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Impairments of Speech Production and Speech Perception in Aphasia

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Phil. Trans. R. Soc. Lond. B 1994 **346**, 29-36
doi: 10.1098/rstb.1994.0125

Impairments of speech production and speech perception in aphasia

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SUMMARY

The basis of speech production and speech perception deficits in aphasia relates to implementation and access rather than to the underlying representation or knowledge base of the sound structure of language. Speech production deficits occur on the phonological level in which the incorrect phonological form of the word is selected but is implemented correctly, and the phonetic level in which the correct sound segments are selected but articulatory implementation is impaired. Phonological deficits emerge regardless of lesion site, whereas phonetic deficits have a specific localized neuroanatomical substrate. Phonetic deficits are not linguistic but affect particular articulatory movements. Speech perception impairments emerge in nearly all aphasic patients, suggesting that the neural basis for speech perception is broadly distributed in the language hemisphere. The impairment reflects the misperception of phonetic features rather than a deficit in the auditory processing of speech and emerges particularly as the sound properties of speech contact the lexicon.

1. INTRODUCTION

The sound structure of language provides the vehicle for conveying the meaningful attributes of language. As such, deficits in either speech production or speech perception contribute to the disruption of the language communication process. In recent years, research has focused on the mechanisms and processes contributing to speech deficits accompanying the adult aphasias. Much as linguistic research has been guided by considerations of the structural levels of the linguistic grammar such as phonology, lexicon–semantics and syntax, so have studies of speech and language deficits in aphasia. Of particular interest has been determining the extent to which deficits in aphasia are selective, affecting a particular component, and additionally determining whether such deficits reflect impairments to the linguistic representations, i.e. the structural properties of language, or alternatively, impairments to the mechanisms responsible for accessing or processing these representations. In this chapter, we will consider the nature of speech production and speech perception deficits in aphasia, specifically addressing these issues. We will also consider the evidence pertaining to the neural substrates for speech production and speech perception.

Models of speech production and speech perception generally share a number of assumptions. The first is that there is a single lexicon subserving both speech production and speech perception. That is, words to be produced or perceived ultimately contact a common representation. The second is that the nature of this sound-shape representation is in terms of abstract linguistic units such as segments, features, and rules for their combination that are specific to the sound structure of language. The third is that these representations are realized in terms of patterns of neural activity of units determined by both excitatory and inhibitory signals from other units. Thus, the identity of a sound or word is determined by the pattern of activity of a number of units.

Although there are a number of shared assumptions, it is also the case that speech production and speech perception are ultimately served by very different mechanisms. In speech production, the representation of a word must be ultimately realized in terms of a set of motor commands to the speech apparatus; in speech reception, the representation of a word must be derived from the acoustic waveform and its various transformations along the auditory pathway. Thus, the study of speech production and speech perception deficits must take account of these different mechanisms. For this reason, the discussion of speech production and speech perception will be considered separately.

2. CLINICAL AND NEUROLOGICAL FRAMEWORK

Before considering deficits in the sound structure of language, it may be useful to provide a framework from which to explore the underlying bases of these

Phil. Trans. R. Soc. Lond. B (1994) **346**, 29–36
Printed in Great Britain

deficits. Classical approaches to the clinical and neurological basis of language disorders in adult aphasics have typically characterized the aphasia syndromes in broad anatomical (anterior and posterior) and functional (expressive and receptive) dichotomies (see Geschwind 1965). The two aphasia syndromes that best characterize the anterior–posterior and expressive–receptive dichotomy are Broca's and Wernicke's aphasia. Broca's aphasics show a profound expressive deficit in the face of relatively good auditory language comprehension. Speech output is typically non-fluent in that it is slow, laboured and often dysarthric, and the melody pattern seems flat. Furthermore, speech output is often agrammatic, characterized by the omission of grammatical words such as 'the' and 'is' as well as the incorrect usage of grammatical endings. Syntactic structures are generally limited to simple sentences with few embeddings and dependent clauses. As to other language abilities, naming an object to confrontation is generally fair to good, and repetition of language is usually as good as or better than spontaneous speech output. The underlying neuropathology includes the frontal operculum as well as premotor and motor regions posterior and superior to the frontal operculum, extending to the white matter below and including the basal ganglia and insula (Damasio 1991).

The syndrome of Wernicke's aphasia is characterized by both language input and output impairments. Auditory language comprehension is severely impaired. Speech output is fluent and well-articulated. Grammatical structures seem relatively intact, although syntactic phrases may be inappropriately juxtaposed. Most characteristically, the semantic content of the output is severely compromised. Language is empty of semantic content, often difficult to understand, with the overuse of high-frequency, low-content words such as 'thing', 'be', 'have', 'this'. In addition, literal paraphasias (sound substitution errors) and verbal paraphasias (word substitution errors) occur in speech output. There are some Wernicke's patients who also produce neologisms or 'jargon'. Another frequent characteristic of this disorder is 'logorrhea' or press for speech, whereby patients, even in a conversational setting, talk on and on without stopping. Other associated language impairments include a moderate to severe naming deficit and a severe repetition disorder. The neuropathology associated with Wernicke's aphasia involves the posterior region of the left superior temporal gyrus, often extending to the supramarginal and angular gyrus (Damasio 1991).

To a first approximation the anterior–posterior dichotomy corresponds well with the functional expressive–receptive dichotomy. After all, it is not surprising to find expressive speech deficits with damage to the motor areas and receptive speech deficits with damage to the auditory association areas. Nevertheless, as we will show, the functional anatomical dichotomy is not upheld with respect to speech production and speech perception impairments in aphasia.

3. SPEECH PRODUCTION

To produce a word or group of words, the speaker must select the word candidate(s) from the lexicon, including its abstract phonological representation, plan the production of the utterance by encoding the abstract phonological representation of the word in terms of the phonetic parameters required for realizing the sound structure in particular contexts, and ultimately implement this phonetic string via a set of motor commands or motor programs to the vocal tract. Analyses of the patterns of speech production in aphasic patients suggest that nearly all aphasic patients, regardless of the aphasia syndrome, display impairments in both the selection and the planning. In particular, patients show four types of error in the speech output (Blumstein 1973). These errors include: phoneme substitution errors in which a phoneme is substituted for another, e.g. 'teams' → 'keams'; simplification errors in which a phoneme or syllable is deleted, e.g. 'green' → 'geen'; addition errors in which an extra phoneme or syllable is added to a word, e.g. 'see' → 'stee'; and environment errors in which the occurrence of a particular phoneme is influenced by the surrounding phonetic context. These errors include metathesis, e.g. 'degree' → 'gedree', and progressive and regressive assimilation errors, e.g. 'Crete' → 'kreke' and → 'trete', respectively. The stability of these patterns is evidenced by their occurrence across languages: French (Bouman & Grunbaum 1925; Lecours & Lhermitte 1969), German (Bouman & Grunbaum 1925; Goldstein 1948), English (Green 1969; Blumstein 1973), Turkish (Peuser & Fittschen 1977), Russian (Luria 1966) and Finnish (Niemi *et al.* 1985).

The pattern of sound substitutions is consistent with the view that the incorrect features have been selected or activated, but have been correctly implemented by the articulatory system. Thus, most substitution errors involve a change in value of a single feature. For example, the production of 'doy' for 'toy' reflects a change in the feature of voicing. Similarly, the production of 'dut' for 'nut' represents a change in the feature of nasality. Relatively few substitution errors produced by aphasic patients involve changes in more than one feature. Similarly, addition and simplification errors show that there has been an incorrect selection of a segment or segments in the particular word. Environment and metathesis errors show that the selection of the phonological patterns has been correct, but that there has been an error in the correct planning of the sequence of the segmental units. Thus, a later sound in a sequence influences the production of an earlier sound segment in assimilation errors, and the order of the sounds in a word or utterance is incorrect in metathesis errors.

Phonological errors also suggest that the nature of the syllable structure (i.e. the organization of consonants and vowels) of the lexical candidate constrains the type and extent of errors made during the selection process (Blumstein 1973, 1990). In particular, the occurrence of phoneme substitution

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

errors is more likely to occur when the syllable contains a single consonant than when it is part of a consonant cluster; for example, [f] is more likely to undergo a phoneme substitution error in the word 'feet' than in the word 'fleet'. Simplification and addition errors are more likely to result in the canonical syllable structure Consonant–Vowel; for example, consonants are more likely to be deleted in a word beginning with two consonants, 'sky' → 'ky', and consonants are more likely to be added in a word beginning with a vowel, 'army' → 'jarmy'. Finally, assimilation errors across word boundaries preserve the syllable structure relations of the lexical candidates. That is, if the influencing phoneme is at the beginning of the target word so is the assimilated phoneme, e.g. 'history books' → 'bistory books'. If the influencing phoneme is at the end of the target word, so is the assimilated phoneme, e.g. 'roast beef' → 'roaf beef'. These results show that information about the syllable structure of a word is represented in the lexicon, and this information is used in the planning buffer for sentence production. If this were not the case, the syllable constraints shown in the assimilation errors would not occur across word boundaries.

Importantly, despite the systematicity and regularity of the phonological errors just described, the particular occurrence of such an error cannot be predicted. That is, sometimes the patient may make an error on a particular word, and other times he or she will produce it correctly. Moreover, the errors are not unidirectional (Blumstein 1973; Hatfield & Walton 1975). A voiced stop consonant may become voiceless, e.g. /d/ → /t/, and a voiceless stop consonant may become voiced, e.g. /t/ → /d/. The fact that patients can correctly select and plan the same utterance that at other times they produce incorrectly, and that the patterns of errors is systematic but multidirectional, suggests that the underlying phonological representations are intact. Instead, there seems to be a failure in either selecting the phonological form from the lexicon or planning the appropriate phonological output. It may be that this mis-selection reflects a change in the level of activation of the nodes corresponding to either features or segments. Whatever the nature of the underlying deficit, however, virtually all aphasic patients regardless of lesion site display phonological output deficits. Importantly, the patterns of impairment among these patients (including Broca's and Wernicke's aphasics) seem to be similar. That phonological disorders occur in the context of these aphasia syndromes implicates the left perisylvian areas. These areas extend from temporal, temporoparietal, and anterior regions including Broca's area and the precentral gyrus, and almost always involve cortical lesions which extend into subcortical areas. Thus, with respect to the neural localization of phonological disorders, there does not seem to be a distinct neural localization.

As discussed above, subsequent to the selection of a lexical candidate or candidates and the planning of the utterance, the phonetic string is ultimately converted into a set of motor commands to the articulatory system. A long-held observation is that one group of patients, Broca's aphasics, produce phonetic errors. The implied basis for these errors is one of articulatory implementation; that is, the commands to the articulators to encode the word are incorrect, poorly timed, and so forth. A number of studies have explored these phonetic patterns of speech by investigating the acoustic properties or the articulatory parameters underlying the production of particular phonetic dimensions. The dimensions investigated include voicing in stop consonants (e.g. /p t k/ vs. /b d g/) and fricatives (e.g. /f s/ vs. /v z/), place of articulation in stop consonants (e.g. /b/ vs. /d/ vs. /g/) and fricatives (e.g. /f/ vs. /s/), and the nasal and stop manner of articulation (e.g. /n m/ vs. /d b/). Results of analyses of the production of the voicing and nasal phonetic dimensions have shown that anterior aphasics evidence significant deficits (Blumstein *et al.* 1977*a*, 1980; Freeman *et al.* 1978; Itoh *et al.* 1979, 1980, 1982; Gandour & Dardarananda 1984*a*; Shewan *et al.* 1984). In particular, anterior patients have shown that they have difficulty in producing phonetic dimensions that require the timing of two independent articulators. These findings have emerged in the analysis of two phonetic dimensions, voicing and nasality. In the case of the feature voicing, the dimension studied is voice-onset time, i.e. the timing relation between the release of a stop consonant and the onset of vocal-cord vibration. The production of nasal consonants also requires appropriate timing between two articulators; in this case, the release of the closure in the oral cavity and the velum opening.

These same patterns emerge across different languages. They occur not only in English and Japanese, for which voice-onset time serves to distinguish two categories of voicing, voiced and voiceless, but also in Thai, for which voice-onset time serves to distinguish three categories of voicing in stop consonants, pre-voiced, voiced, and voiceless aspirated.

Nevertheless, these patients also show normal patterns of production for other phonetic parameters. The constellation of spared and impaired patterns of articulation suggests that their disorder affects impairments in particular articulatory manoeuvres rather than the articulatory implementation of given phonetic features. The evidence comes from studies of voicing in stop consonants. In English, the feature voicing in stop consonants can be cued in several ways. As discussed earlier, voice-onset time provides one measure of voicing for stop consonants occurring in initial position. A second measure is the duration of the vowel preceding a stop consonant. Vowels are short before voiceless stops, e.g. 'write', and long before voiced stops, e.g. 'ride'. If patients have a deficit related to the implementation of the feature voicing, then they should display impairments in both the production of vowel length preceding voiced and voiceless stop consonants as well as voice-onset time. In contrast, if they have a deficit related to particular articulatory manoeuvres, such as the timing of two independent articulators, the production of voice-onset time may be impaired while the produc-

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

tion of vowel length may be normal. Results indicate that although these patients show an impairment in the implementation of the voicing phonetic dimension via voice-onset time, they are able to maintain the distinction between voiced and voiceless stops on the basis of the duration of the preceding vowel (Duffy & Gawle, 1984; Baum *et al.* 1990). Moreover, there is no systematic relation within the same patient between the ability to realize the voicing dimension by means of voice-onset time and vowel duration (Tuller 1984). Thus, these patients do not have a disorder affecting the articulatory production of the feature voicing, but a disorder affecting particular articulatory manoeuvres, namely the timing or integration of movements of two independent articulators.

Although anterior aphasics show a disorder in temporal coordination, their disorder does not reflect a pervasive timing impairment. Fricative durations do not differ significantly from those of normals (Harmes *et al.* 1984) and the patients maintain the intrinsic duration differences characteristic of fricatives varying in place of articulation; for example, /s/ and /š/ are longer in duration than /f/ and /θ/ (Baum *et al.* 1990). English-speaking anterior aphasics maintain differences in the intrinsic durations of vowels; for example, tense vowels such as /i/ and /e/ are longer than their lax vowel equivalents, /I/ and /E/. In addition, Thai-speaking anterior aphasics maintain the contrast between short vowels and long vowels, a contrast which is phonemic in this language (Gandour & Dardarananda 1984*b*).

Finally, recent investigations of coarticulation effects in anterior aphasics show that they produce relatively normal anticipatory coarticulation. For example, in producing the syllable /s/, they anticipate the rounded vowel /u/ in the production of the preceding /s/ (Katz 1988). Nevertheless, they seem to show a delay in the time it takes to produce these effects (Ziegler & von Cramon 1985, 1986) and they may show some deficiencies in their productions (Tuller & Story 1986; but see Katz (1987) for discussion). What these results suggest is that the planning of a segment or utterance in its phonetic context seems to be intact, but it is the ultimate timing or coordination of the implementation of the articulatory movements that is impaired (see Kent & Rosenbek 1983).

Interestingly, although clearly distinguished from anterior aphasics, posterior patients also display a subtle phonetic deficit. Most typically, they show increased variability in the implementation of a number of phonetic parameters (Kent & McNeill 1987; Ryalls 1986), including vowel formant frequencies (Ryalls 1986) and vowel duration (Tuller 1984; Ryalls 1986; Gandour *et al.* 1992). Because these phonetic impairments are not clinically perceptible but emerge only upon acoustic analysis, they are considered to be subclinical. Nevertheless, they indicate that both anterior and posterior brain structures ultimately contribute to the speech production process.

On the basis of the findings just reviewed, several conclusions can be drawn concerning the nature of the phonetic disorders for anterior aphasics and their ultimate underlying mechanisms. In particular, the impairment is not a linguistic one, affecting the implementation of a particular feature. Moreover, the patients have not lost the representation for implementation nor the knowledge base for how to implement sounds in context. Rather, particular manoeuvres relating to the timing of articulators seem to be impaired, ultimately affecting the phonetic realization of some sound segments and some aspects of speech prosody while leaving others intact.

Investigations exploring the potential neuroanatomical structures contributing to the phonetic implementation of speech suggest that there are specific neuroanatomical substrates relating to such phonetic implementation patterns. Computer axial tomography (CAT) scan correlations with patterns of speech production deficits suggest the involvement of Broca's area, lower motor cortex regions for larynx, tongue, and face, and some white matter structures as well in the phonetic implementation of speech (Baum *et al.* 1990). Position emission tomography (PET) studies with normals determining regions of cerebral blood flow activity during speech production also show the importance of these areas as well as the precentral gyrus and premotor areas surrounding Broca's area (Petersen *et al.* 1989).

4. SPEECH PERCEPTION

The process of speech perception and ultimately the auditory recognition of language involves the encoding of the auditory input into a spectral representation based on the extraction of more generalized auditory patterns or properties from the acoustic waveform, the conversion of this spectral representation to a more abstract feature or phonological representation, and then the selection of a word candidate from a set of potential word candidates sharing phonological properties with the target word. Because the primary auditory pathway surfaces in Heschl's gyrus within the temporal lobe, it would not be surprising to find that the auditory association areas in the left temporal lobe were actively involved in speech reception. The classical view of the aphasias, in fact, made this claim, and attributed the language comprehension deficit of Wernicke's aphasia to impairments in the 'sound images' of words (Geschwind 1974) and/or to impairments in 'phonemic hearing' (Luria 1966). As Luria reasoned in explaining his hypothesis, if patients could not perceive phonological contrasts, then they would be unable to process words appropriately for meaning; for example, 'bee' might be misperceived as 'pea', resulting in a severe auditory comprehension disorder. Nonetheless, as we will show, speech perception deficits are not selective with respect to Wernicke's aphasia.

Similar to production studies with aphasic patients, most studies exploring the role of speech perception deficits in auditory comprehension impairments have focused on the ability of aphasic patients to perceive phonemic or segmental contrasts. Studies on segmental perception have indeed shown that aphasic patients evidence deficits in processing segmental

PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

contrasts. These studies have explored patients' abilities to discriminate pairs of words or non-words, e.g. 'pear' vs. 'bear', 'pa' vs. 'ba', or they have asked subjects to point to the appropriate word or consonant from an array of phonologically confusable pictures or nonsense syllables. Results show that nearly all aphasic patients show some problems in discriminating phonological contrasts (Blumstein *et al.* 1977*a*; Jauhiainen & Nuutila 1977; Miceli *et al.* 1978, 1980) or in labelling or identifying consonants presented in a consonant vowel context (Basso *et al.* 1977; Blumstein *et al.* 1977*b*). These problems emerge for the perception of both real words and nonsense syllables. Although there are more errors in the perception of nonsense syllables than in that of real words, the overall patterns of performance are similar, and essentially mirror the patterns found in the analysis of phonological errors in speech production. Namely, subjects are more likely to make speech perception errors when the test stimuli contrast by a single feature than when they contrast by two or more features (Blumstein *et al.* 1977*a*; Miceli *et al.* 1978; Baker *et al.* 1981). Among the various types of feature contrasts, the perception of place of articulation is particularly vulnerable (Baker *et al.* 1981; Blumstein *et al.* 1977*a*; Miceli *et al.* 1978). Interestingly, similar patterns emerge in normal subjects when perceiving speech under difficult listening conditions (see Miller & Nicely 1955).

Importantly, there does not seem to be a relationship between speech perception abilities and auditory language comprehension. Patients with good auditory comprehension skills have shown impairments in speech processing; conversely, patients with severe auditory language comprehension deficits have shown minimal speech perception deficits (Basso *et al.* 1977; Blumstein *et al.* 1977*a*; Jauhiainen & Nuutila, 1977; Miceli *et al.* 1980).

What is not clear from many of the studies exploring the perception of segmental contrasts is whether the failure to perceive such contrasts reflects an impairment in the perception of abstract phonetic features or alternatively an impairment relating to the extraction of the acoustic patterns associated with these features. To explore this issue, several studies have investigated the perception of the acoustic parameters associated with phonetic features by investigating the categorical perception of these parameters. Categorical perception relates to the way normal subjects perceive the phonetic categories of speech. In particular, continuous changes in an acoustic parameter such as voice-onset time associated with a phonetic contrast, in this case voicing, give rise to discontinuous changes in perception; when asked to label or identify the stimuli on an acoustic continuum, listeners perceive them as belonging to discrete categories corresponding to the endpoint, exemplar stimuli, and they show a sharp change in the identification of the categories usually at a particular stimulus along the continuum; when asked to discriminate the stimuli, they accurately discriminate only those stimuli which they labelled as belonging to two different categories, and fail to discriminate those stimuli which they labelled as the same phonetic category, even though all of the discrimination pairs vary along the same physical dimension.

The studies exploring categorical perception in aphasia have investigated two phonetic dimensions: voicing (Basso *et al.* 1977; Blumstein *et al.* 1977*b*; Gandour & Dardarananda 1982) and place of articulation in stop consonants (Blumstein *et al.* 1984). For voicing, the acoustic dimension varied was voice-onset time, and for place of articulation, the dimension varied was the frequency of the formant transitions appropriate for /b d g/, and the presence or absence of a burst preceding the transitions. Results showed that if aphasic patients could perform either of the two tasks (labelling or discrimination), it was the discrimination task. Most importantly, the discrimination functions were generally similar in shape and the locus of the phonetic boundary was comparable to those of normals, even in those patients who could only discriminate the stimuli.

The fact that no perceptual shifts were obtained for the discrimination and labelling functions for aphasic patients, that the discrimination functions remained stable even in those patients who could not label the stimuli, and that the patients perceive the acoustic dimensions relating to phonetic categories in a fashion similar to normals, suggest that aphasic patients do not have a deficit specific to the extraction of the spectral patterns corresponding to the phonetic categories of speech. Rather, their deficit seems to relate to the threshold of activation of the phonetic–phonological representation itself or to its ultimate contact with the lexicon. Consistent with this view are the findings that although patients may show speech perception impairments, their performance is variable; they do not show selective impairments relating to a particular phonetic feature; and the pattern of errors is bi-directional (for example, voiced consonants may be perceived as voiceless, and voiceless consonants may be perceived as voiced). Interestingly, this pattern of results mirrors those found in speech-production studies with aphasic patients.

In contrast to the segmental features of speech, the prosodic cues (i.e. intonation and stress) are consistently less affected in aphasia. Severely impaired aphasics have been shown to retain some ability to recognize and distinguish the syntactic forms of commands, yes–no questions, and information questions when marked only by intonation cues (Green & Boller 1974), even when they are unable to do so when syntactic forms are marked by lexical and syntactic cues. Nonetheless, as with intonation cues, patients' performance is not completely normal. A number of studies have revealed impairments in the comprehension of lexical or phrasal stress contrasts, e.g. 'hótdog' vs. 'hotdóg' (Baum *et al.* 1982; Emmorey 1987), as well as sentential contrasts, e.g. 'he fed her dog bíscuits' vs. 'he fed her dóg biscuits' (Baum *et al.* 1982). Similar findings emerged for the perception of tone contrasts serving as lexical cues in Thai (Gandour & Dardarananda 1983) and Chinese

(Naeser & Chan 1980). Importantly, no differences have emerged in any studies between the performance of anterior and posterior aphasics, a finding consistent with the results for the perception of phonemic contrasts.

Although the results of speech perception experiments suggest that aphasic patients do not seem to have a selective impairment in processing segmental structure underlying the auditory properties of speech, several recent studies have suggested that aphasic patients display impairments in the intersection of the sound properties of speech with lexical access. These studies have shown some interesting dichotomies in the performance of Broca's and Wernicke's aphasics which suggest that patients' impairments do not reflect speech perception deficits *per se* but rather deficits in the interaction of sound structure as it contacts the lexicon (see also Martin *et al.* 1975; Baker *et al.* 1981).

Milberg *et al.* (1988) explored the extent to which phonological distortions affect semantic facilitation in a lexical decision task. They investigated whether a phonologically distorted prime word such as 'gat' or 'wat' would affect the amount of semantic facilitation for target words semantically related to the undistorted prime, e.g. 'cat'. Subjects were asked to make a lexical decision on the second item of stimulus pairs in which the first stimulus was semantically related to the second (e.g. 'cat–dog'), or alternatively, it was systematically changed by one or more features (e.g. 'gat–dog', 'wat–dog'). Fluent patients showed priming in all phonologically distorted conditions (e.g. 'gat–dog', 'wat–dog'), suggesting a reducing threshold for lexical access. In contrast, anterior aphasics showed priming only in the undistorted semantically related condition (e.g. 'cat–dog'), suggesting an increased threshold for lexical access. These results suggest that impairments in the use of phonological information to access the lexicon can manifest themselves in different ways in aphasic patients in the absence of a deficit in processing the phonological properties of speech themselves.

Similarly, the lexical status of a word affects differentially how aphasic patients perform phonetic categorization. Normal subjects typically show a lexical effect. That is, the locus of a phonetic boundary in an acoustic continuum such as voice-onset time (VOT) changes as a function of the lexical status of the endpoint stimuli. When the endpoint /d/ stimulus is a word, e.g. 'dash', and the endpoint /t/ stimulus is a non-word, e.g. 'tash', there are more *d* responses along the continuum; in contrast, when the endpoint /t/ stimulus is a word, e.g. 'task', and the endpoint /d/ stimulus is a non-word, e.g. 'dask', there are more *t* responses along the continuum (Ganong 1980). Broca's aphasics show a larger than normal lexical effect, placing a greater reliance on the lexical status of the stimulus in making their phonetic decisions than on the perceptual information in the stimulus. In contrast, Wernicke's aphasics do not show a lexical effect at all, suggesting that lexical information does not influence phonetic categorization, and perhaps such top-down processing may even fail to guide their language performance (Blumstein *et al.* 1994).

Overall, the findings from speech perception studies of aphasic patients suggest that the neural basis for speech reception is neurally complex, and includes far greater neural involvement than simply the primary auditory areas and auditory association areas in the temporal lobe. Although the number of neurophysiological and electrophysiological studies focusing particularly on speech reception are few, these results provide converging evidence consistent with this view. PET studies have shown that the primary auditory cortex is activated in the processing of simple auditory stimuli (Lauter *et al.* 1985) and that both the primary auditory cortex and the superior temporal gyrus are activated in passive word recognition (Petersen *et al.* 1988, 1989). These results are of no surprise. What is of interest, however, is that other cortical areas seem to be activated as well. For example, Zatorre *et al.* (1992) showed increased activity in Broca's area near the junction with premotor cortex as well as the superior parietal area near the supramarginal gyrus when subjects were required to make a phonetic decision or phonetic judgment about auditorily presented consonant-vowel-consonant (CVC) stimuli. Further, Ojemann (1983) has shown impairments in the ability of patients to identify auditorily presented sound segments embedded in a phonetic context /a_ma/ during electrical stimulation to a wide range of cortical areas including the inferior frontal cortex.

The exact role of these anterior areas in speech reception is not clear. However, these areas seems to be a part of a single neural system. Research with monkeys is consistent with this view. Auditory stimulation studies have shown direct ipsilateral projections to prefrontal regions, and ablation studies of these areas have shown diminished auditory discrimination skills (Pandya *et al.* 1969).

Further research will be required to determine the neural substrates of speech perception and auditory language reception. What is clear, however, is that the classic view of both speech perception and speech production deficits, their underlying bases as well as their neural substrates, may be incorrect, and that the two systems are complex probably requiring a distributed network with the broad involvement of the left cerebral hemisphere.

This research was supported in part by NIH Grant DC00314 to Brown University.

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