≪Preliminary Reports≫

Phonological Deficits in Aphasia Syndromes : A Neurolinguistic Approach

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Abstract : The purpose of the present study was to explore the notion that there are differential sources of phonological breakdown among the major aphasia syndromes. On the basis of a review of the relevant literature, the following neurolinguistic hypotheses were generated. Wernicke's aphasics have a deficit in accessing complete phonological representations. Conduction aphasics have a deficit in constructing phonemic strings. Broca's aphasics have a deficit in phonetically recording phonemic strings.

Key words : Phonological breakdown, aphasia syndromes, neurolinguistic hypotheses

Introduction

Because virtually all aphasics produce phonological errors in their speech, and the clinical impression is that aphasia syndromes differ with respect to the cause of such errors, a central issue in aphasia research has been to characterize the nature of phonological breakdown across the major aphasia syndromes. Various methods of investigation have been used, with mixed results. For example, phonological analyses of spontaneous speech have found considerable homogeneity across the major aphasia syndromes. By contrast, acoustical analyses of oral reading have found marked differences in the phonological errors of these syndromes.

Discrepancies in the results of phonological studies on aphasia can often be traced to differences in methods of data elicitation and analysis. In fact, it will be argued that a consistent picture emerges when such methodological differences are acknowledged. Specifically, one finds tentative support for the following hypotheses concerning phonological breakdown in the speech of the major aphasia syndromes :

1. Wernicke's aphasics have difficulty at the level of accessing complete phonological representations.
2. Conduction aphasics have difficulty at the level of developing phonemic strings.
3. Broca's aphasics have difficulty at the level of developing a phonetic recording of phonemic strings.

The goal of the present study is to explore these tentative hypotheses about phonological breakdown in aphasia. Accordingly, the first section will involve an examination of past research on phonological production in aphasia, organized in a way that ultimately argues for the method of phonological elicitation employed in the present study. The next section will develop a model of sound production for single words. This model will be employed to generate a set of analyses to identify signs of phonological breakdown at each of the three levels specified in the single word sound production model.

Neurolinguistic Studies in Phonological Breakdown

Typically, aberrant phonological behavior in aphasia has been studied examining phonemic (or lit-

eral) paraphasias. It is difficult to strictly define such speech errors. However, the general consensus is that a phonemic paraphasia phonologically resembles the target to the extent that the error (e.g., "tsutsuri" for /kusuri/). Moreover, phonemic paraphasias are typically studied as isolated events via a phonemic comparison of each paraphasia with its intended target word .

Phonological Analyses

Phonological analyses of spontaneous speech have revealed a great similarity among the errors of the major aphasia syndromes. Blumstein (1973) found that the phonemic paraphasias in the spontaneous speech of a group of Broca's, Wernicke's, and conduction aphasics were qualitatively indistinguishable in terms of such properties as phoneme substitution and addition. For example, for all three subgroups, phoneme substitutions were the most prevent type of phonemic distortion. Moreover, for all three subgroups, these errors generally differed from the target phonemes by one feature, and occurred more often with single consonants than with clusters. The distribution of Blumstein's three remaining types of phonemic errors was also similar for the three aphasia subgroups : simplification (omission) errors occurred most often, environment errors (substitutions influenced by surrounding phonemes) were less frequent, and addition errors were the least frequent. Moreover, across the three subgroups, most simplifications and additions involved clusters as opposed to single consonants. (**Fig.1**)

Three results were somewhat surprising, insofar as it is generally held that the underlying deficit responsible for phonological error production differs among these subgroups. Blumstein confronts this issue by positing that her data reflect general phonological principles that constrain patterns of phonological breakdown at various levels of sound production. Nevertheless, she leaves open the possibility that phonological errors in aphasia could be caused

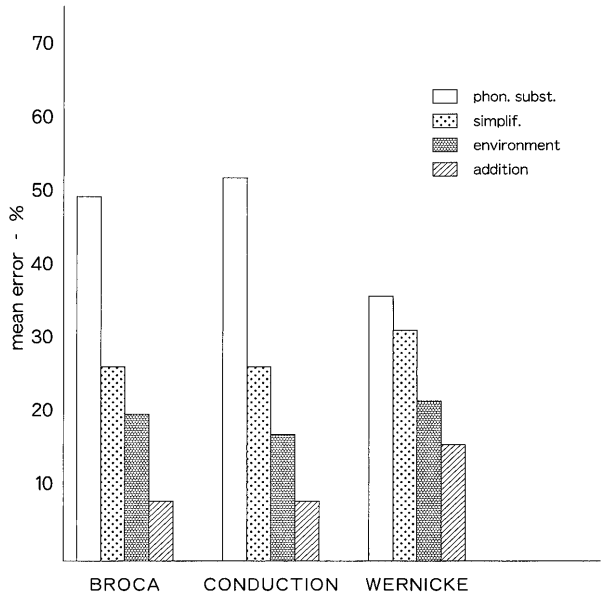

Fig.1. Distribution of aphasic errors (from Blumstein, 1973)
Error categories :
phon.subst. ; the substitution of one phoneme for another
simplif. ; the loss of a phoneme or syllable in a word
environment ; phoneme substitution which can be accounted for by the influence of other surrounding phonemes
addition ; the addition of a phoneme or a syllable in a word

by different types of deficits.

However, it still could be the case that the way in which the Blumstein data were collected and analyzed created a misleading degree of homogeneity. For example, a strict phonemic analysis of individual speech errors obscures possible phonetic level differences. Such errors as the lack of aspiration for initial stop consonants are missed. In fact, it will be seen shortly that important differences among aphasia subgroups exist at the allophonic, or phonetic, level.

Phonological analyses of spontaneous speech have several other possible drawbacks. For example, it is often difficult to identify the targets of phonemic paraphasias within free speech, because the utterances that contain such speech errors are experimentally unconstrained. This may be particularly important for examining Wernicke's aphasics. These apha-

sics are known to produce such phonologically distorted utterances that the intended targets are unclear (i.e. neologism). If targets are not provided for some aphasics, analyzable phonemic errors could be omitted from a phonological error analysis. Moreover, one might be omitting that portion of the data that has the most power to distinguish Wernicke's aphasics from other types of aphasics.

Voice onset time Analyses

Subsequent work by Blumstein et al. (1977, 1980) has attempted to control for more of the variables that appear to be important for a phonological investigation of aphasia. Oral reading was used to elicit production, when oral reading was impaired, repetition was used. In this study, clear targets were provided, and phonological processing was not influenced by phrase level processing. In addition, two types of errors were distinguished. Phonemic and phonetic errors were identified on the basis of criteria defined by normal patterns of voice onset time (VOT) : phonetic errors were vocalizations that fell within the normal VOT range for nontarget phonemes. These criteria were used to determine when a phonological error reflected a phoneme substitution or a violation of acoustic-phonetic boundaries between voiced and unvoiced phoneme.

The findings of Blumstein et al. (1980) can be summarized as follows : the Wernicke's aphasics produced a few phonemic and phonetic errors, Broca's aphasics produced a large number of errors that were predominantly phonetic, and the conduction aphasics fell in between. The result of this study indicated that all groups of aphasics evidence some deviations in timing of articulatory movements. These deficits occur in varying degree and, in general, correspond to clinical observations. In particular, Broca's aphasics have a severe output disorder, conduction aphasics have a moderate disorder, and Wernicke's aphasics are minimally impaired. However, the results of this study also suggested that the dichotomy between phonetic and phonemic disintegration ascribed to the anterior and posterior patients respectively is not so easily drawn.

With this orientation toward the Blumstein et al.. (1980) VOT data, several useful conclusions can be drawn. Firstly, because the Wernicke's aphasics produced few errors on the VOT analysis of reading or repetition., produce many phonological errors on less constrained tasks, such as picture naming, these aphasics appear to have a pre-phonetic deficit. By contrast, the relatively poor performance of the Broca's aphasics demonstrates a deficit at a later stage of sound production. Moreover, because the process of VOT involves the temporal programming of speech sounds, such as the timing of the release of a stop consonant with respect to the onset of glottal pulsing, these result suggest that the sound production deficit of Broca's aphasia involves a difficulty in integrating articulatory movements.

The notion that the articulatory deficit of Broca's aphasia is language-based, or represents a phonetic deficit, is supported by other Blumstein et al. (1980) data. They also performed a VOT analysis of a non-aphasic dysarthric patient, and found that the pattern of VOT productions differed qualitatively from that of the Broca's aphasics. Such data substantiates the clinical impression that there is a distinction between a low-level muscular deficit in articulatory programming (e.g., dysarthria) and a language-based deficit in articulatory programming (e.g., the phonetic disturbance in Broca's aphasia).

The nature of the phonetic production deficit in Broca's aphasia is further clarified by Shinn and Blumstein (1983). Specifically, they found that Broca's aphasics are relatively accurate in reaching the target configuration for a particular place of articulation. This was demonstrated in the following way. A spectral analysis was performed of the labial, alveolar, and velar places of articulation using templates developed from normal speech for the onset

spectrum of each place of articulation. Thus, the report of intact configurations for place of articulation, abnormal VOT's in Broca's aphasia suggests that the phonetic deficit of these aphasics involves the timing between articulatory configurations, not the programming of the articulatory configurations, themselves.

Itoh et al. (1982) have made similar discoveries about VOT production in aphasia as Blumstein et al. However, these purported similar require explication. Itoh et al. (1982) claim that the VOT characteristics of fluent aphasic speech (i.e., the speech of Wernicke's, and conduction aphasics) reflect intact articulatory programming, but impaired phonological processing (phoneme selection or retrieval). Thus, they consider the VOT production of conduction aphasics to be similar to that of Wernicke's aphasics, while Blumstein et al. (1980) consider the VOT production of conduction aphasics to be intermediate to Wernicke's and Broca's aphasics.

Various techniques for examining articulatory process indicate that a deficit in articulatory programming contributes to the phonological breakdown in Broca's aphasia. However, these techniques appear to be unable to identify earlier stage of phonological breakdown. Consequently, these studies do not help characterize the source of phonological breakdown in conduction and Wernicke's aphasia, in both cases, it is prior to the phonetic level.

Breakdown in Conduction Aphasia

Nespoulous et al. (1987) have reported differences between the single word repetition of Broca's and conduction aphasics. Conduction aphasics produced more contextually determined errors (e.g., metatheses), while Broca's aphasics produced substitutions that tended to be phonetically closer to the target. Moreover, the errors of Broca's aphasics displayed a preferential error pattern (e.g., a tendency to devoice voiced phonemes), while those of the conduction aphasics did not. These distinct error pattern were used to argue that the speech production of Broca's aphasics tends to break down at the feature level, while the speech production of conduction aphasics tends to break down at the level of organizing phonemic strings.

Nespoulous et al. (1987)'s notion that conduction aphasia involves a deficit in developing phonemic strings is also consistent with the results of other phonological studies involving this syndrome. Firstly, this interpretation is consistent with such studies as Yamadori and Ikumura (1975), and Kohn (1984), which locate the source of phonological production errors in conduction aphasia at a pre-articulatory stage of sound production. Secondly, other studies indicate the preservation of other aspects of phonological production in conduction aphasia. As discussed earlier, conduction aphasics appear to be relatively intact on several measures of articulatory programming. Moreover, preserved ability to access abstract phonological information is suggested by the frequency of tip-of-the-tongue (TOT) experiences.

Breakdown in Wernicke's Aphasia

The source of phonological breakdown in Wernicke's aphasia has received less attention. Nevertheless, a tentative hypothesis can be developed. Intact co-articulation points to a deficit prior to the phonetic level. The greater number of errors involving phoneme addition and complex substitution relative to conduction aphasics points to a more abstract phonological deficit than that of the conduction aphasics, perhaps at the level of accessing a complete phonological representation of a word. This hypothesis is further suggested by their tendency to produce neologisms, and poor phonemic cuing relative to other aphasics.

A recent examination of the picture naming of one Wernicke's aphasic supports the present hypothesis of the nature of the phonological deficit in Wernicke's aphasia. Miller and Ellis (1987) compared picture naming errors involving phoneme transposition and substitution to artificially generated pseudo-

transpositions and pseudo-substitutions, and found no significant difference. These results were taken to indicate that the phonological errors of their patient were randomly generated, and that such randomness reflected a deficit in lexical access.

A Neurolinguistic Model of Single Word Production

Most neurolinguistic models of sound production are incomplete, because they concentrate on particular aspects of sound production, and describe particular process in terms that are too vague to either prove or disprove. Nevertheless, a schematic model can be synthesized from the relevant literature that specifies enough detail to test the proposed source of phonological disruption in the three major aphasia syndromes. The model will attempt to elucidate three basic, successive stages :

1) the accessing of abstract phonological information, in the form of planning frame with slots coded for syllable number, CV structure, and reduced phonemic information in the form of non-redundant features
2) the construction of a phonemic string, by filling in redundant features, guided by the planning frame
3) the integration of allophonic information, in preparation for motor encoding

Because the present model is tailored to an investigation of phonological picture naming errors, its scope can best be described as a "single word sound production model". It is developed at the "single word" level, in order to examine phonological production stripped of the influences of phrase level semantic and syntactic processing. Nevertheless, it is intended that this model can be integrated into a broader-based language model.

The Model

The model is limited to phonological aspects of word production. Hence, it will pick up the process of word production after the permanent phonological representation of the target has been located within the "mental lexicon" (i.e., a permanent store of semantic and phonological information). It comprises three basic stages :

1) Morphophonemic : the accessing of abstract phonological information, in the form of a planning frame with slots code for syllable number, CV structure, and reduced phonemic information in the form of non-redundant features
2) Phonemic : the construction of a phonemic strings, by filling in redundant features, guided by the planning frame
3) Phonetic : the integration of allophonic information, in preparation for motor encoding

The first stage will be referred to as the "morphophonemic" stage, and the planning frame employed at this stage will be referred to as a "morphophonemic" frame, or representation. This terminology was chosen because in phonological theory, "morphophonemic" refers to "that component in linguistic description that relates morphemes and phonemes, and this term is employed in the present study at an analogous point. The second stage will be referred to as the "phonemic" stage, and the third stage will be referred to as the "phonetic" stage. These latter terms are employed in the present model in a way that is analogous to standard linguistic usage.

Phonological Breakdown as Viewed by the Model

On the basis of the present model for single word sound production, preliminary predictions can be made about the stage that is most likely damaged in Broca's, Wernicke's, and conduction aphasia. Most of the relevant data have already been discussed.

Wernicke's aphasics appear to have a stage 1 deficit, involving impaired ability to access morpho-

phonemic frames. Impaired phonological access is suggested by the following characteristics of Wernicke's aphasics : a) the tendency to produce neologisms, b) poor phonemic cuing, c) the random nature of their phoneme substitution and transposition errors. By contrast, the rarity of VOT errors during simple reading or repetition tasks indicates the preservation of phonetic processes. This scenario is consistent with an examination of velar movements in Wernicke's aphasia, insofar as these aphasics displayed normal patterns of anticipatory co-articulation (i.e., preserved stage 3), and errors that reflected impairment at the level of selecting target phonemes.

Conduction aphasics appear to possess a sound production deficit that is primarily at stage 2. This is the stage that is comparable to that identified by Kohn (1984) as the source of their phonological breakdown (i.e., pre-articulatory). The often cleanly articulated, yet halting spontaneous speech of these aphasics suggests difficulty organizing phonemic level information. A deficit in constructing phonemic strings is also consistent with their tendency to produce : a) more word fragments than other aphasics, b) more contextually determined phonemic errors (e.g., metatheses) on repetition tasks than Broca's aphasics. Relatively intact stage 1 processes are suggested by their good TOT performance and phonemic cuing. Relatively intact stage 3 processes are suggested by their relatively normal VOT productions.

Broca's aphasics seem to processes a stage 3 deficit. It is this level that best captures the "phonetic disintegration" that is characteristic of these aphasics. The abnormal VOT's of Broca's aphasics suggests difficulty at the allophonic level . By contrast, the relative preservation of stage 1 is suggested by : a) their relatively intact phonemic cuing, and b) their rarity of phonological errors in identifying picture homonyms that they cannot articulate. The relative preservation of place of articulation for Broca's aphasics also suggests a well conceived ordering of segments (i.e., the relative preservation of stage 2).

Finally, the above association of each aphasia subgroup with breakdown at a particular stage of sound production has obvious implications for the present study. To verify the present hypotheses of phonological breakdown in aphasia, one needs to find a predominance of : a) stage 1 type errors in the picture naming performance of the Wernicke's aphasics, b) stage 2 type errors for the conduction aphasics, and c) stage 3 type errors for the Broca's aphasics. If these predictions hold, one has a set of feature that can be employed clinically to help identify different sources of phonological breakdown in aphasia.

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