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ON CATEGORIZING APHASIC SPEECH ERRORS

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Abstract—Acoustic studies of voice-onset-time in aphasics' speech suggest that fluent aphasics' errors are misselected phonemic targets whereas nonfluent aphasics' errors are of articulatory origin. However, we must be cautious when extrapolating a theory from only one measure of articulation. In this experiment, I examined utterances produced by five fluent aphasics, five nonfluent aphasics and two controls. First, the voice-onset-time findings were replicated. Second, I examined the duration of vowels preceding word-final stop consonants as an index of the consonant's voicing category. The pattern of voice-onset-times produced did not predict the pattern of vowel durations. Thus, voice-onset-time cannot be used to characterize more generally the output of the speaker.

INTRODUCTION

TRADITIONAL clinical descriptions of aphasia consider the errors in speech produced by posterior, fluent aphasics to originate at the phonemic or phonological planning levels, whereas phonetic or articulatory errors are thought to be more typical of anterior, nonfluent aphasia [1, 17, 21]. Though it is often difficult to disambiguate so-called planning and execution deficits (or phonemic and phonetic deficits), a fine-grained acoustic analysis has great potential for describing the nature of the underlying speech disorder.

Segmental analyses of aphasic speech have typically proceeded by examining one parameter of the acoustic complex that signals a shift in one phonetic dimension. A commonly used measure is voice-onset-time (VOT), a parameter that distinguishes voiced from voiceless stop consonants in syllable-initial position [e.g. 2, 3, 7, 9, 12, but see 22, for an analysis of place of articulation errors]. VOT is the acoustic representation of the time between the burst at release of supraglottal occlusion and the onset of glottal pulsing. For voiced stop consonants in syllable-initial position, glottal pulsing might begin before the release burst, or lag as much as 25 msec after release. In voiceless stop consonants, the onset of glottal pulsing might lag behind supraglottal release by approx. 35–80 msec [15, 16]. Although in normal English speakers the actual VOT values vary somewhat as a function of, for example, place of articulation, speaking rate and phonetic context, the distribution of VOT values for voiced and voiceless cognates is bimodal and more or less nonoverlapping. It is often assumed that the discrete acoustic categories reflect the linguistic intuition that voice cognates are separate phonemic targets.

In contrast to normal speakers, nonfluent aphasics are reported to produce voiced and voiceless stop consonant cognates having about the same VOT values [7, 9], so that the resulting distribution of VOT is unimodal. These data have been interpreted as indicating that the underlying phonological categories have merged. However, the data are also compatible with the view that these speakers select the correct phonemic targets for production but the articulation itself is so distorted that the difference between cognates is not maintained (at least on the VOT dimension). BLUMSTEIN *et al.* [2] attempted to examine this question directly by operationally defining a production error as an error in selecting the phonemic target when the VOT value of the utterance fell within the range of the opposite voice category, as when a required [b] was produced with a VOT value longer than 35 msec. A production error was considered to be of phonetic origin when its VOT value fell between the normal distributions for the voiced and voiceless categories, as when a required [b] was produced with a VOT value between 15 and 35 msec. In accord with previous work, Blumstein *et al.* found a large overlap of VOT values for voiced and voiceless productions by nonfluent (Broca's) aphasics, suggesting a pervasive deficit in the timing of articulatory movements. They note, however, that nonfluent aphasics produced some apparent phonemic errors as well, particularly on voiceless stop consonants. In contrast, errors produced by fluent (Wernicke) aphasics tended to fall within the VOT range of the opposite voice category, suggesting that their errors were primarily errors in selecting the appropriate phonemic target, although some apparent phonetic errors were also noted.

This description is intuitively satisfying in that it agrees with subjective clinical impressions. However, as Blumstein *et al.* recognize, we must be cautious when hypothesizing differences in the mechanisms for production errors from only one measure of articulation. For example, even when restricting discussion to the voicing feature, we find at least 16 cues that potentially influence perception [14]. If the pattern of errors on the VOT dimension is truly indicative of a more general speech disorder then some predictions should hold true. Specifically, a speaker producing apparent phonemic errors as reflected in VOT values might be expected to produce a similar distribution of errors when the same phonemic target appears in different positions in a syllable, even though the phonetic realization of the phoneme may be quite different. For example, in English one strong cue to stop consonant voicing in syllable-final position is the duration of the preceding vowel, which tends to be longer before voiced than before voiceless consonants for both adults [10, 11, 13, 18, 19] and children [20]. Thus if the errors are truly of phonemic selection and have no phonetic component, aphasic speakers who produce voicing errors that fall within the range of VOT values for the opposite voice category in syllable-initial position, should show voicing errors in syllable-final position characterized by preceding vowel durations that fall within the range of vowel durations occurring for the opposite voice category (i.e. a bimodal distribution of vowel durations).

The predictions regarding apparent phonetic errors are much less clear. Basically, the number of errors produced should be a function of the difficulty of articulation, which might be affected by a segment's position within a word. Unfortunately, it is as yet impossible to quantify the complexity of articulation involved in producing quite different acoustic results. If, however, the articulations involved in producing changes in VOT and vowel duration are of the same order of difficulty, nonfluent speakers should show the same distribution pattern for voicing errors in syllable-initial and syllable-final position. If voicing production in initial position is more difficult than in final position (as one might perhaps expect from the difficulty aphasics often have initiating speech), we would expect a greater number of phonetic errors in initial position than in final position. Another possibility is that "articulatory complexity" differs across speakers. If this is so, individual speakers might show a coherent pattern of phonetic errors across syllable positions that is not evidenced by the clinical group.

The study reported here is an attempt to determine whether the pattern of production errors indexed by VOT can be used to characterize more generally the output of the aphasic speaker as containing primarily "phonetic" or "phonemic" errors. To this end, the VOT findings of BLUMSTEIN *et al.* [2] and ITOH *et al.* [12] are first replicated. Next, for the same speakers, the duration of the vowel preceding a final stop consonant is examined. Both acoustic dimensions are interpreted with regard to "apparent phonemic" and "apparent phonetic" errors. In this study, errors are operationally-defined as "apparent phonemic" errors when categories are misplaced along some acoustic dimension, though contrast is maintained. "Apparent phonetic" errors are operationally-defined as those instances of production that fall between categories.

METHOD

Subjects

The subjects in this study included five (Wernicke) fluent aphasics (referred to hereafter as F1–F5), five (Broca's) nonfluent aphasics (referred to as NF1–NF5), and two normal controls. The fluent aphasics were articulatorily agile and used phrases of normal length. However, their speech often made no sense. All of the nonfluent aphasics spoke hesitantly, with long pauses between words, that is, in a manner that was impressionistically effortful. Three of the nonfluent speakers would be characterized as agrammatic (NF1, NF2 and NF5) and three were apractic (NF3, NF4 and NF5). The diagnostic category of each patient was determined by performance on the Boston Diagnostic Aphasia Examination [8] and other neurological and neuropsychological tests. A list of 35 monosyllabic and polysyllabic words and sentences (selected from a larger list provided by DARLEY, ARONSON and BROWN [5]) was used to assess the presence of speech apraxia. A speaker was diagnosed as "apractic" when production of the list contained numerous but inconsistent phonetic errors of various types, as well as attempts at self-correction. The errors were judged by a linguist who had no information concerning the individual patients. In all cases, etiology was vascular and involved only the left hemisphere (see Table 1 for additional information). No tumor or trauma cases were included. All of the subjects were right-handed premorbidly.

Stimuli

The stimuli were 30 prepausal stressed consonant–vowel–consonant words whose vowel was always [æ]; however, slight vowel quality changes across words did occur for some speakers. The test words included minimal pairs differing on the voicing of either the initial or final consonant (e.g. bat vs pat and bat vs bad). Each word, preceded by the word "THE", was printed in large capital letters on an index card and presented to the subject in random order.

Table 1. Descriptive data for aphasic subjects

	Age	Sex	Years of schooling	Year of onset	Auditory* comprehension	Hemiplegia
Fluent speakers						
1	57	F	16	1972	+0.7	No
2	67	F	16	1969	−0.3	No
3	49	M	16	1977	+0.06	No
4	55	M	10	1976	−0.12	No
5	43	M	14	1972	−0.6	No
Nonfluent speakers						
1†	61	F	16	1979	+0.2	No
2†	66	M	12	1980	0.0	Yes
3‡	67	M	4	1979	+0.7	Yes
4‡	69	M	20	1980	+1.0	Yes
5†‡	52	M	8	1974	+1.0	Yes

*Mean of the four auditory comprehension subtests of the Boston Diagnostic Aphasia Examination (GOODGLASS and KAPLAN, 1972).

†Agrammatic speakers.

‡Speech apraxia.

Procedure

Subjects were tested individually in a sound-insulated room. On presentation of the stimulus card, subjects were required to read the phrase aloud at least twice. If the subject was unable to read the card easily, the experimenter would say the phrase for the subject to repeat. The randomized list of phrases was presented a minimum of eight times so that each subject attempted to produce at least 16 tokens of each stimulus word. Subject responses were recorded onto a high-quality tape recorder for later analysis.

Data analysis

Broad phonemic transcriptions of all utterances were made by a trained linguist. Target segments produced with a different manner (e.g. [m] instead of [b] or place of articulation (e.g. [d] instead of [b] than the required utterance are excluded from further report. Substitutions of, for example [bʰ] were included in the analyses. VOT and vowel duration of the remaining utterances were measured using an interactive computer program that displays the acoustic waveform. VOT was defined as the time from the energy burst representing initial stop consonant release to the onset of acoustic periodicity representing vocal fold vibration. Vowel duration was defined as the interval from the onset of acoustic periodicity (excluding any initial aspiration) to the first acoustic evidence of closure for the final stop consonant (the time when the high frequency components of the periodic wave disappear). Spectrograms were also used when VOT or vowel duration could not be measured from the acoustic waveform.

RESULTS

Voice-onset-time

The frequency distribution of the VOT values was plotted individually for each subject. Figure 1 shows examples of the distribution of VOT values for a normal control, a fluent

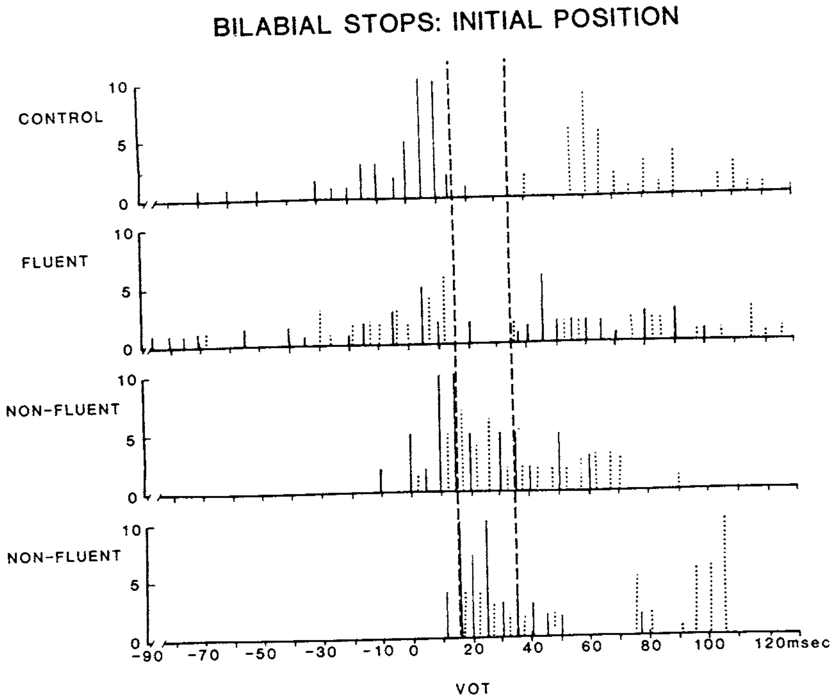

FIG. 1. The distribution of VOT values for bilabial stop consonants in word-initial position for a normal control, a fluent aphasic, and two nonfluent aphasics. VOT is plotted on the abscissa of each graph, number of productions on the ordinate. The vertical lines crossing the four graphs at 15 and 35 msec represent the upper and lower boundaries of voiced and voiceless bilabial stops, respectively. (———) The required production was [b]; (......) the required production was [p].

aphasic (subject F3) and two nonfluent aphasics (subjects NF2 and NF4). Data from these particular aphasics are shown because F3 and NF2 produced the expected patterns of VOT distribution but NF4 did not. The distributions were analyzed in two ways. First, apparent phonetic and apparent phonemic errors were catalogued using the procedure described by BLUMSTEIN *et al.* [2]. Briefly, if at least two instances of crossover of VOT values between the voiced and voiceless distributions occurred, then all VOT values within this middle range were counted as apparent phonetic errors. The boundaries for this middle range were taken from earlier studies of VOT values in normal speakers [15, 16] and were +15 to +35 msec VOT for bilabial stops, +20 to +40 msec for alveolar stops, and +25 to +45 msec for velar stops. For a production to be counted as an apparent mistargeting error, its VOT value had to fall in the range appropriate for its voicing cognate.

The results of this analysis are shown in Table 2 and are in fairly good agreement with other reports [2, 7, 9, 12]. A two-way ANOVA resulted in no significant main effects of group (F 1,8 = 0.31, $P > 0.1$) or error type (F 1,8 = 0.05, $P > 0.1$), but a significant group by error type interaction (F 1, 8 = 5.69, $P < 0.05$). As can be seen from the totals column in Table 2, this interaction occurred because the nonfluent aphasics as a group produced more apparent phonetic than phonemic errors whereas the fluent aphasics as a group produced more apparent phonemic than phonetic errors. The columns representing the different target sounds indicate that this differential pattern of errors occurred for nonfluent aphasics on all of the six target sounds but on only four of the six target sounds for fluent aphasic speakers. Moreover, the tendency for nonfluent speakers to produce more apparent phonemic errors on voiceless than voiced stops was not replicated. The two control subjects produced no errors of any sort.

Table 3 shows the error patterns for individual speakers. Four of the five fluent aphasics showed mostly bimodal distributions of voice-onset-time with the majority of errors falling within the range of the other voice category (apparent phonemic errors). For one fluent aphasic (F4), the voiced and voiceless categories were overlapped considerably, with many errors produced in both the apparent phonemic and apparent phonetic ranges. It is not clear from results of the diagnostic battery why this subject differed so considerably from the other fluent aphasics.

Although all five nonfluent aphasics produced some voice-onset-time values that fell well within the range of the target's voice cognate (apparent phonemic errors), four of the five produced proportionally more errors having intermediate voice-onset-time values (apparent phonetic errors). In contrast, one nonfluent speaker (NF1) produced no voice-onset-time errors that could be characterized as apparently of phonetic origin.

Table 2. Apparent "phonemic" and apparent "phonetic" errors expressed as a percent of total productions for each target consonant, across speakers

Target:	p	b	t	d	k	g	Total
"Phonemic" errors							
Fluent	22.7	24.6	9.7	8.4	12.5	42.2	20.0
Nonfluent	12.0	15.9	3.2	15.8	2.1	19.6	11.4
"Phonetic" errors							
Fluent	8.3	8.2	13.7	10.7	3.8	5.5	6.7
Nonfluent	22.9	29.9	13.8	21.8	22.8	23.9	22.3

Table 3. "Phonetic" and "phonemic" errors expressed as a percent of each speaker's total production of each consonant*

Target	p	b	t	d	k	g	Total
"Phonemic" errors							
F1	0.0	0.0	0.0	0.0	0.0	27.8	4.8
F2	9.1	8.0	21.7	0.0	0.0	12.5	8.5
F3	52.8	56.8	5.3	0.0	20.0	59.1	32.3
F4	20.8	25.6	0.0	41.2	0.0	68.9	26.0
F5	30.8	32.4	20.9	0.0	41.4	42.6	28.0
NF1	3.0	14.3	2.8	23.3	0.0	38.2	13.4
NF2	13.3	27.8	5.6	40.9	0.0	47.6	22.5
NF3	33.3	1.8	2.8	6.1	0.0	5.1	7.9
NF4	0.0	27.1	4.8	8.0	2.4	5.1	7.9
NF5	10.2	8.5	0.0	0.0	8.1	2.0	4.8
"Phonetic" errors							
F1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
F2	0.0	0.0	0.0	0.0	0.0	0.0	0.0
F3	0.0	0.0	0.0	0.0	8.3	0.0	1.3
F4	37.5	41.0	18.6	50.0	9.3	26.7	30.5
F5	4.2	0.0	0.0	3.4	0.0	0.0	1.3
NF1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
NF2	42.2	37.0	22.2	54.5	33.0	38.1	37.8
NF3	15.4	23.2	5.6	18.2	0.0	0.0	10.3
NF4	26.5	63.8	6.0	7.4	20.2	41.3	27.3
NF5	28.2	25.1	33.2	29.1	60.7	40.2	36.1

*Criteria: LISKER and ABRAMSON, 1964; LISKER and ABRAMSON, 1967.

One shortcoming of this analysis is that it does not accurately reflect a situation in which voice-onset-time of the two voice categories are shortened or lengthened relative to normal, whether or not they overlap. For this reason we re-examined the voice-onset-time data to determine simply whether the distribution for a given place of articulation was unimodal or bimodal. If the distribution was unimodal, no delineation of apparent phonetic and phonemic errors could be drawn. If the distribution was bimodal, we determined whether an interval of at least 15 msec without a token separated the two concentrations of data. If so, then all tokens which fell in the opposite voice distribution were termed apparent phonemic errors. If the distribution was strongly bimodal but two or three tokens occurred within the interval between modes, 30 msec of overlap midway between the two modes was ignored when counting apparent phonemic errors. The results of this analysis are shown in Table 4.

Notice first that as a group the fluent aphasics still seem to produce more apparent phonemic errors than the nonfluent group (15.0 vs 4.3%). However, this analysis changes one's conclusions concerning the actual number of targeting errors that occur. For example, in the fourth plot in Fig. 1 (subject NF4), the VOT values for voiced and voiceless stop consonants are longer than those measured for normal speakers so that the aphasic category boundaries do not fall at the normal category boundaries. This does not necessarily mean, however, that the categories have merged. Thus, errors in producing word-initial [p] that appeared in our first analysis to be of phonetic origin appear, with this less stringent criterion, as phonemic errors.

In both analyses of VOT, no errors were produced by the control subjects. Interestingly, the one nonfluent speaker who produced only apparent phonemic errors is severely agrammatic but would not be characterized as having speech apraxia.

Table 4. Percent of intended target and total productions categorized as "phonemic" errors*

	p	b	t	d	k	g	Total
F1	0.0	0.0	0.0	0.0	0.0	27.8	4.8
F2	9.1	8.0	21.7	0.0	0.0	12.5	8.5
F3	52.8	56.8	5.3	0.0	25.0	59.5	33.2
F4	†	†	†	†	†	†	†
F5	33.2	32.4	20.9	2.2	41.4	42.2	28.7
NF1	3.0	6.1	3.3	19.4	5.9	14.7	8.8
NF2	†	†	†	†	†	†	†
NF3	†	†	8.3	6.1	2.8	5.1	4.3
NF4	36.7	6.4	0.0	0.0	2.4	5.1	8.6
NF5	†	†	†	†	†	†	†

*Criterion: bimodality of VOT.
†Unimodal distribution.

Vowel duration

The duration of the vowel preceding voiced and voiceless final stop consonants was measured to determine whether the resulting pattern of errors is similar to the pattern of VOT errors. Figure 2 shows examples of the distribution of vowel durations measured for the same normal control, fluent aphasic (F3) and one of the nonfluent aphasics (NF2) shown in Fig. 1. However with vowel duration, unlike VOT, one does not have a predetermined cut-off value for accepting a token as correct or in error. Rather than arbitrarily defining a range of durations as apparent phonetic errors, I determined only whether for a given place of articulation, the distribution of vowel durations was unimodal or bimodal. As for our second analysis of VOT, when bimodal distributions were separated by at least 15 msec, apparent phonemic targeting errors were counted. When seemingly bimodal distributions were not separated by at least 15 msec, the 30 msec between the two distributions were ignored. If the VOT results are indicative of a "phonemic" speech disorder, then those patients who produced bimodal distributions of VOT values (primarily apparent phonemic errors) should show bimodal distributions of vowel durations.

Table 5 shows the results of this analysis. Notice first that as a group the fluent aphasics produced more bimodal distributions of vowel duration (irrespective of number of errors) than did the nonfluent group, although many individual differences within groups are apparent. Also obvious from a comparison of Tables 4 and 5, is that the distribution of apparent phonemic errors produced by both fluent and nonfluent speakers is not equivalent for word-initial and word-final positions. F1 produced bimodal distributions of both VOT and vowel duration. However, she produced no apparent phonemic errors on [t] and [d] in word-initial position, yet in word-final position, 8.6% of her productions in which [t] was the required target, and 11% of productions in which [d] was the required target were apparent phonemic errors. F2 also produced bimodal distributions of both VOT and vowel duration, with the [t]-[d] distinction producing the most apparent phonemic errors on both measures. However, in word-initial position the voiced alveolar was substituted for the voiceless whereas in word-final position the voiceless alveolar substituted for the voiced. Interestingly, this reversal is the consequence of an inappropriate shortening of both VOT and vowel duration. F3 produced bimodal VOT distributions for all three places of articulation, but a bimodal distribution of vowel duration only for the alveolar stops (the category with fewest

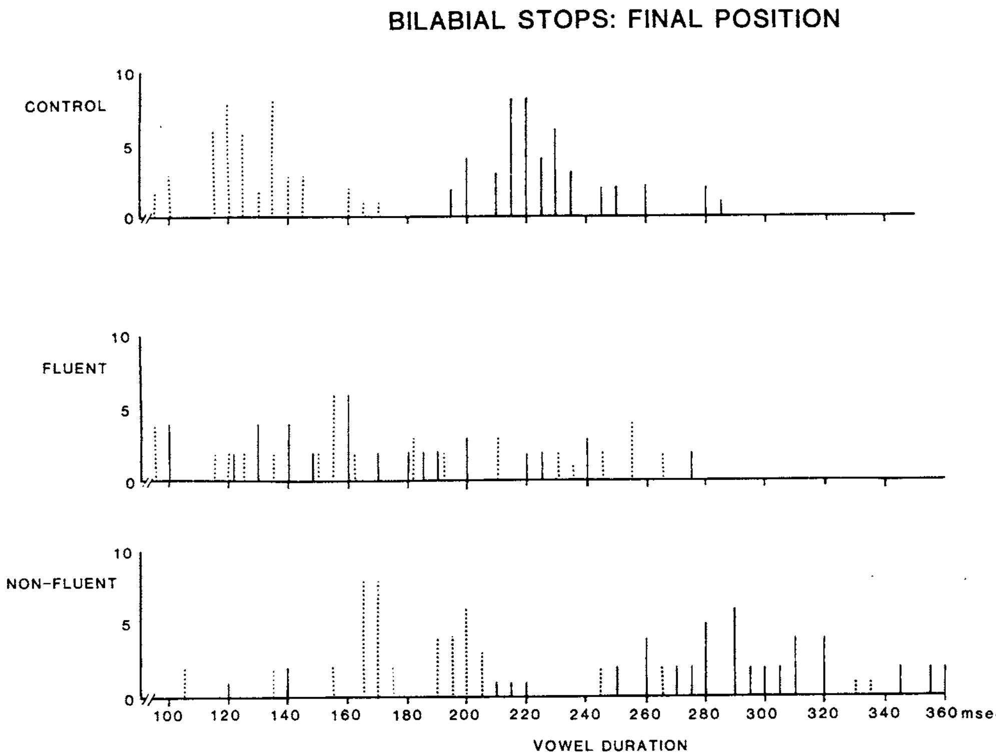

FIG. 2. The distribution of vowel duration measures for bilabial stop consonants in word-final position for a normal control, a fluent aphasic, and a nonfluent aphasic. Vowel duration is plotted on the abscissa, number of productions on the ordinate. (———) The required production was [b]; (......) the required production was [p].

errors on VOT). F4 produced only unimodal distributions of VOT but bimodal distributions of vowel duration for the velar and alveolar stops. Moreover, the errors in word-initial position greatly outnumbered errors in word-final position. F5 produced many apparent phonemic errors at all places of articulation, as indexed by VOT. In contrast, vowel duration measures indicated apparent phonemic errors only for the velar stops.

With regard to the nonfluent speakers, one agrammatic speaker (NF1) produced bimodal distributions of VOT values for all places of articulation but she produced only unimodal distributions of vowel duration. The two other agrammatic speakers (NF2 and NF5) showed the opposite pattern, with unimodal distributions of VOT and bimodal distributions of vowel duration. NF3 produced a unimodal distribution of vowel duration for all places of articulation but a unimodal distribution of VOT only for bilabial stops. NF4 produced bimodal distributions of VOT and vowel duration values for bilabial, alveolar and velar stops. However, errors in word-final position occurred predominantly on voiced consonants, a pattern not reflected in word-initial errors. Again, for many of these errors the measured acoustic duration was inappropriately short. As expected, the two normal speakers produced error-free bimodal distributions of vowel duration in these word lists.

In summary, those patients (fluent and nonfluent) who produced apparent phonemic errors in word-initial position did not necessarily produce those errors in word-final

Table 5. Percent of intended target and total productions categorized as "phonemic" errors on the basis of vowel duration

	p	b	t	d	k	g	Total
F1	8.6	0.0	8.6	11.1	3.1	8.6	6.7
F2	8.7	0.0	0.0	13.8	4.2	4.2	5.2
F3	*	*	6.7	11.1	*	*	3.3
F4	*	*	3.3	9.4	7.1	0.0	3.6
F5	0.0	0.0	*	*	10.2	12.5	5.7
NF1	*	*	*	*	*	*	*
NF2	12.5	12.8	6.7	7.7	8.7	15.0	10.6
NF3	*	*	*	*	*	*	*
NF4	0.0	47.4	0.0	9.4	2.8	26.3	14.5
NF5	0.0	0.0	0.0	12.4	21.3	13.5	7.9

*Unimodal distribution.

position. The result sheds doubt on the conclusion that a production whose value on one acoustic dimension is appropriate to its cognate is indicative of a general impairment in phonemic targeting.

The regularity of apparent phonetic errors can also be questioned given the data in Tables 3, 4 and 5. As previously mentioned, it is possible to demarcate only an arbitrary region of vowel durations, within which productions are categorized as apparent phonetic errors. Thus, a unimodal distribution of measured vowel durations was considered to have "many" apparent phonetic errors, a bimodal distribution to have "none", and a primarily bimodal distribution with scattered intermediate data points to contain "few" apparent phonetic errors. By these rather loose criteria, no consistency was apparent either within or across speakers. Two of the five fluent aphasics (F1 and F2) produced no apparent phonetic errors on word-initial or word-final stop consonants. Speaker F3 produced only a few apparent phonetic errors on voiceless velar stops in initial position but many apparent phonetic errors on final bilabial and velar stops. Speaker F4 produced many apparent phonetic errors on word-initial stop consonants at all three places of articulation but only on word-final bilabial stops. F5 produced many apparent phonetic errors only on alveolar stops in word-final position. Of the five nonfluent speakers, two (NF1 and NF3) produced more apparent phonetic errors on final than initial stops. This occurred at all places of articulation for NF1, but only for alveolars and velars for NF3. In contrast, NF2 and NF5 produced many apparent phonetic errors on word-initial stops but none on word final stops.

DISCUSSION

The results of the first part of this study converge with previous reports of voice-onset-time production by aphasic speakers [2, 7, 9, 12]. Using the VOT boundaries established by Lisker and Abramson [15, 16], it was determined that nonfluent aphasics as a group produced more apparent phonetic than apparent phonemic errors whereas fluent aphasics as a group produced more apparent phonemic than phonetic errors. It does not necessarily follow, however, that those speakers who produce primarily apparent phonetic errors have merged the voicing categories. When VOT values were examined to determine simply whether the resulting distribution was unimodal or bimodal (ignoring the absolute VOT value) four of the five fluent aphasics and three of the five nonfluent aphasics showed evidence of bimodal patterns. Thus it appears that for these speakers separate voicing categories were preserved.

The major result of this study is that each speaker's pattern of errors on word-initial stop consonants (as measured by VOT values) is not a good predictor of the error pattern on word-final stops (as indexed by vowel duration). For each subject, the number of apparent phonemic errors differed radically across positions. In order to attribute the bulk of the errors produced by fluent aphasics to incorrect selection of phonemic targets one would have to suppose that the selection of phonemic targets is sensitive to the phoneme's position within a word. There are, in fact, theories that consider a word's representation in the mental lexicon to be phonologically ordered in a left-to-right manner (e.g. [4, 6]). However, this accounts for neither the unimodal distributions of VOT and vowel duration produced by fluent aphasics nor the lack of consistency across subjects as to whether more apparent phonemic errors were produced on word-initial or word-final stops.

With regard to apparent phonetic errors, I had hoped to find some consistent pattern, at least for the nonfluent speakers, indicating that adequate control of the interval between release of supraglottal occlusion and the onset of glottal pulsing was more difficult than control of the duration of voicing, or vice versa. However, the pattern and number of errors on initial stop consonant production was unrelated to the pattern and number of errors on final stop production. This may be because (1) the apparent phonetic errors are independent of articulatory complexity, (2) these speakers are brain-damaged so that our (admittedly weak) "metric of articulatory complexity" for normal speakers is not appropriate, or (3) articulatory complexity varies among speakers. Furthermore, the nonfluent speakers did not group on the basis of presence or absence of speech apraxia or agrammatism.

In conclusion, it appears that (at least for this small sample of aphasic speakers) the pattern of errors on the voice-onset-time dimension cannot be used to characterize more generally the output of the speaker. These data also indicate that the traditional alignment of fluent aphasics with phonemic errors and nonfluent aphasics with phonetic errors is inadequate as a description of aphasic output. More generally, we should recognize that phonetic and phonemic aspects of speech are not necessarily independent. Clearly much more acoustic and physiological information is needed before we can ascribe the constellation of fluent and nonfluent aphasic errors as primarily phonetic or phonemic in origin.

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