ON THE NEUROBIOLOGY OF THE SOUND STRUCTURE OF LANGUAGE: EVIDENCE FROM APHASIA

S.E. Blumstein

Brown University, Providence, RI 02912, USA

ABSTRACT

The patterns of both speech production and speech perception deficits in aphasia suggest that the disorders reflect impairments to the processes involved in accessing the sound structure rather than selective impairments to the sound properties of speech or to their representations. Speech production deficits occur at both the phonological level, reflecting selection or access impairments, as well as at the phonetic level, reflecting articulatory implementation impairments. Phonological deficits emerge regardless of lesion site, whereas phonetic deficits emerge with damage to specific neuroanatomical structures. Speech perception impairments reflect misperceptions of phonetic features rather than deficits in extracting the acoustic patterns associated with these features. Such impairments emerge particularly as the sound properties of speech contact the lexicon.

One of the most challenging issues in the study of the neurobiology of speech is understanding the neural basis of speech production and speech perception mechanisms. This domain of inquiry has largely focused on investigations of adult aphasic patients, exploring the clinical and behavioral manifestations of their disorder and the accompanying lesion localization. Just as the study of the sound structure of language has been guided by considerations of the structural properties of the speech-language system in normals, both phonological and phonetic, so has the study of speech production and speech perception deficits in aphasia. Two major questions have shaped research in the field. The first centers on whether speech production and speech perception deficits reflect selective impairments to the sound properties of speech and their representations, or alternatively, impairments to the processes involved in accessing these representations. The second centers on whether speech production and speech perception impairments reflect deficits that are primarily phonological in nature, affecting the structural properties of language, or whether they are phonetic in nature, affecting, on the one hand, articulatory implementation in speech production, or on the other hand, acoustic decoding in speech perception. It is these two issues which are the primary focus of this paper.

SPEECH PRODUCTION

In order to produce a word or an utterance, the speaker must select the word candidate(s) from the lexicon including its phonological form (selection), and then encode the abstract phonological representation of the word in terms of the articulatory parameters required for realizing the phonological properties in the particular context in which they appear (articulatory planning). Subsequent to the selection of a lexical candidate or candidates and the articulatory planning of the utterance, the phonetic string is ultimately converted into a set of motor commands or motor programs to the articulatory system. This set of 'instructions' to the articulators relates to the phonetic implementation of speech. The final speech output must conform to the phonological rules of the language including the correct production of the sound segments in their phonetic environment, the appropriate stress pattern of the word, and in larger contexts the appropriate prosodic structure of the utterance including both stress and intonation.

Linguistic theory makes a distinction between phonology and phonetics, and the facts of aphasia support such a distinction. In some cases, patients may produce a wrong sound segment, but its phonetic implementation is correct, i.e. for 'teams' the patient says 'keams'. In other cases, patients may produce the correct sound segment but its phonetic implementation is distorted, i.e. for 'teams' the patient produces an initial /t/ that is overly aspirated.

Nearly all aphasic patients, regardless of the aphasia syndrome and underlying neuropathology, display speech production impairments that implicate a deficit at the phonological level. The patterns of impairment are similar across patients, suggesting that a common mechanism is impaired. The mechanism relating to this phonological impairment most likely relates to the selection and/or organization of the features comprising the candidate lexical entries.

The evidence for this comes from investigations of the patterns of speech production errors produced by aphasic patients [1]. For example, most sound substitution errors, e.g. 'teams' -> 'keams', involve a change in value of a single phonetic feature. This pattern of errors is consistent with the view that the incorrect phonetic feature has been selected or activated, but has been correctly implemented by the articulatory system. It also supports the view that phonetic features are organized in terms of tiers. Tiers have been defined in phonological theory to reflect the fact that phonetic features correspond to independent articulatory gestures (and consequently acoustic events) such as tongue placement and movement, lip movement, laryngeal activity, and height of the velum. Phoneme substitution errors in aphasia rarely involve more than one tier at a time, with feature changes typically relating to place of articulation, e.g. 'teams' -> 'keams', voicing, e.g. 'toy' -> 'doy', nasality, e.g. 'nut' -> 'dut', and manner of articulation, e.g. 'sun' -> 'tun' [2].

Phonological errors also suggest that the nature of the syllable structure of the lexical candidate constrains the type and extent of errors made during the selection process [1,2]. Phoneme substitution errors are more likely to occur in singleton consonants than in clusters, e.g. [f] is more likely to undergo a phoneme substitution error in the word 'feet' than in 'fleet'. Simplification and addition errors are more likely to result in the canonical syllable structure, CV, e.g. a consonant is more likely to be deleted in a cluster, 'sky' -> 'ky', and is 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, e.g. 'history books' -> 'bistory books' and 'roast beef' -> 'roaf beef'. These results show that the syllable structure of a word is part of its lexical representation, 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.

While phonological patterns are similar across aphasic patients, phonetic deficits seem to be more selective. A long-held observation is that aphasic patients with anterior brain-damage produce phonetic errors. The implied basis for these errors is one of articulatory implementation: that is, commands to the articulators to encode words 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. These studies have shown that anterior patients have difficulty producing phonetic dimensions that require the timing of two independent articulators, e.g. voicing (i.e. voice-onset time) and nasality (i.e. the timing of the release of the closure in the oral cavity and the velum opening) [3,4,5,6]. In particular, there is considerable overlap between the target productions in the area of the phonetic boundary. Similar patterns emerge across different languages, occurring 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.

That the phonetic output disorder of these patients likely reflects an articulatory implementation deficit rather than a failure to encode appropriately the phonetic feature such as voicing comes from acoustic analyses of the production of vowel length as a cue to final stop consonant voicing. Results indicate that while anterior aphasic patients show an impairment in the implementation of the phonetic dimension of voicing using voice-onset time, they maintain the distinction between voiced and voiceless stops on the basis of the duration of the preceding vowel [7,8].

Kent and Rosenbek [9] have suggested that the timing problem found for individual segments and their underlying features is a manifestation of a broader impairment in the integration of articulatory movements from one phonetic segment to another. Nonetheless, investigations of coarticulation effects in anterior aphasics show that they produce relatively normal anticipatory coarticulation [10]. For example, in producing the syllable [su], anterior aphasics anticipate the rounded vowel [u] in the production of the preceding [s]. Nonetheless, they may show a delay in the time it takes to produce these effects, and they may show some deficiencies in their productions [11, 12]. These results suggest that phonological planning is relatively intact, but the timing or coordination of the implementation of the articulatory movements is impaired.

While still premature, results exploring the neuroanatomical basis of these phonetic patterns of speech suggest the involvement of specific neuroanatomical substrates. These areas include Broca's area, the lower motor cortex regions for larynx, tongue, and face, and some white matter structures as well [7].

Several conclusions can be made concerning the nature of the phonetic disorders and their ultimate underlying mechanisms. The impairment is selective for patients with specific underlying neuropathology. The deficit is not a linguistic one affecting the implementation of a particular phonetic feature. Moreover, the patients have not lost the representation for implementation nor the knowledge base for how to implement sounds in context, but rather particular maneuvers relating to timing of articulators seem to be impaired.

Interestingly, posterior patients display a subtle phonetic deficit showing increased variability in the implementation of a number of phonetic parameters including vowel formant frequencies [13] and vowel duration [12, 13, 14]. Because these phonetic impairments are not clinically perceptible but emerge only upon acoustic analysis, they are considered to be subclinical. These results suggest that both anterior and posterior brain structures ultimately contribute to the speech production process.

SPEECH PERCEPTION

Current views of auditory language comprehension and specifically the auditory perception of words suggest that contact with the lexicon (and ultimately meaning) requires the encoding of the auditory input into a spectral representation based on the extraction of more generalized auditory patterns of properties from the acoustic waveform, the conversion of this spectral representation to a more abstract feature/phonological representation, and then the selection of a word candidate from a set of potential word candidates sharing phonological properties with the target word.

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. Results show that nearly all aphasic patients show some problems in discriminating phonological contrasts [15, 16, 17] or in labeling consonants presented in a consonant-vowel context [18, 19]. The overall patterns of performance are similar across patients and essentially mirror the patterns found in the analysis of phonological errors in speech production. Namely, subjects are more likely to make perception errors when the test stimuli contrast by a single phonetic feature than when they contrast by two or more features, and the perception of place of articulation is particularly vulnerable [15, 17, 20].

What is not clear from these studies is whether the failure to perceive segmental contrasts reflects an impairment in the perception of phonetic features or alternatively an impairment relating to the extraction of the acoustic patterns associated with these features. To investigate this issue, several studies have explored categorical perception (both labeling and discrimination) of the acoustic parameters associated with voicing [18, 19, 20,21] and place of articulation in stop consonants [22]. Results showed that if aphasic patients could successfully complete one of the two labeling or discrimination tasks, it was discrimination. Most importantly, the shape of the discrimination functions and the locus of the phonetic boundary were 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 labeling functions compared to normals, and that the discrimination functions remained stable even in those patients who could not label the stimuli suggests that aphasic patients do not have a deficit specific to the extraction of the acoustic 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.

Several recent studies have suggested that speech perception problems manifest themselves most strongly when the sound properties of speech contact the lexicon. For example, nonwords, e.g. 'gat', phonologically related to real words, e.g. 'cat', do not seem to access the lexicon in Broca's aphasics as they do in normals [23]. In contrast, for Wernicke's aphasics, such nonwords seem to activate the lexicon more so than they do in normals. Similarly, the lexical status of a word affects differentially how aphasic patients perform phonetic categorization. Broca's aphasics show a larger than normal lexical effect, seeming to place 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, suggesting that top-down information does not influence phonetic categorization, and may even fail to guide their language performance [24].

Overall, the findings from speech perception studies of aphasic patients suggest that the neural basis for speech reception is broadly represented, and includes far greater neural involvement than the primary auditory areas and auditory association areas in the temporal lobe. In fact, anterior as well as posterior brain structures are implicated in the auditory processing of speech. Although the number of neurophysiological and electrophysiological studies focusing particularly on speech reception are few, they provide converging evidence consistent with this view [25, 26, 27, 28, 29].

ACKNOWLEDGMENTS

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

REFERENCES

[1] Blumstein, S.E. (1973), *A Phonological Investigation of Aphasic Speech*, The Hague: Mouton.

[2] Blumstein, S.E. (1990), Phonological deficits in aphasia: Theoretical perspectives, In A. Caramazza (Ed.), *Neurolinguistics: Advances in Models of Cognitive Function andCognitive Neuropsychology and Impairment*, Hillsdale: Lawrence Erlbaum.

[3] Blumstein, S.E., Cooper, W.E., Goodglass, H., Statlender, S., and Gottlieb, J. (1980), Production deficits in aphasia: A voice-onset time analysis, *Brain and Language, 9*, 153-170.

[4] Gandour, J. and Dardarananda, R. (1984), Voice-onset time in aphasia: Thai, II: Production, *Brain and Language, 18*, 389-410.

[5] Itoh, M., Sasanuma, S., Tatsumi, I., Murakami, S., Fukusako, Y., and Suzuki, T. (1982), Voice onset time characteristics

in apraxia of speech, *Brain and Language, 17,* 193-210.
[6] Shewan, C.M., Leeper, H., and Booth, J. (1984), An analysis of voice onset time (VOT) in aphasic and normal subjects, In J. Rosenbek, M. McNeill, and A. Aronson (Eds.), *Apraxia of Speech,* San Diego, Ca.:College-Hill Press.
[7] Baum, S.R., Blumstein, S.E., Naeser, M.A., and Palumbo, C.L. (1990), Temporal dimensions of consonant and vowel production: An acoustic and CT scan analysis of aphasic speech, *Brain and Language, 39,* 33-56.
[8] Duffy, J. and Gawle, C. (1984), Apraxic speakers' vowel duration in consonant-vowel-consonant syllables, In J. Rosenbek, M. McNeil, and A. Aronson (Eds.), *Apraxia of Speech,* San Diego, Ca.: College-Hill Press.
[9] Kent, R. and Rosenbek, J. (1983), Acoustic patterns of apraxia of speech, *Journal of Speech and Hearing Research, 26,* 231-248.
[10] Katz, W.F. (1988), Anticipatory coarticulation in aphasia: Acoustic and perceptual data, *Brain and Language, 35,* 340-368.
[11] Ziegler, W., and von Cramon, D. (1985), Anticipatory coarticulation in a patient with apraxia of speech, *Brain and Language, 26,* 117-130.
[12] Tuller, B. (1984), On categorizing aphasic speech errors. *Neuropsychologia, 22,* 547-557.
[13] Ryalls, J. (1986), An acoustic study of vowel production in aphasia, *Brain and Language, 29,* 48-67.
[14] Gandour, J., Ponglorpisit, S., Khunadorn, F., Dechongkit, S., Boongird, P., and Boonklam, R. (1992), Timing characteristics of speech after brain damage: Vowel length in Thai, *Brain and Language, 42,* 337-345.
[15] Blumstein, S.E., Baker, E., and Goodglass, H. (1977), Phonological factors in auditory comprehension in aphasia, *Neuropsychologia, 15,* 19-30.
[16] Jauhiainen, T., and Nuutila, A. (1977), Auditory perception of speech and speech sounds in recent and recovered aphasia, *Brain and Language, 4,* 572-579.
[17] Miceli, G., Caltagirone, C., Gainotti, G., and Payer-Rigo, P. (1978), Discrimination of voice versus place contrasts in aphasia, *Brain and Language, 2,* 434-450.
[18] Basso, A., Casati, G., and Vignolo, L.A. (1977), Phonemic identification defects in aphasia, *Cortex, 13,* 84-95.
[19] Blumstein, S.E., Cooper, W.E., Zurif, E.B., and Caramazza, A. (1977), The perception and production of voice-onset time in aphasia, *Neuropsychologia, 15,* 371-383.
[20] Baker, E., Blumstein, S.E., and Goodglass, H. (1981), Interaction between phonological and semantic factors in auditory comprehension, *Neuropsychologia, 19,* 1-16.
[21] Gandour, J. and Dardarananda, R. (1982), Voice onset time in aphasia: Thai, I. Perception, *Brain and Language, 17,* 24-33.
[22] Blumstein, S.E., Tartter, V.C., Nigro, G., and Statlender, S. (1984), Acoustic cues for the perception of place of articulation in aphasia, *Brain and Language, 22,* 128-149.
[23] Milberg, W., Blumstein, S.E., and Dworetzky, B. (1988), Phonological processing and lexical access in aphasia, *Brain and Language, 34,* 279-293.
[24] Blumstein, S.E., Burton, M., Baum, S., Waldstein, R., and Katz, D. (1993), The role of lexical status on the phonetic categorization of speech in aphasia, *Brain and Language, 46,* 181-197.
[25] Lauter, J., Herscovitch, P., Formby, C., and Raichle, M.E. (1985), Tonotopic organization in human auditory cortex revealed by positron emission tomography, *Hearing Research, 20,* 199-205. 1985.
[26] Petersen, S.E., Fox, P.T., Posner, M.I., Mintun, M., and Raichle, M.E. (1988), Positron emission tomographic studies of the cortical anatomy of single-word processing, *Nature, 331,* 585-589.
[27] Petersen, S.E., Fox, P.T., Posner, M.I., Mintun, M., and Raichle, M.E. (1989), Positron emission tomographic studies of the processing of single words, *Journal of Cognitive Neuroscience, 1,* 153-170.
[28] Zatorre, R.J., Evans, A.C., Meyer, E., and Gjedde, A. (1992), Lateralization of phonetic and pitch discrimination in speech processing, *Science, 256,* 846-849.

[29] Ojemann, G.A. (1983), Brain organization for language from the perspective of electrical stimulation mapping, *Behavioral and Brain Sciences, 6,* 189-230.