goffman & Smith, 1999; Smith & Goffman, 1998; Solomon & Charron, 1998).

Whereas traditional studies of speech development relied heavily on perceptual descriptions, much of the contemporary research uses quantitative methods that offer greater objectivity and sensitivity. In addition, the stage is being set for promising investigations into the coordination between different aspects of development and the potential for defining predictive measures that operate across behavioral domains. One example is the relationship between performance on a task of nonword repetition and language abilities in typically developing children (Baddeley, Gathercole, & Papagno, 1998). It also has been shown in several studies that children with language impairment perform more poorly than their typically developing peers on the task of nonword repetition (Bishop, North, & Donlan, 1996; Dollaghan & Campbell, 1998;
Sahlen, Wagner, Nettelbladt, & Radeborg, 1998; Stark, & Blackwell, 1997). To be sure, nonword repetition represents the combined influence of several speech and language processes, including auditory memory and phonologic processing. But it also entails preparation and execution of a speech movement sequence—which is to say, speech motor control.

**Young to Late Adulthood**

Much of adulthood pertains primarily to the maintenance and deployment of well established processes of speech motor control. Speech is vital to a wide range of vocations, and it is the means to various social and recreational pleasures. It is expected that speech will increasingly become a major form of human–machine interaction, and as it does so, productivity gains will surely follow. That is, the machines of the future will increase, rather than reduce, the need for speech as the means of communication for commercial and noncommercial pursuits. Machines will become more and more humanized, and a major dimension of humanization will be the sharing of speech between human and machine (Syrdal, Bennett, & Greenspan, 1995). Speech also will meet the need for miniaturization, because it will require only a small microphone for user input. Furthermore, speech can be used even as the arms and hands are devoted to other tasks.

With advancing age, speech changes in its precision, fluency, voice quality, and communicative effectiveness (Linville, 1996; Weismer & Liss, 1991; Wohlert & Smith, 1998). These changes may be similar to the more remarkable changes in speech that accompany a variety of diseases that occur most frequently in older adults. At the least, the age-related changes in speech mandate the use of age-appropriate normative data in the assessment of speech disorders in older adults. Although robust, speech is also intricate. The cognitive, sensory, and motor demands of speech production can be compromised by apparently normal aging processes and by a variety of diseases that become more common with advancing age.

Recent physiologic studies have provided much new information on non-speech oromotor behaviors such as swallowing (Chi-Fishman, Stone, & McCall, 1998; Rademaker, Pauloski, Colangelo, & Logemann, 1998). These data should help considerably in determining the extent of similarity or dissimilarity between the regulation of speech and nonspeech movements and they also contribute to an understanding of the effects of aging on general motor functions of the oral and perioral musculature.

Finally, neuroimaging methods have opened the door to many exciting discoveries about neural activity associated with speech and language. Lauter (1995) reviewed these methods and described their potential for fundamental advances in identifying the neural structures and circuits involved in the perception and production of spoken language.
NORMAL SPEECH MOTOR CONTROL: PROMISING LINES OF RESEARCH

Speech Motor Control Models

Speech has been modeled at various levels (e.g., information, neural, motor, acoustic) and with various methods (e.g., stage models, computational networks; Kent, Adams, & Turner, 1996). Each of these contributes to an overall understanding of speech as human behavior. Although a review of these efforts cannot be attempted within this brief paper, it is important to note at least a few recent advances that have the potential for understanding both normal and disordered speech motor control.

As is discussed later, one of the challenges in understanding speech motor disorders generally is to distinguish impairments of phonology from impairments of motor control per se. Possibly, impairments at both levels coexist in some disorders, and it is therefore important to determine the severity and nature of the two types of impairment. Phonologic theory can be relevant to clinical assessment and treatment and also to the theoretical understanding of the nature of a disorder. In some important respects, several contemporary phonologic theories emphasize the phonetic or articulatory bases of phonologic representations. Indeed, gestural (articulatory) phonology goes so far as to propose that abstract specifications of movements are phonologic primitives, that is, movements themselves are the means of phonologic representation (Browman & Goldstein, 1986, 1992). This proposal can have profound implications for understanding motor plans or programs in speech production. Another example of changing views in speech production research is a move away from the traditional segment-concatenation approach in which speech is thought to be controlled primarily with respect to concatenations of phonemic or phonetic segments. An alternative approach emphasizes a syllable organization, as implemented in the converter/distributor model (Fujimura, 1992, 1994).

Inquiries into the control of speech production encounter an immediate question: What is (are) the primary variable(s) controlled by the nervous system? An answer to this question is central to theories of speech motor control and also can be highly important in assessing and treating disorders of speech motor control. Several regulated variables have been proposed, including acoustic targets, aerodynamic values, positions of individual structures, overall vocal tract configuration, and rates of muscle contraction. It is possible that more than one variable ultimately is needed to understand the neural control of speech, and perhaps the robustness of speech derives in part from a speaker’s flexibility in choosing among these variables. In the case of velopharyngeal inadequacy in craniofacial disorders, a theory has been advanced to explain the regulation of speech in terms of the air pressure head (Warren, 1986; Warren, Dalston, & Dalston, 1990; Warren, Dalston, Morr, Hairfield, & Smith, 1989). This theory has a definite unifying value for a number of clini-
cal observations, including the speaker’s selection of compensatory articulations. Whereas Warren and associates emphasized the role of active reflexive compensatory responses in maintaining air pressure for speech, Moon, Follkins, Smith, and Luschei (1993) explained air pressure regulation in terms of intrinsic physiologic properties of the respiratory system. Although these ideas on air pressure regulation were introduced especially with regard to cleft palate, they could be applied more widely, for example, to the motor speech disorders.

Another way of stating the controlled variable problem is to ask what kind of target reference frame is used by the neural system to govern speech articulation. Currently, two primary alternatives are considered. One type of reference frame is a gestural target based on the degree of constriction at various locations in the vocal tract (Saltzman & Munhall, 1989). The other is an auditory or acoustic target that is used to derive the articulatory movement (Guenther, 1995; Guenther, Hampson, & Johnson, 1998; Perkell, Matthies, Lane, Guenther, Wilhelmms-Tricarico, Wozniak, & Guiod, 1997). Internal models have been particularly important in recent speech production theories, and they may hold value in understanding the motor speech disorders.

An important area of progress is the development of biomechanical models of the speech production system (Lucero & Munhall, 1999; Sanguineti, Laboissiere, & Ostry, 1998). As these models become increasingly sophisticated and accurate, they will offer insights not only into normal speech production but also into various speech disorders.

### Animal Models

There are no universally accepted animal models for spoken language, because no other species exhibits vocal behaviors that embrace the complexity and power of human speech. However, it is possible to use animal models for language-like behaviors in other methods (such as signing) or for certain components of speech production, such as phonation or vocal learning. Birds have been of particular interest because many species of birds learn the conspecific song through auditory exposure and a period of extended practice (Doupe & Kuhl, 1999; Hauser, 1996). Moreover, like human speech, “birdsong requires complex learned motor skills involving the coordination of respiratory, vocal organ and craniomandibular muscle groups” (Suthers, Goller, & Pytte, 1999, p. 927). Progress also has been made in understanding the acoustics of the avian vocal tract (Fletcher & Tarnopolsky, 1999). Certainly the biology and physics of birdsong differ in some respects from human speech, but there is nonetheless a potential benefit from studies of song learning and from research on the neural substrates of birdsong perception and production. It may even be possible that certain disorders of human communication have parallels in birdsong (e.g., deafness and stuttering, the latter being discussed later in
this paper). Although human vocalizations may differ in number and complexity from those of other species, some issues can be addressed in comparative studies, one example being the neural control of the muscle system coordination required for vocalization. Studies of vocalization in cats led to the proposal that the periaqueductal gray matter is an essential brain site for mammalian voice production (Davis, Zhang, Winkworth, & Bandler, 1996).

**DISORDERS OF SPEECH MOTOR CONTROL**

The disorders considered here are those that have been defined or studied in terms of speech motor control dysfunction, beginning with dysarthria and apraxia of speech (often termed the motor speech disorders; Duffy, 1995) and concluding with stuttering and cluttering, which are disorders of fluency. Classification of this broad range of disorders as motor speech disorders does not meet with universal approval. The fluency disorders, in particular, are not always classified in this way, although they have been studied extensively with methods and models from speech motor control. However, there is precedent for the classification used here (Caruso & Strand, 1999), and furthermore, the purpose of this paper is not to insist on a classification so much as it is to explore what a particular classification means in the contemporary understanding of a speech disorder.

**Dysarthria**

Dysarthrias are speech disorders that result from neurologic impairments associated with weakness, slowness, or incoordination of the musculature used to produce speech. These speech disorders occur with considerable frequency in individuals with Parkinson disease, stroke, cerebellar disease, amyotrophic lateral sclerosis, multiple sclerosis, cerebral palsy, and traumatic brain injury (Duffy, 1995; McNeil, 1997). Dysarthrias can be (a) congenital or acquired; (b) static, improving, or worsening in their course; (c) the consequence of pathologic conditions in multiple areas of the central or peripheral nervous system; and (d) associated with various causes (Duffy, 1994). The modern classification of the dysarthrias rests largely on two articles published three decades ago (Darley, Aronson, & Brown, 1969a, 1969b). These articles described the perceptual dimensions for the rating of dysarthria and identified seven types of dysarthria (Table 1) associated with distinctive clusters of deviant perceptual dimensions (Table 2). The authors also commented on the neuropathologies underlying each type of dysarthria, thereby contributing to a clinicoanatomic description. Remarkably, the seminal papers by Darley et al. have never been thoroughly replicated with an independent sample of individuals with dysarthria and an independent panel of judges (Kent, Kent, Duffy, & Weismer, 1998). The hegemony of the Darley et al. articles is testimony to the
seminal nature of their work and, unfortunately, to the limited scope of investigation during the last 30 years. But there is evidence of increased national and international scientific effort. This renewal is aided by several improvements in laboratory methods including acoustics, electromyography, movement transduction, electropalatography, and neuroimaging.

Generally, the dysarthrias have global, rather than focal, effects on the speech production systems of respiration, phonation, and articulation and resonance (Auzou, Ozsancak, Jan, Leonardon, Menard, Gaillard, Eustache, & Hannequin, 1998; Kent et al., 1998). Accordingly, they often affect multiple dimensions of spoken language (voice quality, intelligibility, prosody, and affect), and they present with challenges in clinical and scientific description. Significant

<table>
<thead>
<tr>
<th>Dysarthria type</th>
<th>Primary lesion site</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flaccid</td>
<td>Lower motor neuron (one or more cranial nerves)</td>
</tr>
<tr>
<td>Spastic</td>
<td>Upper motor neuron (pyramidal tract)</td>
</tr>
<tr>
<td>Spastic-flaccid</td>
<td>Both upper and lower motor neurons</td>
</tr>
<tr>
<td>Ataxic</td>
<td>Cerebellum or its outflow pathways</td>
</tr>
<tr>
<td>Hypokinetic</td>
<td>Basal ganglia, especially substantial nigra</td>
</tr>
<tr>
<td>Hyperkinetic</td>
<td>Basal ganglia, especially putamen, caudate, or both</td>
</tr>
</tbody>
</table>

Table 1. Clinicoanatomic Relationships for Major Types of Dysarthria. Shown for Each Type of Dysarthria (Determined by Auditory Perception) is the Primary Lesion Site

Table 2. Major Clusters of Deviant Perceptual Dimensions for Dysarthria, as Reported by Darley, Aronson, and Brown (1969a, 1969b)

<table>
<thead>
<tr>
<th>Type of Dysarthria</th>
<th>Clusters of Deviant Dimensions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ataxic dysarthria</td>
<td>Articulatory inaccuracy, prosodic excess, phonatory-prosodic insufficiency</td>
</tr>
<tr>
<td>Spastic dysarthria</td>
<td>Prosodic excess, prosodic insufficiency, articulatory-resonatory incompetence</td>
</tr>
<tr>
<td>Flaccid dysarthria</td>
<td>Phonatory incompetence, resonatory incompetence, phonatory-prosodic insufficiency</td>
</tr>
<tr>
<td>Spastic-flaccid dysarthria</td>
<td>Prosodic excess, prosodic insufficiency, articulatory-resonatory incompetence, phonatory stenosis, phonatory incompetence, resonatory incompetence</td>
</tr>
<tr>
<td>Hypokinetic dysarthria</td>
<td>Prosodic insufficiency, phonatory incompetence</td>
</tr>
<tr>
<td>Hyperkinetic dysarthria  (Chorea)</td>
<td>Articulatory inaccuracy, prosodic excess, prosodic insufficiency, articulatory-resonatory incompetence, phonatory stenosis</td>
</tr>
<tr>
<td>Hyperkinetic dysarthria  (Dystonia)</td>
<td>Articulatory inaccuracy, prosodic excess, prosodic insufficiency, phonatory stenosis</td>
</tr>
</tbody>
</table>
progress has been made in the description of the dysarthrias through acoustic analyses (Kent, Weismer, Kent, Vorperian, & Duffy, 1999; Weismer, 1997) and physiologic methods (Barlow, 1999; McNeil, 1997) as well as continued perceptual descriptions. In addition, now on the research horizon are contributions from neuroimaging techniques, especially positron emission tomography (PET) and magnetoencephalography, and perhaps eventually functional magnetic resonance imaging (fMRI) as soon as the problem of movement artifacts in the hemodynamic response is resolved. In addition, recent studies have applied transcranial magnetic stimulation, electrical stimulation, or both (Chen, Wu, & Chu, 1999; Ghezzi & Baldini, 1998; Trompetto, Caponnetto, Buccolieri, Marchese, & Abbruzzese, 1998; Urban, Vogt, & Hopf, 1998). It is expected that the use of neuroimaging methods and stimulation methods such as transcranial magnetic stimulation, combined with quantitative analyses of speech, will lead to rapid advances in the understanding of the dysarthrias. However, it should be noted that transcranial magnetic stimulation results are subject to some questions of interpretation (Epstein, 1998; Epstein, Meador, Loring, Wright, Weissman, Sheppard, Lah, Puhalovich, Gaitan, & Davey, 1999).

Assessment and classification of dysarthria. Dysarthria typically is assessed perceptually (especially by auditory pattern) and classified either with respect to the neurologic diagnosis (e.g., Parkinson disease) or a perceptual classification system (Edwards, 1984; Gerratt, Till, Rosenbek, Wertz, & Boysen, 1991; McNeil, 1997; Simmons & Mayo, 1997; Strand & Yorkston, 1994). Frequently used perceptual classifications are those of Darley et al. (1969a, 1969b) or Enderby (1983, 1986). One of the limitations of perceptual assessment is that it can be difficult even for highly trained auditors to differentiate the multiple dimensions of dysarthric impairment. It is not uncommon for an individual with dysarthria to have concurrent disorders of respiration, phonation, articulation, and resonance. Consequently, perceptual methods often are supplemented with acoustic analyses, imaging techniques, aerodynamic recordings, or movement transduction. These supplemental tools not only help to ascertain the function of individual components of speech production, but they also can be used to detect or infer pathophysiologic features such as weakness, rigidity, slowness, or dyscoordination. Ideally, these methods would be used in a thorough replication and extension of the original studies by Darley et al. (1969a, 1969b). Certainly, the stage is set for an expanded program of research that incorporates acoustic and physiologic measures.

Research has shown both age- and sex-related variations in dysarthria. One of the few extensive reviews of childhood dysarthria concluded that “definite similarities to adult dysarthria were not evident [and that] acquired childhood dysarthria requires its own classification” (Van Mourik, Catsman-Berrevoets, Paquier, Yousef-Bak, & van Dongen, 1997, p. 299). Additional support for this conclusion appeared in a study of children with cerebellar or brainstem tumors (Van Mourik, Catsman-Berrevoets, Yousef-Bak, Paquier, & van Don-
Strong similarities were not seen between the children’s dysarthric features and those that have been reported for adults. The recent literature attests to an increasing awareness of the need for developmentally appropriate assessments and treatments, together with a theory of disorder that incorporates various types of developmental information (Caruso & Strand, 1999; Paul & Marans, 1999).

Sex-dependent features of dysarthria only recently have been identified (Hertrich, Spieker, & Ackermann, 1998; Kent, Kent, Rosenbek, Weismer, Martin, Sufit, & Brooks, 1992). In some respects, sex-related differences are not surprising given the sexual dimorphism of the speech production system and sex-related differences in the effects of aging on speech and voice. Sex differences may emerge more powerfully in communication disorders than in other aspects of impaired function, such as control of the limbs and fingers. The further description and classification of the dysarthrias may well entail considerations related to both age and sex of the speaker.

**Treatment of dysarthria.** Many treatments have been used for individuals with dysarthria. The selection of treatment varies with type and severity of the dysarthria, and with the general cognitive, affective, and sensorimotor status of the individual patient. Treatment outcomes have not been studied extensively, but Yorkston’s (1996) review indicated that several interventions have beneficial effects. However, she also noted that a given treatment is not uniformly effective for all types of dysarthria. Therefore, “Guidelines are needed that specify which treatments are most effective for the various dysarthrias” (Yorkston, 1996, p. S53). One particular behavioral intervention designed especially for individuals with Parkinson disease, the Lee Silverman Voice Treatment (Ramig, Bonitati, Lemke, & Hori, 1994; Ramig, 1998), probably has been more systematically investigated than any other. The series of studies on this treatment may well serve as a model for other treatments for other types of dysarthria.

Many dysarthrias result from chronic conditions that cannot be treated successfully with surgery or medications. Accordingly, there is a strong need for behavioral interventions with demonstrated effectiveness. For many individuals with dysarthria, a combination of behavioral and drug or prosthetic treatments should be beneficial, but there is very little systematic research on this issue. Perhaps future work will demonstrate the value of “supportive drug therapy” such as the use of piracetam in conjunction with language therapy in poststroke aphasia (Huber, 1999; Poeck, 1998). However, at least with respect to some current drug and surgical treatments for Parkinson disease, it is does not appear that speech derives the same benefit as nonspeech motor control. For example, recent studies of levodopa treatment, pallidotomy, and pallidal stimulation have shown positive effects on finger movements, handwriting, or overall performance on the Unified Parkinson’s Disease Rating Scale but neutral or even negative effects on speech (Gentil, Tournier, Perrin, & Pollak,
Acquired Apraxia of Speech (Verbal Apraxia)

Like dysarthria, apraxia of speech can appear developmentally as a childhood disorder or it can appear as an acquired disorder in adults. Both the developmental and acquired forms have engendered a literature that defies easy summary. One major obstacle is the lack of agreement on how (and even if) apraxia of speech should be recognized as a clinical entity. It is also uncertain if the developmental form of the disorder parallels the acquired form in its symptomatologic features and cause. The two forms are distinguished in this review, which begins with the acquired form in adults.

Assessment and classification of apraxia of speech. Acquired apraxia of speech in adults is generally thought to result from focal brain (especially cerebral) damage that impairs especially the processes of planning or programming speech movements in the face of essentially normal strength, speed, and coordination of the speech musculature (especially for nonspeech tasks but also for some speech tasks; Duffy, 1995). With some risk of oversimplification, it may be said that apraxia of speech impairs the programming of speech movements, whereas the dysarthrias affect the execution of movements. That is, it often is assumed that in apraxia of speech, the movement plan is disturbed but the muscular system itself may be essentially intact. In dysarthria, the movement plan is intact, but the muscular systems of speech production cannot realize the movement plan. Early perceptual descriptions of apraxia of speech remarked on the large number of substitution errors, the impression of a slow and groping articulatory pattern, and pronounced difficulty with long or phonetically complex sequences. The disorder has been described variously as phonologic, motoric, or cognitive, and perhaps all three will be subsumed in an eventual understanding. Contemporary articles reflect the continuing effort to define the nature and cause of apraxia of speech (Code, 1998; Dogil & Mayer, 1998; Rogers & Storkel, 1999; Whiteside & Varley, 1998; Varley, Whiteside, & Luff, 1999).

Several recent studies indicate that the lesion responsible for apraxia of speech may be quite discrete. Dronkers (1996) reported 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 without lesions in this structure did not have deficits in articulatory planning. A confirmatory case report described apraxia of speech occurring after an acute
infarct limited to the precentral gyrus of the left insula (Nagao, Takeda, Komori, Isozaki, & Hirai, 1999). A role of the insula in speech planning also is supported by studies of normal subjects using the functional imaging methods of magnetoencephalography (Kuriki, Mori, & Hirata, 1999) and PET (Wise, Greene, Buchel, & Scott, 1999).

Assessment and classification of developmental apraxia of speech. Developmental apraxia of speech was first described by Yoss and Darley (1974), who apparently observed similar symptomatologic features between a speech disorder in children and a speech disorder in adults that had been previously described. Whether the symptomatologic features are, in fact, similar remains in question. A major difference between the two forms is that the developmental form may affect the phonologic or motoric processes, or both, by which spoken language is learned, whereas the acquired form is an impairment of previously acquired processes.

A central issue in research on developmental apraxia of speech is whether this disorder can be reliably distinguished from more general speech impairments in children (Davis, Jakielski, & Marquardt, 1998; Hall, Jordan, & Robin, 1993; McCabe, Rosenthal, & McLeod, 1998; Ozanne, 1995). Recent articles on developmental apraxia of speech have identified characteristics of the disorder that could be used as diagnostic markers. The characteristics vary somewhat across studies, and they include limitations in maximum performance tasks (Murdoch, Attard, Ozanne, & Stokes, 1995; Thoonen, Maassen, Gabreels, & Schreuder, 1999; Thoonen, Maassen, Gabreels, Schreuder, & de Swart, 1997); distinctive patterns of segmental errors (Groenen, Maassen, Crul, & Thoonen, 1996; Forrest & Morrisette, 1999); perception or production of rhyme (Marion, Sussman, & Marquardt, 1993); or developmentally inappropriate patterns of stress (Shriberg, Aram, & Kwiatkowski, 1997a, 1997b, 1997c; Skinder, Strand, & Mignerey, 1999). Given (a) the typically small number of participants recruited in these studies, (b) differences in results across studies, and (c) the possibility of nonhomogeneity in the clinical population, it is premature to assert a single diagnostic marker. Progress would be facilitated by data collection at multiple centers using a well-defined protocol of clinical examination. Suggestions for diagnostic criteria have been proposed, and these would be helpful in assuring a common diagnostic system (Hodge, 1994; Hodge & Hancock, 1994).

Developmental apraxia of speech presumably is one form of a more general developmental apraxia, although this assumption should not be accepted uncritically. Even the more general form of the disorder, which has been recognized for almost 100 years, is by no means straightforward in its definition and description (Dewey, 1995). One simplification that may apply to non-speech apraxia is that the developmental and acquired forms appear to be similar (Poole, Gallagher, Janosky, & Qualls, 1997). It is not at all clear that a similar statement can be made for apraxia of speech.
Neuroimaging has rarely been used to study developmental apraxia of speech, but PET and MRI results were reported for members of a large, three-generation pedigree (the KE family), half of whom had a pronounced apraxia of speech presenting especially as difficulties in sequential articulation and orofacial praxis (Vargha-Khadem, Watkins, Price, Ashburner, Alcock, Connelly, Frackowiak, Friston, Pembrey, Mishkin, Gadian, & Passingham, 1998). Abnormalities were detected in both cortical and subcortical motor-related areas of the frontal lobe. Magnetic resonance imaging revealed 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). These results are particularly interesting in that they are among the first to show a specific genetic mutation or deletion that is associated with developmental apraxia of speech and with specific neural abnormalities. (A similar effort has been described for a family with autosomal dominant rolandic epilepsy and speech dyspraxia [Scheffer, Jones, Pozzebon, Howell, Saling, & Berkovic, 1995].) If future studies confirm involvement of the caudate nucleus in developmental apraxia, it may be significant that the volume of both the caudate and lenticular nuclei declines with maturation in males but not females (Giedd, Snell, Lange, Rajakapakse, Casey, Kozuch, Vaituzis, Vauss, Hamburger, Kaysen, & Rapoport, 1996). Possibly, sex-related differences in brain maturation account at least in part for the preponderance of males in populations with developmental apraxia of speech (and stuttering as well).

**Treatment of apraxia of speech and developmental apraxia of speech.** Because rather similar treatments have been recommend for both the acquired and developmental forms of this disorder, the two forms are considered together. A range of treatments for the acquired form are described by McNeil, Robin, and Schmidt (1997). Generally, these treatments focus on articulatory placement, temporal sequencing, or integral (cross-method) stimulation combined with progressive task difficulty. Efficacy data for most of these approaches are rare or nonexistent, although some treatment outcomes have been described (Wambaugh, Kalinyakfiszar, West, & Doyle, 1998). Treatments for developmental apraxia of speech have been described by Pannbacker (1988), Square (1994, 1999), and Strand and Skinder (1999). Among the treatments are integral stimulation, tactile-kinesthetic facilitation, rhythmic and melodic facilitation, and gestural cuing. Data on treatment outcome, effectiveness, and efficacy are limited.

**Fluency Disorders**

The fluency disorders include developmental stuttering, cluttering, and acquired stuttering in the older child or adult. Terminology and diagnostic criteria are not uniform, but recent efforts to clarify and systematize the terminol-
ogy and defining characteristics have been made ("Terminology pertaining to fluency and cluency disorder," 1999; Perkins, 1994). The developmental and acquired forms are distinguished here because they are associated with somewhat different symptomatologic features and perhaps causes. The present discussion emphasizes developmental stuttering (which typically appears in early childhood) and acquired neurogenic or psychogenic stuttering (which occurs especially in adults). Only passing reference is made to cluttering, which is not well represented in the scientific literature and is open to much debate concerning its nosologic status.

Assessment and classification of developmental stuttering. The following quotation from a recent article in the *American Family Physician* is a good point of departure for a discussion of developmental stuttering:

The etiology of stuttering is controversial. The prevailing theories point to measurable neurophysical dysfunctions that disrupt the precise timing required to produce speech. Stuttering is a common disorder that usually resolves by adulthood. Almost 80 percent of children who stutter recover fluency by the age of 16 years. Mild stuttering is self-limited but more severe stuttering requires speech therapy, which is the mainstay of treatment. Delayed auditory feedback and computer-assisted training are currently used to help slow down speech and control other speech mechanisms. Pharmacologic therapy is seldom used although haloperidol has been somewhat effective. (Lawrence & Barclay, 1998, p. 2175)

The cause of developmental stuttering is in fact unclear, but a major line of research over at least three decades has investigated the possibility of a motor control disorder as at least one component (Ingham, 1998; Peters, 2000; Postma, Kolk, & Povel, 1990). Evidence for a motor control involvement in the cause of stuttering is reviewed here, but it should be noted that this involvement is controversial, and some authors have questioned if research to date has securely established that stuttering should be viewed as a disorder of speech motor control (Ingham, 1998).

Part of the theoretic uncertainty hinges on the degree to which stuttering is a motoric disorder or a linguistic disorder. In particular, three major hypotheses have been advanced regarding motoric and linguistic factors in stuttering (Peters, 2000; Peters & Starkweather, 1990). The first hypothesis is that at least two subgroups should be recognized among stutterers, one subgroup with a predominant motoric deficit and another with a predominant linguistic deficit. Confirmation of this hypothesis requires epidemiologic studies of a suitably large number of subjects with well-defined criteria to distinguish the hypothesized motoric and linguistic subtypes. A variant of this first hypothesis is that a developmental imbalance between motoric and linguistic factors accounts for stuttering. That is, as the child acquires language, the skills for language and speech motor control may not be commensurate. The second hypothesis is that language formulation and the motor processes of speech are
mutually interfering, and it is this interference that explains stuttering. According to this view, both motoric and linguistic abnormalities should be evident. The third hypothesis is that speech fluency is affected by various language processes, including aspects of language competence (such as lexicon size and number of syntactic forms) and language performance (for example, word finding and sentence construction). In this view, various linguistic stressors may lead to dysfluencies, and the identification of these stressors is a prime objective in research on stuttering. In addition to these hypotheses, it also should be acknowledged that the movement abnormalities observed in individuals who stutter may not be core motor disturbances, but rather compensations used in an attempt to produce fluent speech (Jancke, Bauer, Kaiser, & Kalveram, 1997; Van Lieshout, Hulstijn, & Peters, 1996a, 1996b).

Evaluation of these hypotheses requires the investigation of both motoric and linguistic factors in stuttered speech, along with paradigms such as concurrent attention-demanding tasks (Bosshardt, 1999), detailed analysis of language associated with dysfluencies (Howell, Au-Yeung, & Sackin, 1999; Logan & LaSalle, 1999; Ratner, 1997a, 1997b; Yaruss, 1999), and further assessments of nonspeech motor skill (Ward, 1997; Zelaznik, Smith, Franz, & Ho, 1997).

Recent advances in stuttering research include applications of neuroimaging and the use of acoustic and physiologic measurements of motor coordination. In addition, studies have been carried out to examine the genetics, epidemiologic, comorbid, predispositional, and neurologic correlates of stuttering. These are complex topics that can be mentioned only briefly and selectively here.

Neuroimaging results point to atypical patterns of activation in the brains of stutterers compared with nonstuttering control participants. However, differences in methods and results across studies make it difficult to draw a unified conclusion at this time. A PET study of three individuals who stutter indicated increased 6-FDOPA uptake in ventral limbic cortical and subcortical regions, which was interpreted as evidence for an overactivity in the presynaptic dopamine system in brain structures that control speech (Wu, Maguire, Riley, Lee, Keator, Tang, Fallon, & Najafi, 1997). Another PET study showed increased activity in anterior forebrain regions but reduced activation in postrolandic regions concerned with perception and sensory decoding (Braun, Varga, Stager, Schulz, Selbie, Maisog, Carson, & Ludlow, 1997). Neuromagnetic responses to monaural tones also distinguished individuals who stutter from those who do not, and it was suggested that a nonoptimal processing of auditory input may contribute to dysfluencies (Salmelin, Schnitzler, Schmitz, Jancke, Witte, & Freund, 1998). In a PET functional activation study by Fox and associates (Fox, Ingham, Ingham, Hirsch, Downs, Martin, Jerabek, Glass, & Lancaster, 1996), individuals who stutter had distinguishing and significant activations in the supplementary motor area, superior lateral premotor cortex, and primary motor cortex with right dominance. Those who stutter also showed a prominent cerebellar activation and, interestingly, nonactivation or deactivation of auditory areas of superior temporal cortex.
Interpretation of the neuroimaging data is not straightforward. A skeptic may conclude that the studies simply show that behavioral disturbances are associated with abnormal patterns of neural activation, much as different neural activations may be seen for limping versus normal walking. For that matter, fundamental questions remain to be answered, such as whether additivity of activation patterns can be assumed (Sidtis, Strother, Anderson, & Rottenberg, 1999). However, neuroimaging studies eventually may point to inherent differences in brain organization as well as showing how differential neural activations are associated with fluency versus dysfluency. There is also potential for a synthesis of evidence from studies of brain activation, stuttering-related brain lesions, and cortical stimulation that either induces or prevents stuttering. Recently, for example, Muroi, Hirayama, Tanno, Shimizu, Watanabe, and Yamamoto (1999) reported on an individual who stopped stuttering after bilateral thalamic infarction. They pointed out that the infarction affected two thalamic nuclei that have been implicated in stuttering in previous activation, lesion, and stimulation studies. It was concluded that both developmental and acquired stuttering are associated with disturbed function of one or both of two reciprocal thalamocortical circuits, one between supplementary motor area and the thalamic centromedian nucleus, and the other between the dorsomedial nucleus of thalamus and the lateral prefrontal area. It is noteworthy that a dopaminergic association with stuttering has been proposed (Anderson, Hughes, Rothi, Crucian, & Heilman, 1999; Canter, 1971) and that the thalamus has been implicated as a central generator for parkinsonian tremor, with involvement of peripheral inputs as either part of an unstable reflex loop or as a modulator of the central oscillator (Hua, Reich, Zirh, Perry, Dougherty, & Lenz, 1998). Still another neural correlate was reported for an individual who had Tourette syndrome with comorbid obsessive-compulsive disorder, attention-deficit hyperactivity disorder, stuttering, and gait disturbance (Demirkol, Erdem, Inan, Yigit, & Guney, 1999). Magnetic resonance imaging scans revealed bilateral and symmetrical globus pallidus lesions with a specific “tiger’s eye” appearance.

The identification of specific structures or pathways involved in the cause of stuttering is problematic because of the lack of agreement among published studies. However, the foregoing discussion chronicles recent progress that may herald a new understanding of neurologic abnormalities associated with developmental stuttering. At least three general interpretations may be drawn from the available data. First, stuttering can result from a variety of neurologic disturbances and is not necessarily related to damage to any one structure or neural pathway. Second, stuttering is particularly associated with anomalies in hemispheric asymmetry. Third, stuttering reflects damage to the extrapyramidal motor pathway, especially the basal nuclei, thalamus, or thalamocortical connections.

Several writers have reported on both progress and potential in epidemiologic and genetic studies of stuttering (Felsenfeld, 1996, 1997; Kidd, 1980;
Several intriguing results have been reported, but consensus has not been reached on some critical issues. Felsenfeld (1997) suggested that the course of research should consider: (a) the development of a standard assessment battery that can be used to diagnose stuttering through the lifespan; (b) examination of the relationship between family history status and epidemiologic variables (e.g., stuttering severity and probability of spontaneous remission); (c) exploration of extrinsic (environmental) factors that may precipitate or maintain the disorder; and (d) increase the pool of high-density pedigrees available for genetic modeling and linkage analyses.

It has long been known that children who stutter are more likely than non-stuttering control children to have other speech and language disorders. Certainly, it can be argued whether the comorbidity reflects a general susceptibility to communication disorder, or if the associated problems are secondary to the stuttering (e.g., the stuttering interferes with speech and language development, so children who stutter are disadvantaged). Recent work has helped to demonstrate associations between stuttering and a variety of other conditions, including Tourette syndrome features (Abwender, Trinidad, Jones, Como, Hymes, & Kurlan, 1998), specific language impairment with phonologic involvement (Hall, 1999), phonologic delay (for children whose stuttering is persistent; Yairi & Ambrose, 1999), and hyperfunction of auditory self-monitoring (Ratner, 1997a). Perhaps none of these accompanying features points to a definite causal link, but all of them together could possibly define a neurobehavioral composite.

It also may be critical to take a lifespan perspective to create a coherent view of stuttering and its correlates. For example, the apparent hyperfunction of auditory self-monitoring observed in children’s stuttering (Ratner, 1997a, 1997b) stands in contrast to the reduced or atypical activation of auditory cortex seen in adults who stutter (Braun et al., 1997; Fox et al., 1996; Salmelin et al., 1998). It is known that adults who do not stutter tend to show an inhibition of auditory cortex during self-produced speech (Numminen, Salmelin, & Hari, 1999), but it is not clear when and how this inhibitory effect emerges. One interpretation of the data on persons who stutter is that children who experience stuttering initially have a highly activated auditory cortex during stuttering but gradually learn to inhibit activity strongly in this cortex, especially during episodes of dysfluency. It also should be noted that neuroanatomic studies suggest that there is a protracted structural maturation of the fiber pathways associated with auditory and motor speech functions (Paus, Zijdenbos, Worsley, Collins, Blumenthal, Giedd, Rapoport, & Evans, 1999), which means that early sensorimotor experience with speech could shape the neural pathways. Structural maturation of the brain is accomplished during childhood, as speech and language are acquired.

The value of a lifespan perspective is shown by other recent work as well. It appears that the loci of dysfluencies change with language maturation. A
change in stuttering locus from function words to content words was reported by Howell, Au-Yeung, and Sackin (1999). In a latent trait study of the development of persistent stuttering, it was concluded that stuttering progressed through four general stages that reflected an increasing integration and stabilization (Kalinowski, Kalinowski, Stuart, & Rastatter, 1998).

Is it possible that stuttering-like behaviors occur in animals? Leonardo and Konishi (1999) reported that perturbations of auditory feedback in adult zebra finches caused a decrystalization (disintegration of mature song) that included a “stuttering” effect. This demonstration of a stuttering-like phenomenon in birds joins studies of deafening in birds as routes to the use of animal models for certain kinds of communication disorders in humans. As a highly speculative example, consider the following. It has been proposed that developmental stuttering is related to transmission of a single major genetic locus (Ambrose, Yairi, & Cox, 1993). It has also been reported that motor-driven gene expression occurs in songbirds (Jarvis & Nottebohm, 1997). Perhaps a motor-driven gene expression is involved in the explanation of stuttering in humans.

**Treatment of developmental stuttering.** Because most of the literature on treatment of stuttering pertains to the developmental form, discussion of treatment is limited here to developmental stuttering. Given that most children who stutter will recover, what distinguishes the child who will recover from the child who will not? An answer to this question could determine the use of clinical intervention. It has been suggested that resistant and recovered stuttering have a common genetic cause, with persistence being related partly to additional genetic factors (Ambrose, Cox, & Yairi, 1997). It is not only children who recover from stuttering, but some adults as well (Finn, 1996, 1998). The distinction between resistant and recovered stuttering is important both to efficient clinical intervention but also to an understanding of stuttering itself.

A variety of treatment approaches have been developed for stuttering, and progress also has been made in defining criteria for studies of treatment outcomes and efficacy (Cordes, 1998; Ingham & Riley, 1998). Conture (1996), who reviewed published data on treatment outcomes, concluded that “[th]e average number of people estimated to stutter who benefit from treatment (7 of 10) undoubtedly varies with age (younger clients appear to improve somewhat more quickly and more easily than older clients), severity, type, and/or length of stuttering (longer history of stuttering appears to increase the duration of treatment and decrease likelihood of a complete recovery)” (p. S24). A more recent study demonstrated that the benefits of treatment for children and adolescents are evident for several years after intervention (Hancock, Craig, McCreany, McCaul, Costello, Campbell, & Gilmore, 1998).

**Assessment and classification of acquired stuttering.** Acquired stuttering is associated with various conditions, but especially brain lesions, pharmacologic effects, and psychogenesis. Studies in the first two of these areas do not point to discrete or focal causes of neurogenic stuttering. In a study of four
individuals with stuttering occurring after stroke at presentation, Grant, Bisousse, Cook, and Newman (1999) concluded that both the clinical presentation and lesion sites were variable across the four individuals. Other lesion sites have included either bilateral diffuse lesions or a unilateral lesion (Heuer, Sataloff, Mandel, & Travers, 1996), a hemispheric lesion in addition to a callosal lesion (Tsumoto, Nishioka, Nakikita, Hayashi, & Maeshima, 1999), and striatocapsular infarction (Kono, Hirano, Ueda, & Nakajima, 1998). As noted earlier, thalamocortical pathways have been proposed as primary lesion sites in both developmental and acquired stuttering, and these should receive attention in future studies. With respect to drug-induced stuttering, Brady’s (1998) review did not identify a single neurotransmitter system as causative, but rather suggested that stuttering results from multiple, interacting neurotransmitter systems. Psychogenic stuttering is distinguished from neurogenic stuttering primarily in response to treatment, with the former showing a relatively rapid and favorable response to behavioral therapy and associated management strategies (Baumgartner & Duffy, 1997). Possibly, dysfluent speech is a behavioral consequence of a variety of neural disturbances, and there may not be a common neuropathologic characteristic across the various reports of acquired stuttering.

Assessment and classification of cluttering. Cluttering is recognized by some authorities as another dysfluency disorder, distinct from stuttering. The literature on cluttering is quite small and, even for its limited quantity, addresses uncertainties of diagnosis and cause similar to those already considered for stuttering. A special issue of the Journal of Fluency Disorders (1996, Volume 21, Numbers 3–4) is devoted to cluttering. One of the contributions is a bibliography for papers published since 1964 (Myers, 1996). Cluttering would benefit from the same general research directions summarized for stuttering and apraxia of speech.

CONCLUSIONS

Research progress has been made on several fronts, and it is clear that the various areas of research have the potential for a powerful convergence on a deeper understanding of how speech is controlled and how the desired regulation is affected by disorders such as dysarthria, apraxia of speech, and fluency disorders. General conclusions regarding the research literature on these disorders are as follows:

1. There are continuing uncertainties with regard to cause and assessment.
2. There is a limited research effort on treatment outcomes and efficacy, with a nearly total absence of randomized clinical trials.
3. Quantitative instrumental methods (acoustic and physiologic techniques) are being increasingly applied in the study of these disorders, and these
methods complement the perceptual descriptions that have formed much of the clinical literature.

4. Neuroimaging methods are beginning to be used to detect neuropathologies, but the numbers of participants in such studies are quite small and the results are not always consistent across studies.

5. Particularly for dysarthria and apraxia of speech, the literature reflects a limitation in the amount and variety of data. Developmental stuttering is associated with an immense literature, but fundamental questions remain about this disorder.

6. Each of these disorders will very likely be illuminated by research methods and technologies now at hand. A particularly promising direction is for multidisciplinary and multitechnology studies. For example, several writers have endorsed a program of research for stuttering that embraces epidemiology, genetics, neurology, speech pathology, and speech science. The same combination of specialties are needed for research on dysarthria and apraxia of speech.

7. For all of these disorders, both a developmental form and an acquired (adult) form have been recognized. However, despite some general similarities in symptomatologic factors, it is not clear that the developmental and adult forms reflect the same mechanisms of impairment. It may be unwise to impose clinical nosology for adults unto children. Disorders that affect speech motor learning in children may be fundamentally different from the disorders that disrupt previously acquired speech motor skills in adults.

8. Finally, research on both normal and disordered speech confronts the question of how linguistic and motoric aspects can be distinguished in the planning and execution of speech movements. Important advances could well turn on the resolution of this question, which is a contemporary issue in both theory and empirical research.

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REFERENCES


ing speech and language production in developmental stuttering—an H20-15 positron emission tomography study. *Brain, 120*, 761–784.


speech dimensions in the dysarthrias. *Journal of Speech and Hearing Research, 12*, 462–496.


Fox, P.T., Ingham, R.J., Ingham, J.C., Hirsch, T., Downs, J.H., Martin, C.,


pallidotomy for Parkinson’s disease: Results after more than 1 year. *Journal of Neurology, Neurosurgery & Psychiatry, 67*, 511–517.


CONTINUING EDUCATION

Research on Speech Motor Control and Its Disorders: A Review and Prospective

1. Compared with other motor skills in humans, speech:
   a. Is relatively slow in the rate of movements performed
   b. Is produced at rates faster than any other discrete human motor performance
   c. Involves more motor fibers than any other human mechanical activity
   d. Both b and c

2. Recent research reviewed in this article indicates that the motor behavior in babbling and other infant vocalizations:
   a. Derives directly from other oral behaviors such as chewing and swallowing
   b. Has little or no value in identifying children with health problems or with potential communication problems
   c. Is continuous with some aspects of later speech development
   d. Is completely unaffected by the child’s hearing status

3. Dysarthrias are defined as speech disorders that:
   a. Involve disturbances in the motor programs that specify movement sequences
   b. Are characterized primarily by excessive or exaggerated movements in the production of speech
   c. Result from neurologic impairments associated with weakness, slowness, or incoordination of the musculature used to produce speech
   d. Result exclusively from damage to the peripheral nervous system (the cranial and spinal nerves)

4. Children who stutter are:
   a. More likely than children who do not stutter to have other speech and language disorders
   b. Very unlikely to overcome the speech disorder without therapy
   c. Less likely than adults who stutter to improve with therapy
   d. Both b and c

5. Which of the following structures has been shown by recent neuroimaging studies to be associated with articulatory planning and the disorder of apraxia of speech in adults?
   a. Cerebellum
   b. Insula
   c. Putamen
   d. All of the above