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Relating Selective Brain Damage to Impairments with Voicing Contrasts

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Research is reviewed concerning the performance of several neurological groups on the perception and production of voicing contrasts in speech. Patients with cerebellar damage, Parkinson's disease, specific language impairment, Broca's aphasia, apraxia, and Wernicke's aphasia have been reported to be impaired in the perception and articulation of voicing. The types of deficits manifested by these neurologically impaired groups in creating and discriminating voicing contrasts are discussed and the respective contributions of separate neural areas are identified. A model is presented specifying the level of phonemic processing thought to be impaired for each patient group and critical tests of the model's predictions are identified.

Key Words: speech; aphasia; apraxia; LI; cerebellum; Parkinson's.

People speak without apparent effort, focusing on the message they wish to communicate rather than the actions necessary to convey that message. Conversation would be tedious if remembering how to coordinate our lips, tongue, jaw, and larynx to produce words was not effortless. In the same way, listeners normally busy themselves with interpreting the content of an utterance rather than attending to the acoustic composition of each sound. People seem to effortlessly perceive what has been said even if they do not always understand the content. The ease with which people produce and perceive speech, however, belies the complex processes needed to perform these tasks. Only when these processes are disturbed does the difficulty of speech production and perception become apparent. Difficulties in producing and perceiving phonemes, the distinctive sounds used to differentiate words (Ladefoged, 1993), can occur even if grammatical and semantic functions are preserved.

Impairments of phonemic processing are associated with a variety of neurological conditions ranging from cerebellar and basal ganglia pathology to left frontal and left posterior temporal lobe lesions. The diversity of cortical and subcortical areas linked with phonemic deficits seems paradoxical to modular accounts of language. It is proposed here that traditional linguistic theory can provide a taxonomy useful in classifying these disorders. The taxonomy not only summarizes and clarifies existing data, but predicts the pattern of phonemic impairments expected for patients with lesions to specific neural areas.

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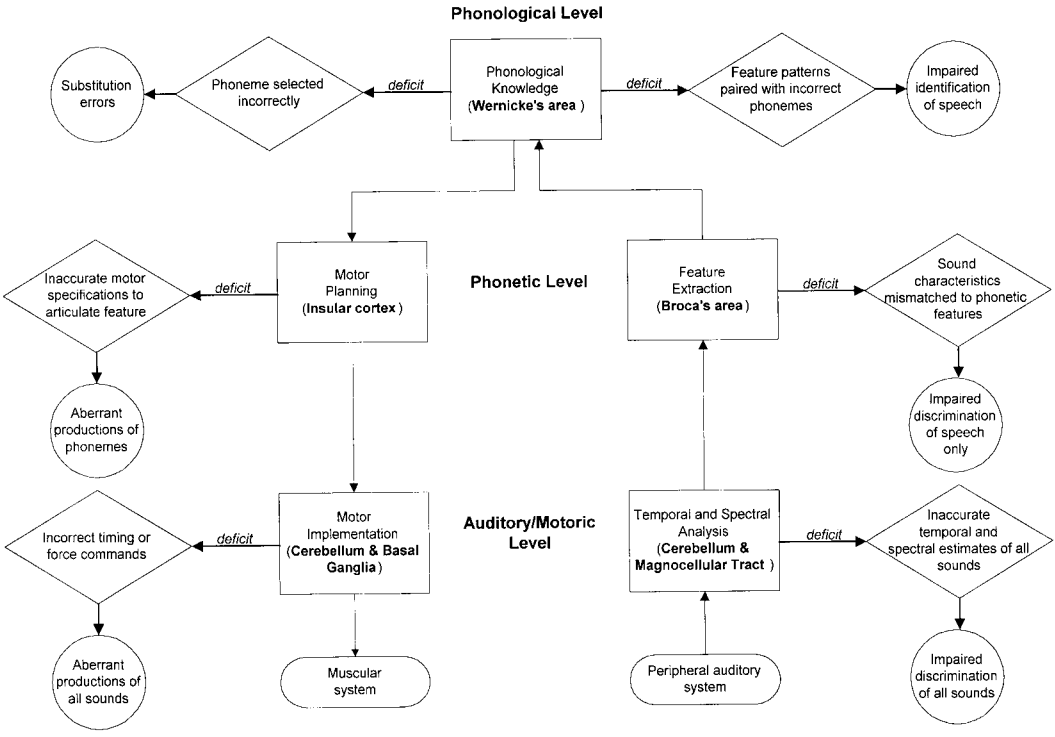

FIG. 1. Neural areas affecting performance at each level of phonemic processing. Each phonemic level is split into production and perceptual domains on the left and right side of the model, respectively.

The taxonomy classifies phonemic impairments based on the level of computation thought to be compromised (see Fig. 1) and whether the deficit is manifest in production or perception. The auditory/motoric level refers to processing that is not language-specific but is crucial for the production of any action or the perception of any sound. The phonetic level is specifically linked to speech and consists of the computations needed to produce or perceive critical linguistic features that comprise phonemes. Finally, the ability to match feature bundles to the phonological store or to select the appropriate phoneme to produce the correct word are claimed to be phonological-level computations.

The model in Fig. 1 displays production tasks on the left and perceptual tasks on the right. Starting with the phonological level of computation on the production side, the model claims that speakers need to be able to select the correct phonemes for the word they wish to utter and then be able to match that phoneme to a representation that specifies how the phoneme is to be produced (phonetic level of processing). The phonetic representation may include information such as the voicing, place, and manner of a phoneme and a plan of how to perform such movements. Implementation of phonetic parameters such as the force and timing of the articulators would be considered a motoric skill as problems of execution would effect both speech and nonspeech.

Listeners as well as speakers need to perform auditory, phonetic, and phonological computations. After determining the temporal and spectral parameters of the incoming sound, listeners need to match their results to a stored phonetic representation. At this level, listeners may match certain parameters of the sound to feature detectors that determine whether voicing has occurred or whether the sound was most like a stop or a nasal. These computations may not be purely linguistic in nature, but could

be used to discretely categorize any continuously varying feature. Even if listeners are able to determine phonetic features, they still need to label the sound phonologically as a /d/ rather than, say, a /b/. For illustrative purposes, I have implied that each phoneme is identified serially, although this is probably not the case. In truth, the offset of one phoneme and the onset of another are not discrete as the vocal tract is moving continuously (Ladefoged, 1993). What is more probable is that listeners parse words into larger segments without identifying each phoneme separately. Nevertheless, phonetic representations need to be linked to phonological knowledge regardless of whether segments are analyzed as individual phonemes, diphones, or syllables.

In Fig. 1, I have listed what deficits can be predicted from a disturbance in a particular type of computation at each level of processing. In theory, auditory impairments should result in a deficit in discriminating and identifying nonspeech stimuli whereas a loss of phonetic computations should be evidenced by discrimination and identification difficulties with speech alone. If phonological processing is impaired, patients should have no trouble detecting the features of a sound and thereby be able to discriminate between two phonemes. When asked to label phonemes, however, these patients should be at a disadvantage. Similar predictions can be made for production depending on the level of processing thought to be impaired. At the motoric level, problems with implementation should result in aberrant productions of both speech and nonspeech sounds. The lack of phonetic computations should have this result only with speech. Deficits in phonological selection, however, should result in perfectly uttered phonemes that were not desired by the speaker.

In light of the varied types of processing needed to produce and perceive phonemic contrasts, it is not surprising that several neurologically impaired populations exhibit deficits in these tasks. Given that these deficits have been linked with damage to a distributed set of brain structures, it would be helpful to clear up the nature of the impairments. It is unparsimonious to suggest that the task of phonemic perception and production is located redundantly in each of these areas. More likely, these tasks consist of a number of phonological, phonetic, auditory, or motoric subprocesses that are instantiated in different neural areas, and that the type of breakdown in voicing perception and production will reflect the lack of computations attributable to these areas. The purpose of this article is to describe the type of deficits manifested by several neurologically impaired groups of patients in creating and discriminating phonemic contrasts and to identify the respective contributions of different neural areas.

Although the ability to perceive and produce a wide array of phonemic contrasts may be impaired by neurological damage, most research has focused on the perception and production of initial stop consonants such as /b/ and /p/ that differ in voicing (state of the vocal cords) rather than phonemes that are contrastive of place (the configuration of the articulators) or manner (conditions of airflow). Such a focus may be warranted because of the difficulty in perceiving and especially producing such contrasts. Moreover, voicing is used to contrast stop consonants in most languages (Ladefoged & Maddieson, 1996). Thus, this review focuses on the ability of specific patient groups to perceive and produce voicing contrasts.

When the vocal cords are adducted, air passing through the larynx will cause them to vibrate. Phonemes produced when the vocal cords are vibrating are said to be ‘‘voiced’’ while ‘‘voiceless’’ sounds occur when the vocal cords are apart. Although this review concentrates on the voicing of stop consonants, voicing is also used to contrast phonemes produced in other manners such as affricates (ch/j) and fricatives (s/z). In English, three pairs of stop consonants, varying in place of articulation, are contrasted by voicing—bilabial (b/p), alveolar (d/t), and velar (g/k).

To produce a voicing contrast between initial stop consonants, a speaker needs to

temporally coordinate respiratory, laryngeal, and orofacial muscles (Ackermann & Hertich, 1997). In producing a /b/, the lips are brought together and the vocal tract is closed. When the lips are opened again after some tens of milliseconds, a burst of acoustic energy is produced called the ''release burst'' and the vocal cords start to vibrate almost immediately once airflow has been resumed. The time interval between the release burst and the onset of vibration is termed ''voice onset time'' (VOT) and lasts less than 20 ms for voiced stops in English. VOTs for voiceless stops like /p/ are longer because the vocal cords remain abducted for some time (>40 ms) after the release of the closure. This produces a turbulent sound called ''aspiration'' which continues until the vocal cords are narrowed.

Most American English speakers do not actually produce vocal cord vibration during ''voiced'' stops, so that all the stop consonants in English are technically ''voiceless'' with /b/d/g/ being voiceless, unaspirated stops and /p/t/k voiceless aspirated stops. However, the former consonants are still commonly referred to as voiced and the latter as voiceless given that the onset of voicing occurs almost immediately after the release of the closure for voiceless, unaspirated stops.

To discriminate between initial stop consonants, several computations need to be undertaken. Auditory cues that differentiate voicing contrasts include the presence or absence of aspiration (Repp, 1979, 1982), VOT, the relative pitch of the following voiced period (Haggard, Summerfield, & Roberts, 1981), the transition rate and/or the frequency of the first formant (F1) at voicing onset (Stevens & Klatt, 1974; Summerfield & Haggard, 1974), and the intensity of the burst (Repp, 1984). Of course, not all of these cues are equally important (Repp, 1982). ''If two cues, x and y, are relevant for a distinction, it may turn out that for any value x, a variation of y will effect a significant shift in listeners' phonetic judgments but that there will be some values of y for which varying x will have negligible effect on phonetic judgments'' (Abramson & Lisker, 1985, p. 25). The presence or absence of aspiration appears to be the most important determinant of voicing followed closely by VOT. Revoile et al. (1987) found that the addition of a long silent VOT between the burst and the vowel of a voiced stop still allowed listeners to correctly classify the consonant 78% of the time, but replacing that silence with aspiration caused accuracy to drop to 4%. Moreover, accuracy was only 70% for voiceless stops with long, but unaspirated VOTs. The other three cues to voicing can effect perceptual boundaries but only for ambiguous values of aspiration and VOT (Abramson & Lisker, 1985; Revoile, Pickett, Holden-Pitt, & Talkin, 1987).

Although a number of cues differentiate voicing cognates, most research has focused on speakers' and listeners' ability to produce and perceive VOT. This may be because VOT is easy to measure and manipulate while also acting as an important cue to voicing. Thus, discussion of voicing contrasts will primarily center around patients' ability to process VOT.

Of concern here are groups that show deficits with voicing contrasts that are not due to cochlear, primary auditory cortex, or primary motor cortex abnormalities, or muscular weakness. Groups that have shown difficulty with voicing contrasts in previous studies that are not due to these factors include Broca's and Wernicke's aphasics (Basso, Casati, & Vignolo, 1977; Baum, Blumstein, Naeser, & Palumbo, 1990; Blumstein, Baker, & Goodglass, 1977; Blumstein, Cooper, Goodglass, Statlender, & Gottlieb, 1980; Blumstein, Cooper, Zurif, Caramazza, 1977; Gandour & Dardarananda, 1984), language-impaired (LI) children (Stark & Tallal, 1979; Tallal & Stark, 1981; Tallal, Stark, & Curtiss, 1976), and adults suffering from apraxia (Hoit-Dalgaard, Murry, & Kopp, 1983; Itoh et al., 1982; Itoh, Tatsumi, Sasanuma, & Fukusako, 1986; Square-Storer, Darley, & Sommers, 1988), Parkinson's disease (Forrest, Weismer, & Turner, 1989; Lieberman et al., 1992), and cerebellar dysarthria (Gan-

dour & Dardarananda, 1984; Ivry & Gopal, 1993; Ackermann & Hertich, 1997). I will organize this discussion based on the level of processing (phonological, phonetic, or auditory/motoric) proposed to be impaired in each group. Toward this end, I will review reports of both perceptual and production deficits. Depending on the type of computation impaired, a group of patients may show analogous deficits in perception and production. This idea will be more fully explicated in following sections, but briefly, the idea is that a store of knowledge or type of computation is shared and accessed by speech production or perceptual mechanisms. Although speech production and perception do not require completely overlapping brain areas—for example, speech perception tasks probably do not require motor planning areas—the deficits shown by some of the groups reported here appear to be in computations or stores that are independent of the type of task, either listening or speaking, performed. For each group, I will first discuss production deficits followed by perceptual deficits. I will then close by discussing whether any correlation between the two exists and, if so, why.

AUDITORY/MOTORIC LEVEL OF PROCESSING

At the most fundamental level of speech production, speakers need to implement the correct force and timing specifications of the phoneme. For example, if the duration between the onset of the opening of the lips to the start of vocal cord vibration is not timed properly, a speaker may produce a /ba/ instead of a /pa/ or vice versa. Three neurological groups exhibit deficits at the auditory and/or motoric level of processing—those with cerebellar dysfunction due to stroke or atrophy, those with basal ganglia abnormalities such as Parkinson's disease, and children diagnosed with language impairment.

Cerebellar Dysarthria

Cerebellar patients have been shown to perform poorly on tasks that require precise temporal computations on the order of milliseconds (Ivry & Keele, 1989; Ivry, Keele, & Diener, 1988). In terms of motor tasks, these patients have shown deficits in the timing of rhythmic movements (Ivry & Keele, 1989). Cerebellar patients are more variable in the duration they produce between successive taps, and this variability is not due to strictly motor implementation factors, but to abnormalities of a central timing process (Ivry et al., 1988). In conjunction with their temporal deficits in production, cerebellar patients also exhibit impairments in judging the duration of tonal stimuli (Ivry & Keele, 1989). Given their general difficulties in tasks that require fine temporal processing, it is reasonable to ask whether these deficits will carry over into the domain of speech. As stated previously, fine temporal coordination is required to produce voicing contrasts and, as VOT is an important cue in perceiving voicing, discriminating duration may also be impaired.

Production. A failure to produce VOT correctly may manifest itself in abnormal mean durations, variability, or both. Hence, the results of both measures are reported and any dissociations of VOT patterns between groups are considered in the General Discussion.

Three studies have reported that cerebellar patients exhibit difficulty in producing voicing contrasts and, specifically, in producing normal VOT distributions. Ivry and Gopal (1993) tested six German-speaking patients with cerebellar atrophy while Gandour and Dardarananda (1984) observed one subject with ischemic encephalopathy involving the cerebellum, and all reported similar results. Patients in these studies

were required to produce between 10 and 24 repetitions of initial stop consonants as part of a word (Gandour & Dardarananda, 1984) or syllable (Ivry & Gopal, 1993). The average VOTs for cerebellars tended to be longer for all stop consonants, but especially voiceless, aspirated stops. Ivry and Gopal reported that cerebellars' VOTs were 7 ms longer than controls for voiced stops and 15 ms greater for voiceless stops. Gandour and Dardarananda also reported that their Thai patient's VOTs were longer for both voiced, unaspirated and voiceless, aspirated stops, but not voiceless, unaspirated (the equivalent of English ''voiced'') stops. This finding corresponds to other reports of a general slowing of cerebellar speech (Kent, Netsell, & Abbs, 1979). Given that VOT lengthens as speech rate slows (Miller, Green, & Reeves, 1986), it is not surprising that VOTs would be lengthier for these subjects. Despite this general slowing, differences in the average VOT between each voicing cognate were maintained.

In terms of variability, both studies reported that cerebellar subjects exhibited much less consistency in VOT productions than controls, especially for voiceless stops. Ivry and Gopal (1993) report an average standard deviation of about 5 ms for controls for each of the voicing cognates whereas cerebellars exhibit standard deviations of about 14 ms for voiced stops and 22 ms for voiceless stops. Moreover, Ivry and Gopal had a healthy listener identify the syllables produced by the cerebellar patients. Patients produced tokens of /ba/, /pa/, /da/, and /ta/ in order to assess whether voicing contrasts (/ba/-/pa/, /da/-/ta/) were more affected by cerebellar dysarthria than place contrasts (/ba/-/da/, /pa/-/ta/). They report that the judge made many more confusions along the voicing than the place dimension.

Given the more lengthy VOTs, however, it is possible that greater variability is not due to the malfunctioning of a central timer, but simply a function of the overall duration. In other words, the greater variability displayed by cerebellar patients may be the result of a general slowness in their speech that may not be related to central timing abnormalities. Neither study reported variability as a function of duration, however, in examining the average VOT and SDs reported in Ivry and Gopal, it is apparent that the ratio of variability to average VOT duration is much greater for the cerebellars (voiced: ~.93; voiceless: ~.5) than controls (voiced: ~.66; voiceless: ~.19).

Ackermann and Hertich (1997) report findings that may shed some light on this issue. They asked eight German patients with cerebellar atrophy to utter the words /daten/ and /taten/ 10 times each as part of a carrier phrase. They then calculated average VOTs, standard deviations (*SD*s), and sentence durations for each subject. Half of the cerebellar subjects had longer than normal sentence durations while the other half did not exhibit this general slowing. Of the four subjects with normal sentence durations, half displayed average VOTs for the voiced stop within the normal range while the other two produced VOTs that were slightly longer (1 ms) than the maximum VOT produced by the controls. All of the VOTs for the voiceless stops were within the normal range, but two subjects showed higher variability than controls. For these four subjects then, VOTs were relatively unimpaired, with two subjects showing higher variability on the voiceless cognate. This finding provides some evidence that the variability of VOT is disrupted because of impaired temporal computations and is not simply a function of overall duration.

Of the remaining four subjects with longer than normal utterance durations, one subject showed normal mean VOTs, but higher variability on both voicing cognates while another showed abnormal mean VOT and *SD* for only the voiceless cognate. Only the two most severely dysarthric subjects displayed disrupted VOT durations while variability remained normal.

The common finding across these studies is the association of cerebellar pathology

with greater variability of VOT production. Variability seems most affected in the production of voiceless stops, possibly because these phonemes pose higher demands on temporal control (Cooper, 1977). Voiced stops may simply reflect the coupling of the burst release and vocal cord vibrations with the time point of adduction being less critical than for voiceless stops. For instance, the vocal cords may be adducted at any point before the closure is released for voiced phonemes in English. Since the vocal tract is closed off, vibration does not occur even when the vocal cords are narrowed early; thus, the speech signal is unaffected. However, adducting the vocal cords too early for voiceless stops would result in audible voicing and, if VOT is too short, be taken for a voiced rather than voiceless phoneme.

Of course, these results do not exclude the possibility that VOT is disrupted due to factors other than timing. For example, too little force applied to the adduction of the vocal cords may result in aspiration thereby producing a longer VOT. Given these patients' temporal-processing impairments in nonspeech domains as well as their results in perception tasks, the timing hypothesis becomes more likely.

Perception. As VOT acts as an important cue to voicing, researchers felt justified in asking whether patients with temporal perception deficits would show impairments in identifying voicing contrasts. Ivry and Gopal (1993) tested 15 native German cerebellar patients' and 7 controls subjects' perception of two stop consonant continua varying in either voicing or place. Each continuum consisted of nine syllables, and either varied in VOT by 10-ms steps (/ba/-/pa/) or the onset frequencies of the second (F2) and third (F3) formant (/ba/-/da/). Each syllable of the continua was repeated 32 times in a random order and subjects were asked to identify what they heard. Results indicated that the identification functions of the cerebellar patients were quite similar to that of the controls for both continua. In other words, cerebellars and controls exhibited a steep decline in the mean number of responses to one endpoint midway through the continuum. Consistency of responses to each syllable was also calculated and found to be no different for control and cerebellar subjects. The authors conclude either that the timing system impaired by cerebellar damage is not involved in speech timing perception or that the multiple cues to voicing, such as the presence or absence of aspiration, allowed the cerebellar subjects to perform as well as controls.

Interested in exploring these alternative explanations, Ackermann, Graber, Hertich, and Daum (1997) tested 10 German cerebellar patients on a purely durational phonemic contrast. They varied the occlusion time of the medial stop consonant /t/ in ''boten'' to create the word ''boden'' in 10 steps of 10 ms each. They also used another minimal pair that varied the VOT of the initial stop consonant (tick/dick). All tokens in the continua were presented 10 times and listeners were asked to identify what they heard. As in Ivry and Gopal, both patients and controls exhibited an abrupt transition from one response type to another for the tick/dick contrast. However, subjects with cerebellar atrophy did not show a phonemic boundary for the boten/boden series. The function is essentially flat with cerebellar subjects identifying every token of the continuum as /boten/ between 30 and 40% of the time. Thus, using a purely durational cue, these researchers found that cerebellar patients were unable to accurately identify these words.

There is a possibility, however, that it is simply the number of available cues that influences the performance of cerebellar subjects. As stated previously, VOT is only one cue available in identifying initial stops. In fact, it is difficult, if not impossible, to synthesize an identifiable VOT continuum while keeping aspiration parameters constant. Thus, even with synthetic stimuli, multiple cues to voicing in initial stop consonants are present. It is possible, as Ackermann et al. have shown, to synthesize a medial stop consonant contrast that varies in only temporal parameters. It would

be helpful to know whether cerebellar patients will experience difficulty if only a single spectral, rather than temporal, cue is available. Ivry and Gopal's control task using a place continuum provides evidence that cerebellars can perform accurately with only spectral information. As Ivry and Gopal manipulated both the onset frequencies of F2 and F3, it may be that patients' performance will decline relative to controls if only one spectral cue (either F2 or F3 onset frequency) were present. This seems unlikely, however, as spectral cues are probably not analyzed separately.

Conclusions. As a whole, the results indicate that cerebellar patients' problems in computing temporal parameters underlie their deficits in certain speech tasks. Selective deficits in timing seem to be the simplest explanation of the pattern of impairments displayed by these patients such as their difficulties in (a) judging medial occlusion durations or (b) producing VOTs consistently within the normal range. Cerebellar patients, however, exhibit deficits in other aspects of speech such as irregular articulation, fluctuating pitch, irregular rate and stress, and erratic phonation (Darley, Aronson, & Brown, 1969) and even Ivry and Gopal (1993) found that frequency onsets for vowels were more variable for cerebellars. It is unclear at this point whether timing impairments could underlie all of these problems.

These results also indicate that cerebellar deficits in phonemic tasks are auditory/motoric in nature. The fact that these patients display impairments in nonspeech motor tasks and that they have difficulty in judging tonal durations indicates that phonetic knowledge per se is most likely intact. Most cerebellar patients' mean VOTs are within normal ranges and they seem to be unimpaired on perceptual speech tasks that do not involve temporal judgments. Given that temporal computations in general are impaired, it is easy to see why difficulties would arise in the same individual for both perceptual and articulatory speech tasks involving precise timing. However, no one has determined the correlation between production and perception deficits in the same group of cerebellars.

Parkinson's Dysarthria

Parkinson's disease is a disorder caused by atrophy to the dopaminergic nigrostriatal pathway in the basal ganglia. Characteristic symptoms of this disease include tremor, rigidity, bradykinesia, and disturbances of posture that result in limitations of speed and range of movement as well as difficulties in movement initiation. Studies have reported that these patients' motor impairments affect the production of voicing contrasts and, specifically, VOT.

Production. Lieberman and colleagues (1992) asked 40 patients with mild to moderate Parkinson's disease to produce three to five repetitions of voiced and voiceless initial stop consonants at each place of articulation. Nine of the 40 subjects exhibited some overlap in the VOT distributions of the voicing cognates. Unfortunately, neither the average VOT nor variability was reported. It is impossible to conclude, then, whether the overlap is due to one or both of the VOT means shifting up or down or whether increased variability is causing the overlap. In any case, overlap was computed for each place of articulation based on only 10 productions—five of each voicing cognate so percentage overlap would increase dramatically for each phoneme falling above or below the VOT boundary.

A more rigorous study was conducted by Forrest et al. (1989), who recorded the productions of initial and medial stop consonants by nine mild to moderately impaired Parkinson patients. Subjects uttered two sentences (''Buy Bobby a poppy'' and ''Build a big building'') 25 times each. Interestingly, VOTs for all five initial /b/s were longer for Parkinson patients than controls, although only the VOTs for the very first /b/ of each sentence were significantly longer (7–9 ms). Moreover, the

formant transition durations of /b/ in the initial word ''Buy'' were significantly shorter for Parkinson patients by about 22 ms compared to controls. There were no significant differences between groups in the production of /p/, the voiceless cognate, although Parkinson patients tended to have shorter VOTs than controls. Individual variances were not reported.

The authors point out that the longer VOTs exhibited by Parkinson patients for the voiced stop may simply be due to their general difficulty in initiating movements of all kinds. When the initial stop is part of a word in the middle of a sentence, Parkinson patients seem to produce well-articulated phonemes. In fact, a study by Ackermann et al. (1999) found Parkinson patients to perform similarly to controls when asked to produce /daten/ and /taten/ as part of the sentence ''Ich habe _____ gelesen''. Moreover, Forrest et al. (1989) reported that sentence-initial stops contained shorter formant durations than other stops. The cooccurence of long VOTs with shorter formant transition durations may be evidence that the patients maintain the overall duration of the segment but simply experience difficulty in initiating vocal cord vibration. It seems unlikely, then, that Parkinson's disease results in higher-level speech disorders, but more probably impacts speech production at an articulatory level. Only one experiment directly assessed this question by measuring reaction times between the presentation of a click and the onset of speech (Ludlow, Connor, & Bassich, 1987). These authors reported no significant differences in phonation onset between Parkinson patients and controls. However, the stimuli (/a/, /ba/, and /ga/) were uttered only once by each of the patients.

Perception. Although Lieberman et al. (1992) tested the ability of Parkinson patients to perceive grammatically complex sentences, no studies have assessed phonemic perception for this group. In addition to motor impairments, Parkinson patients have exhibited difficulty in cognitive tasks that require the shifting of attention or type of processing (Hayes, Davidson, Keele, & Rafal, 1998). However, it is doubtful that such an impairment would impact speech perception.

Conclusions. Parkinson patients produce overlapping VOT distributions that appear to be primarily due to the longer VOTs of the voiced cognate. As these patients are known to have trouble initiating movements in general, it is reasonable to conclude that they would also display difficulties in initiating speech. Parkinson patients may be relatively unimpaired at producing voiceless stops because they have more time before vocal cord adduction and, thus, sufficient time to prepare this sequence of gestures.

Unlike cerebellar patients, we would expect speech processing difficulties to be manifest only in the production of phonemic contrasts. Parkinson patients' general slowness in initiating movements is unlikely to interfere with their ability to perceive sound as these processes are performed by separable motor and perceptual neural areas.

Language-Impaired Children

Another group displaying impairments of voicing perception and production is children with specific language impairment. LI is defined as ''a developmental disorder in which severe problems of comprehension of language and of language output are manifested in the absence of hearing loss, cognitive impairment, or emotional problems'' (Stark & Tallal, 1979, p. 1703). Tallal and colleagues have proposed that this disorder is the result of a general impairment in processing rapidly presented stimuli and, therefore, define this disorder as auditory rather than phonological or phonetic (Tallal & Piercy, 1973, 1974; Tallal, Miller, & Fitch, 1993). In support of the rapid processing hypothesis, is a study showing that adult dyslexics (see Tallal,

Miller, & Fitch, 1993, for a discussion of the high coincidence of dyslexia and LI) have cellular defects of the magnocellular pathway—cells that respond primarily to transient or moving stimuli (Galaburda & Livingstone, 1993). There is evidence, however, suggesting that these children may actually have a speech-specific deficit rather than a general auditory disorder. These arguments will be presented after a discussion of LI children's performance with voicing contrasts.

Production. Although most studies with LI children have focused on speech perception, two studies by the Tallal group have reported how LI children fare when producing initial stop consonants varying in place and voicing (Tallal et al., 1976; Stark & Tallal, 1979). Tallal et al. (1976) asked 12 children diagnosed with LI and 12 age-matched control children to repeat initial stop consonants six to eight times each as part of a syllable or syllable cluster (e.g., /ble/). The authors hypothesized that LI children should have the most trouble with stop consonants spoken as part of a cluster because formants change more rapidly in this context than as part of a simple consonant + vowel syllable. Acoustic data were not reported in this study; instead, results were displayed as the percentage of errors made by two transcribers in identifying the utterances of LI children and controls. Transcribers made more errors for all children's productions when consonants occurred in clusters, but the LI children showed a greater difference between the two conditions. Unfortunately, errors were not identified as either place or voicing errors, so it is impossible to know whether LI children were selectively impaired on one feature or whether their difficulty was manifest with both features.

A follow-up experiment conducted by Stark and Tallal (1979) provided much more data. They examined the performance of 12 LI and control children on their abilities to produce initial stop consonants as part of syllables and words. Each consonant was repeated between four and six times. Again, two transcribers counted the number of errors they made in identifying the productions of the children. Both place and voicing errors were equally likely. The authors also measured VOT, and reported that LI children had much greater variability for both voiced and voiceless initial stops. Modal values of both voicing cognates appeared to be similar between groups. Further, the severity of LI was highly correlated with the degree of variability displayed in VOT indicating that LI children with more severe perceptual problems were more likely to produce highly variable VOTs.

Perception. On the perceptual front, most work has been done on the perception of place rather than voicing and, specifically, with the perception of two consonants /b/ and /d/. In a number of studies, Tallal and colleagues have reported that LI children experience difficulty in discriminating between these two initial stop consonants. However, when the formant transitions are lengthened to 80 ms, these subjects perform as well as controls (Tallal & Piercy, 1973, 1974, 1975). The exclusion of other phonemic contrasts by the Tallal group has been criticized by others because of the importance of knowing how LI children would fare on contrasts that may or may not require rapid processing (Studdert-Kennedy et al., 1994). Only one study to date has examined the performance of LI children on voicing contrasts (Tallal & Stark, 1981). This is especially striking given the results indicating VOT production difficulties for LI children.

As part of an experiment observing performance on a number of speech contrasts, Tallal and Stark (1981) assessed 35 LI children and 38 age- and IQ-matched controls on voicing perception. Subjects identified the syllables /da/ and /ta/ synthesized with a 60-ms difference in VOT using a go/no go procedure. They pushed a button when hearing /da/, but were instructed to withhold button presses and shake their heads ''no'' when presented with /ta/. LI children made more errors in identifying voicing contrasts and place of articulation contrasts (/ba/ vs. /da/) than control children.

Voicing errors were not as common as place of articulation errors for all subjects, but the authors point out that there are more cues available to distinguish voicing pairs.

The basis for the Tallal group's claims of an auditory disorder of speech comes from the results of nonspeech tasks that ask subjects to discriminate between sequences of tones or touches (Johnston, Stark, Mellits, & Tallal, 1988; Tallal, Stark, Kallman, & Mellits, 1981; Tallal, Stark, & Mellits, 1985). For example, a subject may be asked whether the low tone came before the high tone or vice versa. LI children show impairments at the task when the interstimulus interval (ISI) is small, but perform normally when the ISI is lengthened (Tallal & Piercy, 1973, 1974). It has been argued, however, that impairments with nonspeech stimuli are not really a problem with rapid signal processing but with temporal order judgments. In contrast, the speech problems exhibited by LI children do not appear to be temporal order impairments. No one has ever shown, for example, that LI children have difficulties discriminating /ba/ from /ab/. Without an adequate nonspeech control experiment, it is impossible to tell whether speech impairments are auditory in nature or speech-specific.

Mody, Studdert-Kennedy, & Brady, (1997) attempted to provide such a control using syllable-like stimuli composed of two sine waves. They also tested discrimination with a /ba/–/da/ continuum synthesized to contain differences in two formants. Unlike the Tallal group, Mody et al. were primarily interested in the performance of good and poor readers. Although poor reading skills are often associated with LI, these subjects were not diagnosed with LI per se. However, these subjects did show impairments in the discrimination of the /ba/–/da/ continuum presented at the fastest ISI like LI children. More importantly, poor readers were unimpaired in the nonspeech condition, outperforming the control subjects.

Those who view LI as a speech-specific disorder claim that these children have a higher-level deficit that only becomes apparent when having to process phonemes quickly (Mody et al., 1997; Studdert-Kennedy et al., 1994, Studdert-Kennedy & Mody, 1995). There is some evidence that LI children are impaired at phonemic contrasts that do not rely on the perception of brief signals. Tallal and Stark (1981) reported that LI children were impaired in the perception of /sa/ and /sha/ which, unlike other place of articulation contrasts, differ throughout the initial 130 ms of frication noise. As LI children performed equally to controls when the formant transition of /ba/ and /da/ were stretched to 80 ms, it was hypothesized that the LI children should have no problems discriminating this contrast. The authors note, ''This suggests that the perceptual deficit of LI children may not be limited to discriminating specific types of temporal cues. Rather, these children also may have difficulty discriminating certain spectral cues'' (p. 574).

Another concern with the Tallal studies is the presence of ceiling effects which make it difficult to determine whether LI children do improve in tasks that reduce rapid processing demands. Although LI children's performance improves when stop transitions are lengthened to 80 ms, the effect of this manipulation on controls is unclear because they are already performing perfectly on syllables that have transitions of only 40 ms. If controls were not at ceiling, they might show improvements of the same magnitude as LI children when transitions are lengthened.

Fortunately, a recent study conducted by Wright and colleagues does not suffer from ceiling effects and still maintains that LI children suffer from a general auditory deficit. They suggest that the phonemic perception problems experienced by LI children may not be due to impairments in rapid stimulus processing per se, but are caused by their difficulty in separating a brief sound from a rapidly following sound of similar frequency (Wright et al., 1997). They report that LI children suffer from

an excessive amount of backward masking. When having to detect whether a tone was present, LI children's detection thresholds were significantly higher only when the tone preceded the onset of noise, but thresholds were similar to controls if the onset of the tone was presented during or after the noise signal.

This explanation is slightly different than the original claim of deficits in rapid signal processing. The nature of the disorder would still be considered auditory, but is due to impaired perception of brief, successive signals and not to a deficit in processing rapid signals per se.

Conclusions. Children classified as language-impaired have trouble with the discrimination and identification of place of articulation and voicing contrasts as well as being less able to detect a tone followed by noise. Impairments in both speech and nonspeech domains indicate a general auditory disorder although it is unclear whether the difficulty is due to deficits in rapid signal processing or excessive backward masking. Although Mody et al. (1997) reported normal performance on a nonspeech control task, their group of poor readers may not be comparable to those specifically classified as language-impaired.

Most of the research on LI has focused on perceptual abilities; however, these subjects display deficits in production as well. Similar to the cerebellar patients, LI children produce VOTs with normal modal values but disrupted variability indicating implementation problems rather than a loss of phonetic knowledge. Two alternative explanations have been offered by the Tallal group for the presence of implementation difficulties for LI children (Stark & Tallal, 1979). First, the perceptual problems displayed by LI children may cause them to have trouble learning how to produce speech. Both difficulties with rapid signal processing or backward masking may result in perceptual feedback that is not sufficient for learning the motor patterns associated with certain phonemes. Alternatively, a central disorder may underlie both the perceptual and the motor difficulties. In the case of a disorder of rapid signal processing, subjects may have problems producing a series of rapid movements; however, it is difficult to claim a central disorder of masking that would appear in both perceptual and production domains. Thus, the idea that excessive backward masking underlies LI may be ruled out if it is found that LI children have problems making rapid nonspeech movements.

PHONETIC LEVEL OF PROCESSING

After extracting temporal and spectral information from the incoming signal, listeners must match these parameters to certain regularized features associated with phonemes. In the same way, speakers need to be able to retrieve the motor patterns associated with these features in order to accurately reproduce them. Two neurological populations appear to have difficulty with one or the other of these phonetic tasks—Broca's aphasics and speech apractics. Broca's aphasia is characterized by slow, deliberate speech using very simple grammatical structure. It is generally associated with more anterior lesions including the left, frontal operculum (Geschwind, 1965).

The causes of apraxia of speech are more controversial, but evidence has been presented that suggests the involvement of the insular cortex (Dronkers, 1996). Apraxia of speech is believed to reflect a disturbance in the programming of movements for speech separate from aphasia. ''Unlike aphasia, in which there are nearly always multimodality impairments of language, apraxia of speech can exist independent of impairments in verbal comprehension, reading comprehension, and writing . . . and its occasional emergence as the only disturbance of speech justify its

identification as a unique speech disorder (Duffy, 1995, p. 259). Patients with this disorder will ''inconsistently misarticulate words and then struggle for the correct pronunciation in repeated trials'' (Dronkers, 1996, p. 160). A substantial portion of these patients also have nonverbal oral apraxia which is characterized by the inability to perform volitional movements or imitate gestures such as coughing, smacking the lips, clicking the tongue, or blowing (Duffy, 1995). The site of the lesion causing this disorder is very close to the neural area involved in apraxia of speech, although each of these disorders can exist independently of the other.

In general, previous research with these groups has not attempted to dissociate their performance on speech tasks. Instead, speech deficits of Broca's aphasics, or less commonly apractics, have been contrasted with those of Wernicke's aphasics. Subjects in these experiments are classified according to their type of aphasia based primarily on their performance on certain aphasia diagnostic batteries such as the Boston Diagnostic Aphasia Inventory, and not on CT or MRI scans. Thus, it is often unclear whether participants are ''pure'' Broca's aphasics or apractics or have both disorders. In fact, Broca's area is in very close physical proximity to the insular region. It is quite probable, then, that these two disorders often cooccur. Indeed, Duffy (1995) suggests that, in order for Broca's aphasia to be diagnosed, apraxia of speech must be present. For this reason, I have combined the discussions of Broca's aphasia and apraxia of speech in an attempt to dissociate the impairments shown on voicing production and perception by each of these groups.

Production. Several studies have shown that Broca's aphasics are impaired at producing distinct VOTs, an essential cue to voicing. Subjects in these experiments utter anywhere from 5 to 40 tokens each of initial voiced and voiceless stops. The typical pattern of results indicates that Broca's aphasics produce more variable VOTs for both voicing cognates and average VOTs that are closer to the midpoint between the two distributions than controls (Blumstein et al., 1977, 1980; Gandour & Dardaranda, 1984; Baum et al., 1990).

Other studies have measured apractics performance on VOT tasks with the same result (Hoit-Dalgaard et al., 1983; Itoh et al., 1982). Itoh and colleagues (1982) asked four Japanese apractic speakers to produce initial stop consonants, /de/, /te/, /ge/, and /ke/, 25 times each, and found considerable overlap of the VOT distributions. Modal VOTs tended to be closer to the midpoint and variability was greater than for fluent aphasics and controls for both voicing cognates, but especially voiced stops. Hoit-Dalgaard et al. (1983) found similar results of mean VOT with English speakers, although voiced and voiceless stops were equally aberrant. However, variability did not appear abnormal in a perusal of the graphs.

In both these studies, the occurrence of aphasia was noted for some (Itoh et al., 1982) or all (Hoit-Dalgaard et al., 1983) of the subjects along with apraxia of speech. However, the pure apractic results reported by Itoh et al. did not appear to differ from those with a cooccurence of apraxia and aphasia. In contrast, Seddoh and colleagues (1996) did not find any deficits in VOT production for a group of apractic subjects. They asked five pure apractic subjects to repeat words beginning with /b/ and /p/ 10 times as part of a carrier phrase ''That's a _____ a day.'' Apractic subjects had significantly higher mean VOTs for both voicing cognates, but were not more variable. Graphs indicating the number of tokens uttered at a given VOT were not presented but, based on the mean and variability information, it would appear that these apractic subjects were able to produce nonoverlapping VOT distributions albeit at long total durations. Thus, one experiment has found VOT abnormalities for apractics in the absence of aphasia while another has not. Replications of the above experiments with pure apractic subjects would be helpful in concluding

whether VOT abnormalities are a symptom of apraxia of speech only or if aphasia must be present.

Perception. Studies of Broca's aphasics have indicated perceptual abnormalities when hearing voicing contrasts. In an identification task, Basso et al. (1977) reported that 10 of 11 Broca's aphasics showed mild to severe impairments in identifying a /da/–/ta/ contrast. Slight impairment was defined as misclassifying tokens of the continuum at times but still showing a steep categorical function. The other end of the rating scale was termed ''very severe'' and indicated that the perceptual function was flat with no trend toward correct identification. Four of 11 were classified as having a very severe perceptual impairment.

In contrast, Blumstein et al. (1977) found that four of five Broca's aphasics could discriminate voicing contrasts and three of those four could also label them. In another study, Blumstein et al. (1977) found that Broca's aphasics made significantly fewer discrimination errors of voicing contrasts than Wernicke's aphasics. It is unclear whether Broca's aphasics would be impaired at this task relative to controls as healthy subjects were not tested. Broca's aphasics erred in discriminating words differing in their initial stop consonants 10% of the time, and it is easy to imagine that controls would perform perfectly at this task as the words were tokens of natural speech presented clearly.

A study of five apractics with aphasia indicated that they were impaired at the identification of a 38-point continuum of /bees/–/peas/. Errors ranged from 4 to 60% based on normative data (control subjects were not tested). However, Square-Storer et al. (1988) report that ''pure'' apractic patients show no perceptual impairments. Voicing data were not specifically reported but, instead were combined over place and manner contrasts. In general, apractics performed like controls in discriminating spoken real words and syllables. Apractics with aphasia, however, made significantly more errors than controls on this discrimination task. This experiment suggests, then, that pure apractics will not exhibit perceptual difficulties. Only if aphasia is present will subjects be impaired at phonemic contrasts.

Conclusions. The fact that Broca's aphasic/apractic speakers produce VOTs that fall at all points of the continuum indicates that the problem is most likely with motor programming rather than with phonological selection. If phonetic information were intact, these patients should produce clearly distinct VOT distributions with tokens occasionally falling well within the range of the opposing distribution. Instead, the commands to the articulators are incorrect and result in widely divergent VOTs. Of course, these results also fit with the hypothesis that these patients have both selection and programming impairments. It could be that the tokens falling between the two distributions are due to motor programming errors, but that the productions falling within the opposing distribution are errors of phonological selection. Given the close proximity of the neural areas involved in nonverbal oral apraxia and apraxia of speech, it seems quite likely that the substitution-like errors of Broca's aphasics/apractics are simply severe distortions of the correct phoneme. In addition, deficits in VOT production do not appear to be motoric in nature. Although nonspeech movements may be abnormal for apractics, they do not necessarily have to be.

Broca's aphasics/apractics also show impairments in discriminating and identifying voicing contrasts. This deficit is most likely phonetic rather than phonological in nature given that discrimination and identification are both abnormal. If phonetic processing were intact, one would expect preserved discrimination and impaired identification. Instead, it appears that aphasic/apractics have difficulty in extracting phonetic features from the incoming acoustic stream. These perceptual impairments are probably not due to auditory deficits in general given normal performance on nonspeech stimuli. Divenyi and Robinson (1989) found aphasics to be unimpaired in

identifying frequency sweeps and other nonlinguistic stimuli. Thus, it would appear that temporal and spectral parameters are properly extracted but, if required to match those specifications to phonetic features, these patients will be impaired.

Although Broca's aphasics appear to show deficits in the discrimination of voicing contrasts, pure apractics may not have such an impairment. This finding may explain why there has been such a low correlation in these studies for performance in perceptual and production tasks (Blumstein et al., 1977; Gandour & Dardarananda, 1984). It is also reasonable to assume that a phonetic deficit in motor planning would be dissociable from a phonetic perceptual problem. A common store of phonological knowledge could be accessed independently by motor and perceptual areas.

PHONOLOGICAL LEVEL OF PROCESSING

At the phonological level, listeners must match phonetic features of the acoustic signal to stored phonological representations. In a similar fashion, speakers must select phonemes correctly with accurate links between that phoneme and the phonetic knowledge of how to produce that sound. Wernicke's aphasics appear to be impaired at these types of phonological tasks, and this deficit can be observed on both perception and production tasks. This type of aphasia is characterized by fluent speech in conjunction with severe comprehension deficits. It is associated with more posterior lesions including left superior temporal gyrus and often extends to the supramarginal and angular gyrus (Damasio, 1991).

Production. One of the earliest studies of aphasics' productions of voicing contrasts was conducted by Blumstein et al. (1977). They compared the VOT productions of a number of different types of aphasic patients including two Wernicke's aphasics. Each subject uttered 12 tokens of the initial stop consonant /d/ and an equal number of its voiceless cognate /t/ as part of a monosyllabic word. All four control subjects and four non-Wernicke's aphasics produced nonoverlapping distributions, one for the voiced and the other for the voiceless cognate. Wernicke's aphasics' productions were similarly distributed, but with a few notable exceptions. Although there was a clear separation between the voiced and the voiceless distributions, two productions of the voiced token from one of the subjects were well within the voiceless range. Thus, unlike Broca's aphasics, Wernicke's aphasics showed a clear separation between the two distributions with no tokens falling midway between them.

In another study by the Blumstein group (1980), five Wernicke's aphasics were required to utter 40 tokens each of the initial stop consonants, /b/, /d/, and /g/, and their voiceless cognates, /p/, /t/, /k/, as part of a monosyllabic word. The word was produced as part of the carrier phrase ''This _____'' so any problems initiating speech would not affect the results. Controls showed no errors of production while Wernicke's aphasics erred 8% of the time. Half of the errors produced by Wernicke's aphasics were tokens that fell within the VOT range of the opposing distribution (what the authors term ''phonemic errors''). The other half fell between the two distributions, and were termed ''phonetic errors.'' The ratio of phonemic to phonetic errors was higher for the Wernicke's aphasics than for the Broca's or conduction aphasics. Both Broca's and conduction aphasics made about 9–12% more phonetic than phonemic errors and they had more errors overall (Broca's = 40%; conduction = 29%).

The first thing to note here is that Wernicke's aphasics have a much lower incidence of aberrant VOT productions than other aphasics. Indeed, two other studies (Gandour & Dardarananda, 1984; Baum et al., 1990) have reported that Wernicke's aphasics make no errors of VOT production. These aphasics have traditionally been termed fluent aphasics because of their well-articulated speech.

The second result of note is that Wernicke's aphasics still produce some VOTs that fall between the two distributions, although they produce fewer of these types of errors relative to phonemic substitution errors than other aphasics. Moreover, the criteria used to classify phonetic errors was based on normative data provided by Lisker and Abramson (1964), and not on a determination of individual boundary points. Since graphs of subjects' productions were not provided it is impossible to determine whether any overlap between the two VOT distributions occurred. Although Wernicke's aphasics may have produced errors according to Lisker and Abramson's normative VOT boundaries, they still may have produced contrastive voicing tokens. In fact, Blumstein et al. (1980) assessed the VOT distributions for ''correct'' productions of the six consonants and found that the variability of the voiceless distribution for Wernicke's aphasics was similar to that of controls and significantly different from that of Broca's aphasics. On the other hand, Wernicke's aphasics were more variable than controls and Broca's aphasics in the production of voiced stop consonants because of their tendency to prevoice these phonemes.

Thus, there is some evidence that Wernicke's aphasics suffer from voicing production deficits that are phonological in nature. They make few errors overall and a high proportion of these are substitution errors.

Perception. Wernicke's aphasics are known primarily for their severe comprehension deficits, and so more research has dealt with possible phonemic perceptual rather than production impairments. In an early study by Basso et al. (1977), 13 of 18 Wernicke's aphasics were categorized as having slight to very severe difficulty in identifying a 30-point continuum of /da/–/ta/ tokens with VOT durations varying in 10-ms steps. Seven subjects were described as having no perceptual boundary zone and no trend toward correct identification. None of the right-hemisphere-damaged patients exhibited any perceptual difficulties. Itoh et al. (1986) reported a similar result for Japanese-speaking Wernicke's aphasics. Wernicke's aphasics produced less steep categorical functions and made more confusions in identifying a /ga/–/ka/ continuum than controls.

Blumstein et al. (1977) tested both discrimination and identification capabilities of Wernicke's aphasics on a /da/–/ta/ continuum. In one part, subjects were asked to identify the phonemes they heard. In the second part, subjects were asked to discriminate pairs of syllables differing in VOT by 20 ms. Each pair was presented two to three times each. Interestingly, all four Wernicke's aphasics could discriminate the stimulus pairs. They responded ''different'' to stimuli on either side of the VOT boundary and ''same'' to stimuli within a category. In contrast, only one of the four Wernicke's aphasics could correctly identify those same tokens. Correct identification was defined as consistent (70%) identification of stimuli with a steep slope between the two category values. This finding seems to indicate that phonetic information is intact in these patients. They are able to perceive differences in acoustic parameters as well as being able to detect which acoustic variations signal a phonemic opposition. For example, to be deemed a ''correct'' discrimination response, subjects need to determine that a phoneme with a 20-ms VOT is phonologically different than one with a 40-ms VOT, but respond that two syllables are the same if their VOTs are 40 and 60 ms, respectively. Thus, their difficulty lies in linking this phonetic information to a phonological label.

However, discrimination deficits have been reported for Wernicke's aphasics in another study (Blumstein et al., 1977). A group of six Wernicke's aphasics was observed to make more voicing discrimination errors than Broca's aphasics (control subjects were not tested). This result would seem to contradict the finding that Wernicke's aphasics are able to extract phonetic features from incoming speech. However, this study differed from the others in one important aspect. In the previous

studies, the phonemic contrast of interest was part of a syllable, but in Blumstein et al.'s (1977) study the stop consonant was the initial portion of a word. Perhaps what is impaired in Wernicke's aphasia is not only the labeling of isolated syllables, but also the ability to use lexical items in a top-down fashion to aid in phonetic discrimination. Thus, two connections are faulty—the link between phonetic features and phonological knowledge and the connection between lexical items and their phonological constituents. Instead of relying on a bottom-up strategy to discriminate pairs of words differing in their initial stop consonants, they perceive the whole word and then check to see if the phonological constituents are the same. Evidence that subjects use such a strategy comes from their data showing that all aphasic groups make more discrimination errors on nonsense than real words. In cases of ambiguous phonetic information, then, subjects rely on lexical information if it is available.

Further evidence for Wernicke's inability to use lexical information in a top-down fashion comes from studies of word priming and semantic judgments (Milberg & Blumstein, 1981; Milberg, Blumstein, & Dworetzky, 1988). Wernicke's aphasics often show normal priming of semantically and phonologically related words in a lexical decision task; however, when asked to make overt judgments of whether the same two words are related in meaning, they perform very poorly.

Conclusions. Wernicke's aphasics have few problems in articulating voicing contrasts. However, when they do err, their incorrect productions are more likely to be substitution errors than implementation errors. This suggest that the planning of speech movements are intact, but that either phonological selection or the link between phonological knowledge and motor plans is impaired.

With the exception of discriminating pairs of words, Wernicke's aphasics seem to have few problems in discriminating voicing contrasts. However, when the task is to identify those voicing cognates, Wernicke's aphasics are severely impaired. Again, this suggests that the link between phonetic features and phonological knowledge is disrupted with the result that identification, but not discrimination, of voicing contrasts suffers.

GENERAL CONCLUSIONS

Although several neurologically impaired groups have trouble perceiving and producing voicing contrasts, each group has distinct deficits underlying their poor performance (see Table 1). Accurate and reliable processing is required at different levels in order for voicing contrasts to be heard and articulated correctly. The fact that so many neurological groups are impaired at voicing tasks is an indicator of the great number of computations contributed by separable neural structures. Experiments have emphasized the importance of estimating duration accurately, initiating movements rapidly, perceiving brief successive sounds, extracting features that are characteristic of certain phonemes, planning the movements required to produce those features, linking those features to a phonological store, and retrieving the correct motor plans to produce the desired phoneme. Figure 1 displays the model of phonemic processing described earlier with the neural areas thought to be contributing to each of those computations.

Assessment of Model

Starting at the auditory/motoric level, the model claims that damage to the cerebellum, basal ganglia, or magnocellular pathway will result in discrimination and/or production deficits for all sounds. Cerebellar patients' difficulties with temporal pro-

TABLE 1
Summary of Results of Voicing Experiments

Patient group	Perceptual deficits		Production deficits	
	Discrimination	Identification	Contrastive mean VOT	Variability
Cerebellar patients	Not tested	Abnormal (medial closure duration only)	Normal	Abnormal voiceless cognate
Parkinson patients	Not tested	Not tested	Abnormal voiced cognate of initial word	Not reported
LI children	Abnormal	Abnormal	Normal	Abnormal
Pure apraxics	Normal	Normal	Unclear	Unclear
Broca's aphasic/ apraxics	Abnormal	Abnormal	Abnormal	Abnormal
Wernicke's aphasics	Normal (syllables)–abnormal (words)	Abnormal	Normal but occasional substitutions	Normal

duction and perception may affect their abilities to produce and perceive speech accurately. All of the experiments reviewed above support the notion that cerebellar patients suffer from a non-speech-specific deficit. In terms of the production of voicing contrasts, cerebellar patients are able to maintain a distinction in VOT duration between voicing cognates. Note that mean VOTs are rarely disrupted for these patients despite being abnormally variable in their productions. This pattern of results is what we should expect if cerebellar patients have trouble with the implementation of phonetic knowledge rather than phonetic knowledge per se. They have a notion of how voicing cognates differ, but lack the ability to execute motor specifications because of a deficit in temporal computations.

Also consistent with the model is the cerebellar patients' results on identification tasks. Given their general timing impairments, cerebellar subjects only show deficits in identifying stops where the only cue to voicing is durational. If other cues such as aspiration are present, cerebellar patients do as well as control subjects in identifying voicing contrasts. Interestingly, these patients have not been tested on discrimination of initial stops. A prediction of the model would be that these patients should show discrimination impairments only on phonemes that differ by durational cues. If cerebellar patients were unimpaired at discrimination but poor at identification, a more speech-specific deficit would have to be the cause rather than general timing impairments.

For Parkinson patients, it is hypothesized that the bradykinesia associated with basal ganglia lesions results in speech deficits that are primarily motoric in nature. In fact, one study has found problems for Parkinson patients in initiating speech (Forrest et al., 1989). Moreover, studies that have Parkinson patients utter words with an initial stop placed in the middle of a sentence do not find problems with VOT production (Forrest et al., 1989; Ackermann et al., 1999). Importantly, the model predicts that there should be no problems in perceiving phonemes. No studies of speech perception with Parkinson patients have been conducted, but these experiments would constitute a critical test of the model.

The claim that LI children suffer from a general auditory deficit is more controversial, but the model asserts that damage to the magnocellular pathway associated with

LI results in a deficit of processing rapid, successive sounds that influences both the motor and perceptual aspects of speech. Difficulties with rapid, successive sound may be due to problems with rapid signal processing per se or to excessive backward masking. In support of a general auditory disorder, is the fact that LI children have problems perceiving brief acoustic signals associated with both speech and nonspeech (Tallal, 1973, 1974, 1975; Wright et al., 1997). In terms of production, modal VOTs seem to be unimpaired in this group while variability is greater than normal indicating that subjects are attempting to maintain the voicing contrast but display problems in execution. It is unclear whether production difficulties are caused by poor motor learning due to perceptual impairments or whether a central disorder underlies both production and perception deficits. Research focusing on the ability of LI children to produce rapid nonspeech movements would be helpful in resolving this issue. Moreover, the results of such an experiment could rule out the explanation of excessive backward masking as a cause of LI if it were shown that these subjects do have trouble making rapid, successive movements. It would be difficult to posit a central disorder of backward masking that would affect both movement and hearing.

In contrast, impairments at the phonetic and phonological levels are claimed to be selective to speech tasks. Groups impaired at the phonetic level are thought to have deficits in planning speech gestures or extracting phonological features from the incoming signal. Insular cortex lesions are hypothesized to primarily affect the specification of accurate motor commands for speech sounds whereas damage to Broca's area may be more involved in the matching of phonetic features to sound characteristics. There is some support so far that pure apractics without aphasia have no perceptual difficulties with voicing contrasts (Square-Storer et al., 1988), while Broca's aphasics with apraxia have been shown to be impaired at both discrimination and identification. More studies of pure apractics are needed to test the predictions of this model, however.

Broca's aphasics with apraxia also show disrupted VOT means as well as great variability in voicing production. This is in contrast to the pattern of results shown by those groups hypothesized to have impairments at the motoric level. In accordance with the model, Broca's aphasics/apractics display a disturbance of phonetic knowledge that results in the production of VOT means that are not contrastive. It is unknown at this point whether damage to Broca's area alone would result in VOT production deficits, although one study (Itoh et al., 1982) did find aberrant VOT durations for pure apractics.

Lesions that include Wernicke's area are thought to result in phonological processing deficits consisting of incorrect phoneme selection and identification. In line with the model's predictions, Wernicke's aphasics show contrastive VOT durations as well as normal variability. When errors do occur, they are substitution errors indicating a fault in phoneme selection rather than in motor planning or execution. The results of speech perception tasks for these patients conforms to the model's predictions for syllables but not for words. In line with the notion that Wernicke's aphasics are unimpaired at extracting phonetic features from the incoming signal, discrimination of voicing contrasts when part of a nonsense syllable is normal. Wernicke's aphasics only show difficulties when having to match that sound to a phoneme. In contrast, Wernicke's aphasics were impaired at discriminating voicing cognates when presented as the initial stop of a meaningful word. It has been suggested that Wernicke's aphasics are unable to use lexical information in speech perception tasks in a top-down fashion (Milberg & Blumstein, 1981; Milberg et al., 1988). In line with the model, however, Wernicke's aphasics show priming of phonologically related words so the discrimination of phonemes that constitute real words must be intact.

Future Directions

With current technological advances, researchers should be able to conduct more systematic inquiries of phonemic processing in different neurological populations. For example, magnetic resonance imaging allows scientists to more accurately pinpoint sites of neurological damage. Instead of having to rely on standard aphasia or communicative batteries which may provide too gross a classification of speech disorders, researchers can determine the relationship of lesion site to observed impairments. This should help reduce confusion that can result when a supposedly unitary group shows divergent results. For example, parietal and insular apraxics are normally lumped together in speech experiments. However, important differences may exist between the two groups in terms of speech impairments. It may explain why some studies show production deficits for pure apraxics and others do not.

Advances in speech analysis and synthesis programs also allow researchers to control and examine a wider variety of phonemic contrasts. Even so, little work has been done that examines patients' performance on other types of phonemic tasks. This is especially true in the area of production. Although some have explored whether the durations of segments other than VOT are impaired in certain groups (Forrest et al., 1989), very few have examined whether spectral parameters are also affected. It is important to know, however, if any deficits in the production of spectral information vary systematically for each patient group. For example, would Parkinson patients produce aberrant formant onsets for only the first phoneme of each sentence?

In the past, many researchers did not report individual mean VOTs and standard deviations that could be helpful in assessing different kinds of voicing aberrations. These statistics can help in determining what aspects of VOT are produced abnormally. For example, LI children produce modal VOTs that are distinct for each cognate while variability is greater than normal. However, Broca's aphasics/apraxics appear to produce abnormal mean VOTs and be much more variable as well. Parkinson patients appear to have more trouble articulating the voiced cognate while cerebellar subjects find the voiceless cognate more difficult because of the type of motoric deficits experienced by each group. Future studies need to report both statistics.

In the same way, it is important to specify the speech perception impairments of the various groups. For example, it would be helpful to know how consistent subjects are at labeling or discriminating phonemes along a continuum rather than relying on crude measures such as the total number of errors. Are certain neurological groups inconsistent at discriminating phonemes along all points of the continuum or just in the middle? In the same vein, more studies of phonemic perception using a variety of stimuli such as syllables, words, and nonsense words are needed. As stated previously, Wernicke's aphasics may have trouble with phonemic discrimination with words but not syllables. In contrast, this manipulation may affect the other neurological groups differently. For example, using words may help cerebellar patients to discriminate phonemes but impair discrimination for Wernicke's aphasics.

In addition, perceptual studies reporting an improvement in patients' performance under certain conditions need to account for possible ceiling or floor effects. Equating the difficulty of the task for controls and patients is necessary to eliminate ceiling effects and allow for the determination of whether a group actually does improve relative to controls. For example, would controls in the Tallal group's experiments improve just as much as LI children when formant transitions are lengthened if they were not already performing perfectly?

Finally, experiments studying a number of patient groups' performance on the same task would be useful in dissociating the levels of processing impaired. Neurologically impaired control groups should be tested on these same tasks. Although

some studies have attempted provide such a group (Blumstein et al., 1977; Gandour & Dardarananda, 1984), it often consists of a single right-hemisphere-damaged patient. It would be interesting to know whether some of the reported deficits in speech production and perception are due to brain damage in general.

Phonemic processing is a perfect example of the way neural areas function together to perform a task that, on the surface, appears effortless. By assessing the performance of several neurological groups, we are able to determine what types of subprocesses are involved in phonemic tasks and what neurological sites are involved in those computations. In the end, this research should not only allow us to build a model of the computations involved in phonemic processing, but also tell us about the function of several neural areas.

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