Clinicoanatomic Studies in Dysarthria: Review, Critique, and Directions for Research

More than 30 years ago, Darley, Aronson, and Brown (1969) proposed clinico-anatomic correlations for seven perceptual types of dysarthria. These correlations have not been systematically re-examined even though imaging technologies developed in recent years provide the means to do so. This review considers data from published imaging studies as well as data from selected medical interventions to evaluate the current state of knowledge that relates lesion site to the nature of a speech disturbance. Although the extent data are not sufficient to allow a complete evaluation of the seven types of dysarthria described by Darley et al., relevant information has been reported on lesions of the pyramidal pathway, extrapyramidal pathway, and cerebellum. In general, the results are best explained by an equivalence mode of brain-behavior relationship in which a type of dysarthria is associated with a lesion in one of two or more brain structures. Criteria also are proposed for future studies of clinicoanatomic relationships in neurogenic communication disorders.

KEY WORDS: dysarthria, neuroimaging, neurologic disorders, clinicoanatomic studies, speech disorder

A broad goal of research on neurogenic disorders is to establish the relationship between lesion and functional disturbance. In two classic articles, Darley, Aronson, and Brown (1969a, 1969b) proposed a systematic classification of adult dysarthrias including hypotheses that relate site of lesion and type of speech disturbance. Darley et al. described the dysarthrias associated with seven discrete neurological groups and concluded that, “Speech indeed follows neuroanatomy and neurophysiology” (1969, p. 246). Location of the lesion responsible for the dysarthria was explicit in the design of their studies, because the clinical groups were constituted as follows (with site of lesion given in parentheses): bulbar palsy (lower motor neuron lesion), pseudobulbar palsy (upper motor neuron lesion), amyotrophic lateral sclerosis (lesions to both upper and lower motor neurons), cerebellar disorders (lesions to the cerebellum), parkinsonism (extrapyramidal lesion), dystonia (extrapyramidal lesion), and chorea (extrapyramidal lesion). The localization hypotheses were based primarily on clinical observations, especially the perceptual attributes of speech samples considered against classical formulations of neuropathology. Imaging data were rarely available. These clinicoanatomic bases of dysarthria have stood essentially intact for three decades (Duffy, 1995; Kent, Kent, Duffy, & Weismer, 1998).

Since the publication of the classic papers by Darley et al. (1969a, 1969b), many articles have been published on the dysarthrias, and a
number of these include information on site of lesion determined by neuroimaging, medical interventions, experimental stimulation methods, or other procedures. The goal of this review is to examine clinicoanatomic studies and to synthesize the results into a progress statement on the neuropathologic bases of the dysarthrias.

### Information Considered in This Review

This review covers especially those reports in which some form of neuroimaging was used to detect lesions in individuals with dysarthria. The neuroimaging methods of potential interest are computed tomography (CT), magnetic resonance imaging (MRI), functional magnetic resonance imaging (fMRI), positron emission tomography (PET), single photon emission tomography (SPECT), magnetoencephalography (MEG), and electroencephalography (EEG).

The following caveats should be noted:

1. Studies of neural lesions related to dysarthria often are more much informative concerning the size and location of the lesion than on the characteristics of dysarthria. Frequently, the dysarthria is neither classified nor described in a way that adequately portrays the speech disturbance (O’Shanick, 1997). Furthermore, in some studies it is not even clear if the general term dysarthria (let alone its type or features) is accurate because the speech disruptions may have taken other forms, such as apraxia of speech or aprosodia. This shortcomings of the literature has several explanations, one of which is simply that individuals with the expertise needed to make speech assessments were not involved in the studies.

2. Neuroimaging has certain limitations in the study of speech and its disorders. Because fMRI is affected by movement artifacts related to speech articulation, this method is of limited use for speech studies (Kent, 1998). Although improvements are underway that should permit the use of fMRI to study brain activation during speech, these new technologies are only emergent at this time (Birn, Bandettini, Cox, & Shaker, 1999; Lotze, Seggewies, Erb, Grodd, & Birbaumer, 2000) and apparently have not been used in studies of dysarthria. In addition, imaging methods vary in their sensitivity to lesions in different parts of the nervous system. For example, CT is fairly sensitive to lesions in the corona radiata and capsule but has limited sensitivity to brainstem lesions. Therefore, when the results from a single imaging technique are considered, it is possible that the data are biased by considerations of sensitivity. Ultimately, it is valuable to have confirmatory evidence from different techniques. Most of the imaging data on lesions in dysarthria have been obtained from structural imaging methods such as CT and MRI. An additional problem is that many of the neurologic disorders linked with dysarthria do not have lesions that are routinely or readily determined by brain imaging methods. For example, imaging is rarely performed on individuals with Parkinson’s disease or those with various neurodegenerative disorders.

3. Neuroimaging methods have been applied much less frequently to the dysarthrias than to such disorders as aphasia or dementia. The sparseness of the clinicoanatomic literature on dysarthria is immediately evident in a review of the literature. But sufficient progress has been made to warrant a summary of current knowledge of clinicoanatomic relationships and to formulate hypotheses for future study.

Information on lesion sites and neuropathology also can be derived from methods other than neuroimaging. One source of information is surgery or other invasive intervention that either resolves dysarthria or results in dysarthria as a complication. Some examples are stereotactic thalamotomy, pallidotomy, and deep brain stimulation in the treatment of intractable essential tremor or Parkinson’s disease (Kondziolka et al., 1999; Taha, Janssen, & Favre, 1999; Zirh, Reich, Dougherty, & Lenz, 1999). Neuroimaging results are complemented by a small number of stimulation studies, especially those using transcranial magnetic stimulation (TMS) or rapid-rate transcranial magnetic stimulation (rTMS), in which an intense magnetic field induces an electric field that can either interfere with or facilitate behavioral responses such as speech (Epstein, 1998; Epstein et al., 1999; Kuchta, Reuter, Kurthen, Kohler, & Linke, 1997; Michelucci et al., 1994; Pascual-Leone, Gates, & Dhuna, 1991; Topper, Mottaghay, Brugmann, Noth, & Huber, 1998). Results from these procedures are integrated with imaging data in this review.

### Structure of the Review

Clinicoanatomic studies are by no means uniform across the different types of dysarthria; therefore, the extant data do not permit a comprehensive view of the relationship between lesion site and characteristics of the speech disorder. In particular, the published data do not permit a full consideration of the clinicoanatomic relationships proposed by Darley et al. (1969a). As mentioned earlier, the published studies often do not provide a detailed description of abnormal speech characteristics or report classification of any sort, so that the clinical presentation is typically an unspecified dysarthria. This is particularly unfortunate given that the dysarthria types can differ in several respects that may be informative regarding site of lesion. Given the nature
of the source material, this review of the clinicoanatomic literature is organized as follows:

1. Lesions of the pyramidal motor system (upper motor neuron pathways): These lesions are associated especially with spastic or unilateral upper motor neuron (UMMN) dysarthria. These perceptual types of dysarthria are linked with bilateral and unilateral damage, respectively, to the motor pathway. Also included in this category are progressive dysarthria (anarthria), isolated dysarthria, and dysarthria/clumsy hand syndrome. The justification for these inclusions is as follows. Progressive dysarthria is poorly described but often may involve an apraxia of speech with or without dysarthria. It is likely that some cases do warrant the label of dysarthria but that others fall into the classification of apraxia of speech. Because of the frequently mentioned speech disorder, it is included in this category, but in many of the relevant papers it is not possible to exclude with confidence the possibility of apraxia of speech as at least a co-occurring condition. Isolated dysarthria is included in this category because the most frequently reported site of lesion is within the pathway in question. Dysarthria/clumsy hand syndrome is included here (but cross-listed with paragraph 3 below) because it involves a combination of pyramidal and cerebellar signs.

2. Lesions of the cerebellar system (the cerebellum and its inflow/outflow pathways): Cerebellar lesions are usually associated with ataxic dysarthria, but it should be noted that this form of dysarthria can occur in a variety of neurologic conditions (Duffy, 1995).

3. Lesions of the extrapyramidal system/basal ganglia: These lesions are associated with hypokinetic and hyperkinetic dysarthrias. The former is typically observed in Parkinson's disease but may accompany other disorders, such as progressive supranuclear palsy. Hyperkinetic dysarthria is usually classified as either dystonic or choreiform, but may include speech abnormalities associated with a number of different involuntary movement disorders (e.g., athetosis, myoclonus, action mydonus, tremor, tics).

Lesions of the Pyramidal System/Upper Motor Neuron Pathway

The pyramidal system (or what Duffy, 1995, called the direct activation pathway) is generally taken as the primary system of voluntary motor control (although it never operates independently from the extrapyramidal system). The upper motor neuron of the pyramidal motor pathway originates primarily in motor cortex, but some neurons—perhaps as many as 40%—are located in the postcentral gyrus (Davidoff, 1990). The corticobulbar fibers that innervate the nonrespiratory speech musculature originate especially in the inferior precentral gyrus and descend through the corona radiata. The fibers continue in a twisting, screwlike pattern through the centrum ovale until they converge into the internal capsule. Fibers destined to the cranial nerve nuclei are found near the genu, with cortico-lingual and cortico-facial tracts being closely situated. In pure motor stroke, lesions generally occur in one or more of the following: corona radiata, posterior limb of the internal capsule, putamen, globus pallidus, thalamus, pons, and medulla (Schonewille, Tuhrim, Singer, & Atlas, 1999).

Lesions to the pyramidal tract structures are associated with upper motor neuron syndrome, whereas damage to the basal ganglia (extrapyramidal system) is associated especially with hypokinetic or hyperkinetic syndromes. Recent work with diffusion-weighted magnetic resonance imaging (Inoue, Shimizu, & Yoshimoto, 1999) demonstrates that the entire pyramidal tract can be visualized on a single fiber mapping image in which nerve fiber integrity was confirmed for neurologically normal subjects but shown to be disrupted in patients with tumors. This approach holds promise for the description of lesions in stroke-related dysarthria but apparently has not been used for this purpose to date. Complementary advances permit the study of corticobulbar innervation of tongue and orofacial muscles (Terao et al., 2000; Urban, Vogt, & Hopf, 1998).

Most reports on pyramidal pathway damage pertain to a dysarthria associated with supratentorial ischemic stroke. Presumably, the type of dysarthria is either spastic or unilateral upper motor neuron, but actual classification or clear descriptions of deviant speech characteristics has not been commonly reported in lesion studies. It is presumed that the dysarthrias are of either the spastic or unilateral upper motor neuron variety, but it is possible that mixed dysarthrias also are involved. As shown in Table 1, the dysarthria is associated with a variety of lesions distributed along a pathway that extends from motor cortex through the corona radiata to the internal capsule. It has been asserted that a single type of dysarthria (unclassified) is common to this lesion pathway (Urban, Hopf, Fleischer, Zorowka, & Mullerforell, 1997). From a combined study of MRI and rTMS, Urban, Hopf, Zorowka, Fleischer, and Andreas (1996) concluded that “interruption of the corticobulbar pathways to the tongue is crucial in the pathogenesis of dysarthria following extracerebellar lacunar stroke” (p. 1135).

The presence of dysarthria assumes a general prognostic value in that speech disorder and disability emerged as independent predictors of one-year mortality in a study of 89 patients with lacunar stroke (Ryglewicz, Baranska-Gieruszcak, Mendel, Poniatowska, & Kozlowski, 1997). Further, it has been suggested that the presence of dys-
arthria, along with headache and abrupt onset of symptoms, can distinguish hemorrhagic lacunar stroke from other causes of lacunar stroke (Arboix, Garcia-Eroles, Massons, Oliveres, & Targa, 2000).

**Unilateral Upper Motor Neuron Dysarthria**

One of the few studies to provide information on both dysarthria characteristics and site of lesion is Duffy and Folger (1996), who examined patients with dysarthria associated with unilateral upper motor neuron lesions. These usually manifest as a mild to moderate speech involvement characterized by imprecise consonant articulation, slow and irregular speech AMRs, slow speech rate, and phonatory abnormalities (especially harshness). The responsible lesion is summarized in Table 2, which bears a strong similarity to Table 1.

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Cases observed (%)</th>
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<tbody>
<tr>
<td>Lobar</td>
<td>43</td>
</tr>
<tr>
<td>Cortical</td>
<td>7</td>
</tr>
<tr>
<td>Subcortical</td>
<td>7</td>
</tr>
<tr>
<td>Cortical and subcortical</td>
<td>29</td>
</tr>
<tr>
<td>Internal capsule</td>
<td>34</td>
</tr>
<tr>
<td>Internal capsule or pons</td>
<td>4</td>
</tr>
<tr>
<td>Pericapsular</td>
<td>11</td>
</tr>
<tr>
<td>Pericapsular/ subcortical and lobar</td>
<td>5</td>
</tr>
<tr>
<td>Thalamus (bilateral)</td>
<td>2</td>
</tr>
<tr>
<td>Brainstem</td>
<td>2</td>
</tr>
</tbody>
</table>

Unilateral upper motor neuron dysarthria differs from the perceptual dysarthria types identified by Darley et al. (1969a, 1969b) in that it is an anatomic label and may include speech characteristics that represent weakness, spasticity, and even ataxia. In at least 50% of cases with dysarthria, a unilateral upper motor neuron lesion is associated with a lingual weakness that contributes to the dysarthria (Duffy & Folger, 1996). It is not clear why lingual paralysis occurs in some individuals with monohemispheric stroke but not others. Based on the results of a TMS study, Muelbacher, Artner, and Mamoli (1998) suggested that the reason is the variable availability of uncrossed motor projections among different individuals.

**Progressive Dysarthria (or Apraxia of Speech)**

Damage to Broca’s area and surrounding cortical regions is associated with a progressive loss of speech that has been termed primary progressive aphasia (Didic, Felician, Ceccaldi, & Ponzet, 1999; Kertesz & Orange, 2000; Zakzanis, 1999), progressive anarthria (Didic et al., 1999; Infante, Sanchez Guerra, Polo, Berciano, & Oterino, 2000), or progressivedysarthria (Santens et al., 1999). The choice of terminology partly reflects the initial or predominant symptomatology. The disorder of particular interest here is typically characterized by impaired articulation (or apraxia of speech), telegraphic style, and a difficulty in performing complex orofacial and hand movements (Didic, Ceccaldi, & Ponzet, 1998). It is possible that it is labeled more accurately as apraxia of speech or apraxia of speech with dysarthria. The clinical features are most consistent with apraxia of speech, but this term is not used consistently in neurology.

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**Table 1.** Supratentorial stroke lesions associated with dysarthria (typically unclassified as to perceptual type and generally lacking in a description of the speech disturbances).

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower motor cortex</td>
<td>Urban et al. (1997)</td>
</tr>
<tr>
<td>Parietal cortex</td>
<td>Murdoch et al. (1994)</td>
</tr>
<tr>
<td>Corona radiata</td>
<td>Ichikawa &amp; Kageyama (1991), Tonkonagy &amp; Goodglass (1981), Schonewille et al. (1999), Urban et al. (1997)</td>
</tr>
<tr>
<td>Periventricular white matter</td>
<td>Murdoch et al. (1994)</td>
</tr>
<tr>
<td>Internal capsule (not further specified)</td>
<td>Murdoch et al. (1994)</td>
</tr>
<tr>
<td>Posterior limb of internal capsule</td>
<td>Combarros et al. (1992), Decroix et al. (1986), Schonewille et al. (1999), Urban et al. (1997)</td>
</tr>
<tr>
<td>Anterior limb of internal capsule</td>
<td>Ichikawa &amp; Kageyama (1991), Ozaki et al. (1986)</td>
</tr>
<tr>
<td>Thalamus (bilateral)</td>
<td>Karacostas et al. (1994)</td>
</tr>
</tbody>
</table>
In one of the larger studies, eight patients were examined, with long-term follow-up (6 to 10 years) for four cases (Broussolle et al., 1996). CT and MRI 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” (Broussolle et al., 1996, p. 44). The patients studied in follow-up progressed to muteness and bilateral suprabulbar paresis. In five patients studied by Didic et al. (1998), the neural damage associated with the early stage of the disease was thought to be in the ventral compartment of the premotor cortex, but precise localization 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 who had a 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, Kartsounis, 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 et al., 1999). For one patient with progressive dysarthria, Selnes, Holcomb, and Gordon (1997) reported that MRI revealed a localized left-sided perisylvian lesion and that PET showed a left and possibly right perisylvian hypometabolism localized to the area of tissue loss. Didic et al. (1999) concluded from a review of the literature that both primary progressive aphasia and progressive dysarthria typically are associated with nonspecific lesions and Pick-type pathology (i.e., inclusion bodies and swollen neurons). They also noted that “progressive disorders of only one domain of cognition may well be due to preferential involvement of anatomically and functionally defined neural systems and could therefore be considered as ‘system atrophies’” (p. S73).

Progressive dysarthria also has been observed in individuals with primary lateral sclerosis (de Koning, van Doorn, & van Dongen, 1997; Fisher, 1977). The article by de Koning et al. is noteworthy for its relatively detailed account of speech changes over a 5-year period, beginning with initial signs of imprecise consonants (which appears to justify the classification of dysarthria at least in the early stages of the disorder) and culminating in complete anarthria. Although an early MRI was unremarkable, a subsequent MRI obtained 3 years after the initial complaint revealed a lacunar infarct in the right cerebral peduncle. SPECT showed a bilaterally reduced activity especially in the basal motor cortex. A common feature to these patients was the initial impairment of speech (often the earliest sign of disorder), progressive deterioration usually leading to mutism, and involvement of the posterior inferior frontal lobe—especially the operculum. It bears repeating that it is uncertain if the disorder in question is most accurately labeled a dysarthria as opposed to apraxia of speech or a combination of the two.

**Isolated Dysarthria**

Infrequently, dysarthria is the sole or predominant sign of stroke— in which case, it is typically termed isolated or pure dysarthria (Arboix, Massons, Oliveres, & Titus, 1991; Fisher, 1982; Ichikawa & Kageyama, 1991; Kim, 1994; Orefice, Fragassi, Lanzillo, Castellano, & Grossi, 1999; Ozaki, Baba, Narita, Matsunaga, & Takebe, 1986; Tohgi, Takahashi, Takahashi, Tamura, & Yonezawa, 1996). This condition is of particular interest in isolating the neural pathways that control speech. It is likely that in many of the reported cases, the dysarthria was accompanied by mild concomitant deficits, particularly orofacial paresis. The literature is not easily summarized because (a) the lesions are widely distributed (cortical and subcortical, unilateral and bilateral), (b) it is difficult to eliminate the possibility of remote effects such as diaschisis or compensations (Okuda, Kawabata, Tachibana, & Sugita, 1999), and (c) the dysarthria is rarely described in sufficient detail to permit a classification into perceptual types or to provide a clear idea of the speech disturbance. Table 3 shows that the responsible lesions are found primarily in the corona radiata, internal capsule, basal ganglia, and pons (lesion locations already noted in the preceding discussion of nonisolated dysarthrias). Takahashi, Satoh, Takahashi, Chiba, and Tohgi (1995) reported that isolated dysarthria tends to occur with relatively anterior lesions of the corona radiate or junctional zone to the internal capsule. They also noted that dysarthria occurs more often with left than right lesions and that such lesions may simultaneously interrupt the corticobulbar pathway and callosal fibers to the right hemisphere that carry speech information. It has been reported that an isolated, reversible dysarthria can result from a basilar artery balloon occlusion (Hartmann et al., 1999).

**Dysarthria–Clumsy Hand Syndrome**

In some neurologic disorders, dysarthria is characteristically accompanied by a specific nonspeech motor dysfunction. Dysarthria–clumsy hand syndrome, first described by Fisher (1967), is characterized by a combination of ataxic and pyramidal signs affecting speech and hand movements. Fisher (1978) described a similar condition, ataxic hemiparesis, that may be confused with dysarthria–clumsy hand syndrome (Glass, Levey, & Rothstein, 1990). In a study of six patients, Glass et al. (1990) reported that all patients had pontine infarctions
contralateral to the affected limb. Further, in their re-
view and re-evaluation of the literature, they concluded
that pontine lesions were identified in 12 of 15 patients
with dysarthria–clumsy hand syndrome. The remain-
ing patients without pontine damage had lesions in the
capsule, corona radiata, or both. Schonewille et al.
(1999) reported on two cases of this syndrome. For one
patient, the lesions were in the posterior limb of the in-
ternal capsule and the putamen. For the other, the le-
sion was in the caudate nucleus. But it also has been
reported that dysarthria–clumsy hand syndrome may
result from infarction of the cerebral peduncle (Urban,
Hopf, Visbeck, Fleischer, & Andreas, 1996). It may be
concluded that although a pontine lesion is typical of
this syndrome, lesions in the capsule, basal ganglia, or
cerebral peduncle also may be responsible. As noted ear-
lier, this syndrome can be cross-listed with the category
of cerebellar lesions, which is discussed next.

Lesions of the Cerebellar System

Ataxic dysarthria is associated with a large num-
ber of neuropathologies and a variety of focal or diffuse
lesions (Duffy, 1995). It is helpful to review some of the
major hypotheses concerning cerebellar dysfunction in
speech before considering the imaging data. One of the
earliest clinicopathologic hypotheses held that the ver-
mis is particularly important for speech (Holmes, 1917;
Mills & Weisenburg, 1914). Recent support for this idea
was reported by Chiu, Chen, and Tseng (1996), who com-
pared speech and other motor dysfunctions in 15 pa-
tients with cerebellar disease and dysarthria. They
determined that the midline structures of vermis and
fastigial nucleus were the primary focus for the coordi-
nation of motor speech. However, in a review of dysar-
thria in cerebellar disease, Ackermann and Ziegler
(1992) concluded that a speech disorder is particularly
associated with damage to the paramedian regions of
the superior cerebellar hemispheres. In another review,
Timmann, Kolb, and Diener (1999) identified rostral
paravermal lesions as responsible for dysarthria. Barth,
Bogousslavsky, and Regli (1993) concluded from a MRI
study that infarcts in the territory of the superior cer-
ebellar artery are associated with a syndrome of dysar-
thria, vertigo or unsteadiness, limb or trunk ataxia, and
nystagmus.

Lesions Associated With Ataxic Dysarthria

Table 4 summarizes the lesions that have been re-
ported in several studies of ataxic dysarthria. For the
most part, the lesions are limited to the cerebellum but
can affect cerebellar inflow and outflow tracts, includ-
ing crossed cortico-cerebellar pathways, such as (a) the
frontopontocerebellar tract originating in Brodmann
Area 10, and (b) the tract connecting the orofacial area
of motor cortex with the paravermal segment of the con-
tralateral cerebellar hemisphere. The identification of
responsible lesion is complicated by the possibility of
diaschisis, in which a lesion to one brain structure
causes a functional disturbance in a remote structure
that is itself not damaged. SPECT evidence of cerebello-
cerebral diaschisis has been reported in patients with

<table>
<thead>
<tr>
<th>Lesion location</th>
<th>Source</th>
</tr>
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<tbody>
<tr>
<td>Centrum ovale</td>
<td>Arboix et al. (1991)</td>
</tr>
<tr>
<td>Corona radiata (bilateral)</td>
<td>Takahashi et al. (1995)</td>
</tr>
<tr>
<td>Internal capsule (unilateral)</td>
<td>Urban et al. (1999)</td>
</tr>
<tr>
<td>Internal capsule (laterality not specified)</td>
<td>Arboix et al. (1991)</td>
</tr>
<tr>
<td>Corona radiata and/or internal capsule (bilateral)</td>
<td>Okuda et al. (1999), Takahashi et al. (1995)</td>
</tr>
<tr>
<td>Basal ganglia</td>
<td>Kim (1994)</td>
</tr>
<tr>
<td>Pons</td>
<td>Arboix et al. (1991), Kim (1994), Oficilce et al. (1999), Tohgi et al. (1996)</td>
</tr>
<tr>
<td>Cerebellum (paravermal zone of right hemisphere)</td>
<td>Gironell et al. (1996)</td>
</tr>
</tbody>
</table>

When focal lesions have been determined, their distribution includes the vermal, paravermal, and lateral portions (hemispheres) of the cerebellum. Although lesions associated with dysarthria often are on the left side, this is not necessarily the case (Ackermann, Vogel, Peterson, & Poremba, 1992). The composite picture is one of widely scattered lesions in the midline or in either hemisphere. One interpretation of these lesion data is that different speech functions are represented in different regions of the cerebellum, either because of individual variations in regional cerebellar function (population heterogeneity) or because speech is multiply represented in the cerebellum (distributed representation). Possibly, different parts of the cerebellum are involved in the motor control of different motor systems within speech production or they are involved with different functions of motor control. For example, Ackermann et al. (1992) observed that a bilateral infarction appears to be associated with the most severe articulatory deficits and that a lesion of the dentate nucleus is linked with phonatory disturbances. Another factor to consider is the possibility of accompanying lesions in noncerebellar regions. Cisneros and Braun (1995) hypothesized that differences in the severity of ataxic dysarthria could be related to lesions in structures other than the cerebellum. Specifically, they proposed that more severe involvement (both respiratory insufficiency and articulatory/phonatory difficulties) reflected additional damage to pontine and medullary structures. Imaging data are needed to evaluate this hypothesis. In a PET study of olivopontocerebellar atrophy, Kluin et al. (1988) reported a significant inverse correlation between the severity of ataxia in speech and the local metabolic rates for glucose in the cerebellar vermis, cerebellar hemispheres, and brainstem.

A full understanding must account for the remote effects of the lesion as well as for its local effects. TMS results from patients with autosomal-dominant or idiopathic cerebellar ataxia indicated that one consequence of cerebellar disease is a diminished facilitatory influence of the cerebellum on motor cortex (Liepert et al., 1998). Finally, as an example of a promising line of study, Ikuta (1998) used proton magnetic resonance spectroscopy and SPECT in a study of patients with olivopontocerebellar atrophy (OPCA). Compared to controls, patients with OPCA had decreased ratios of N-acetylaspartate to creatine (NAA/Cre). Moreover, patients with a more severe ataxic gait and dysarthria had a slightly lowered NAA/Cre relative to other OPCA patients. Ikuta’s study is one of the very few to apply magnetic resonance spectroscopy to individuals with dysarthria, but it points the way to future investigations.

### Cerebellar Mutism

Although the focus of this review is on dysarthrias in the adult, it is pertinent here to note the syndrome of cerebellar mutism and subsequent (typically ataxic) dysarthria that may occur as a complication of posterior fossa surgery in children or as a consequence of hemorrhages or trauma (Turgut, 1998). The location of the lesion responsible for cerebellar mutism and dysarthria is somewhat unclear. Some studies point to a lesion in the cerebellar hemispheres (Janssen et al., 1998).

#### Table 4. Lesions associated with ataxic dysarthria.

<table>
<thead>
<tr>
<th>Midline regions</th>
<th>Holmes (1917), Mills &amp; Weisenburg (1914)</th>
</tr>
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<tbody>
<tr>
<td>Vermis</td>
<td>Chiu, Chen, &amp; Tseng (1996)</td>
</tr>
<tr>
<td>Vermis and fastigial nucleus</td>
<td>Chiu, Chen, &amp; Tseng (1996)</td>
</tr>
<tr>
<td><strong>Paramedian and lateral regions</strong></td>
<td>Lalone &amp; Botez (1990)</td>
</tr>
<tr>
<td>Superior cerebellar vermis, both cerebellar hemispheres, paravermal and lateral aspects of the hemispheres, and left paravermal area</td>
<td>Chaves et al. (1994); Erdemoglu &amp; Duman (1998); Stangel et al. (1999)</td>
</tr>
<tr>
<td>Regions supplied by rostral basilar artery (includes superior cerebellar artery)</td>
<td>Timmann, Kolb, &amp; Diener (1999)</td>
</tr>
<tr>
<td>Rostral paravermal area</td>
<td>Ackermann &amp; Ziegler (1992)</td>
</tr>
<tr>
<td>Paramedian regions of the superior cerebellar hemispheres</td>
<td></td>
</tr>
<tr>
<td><strong>Noncerebellar regions</strong></td>
<td></td>
</tr>
<tr>
<td>Frontal cerebral cortex</td>
<td>Marie et al. (1998), Terry &amp; Rosenberg (1995)</td>
</tr>
</tbody>
</table>
Lesions of the Extrapyramidal System/ Basal Ganglia

The extrapyramidal system, or what Duffy (1995) termed the indirect activation pathways, includes the basal ganglia (caudate, putamen, globus pallidus, subthalamic nucleus, substantia nigra), thalamus, and related structures. Although this system clearly participates in the neural control of speech, there is disagreement on the role of individual structures. Lesions to this system are associated with two primary types of dysarthria: hypokinetic dysarthria (particularly common in Parkinson’s disease) and hyperkinetic dysarthria (dystonic and choreic forms were studied by Darley et al., 1969a, 1969b); but hyperkinetic dysarthria can also be secondary to other involuntary movement disorders such as tremor, athetosis, myoclonus, action myoclonus, and tics.

Although hypokinetic dysarthria has been described especially in connection with Parkinson’s disease or parkinsonian syndromes, it also has been observed in progressive supranuclear palsy (Metter & Hanson, 1991) and multiple system atrophy (Kluin et al., 1996). A possible difference in pathophysiology between Parkinson’s disease and progressive supranuclear palsy is that both the globus pallidus interna (GPI) and globus pallidus externa (GPe) are damaged in progressive supranuclear palsy, but only the internal segment is affected in Parkinson’s disease (Hardman & Halliday, 1999a, 1999b). Although very few imaging studies have been reported on speech functions in these disorders, the more general literature points to some important hypotheses pertaining to speech vis-à-vis nonspeech motor control.

Imaging Studies

Imaging studies of the dopamine system reveal dichotomous relationships that should lead to an improved understanding of the various effects of Parkinson’s disease and related disorders. Volkow et al. (1996), in a recent review of PET studies, discussed how this technology can answer fundamental questions in pathophysiology and effective treatment. PET has potential for the differential diagnosis of various forms of parkinsonism and for the detection of subclinical dopaminergic deficits (Shinotoh & Calne, 1995). In vivo studies with Fluorodopa 18 can determine dopamine metabolism in parts of the striatum and relate localized metabolic deficiency to either motor or cognitive dysfunctions. Motor impairment has been associated with abnormalities in the putamen, whereas memory impairment has been related to abnormalities in the caudate (Holthoff-Detto et al., 1997). Studies using PET and MRI have shown that lesions of the GPI are associated with hyperkinetic signs, whereas lesions of the GPe or globus pallidus central (Gpc) are associated with hypokinetic or akinetic/rigid signs (Bucher et al., 1996; Hirato, Ishihara, Horikoshi, Shibazaki, & Ohye, 1995).

PET studies are informative regarding over- or underactivity in other regions, including supplementary motor area, dorsal prefrontal cortex, lateral premotor cortex, parietal cortex, and cerebellum (Brooks, 1999). Motor disturbances in Parkinson’s disease may be explained by these patterns of hyperactivity and hypoactivity. Brooks (1999) concluded that rest tremor results from a combination of overactivity of cerebellar connections and loss of dopaminergic function. Ventral thalamotomy or thalamic stimulation reduces tremor and cerebellar activation but also reduces activation of primary motor cortex.

A dysarthria also has been noted to occur in association with anterior thalamic infarction. Ghika-Schmid and Bogousslavsky (2000) reported on 12 patients with an isolated infarct of the anterior thalamus. In addition to a common severe perseverative disorder and anterograde memory impairment, the patients had a variety of other disorders. Dysarthria (unspecified as to salient characteristics or perceptual type) occurred in 8, and hypophonia was observed in 5. Given the relatively high
rate of dysarthria in the sample, it would be valuable to know more about the speech disorder (e.g., perceptual type of the dysarthria or a description of its speech-voice characteristics).

Although very few imaging studies directly address speech production in individuals with Parkinson’s disease, preliminary data indicate that behavioral therapy (intensive speech and voice treatment) produces changes in regional cerebral blood flow (rCBF) that resemble the effect of pallidotomy (Liotti et al., 1999). Specifically, post-treatment rCBF in the globus pallidus was reduced in the resting state, presumably reflecting a reduction of overactivity in the globus pallidus. However, rCBF increased for vocalization relative to rest. However, this is not to say that pallidotomy consistently results in improved speech. As considered in the next section, interventions do not necessarily have the same outcomes for speech and nonspeech functions.

Effects of Parkinson’s Disease on Speech and Nonspeech Motor Systems

It cannot be assumed that speech and nonspeech motor functions are affected in exactly the same way by Parkinson’s disease. Some particularly important questions to be considered in relating speech and other movements in Parkinson’s disease are (a) the degree to which speech and nonspeech motor systems exhibit the same motor abnormalities, (b) the competition between speech and other motor behaviors, and (c) the differential effects of clinical interventions for speech and nonspeech movements. Each of these is considered in the following.

Similarity and Dissimilarity of Disruptions in Speech and Nonspeech Motor Systems

Concerning the question of similar motor abnormalities, Hunker and Abbs (1990) concluded from a spectral analysis that tremor frequency is uniform in the lips, tongue, jaw, and index finger. This conclusion supports the simplifying hypothesis of a single tremor generator. However, a coherence analysis of EEG records was interpreted to mean that multiple oscillators are involved, especially in interlimb comparisons (Raethjen et al., 2000). Differences between speech and other motor systems also have emerged on diadochokineti c tasks. Stebbins and Goetz (1998) reported a factor analysis of the Unified Parkinson’s Disease Rating Scale (UPDRS) in which speech and facial expression loaded on the same factor as balance and gait. This factor was interpreted to reflect axial function, balance, and gait. Speech diadochokinesis was considered by Stebbins and Goetz to be an axial task, accomplished by the bilaterally innervated midline speech structures. Movements in the right and left limbs emerged as two separate factors (right and left bradykinesia). Gurd, Bessell, Watson, and Coleman (1998) concluded that speech diadochokinesis and finger tapping are doubly dissociated in Parkinson disease, which supports the idea that different neural systems are responsible for articulation versus digit control.

Evidence of Competition Between Speech and Nonspeech Motor Control

With respect to the question of competition between speech and other voluntary motor behaviors, it appears that vulnerability to falls may be related to a choice between walking and talking (Lundin-Olsson, Nyberg, & Gustafson, 1997; Nisipeanu & Inzelberg, 1997). An interaction between locomotion and speech also can be seen in the effects of a simultaneous verbal fluency task on walking in individuals with Parkinson’s disease (Camicioli, Oken, Sexton, Kaye, & Nutt, 1998). Gait-speech interaction certainly is important as a factor of patient safety, but it also offers intriguing possibilities to understand the motor control systems that support these two tasks.

Speech and Nonspeech Effects of Ablative Lesions, Deep Brain Stimulation, and Dopamine Transplants

Several studies of individuals with Parkinson’s disease or essential tremor point to differential effects of interventions on speech and nonspeech motor functions. These reports show that various interventions that improve nonspeech motor control (especially in the limbs) often have neutral or even negative outcomes for speech. As summarized in Table 5, this pattern of results has been reported for levodopa therapy, unilateral or bilateral posteroventral pallidotomy, fetal dopamine transplants, and pallidal or thalamic stimulation. To be sure, other studies have shown some benefit for speech from these interventions (Jiang, Lin, Wang, & Hanson, 1999; Leanderson, Meyerson, & Persson, 1972; Robertson & Hammerstad, 1996), but the essential point is that these positive outcomes for speech have not been consistently demonstrated. Some negative effects on speech may be attributable to lesions from surgery. Hariz and De Salles (1997) reported that the side effect of dysarthria in posteroventral pallidotomy resulted from lesions that encroached on the internal capsule. Schulz and Grant (2000) concluded from a review of the literature on various treatments of voice and speech disorders in Parkinson’s disease that speech therapy, along with appropriate medications, is the most efficacious intervention. Possibly, the dissimilar outcomes for speech and nonspeech functions following pharmacologic, surgical,
Table 5. Outcomes for nonspeech and speech functions of various treatments for Parkinson’s disease (PD) and essential tremor (ET). UPDRS = Unified Parkinson’s Disease Rating Scale (Fahn et al., 1989); ADL = activities of daily living; DDK = diadochokinesis.

<table>
<thead>
<tr>
<th>Author</th>
<th>Method</th>
<th>Nonspeech outcome</th>
<th>Speech outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bakker et al. (1997)</td>
<td>Fetal dopamine transplants for PD</td>
<td>Not described</td>
<td>No systematic effect</td>
</tr>
<tr>
<td>Cahill et al. (1998)</td>
<td>Levodopa for PD</td>
<td>Improved labial function</td>
<td>Improved labial function</td>
</tr>
<tr>
<td>Dürif et al. (1999)</td>
<td>Speaking aloud and other cognitive and motor tasks in PD</td>
<td>Worsened dyskinesia</td>
<td>N ot described</td>
</tr>
<tr>
<td>Gamboa et al. (1997)</td>
<td>Dopaminergic drugs for PD</td>
<td>Not described</td>
<td>Persisting voice problems</td>
</tr>
<tr>
<td>Gentil et al. (1998)</td>
<td>Levodopa therapy for PD</td>
<td>Positive for finger movements</td>
<td>Neutral or negative or orofacial movements</td>
</tr>
<tr>
<td>Ghika et al. (1999)</td>
<td>Bilateral posteroventral pallidotomy for PD</td>
<td>Positive for UPDRS</td>
<td>Negative for 1 of 4 patients</td>
</tr>
<tr>
<td>Gross et al. (1997)</td>
<td>GPi stimulation for PD</td>
<td>Positive for akinesia, rigidity, and gait</td>
<td>Positive</td>
</tr>
<tr>
<td>Hariz &amp; De Salles (1997)</td>
<td>Posteroventral pallidotomy for PD</td>
<td>Generally positive for tremor</td>
<td>Dysarthria as side effect in 3 of 138 patients</td>
</tr>
<tr>
<td>Jiang et al. (1999)</td>
<td>L-dopa therapy for PD</td>
<td>N ot described</td>
<td>Positive for voice (acoustic analysis)</td>
</tr>
<tr>
<td>Komolili et al. (2000)</td>
<td>Apomorphine therapy in double-blind, randomized, placebo-controlled cross-over design</td>
<td>Improvement in motor score of UPDRS</td>
<td>N eutral for voice and speech</td>
</tr>
<tr>
<td>Leanderson et al. (1972)</td>
<td>Levodopa therapy for PD</td>
<td>Positive for mobility, activities of daily life, emotional well being, and bodily discomfort</td>
<td>Positive for lip muscle activity</td>
</tr>
<tr>
<td>Martinez-Martin et al. (2000)</td>
<td>Pallidotomy for PD</td>
<td>N ot described</td>
<td>Neutral for communication</td>
</tr>
<tr>
<td>Obwegeser et al. (2000)</td>
<td>Thalamic stimulation for ET</td>
<td>Reduction of tremor (reduction of head, voice, tongue, and face tremor in a subgroup analysis)</td>
<td>Dysarthria as one of the most frequent reversible side effects</td>
</tr>
<tr>
<td>Pahwa et al. (1999)</td>
<td>Bilateral thalamic stimulation for ET</td>
<td>Positive for tremor score and ADL</td>
<td>Worsening of dysarthria in some patients</td>
</tr>
<tr>
<td>Poluha et al. (1998)</td>
<td>Levodopa treatment for PD</td>
<td>Positive for handwriting</td>
<td>N eutral</td>
</tr>
<tr>
<td>Robertson &amp; Hammerstad (1996)</td>
<td>Levodopa treatment for PD</td>
<td>N ot described</td>
<td>Positive for jaw movements</td>
</tr>
<tr>
<td>Schrag et al. (1999)</td>
<td>Unilateral pallidotomy for PD</td>
<td>Positive for UPDRS</td>
<td>Negative</td>
</tr>
<tr>
<td>Schulz et al. (1999)</td>
<td>Unilateral pallidotomy for PD</td>
<td>Positive for limb motor function</td>
<td>Positive for some patients but 3 did not show consistent positive changes</td>
</tr>
<tr>
<td>Scott et al. (1998)</td>
<td>Unilateral or bilateral posteroventral pallidotomy for PD</td>
<td>Positive for UPDRS</td>
<td>Negative, esp. for DDK, but not judged to be functionally significant</td>
</tr>
<tr>
<td>Shima et al. (1996)</td>
<td>Unilateral or bilateral posteroventral pallidotomy for PD</td>
<td>Positive for rigidity, tremor, and movement</td>
<td>Worsened dysarthria in 3 patients</td>
</tr>
<tr>
<td>Solomon et al. (2000)</td>
<td>Unilateral or bilateral pallidal stimulation for PD</td>
<td>Positive for general motor function</td>
<td>Variable across 3 patients</td>
</tr>
<tr>
<td>Taha et al. (1999)</td>
<td>Thalamic deep brain stimulation for ET</td>
<td>Positive for head, voice and bilateral limb</td>
<td>Dysarthria as side effect</td>
</tr>
<tr>
<td>Theodoros et al. (2000)</td>
<td>Unilateral or bilateral pallidotomy for PD</td>
<td>N o information</td>
<td>Reduced intelligibility in 10 of 12 subjects</td>
</tr>
<tr>
<td>Uitti et al. (2000)</td>
<td>Unilateral pallidotomy for PD</td>
<td>Improved motor function</td>
<td>Mild decline of intelligibility in one-third of 57 patients</td>
</tr>
<tr>
<td>Wang et al. (1999)</td>
<td>Dopaminergic therapy for PD</td>
<td>Improved on motor section of UPDRS</td>
<td>N o change in acoustically measured vocal parameters</td>
</tr>
<tr>
<td>Wang et al. (2000)</td>
<td>Dopaminergic therapy for PD</td>
<td>Improved on motor section of UPDRS</td>
<td>Neutral in phonation</td>
</tr>
</tbody>
</table>
or stimulation methods can provide information on the neural regulation of speech as distinct from that for the limbs or for midline structures of the trunk. Although an increasing number of studies have been reported on the effects of ablative lesions or deep brain stimulation on Parkinson's disease, Lang (2000) concluded that "the evidence for benefit from surgical therapies...is relatively weak by today's scientific standards" (p. 1124).

The implications of the data reviewed in this section seem to be, at the minimum, that speech and nonspeech motor behavior are controlled by neural systems that are partially overlapping but also distinctive in important ways. The evidence for competition between speech and nonspeech motor control indicates that these functions either share neural resources (e.g., a largely overlapping neural regulation) or otherwise place a conflict on the simultaneous deployment of speech and nonspeech motor control. The evidence for asymmetric outcomes of intervention points to some basic dissimilarities in the structures or strategies of neural regulation.

**General Discussion**

Within the limitations of the information reviewed here, it is possible to delineate a pathway, but not necessarily a single structure, that is associated with each dysarthria considered in this paper. In general, then, the clinicoanatomic correlation is between a type of dysarthria and a number of structures that are parts of a pathway or network. Examples are given in Tables 1, 2, 3, and 5. Admittedly, some of the pathways are extensive. The same kind of analysis may be applicable to other types of dysarthria once the requisite data are available. The conclusions drawn from clinical studies potentially can be confirmed with data from neuroimaging studies of neurologically normal speakers. Ultimately, the synthesis of results from both clinical and control subjects should define the primary pathways of speech motor control. Some suggestions on this matter have been discussed elsewhere (Kent, Kent, Weismer, & Duffy, in press).

**Limitations and Potentials**

As noted earlier, there are several limitations in the clinicoanatomic studies available for review. Accordingly, the interpretation of the compiled information confronts a number of issues that are not satisfactorily resolved. These include the typical absence of clear descriptions of deviant speech characteristics or classification of dysarthria; the possibility of diaschisis (the remote effects of a lesion); the influence of sensorimotor-cognitive-linguistic aspects of assessment tasks (particularly complicating if a given structure participates in several functions, as may be the case with the cerebellum); and neglect of the severity of dysarthria (variations of severity even with the same dysarthria classification can complicate correlations with anatomic or metabolic abnormality). It appears that the potential for clinicoanatomic studies of dysarthria is considerable, and it is therefore appropriate to consider the directions that future studies might take.

It is beyond the purpose of this review to examine either the overall clinical utility or the broad theoretical foundations of the dysarthria classification system introduced by Darley et al. (1969a, 1969b). Commentaries on these general issues have been published elsewhere (Duffy & Kent, in press; Kent, 1996; Kent et al., 1998). But it is highly likely that the original system should be modified in some respects, such as inclusion of additional types of dysarthria (unilateral upper motor neuron dysarthria being one example considered in this review), the specification of acoustic and physiologic correlates for each perceptual type, and (possibly) the delineation of subtypes to reflect the heterogeneity within major types.

**Directions for Future Research**

There may be serious limitations to the classic approach followed in most clinicoanatomic studies: the group comparison design. With this method, "imaging findings are used for grouping patients according to the lesion location...and the interpretation obeys a subtractive logic, i.e., the poor performance of one group implies that the damaged region defining the group is required for task completion" (Godefroy et al., 1998, p. 1546). As Godefroy et al. pointed out, a problem with this strategy is that lesion distribution is determined by the nature of the pathological process and is not necessarily congruent with the functional organization of the brain. In considering alternatives to the classic approach, these authors proposed four basic modes of brain-behavior relationship: Unicity is the condition in which a deficit corresponds to a lesion in a single structure—"one deficit, one lesion" (p. 1546). Equivalence is the mode in which a deficit depends on a single lesion in one or the other of two or more possible structures. Godefroy remarked that this may be the most frequent pattern in brain-behavior relationships. Association obtains when a deficit requires the combined lesion of two different structures. Summation occurs when a single lesion in one of two possible structures results in a minor deficit and the combined lesion of both structures produces a major deficit. A kind of bilateral summation occurs in dysarthria associated with upper motor neuron lesion. Whereas a unilateral lesion typically results in a mild to moderate dysarthria, bilateral lesions tend to produce a severe dysarthria.
Other modes may be included as well, either as additional categories or as modifications to the categories defined above. Diaschisis is a mode of brain-behavior relationship in which a lesion in one brain structure results in a functional change in another, remote brain structure, which itself does not experience direct damage. This mode may occur with some frequency, but it has seldom been explicitly investigated. Some evidence of diaschisis was considered in this review. Another possible mode is disconnection, in which damage to white matter may disconnect structures that participate in a control objective. For example, Daniels, Brailey, and Foundas (1999) observed that the most common lesion associated with lingual discoordination during swallowing in patients with stroke was in the periventricular white matter.

Many of the clinicoanatomic relationships summarized in the present paper are best described as following the equivalence mode. For example, if ataxic dysarthria is taken as the deficit, then the lesion can be in one of two or three different structures: cerebellum, cerebellar outflow pathways, or the cerebral cortex.

The approach taken by Lefkowitz and Netsell (1994), in a study of post-traumatic speech disorders, was to group subjects according to clinical deficit (delayed initiation, dysprosody, dysarthria) and then to determine the lesions for each group. MRI was used to identify five categories of structural correlation (listed in order of the strength of association between clinical deficit and observed MRI abnormalities): discrete lesions, atrophic lesions, afferent fiber lesions, nonrecognized lesions, and functional lesions. Prototypes were then identified from the results for 11 subjects with post-traumatic speech disorders. The dysarthria prototype had a large area of hyperintense ischemic change that included the left primary sensory and motor cortices, as well as Broca’s area. The dysprosody prototype was associated with extensive hyperintense ischemic changes in the supraventricular white matter bilaterally. Finally, the initiation prototype had an atrophic lesion of the midbrain.

Dysarthria also can be portrayed as a task-based profile, in which functional deficits are described for motor subsystems or specific aspects of speech production (Kent & Kent, 2000). The goal of this approach is to correlate specific dysfunctions (e.g., laryngeal hypofunction, slowness of movement) with damage to particular regions of the neural control system for speech. In this approach, the objective is not so much to determine the lesion site for a perceptual type of dysarthria as it is to identify the lesions that correlate with particular dimensions of speech production difficulty.

Above all, progress in clinicoanatomic correlations in dysarthria will depend on (a) a rich description of the speech disorder in perceptual, acoustic, and physiologic domains, and (b) interpretation of neuroimaging data in terms of carefully specified modes of brain-behavior relationship. Darley et al. (1969a, 1969b) laid the foundation for this work; advances in neuroimaging technology, speech acoustics, and speech physiology should be major factors in future accomplishments.

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