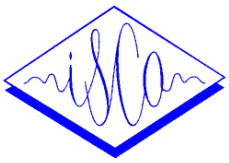

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RATE OF SPEECH EFFECTS IN APHASIA: AN ACOUSTIC ANALYSIS OF VOICE ONSET TIME

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ABSTRACT

The production of voice onset time (VOT) contrasts at two rates of speech was investigated in three groups of subjects: nonfluent and fluent aphasic patients and non-neurological controls. Subjects produced the consonants [b d g p t k] in the environment preceding the vowels [i e a o u] in a carrier phrase at a slow/normal and a fast rate of speech. For the normal speakers, acoustic analyses revealed significantly shorter VOTs at the fast as compared to the slow/normal rate, with a larger change evident in the voiceless relative to the voiced stop consonants. Both groups of aphasic patients produced rate changes that were smaller in magnitude than those of the normal subjects. Further, both brain-damaged patient groups produced VOTs that were shorter in the fast condition compared to the slow condition. However, a number of aberrant patterns emerged. For the nonfluent aphasic subjects, as in previous studies, voiced and voiceless consonants were produced with somewhat overlapping VOT distributions, suggesting a deficit in speech timing in these subjects. For the fluent aphasic patients, no differences in magnitude of VOT change emerged for voiceless relative to voiced consonants. Implications of these findings for theories of the nature of speech production impairments in aphasic patients are considered.

INTRODUCTION

Many investigations have explored temporal parameters of speech in aphasic patients and have demonstrated that nonfluent aphasic patients often display patterns of production that deviate from the norm [1,2,3,4,5,6,7,8]. Of particular relevance to the present study are a number of experiments that focus on the effects of variations in rate of speech on speech production deficits in aphasic patients [9,10,11,7]. Through acoustic analyses, these studies have shown that both nonfluent and fluent aphasics are able to vary their rates of speech to a limited degree. Research has demonstrated that segment to whole word ratios, often referred to as relative timing relations, are unimpaired in aphasic subjects, despite changes in rate of speech [7]. However, it has also been shown that variations in rate of speech may not be appropriately implemented at the segmental level in nonfluent aphasic speakers' vowel production [10]. It is not known to what extent temporal parameters that provide cues to phonemic contrasts are maintained or impaired in aphasic patients.

The goal of the present investigation is to examine the production of voice onset time (VOT) at two rates of speech in aphasic individuals. Past research has shown repeatedly that nonfluent aphasics produce voiced and voiceless stops with overlapping VOT values, in contrast to the non-overlapping bimodal distribution characteristic of normal speech production; fluent aphasic speakers generally maintain a relatively normal pattern of VOT values, with some productions whose VOTs fall within the range of the alternate voicing category [2,3,12,13]. Based on these findings, researchers have attributed to nonfluent aphasic patients a deficit in the implementation of temporal parameters of speech; fluent aphasic patients, in contrast, are thought to exhibit an impairment in phonemic planning [2,3].

In normal speech production, as rate of speech increases, VOT decreases; this decrease is most notable for voiceless stops in English, while VOTs for voiced stops remain relatively unchanged [14,15,16]. The present study was designed to determine whether nonfluent and fluent aphasic patients produce VOT patterns across modifications in rate of speech that are comparable to those of normal speakers. If the aphasics are unable to alter their speaking rates, it would suggest an impairment in the planning of speech timing. If the patients exhibit impairments in the execution of rate changes at the segmental level (in terms of VOT values), an impairment in phonetic implementation would be indicated.

METHODS

Subjects

Ten nonfluent aphasic patients, 7 fluent aphasic patients, and 10 age-matched non-neurological controls participated in this study. All were native speakers of English with no hearing impairments. All aphasic patients were at least 4 months post onset and were diagnosed using the Boston Diagnostic Aphasia Examination [17] and the Apraxia Battery for Adults [18].

Stimuli

The stimuli included the six stop consonants [b d g p t k] in the environment preceding each of the vowels [i e a o u] followed by a final [d]. All stimuli were produced in the context of the carrier phrase "Please say ___". Stimuli were printed in orthographic form on index cards for the elicitation procedures.

Procedure

Stimuli were produced four times each in random order at a slow/normal and fast rate of speech. The two rates were demonstrated prior to recording. If a subject was unable to read a stimulus, a model was provided by the examiner for repetition. Stimuli were recorded using a Sony Professional Walkman (WMD6C) and Sony ECM-909 directional microphone located 10 inches from the speaker's mouth.

Acoustic analyses

Stimuli were digitized at a rate of 20 kHz

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with a 9 kHz low-pass filter and 12-bit quantization using the BLISS speech analysis system [19]. From the waveform display, three duration measures were calculated. Utterance duration was computed and converted to a measure of syllables per second to determine rate of speech. Measures were made from the onset of the burst corresponding to the [p] of "please" through the end of the target word's [d]-final release burst. Syllable duration was also computed, from the onset of the burst of the target syllable's initial stop consonant through the end of the syllable-final [d]. The third measure calculated VOT values from the onset of the target-initial stop burst to the onset of periodicity corresponding to the following vowel. Any periods of voicing during closure for the target-initial stop were excluded, yielding a minimum possible VOT of 0 ms.

RESULTS

We will focus only on results for two of the three measures calculated: utterance duration and VOT. Utterances with paraphasic errors in the target syllable (except for consonant voicing) were excluded from the analyses. Mean speaking rates for each of the subject groups are displayed in Table 1. For the normal subjects, the fast rate was approximately 3.2 times the slow rate; for nonfluent and fluent aphasic subjects, the difference in rates was much smaller with the fast rate approximately 1.7 and 1.8 times the slow rate for the nonfluent and fluent aphasic groups respectively. Statistical analyses (ANOVA) revealed a significant main effect for rate of speech ($F(1,24)=160.550$, $p<.001$) and a significant group x rate interaction ($F(2,24)=15.816$, $p<.001$). Post hoc tests (Newman-Keuls, $p<.01$) yielded significant differences between the slow and fast rates within each subject group. Further, the normals' fast rate was significantly faster than that of both aphasic groups.

Table 1. Mean rates of speech (syllables/second) for each subject group.

Group	Fast	Slow
Normals	3.5	1.1
Nonfluent Aphasics	2.5	1.5
Fluent Aphasics	2.6	1.5

The mean VOT values computed for each consonant at each rate are provided in Table 2 for each of the three speaker groups. Overall, all groups produced voiceless consonants with longer VOTs than voiced at both rates of speech. In addition, all three groups exhibited longer VOTs for velar consonants compared to alveolars and labials, consistent with predictions of normal speech production. Finally, all groups produced VOTs in the fast condition that were shorter than in the slow rate condition. For normal subjects, statistical analyses confirmed the expected patterns just noted, as well as a greater change in VOTs for voiceless consonants relative to voiced and for velars relative to alveolars and labials across rates of speech. Statistical analyses of the nonfluent aphasic subjects' data yielded a very similar pattern to that of the normal subjects with the exception of the differential changes across place of articulation. Results for the fluent aphasic patients revealed significant changes in VOT across rate conditions but no interactions with voicing (i.e., changes in voiced and voiceless VOTs were of comparable magnitude) or place of articulation (i.e., changes in velar consonants were of comparable magnitude to those in alveolars and labials).

Table 2. Mean VOT values (ms) for normal (N), nonfluent (NF), and fluent (F) groups at slow and fast rates of speech.

Group	Rate	[b]	[d]	[g]	[p]	[t]	[k]
N	Slow	9	17	29	86	96	112
	Fast	4	10	16	46	54	66
NF	Slow	12	18	26	83	92	107
	Fast	7	12	29	64	69	83
F	Slow	11	21	33	86	95	99
	Fast	9	17	29	67	76	83

A further analysis was undertaken to examine the distributions of VOTs. Figure 1 provides a representative example of the VOT distribution for labial consonants for one normal, one nonfluent, and one fluent aphasic subject. As may be noted, the normal subjects maintained bimodal VOT distributions at both rates of speech. As expected, the distributions for voiced and voiceless tokens are closer together in the fast condition as compared to the slow. In the slow condition, the nonfluent subjects produced a relatively bimodal VOT distribution with some overlap of individual tokens, consistent with previous research [2,3]. In the fast condition, the degree of overlap is substantially increased for these subjects. The fluent aphasic patients produced bimodal VOT distributions at both rates of speech, comparable to the normal speakers.

DISCUSSION

The results demonstrate that both nonfluent and fluent aphasic subjects are capable of modifying their rates of speech, albeit to a lesser extent than normals. In particular, the aphasic patients may be constrained in the maximum speaking rate that they can produce; this hypothesis is consistent with results of previous studies suggesting slower than normal speech production in nonfluent aphasics [10,5,6,7]. In addition, the findings indicate that VOTs at the fast rate were shorter than those at the slow rate for all speaker groups. The results further show that all three groups of subjects produced voiceless stop consonants with significantly longer VOTs relative to voiced stops in both rate conditions.

Differences between the two aphasic groups and the normals emerged upon closer inspection of the VOT data. Specifically, the magnitude of the change in VOTs from slow to fast conditions was larger for voiceless than voiced stops and for velar relative to alveolar and labial stops in normal subjects, as expected [14,15,16]. For the nonfluent aphasic patients, no differences were found in the magnitude of VOT change with increased speaking rate across different places of articulation. The pattern of results was, in other respects, quite similar to that of the normal subjects; however, the VOT decreases

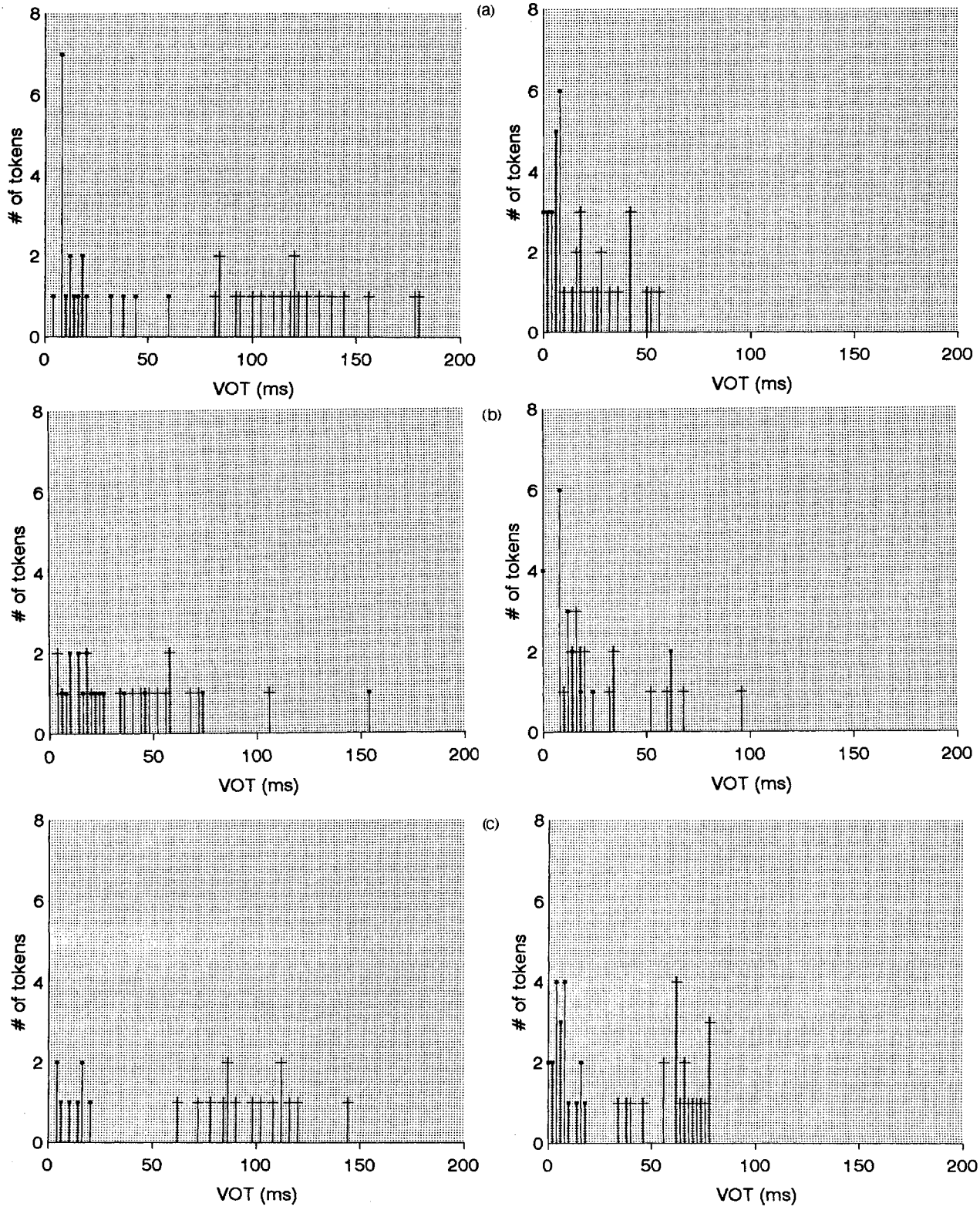

Figure 1. Sample voiced (.) and voiceless (+) VOT distributions for labial consonants at slow (left) and fast (right) rates of speech for 1 normal (a), 1 nonfluent aphasic (b), and 1 fluent aphasic (c) speaker.

with increased rate of speech were far smaller than those of the normal subjects. In addition, the VOT distributions of the nonfluent aphasic subjects revealed a considerable amount of overlap of individual productions, particularly at the fast rate of speech. These findings are consistent with previous results which have suggested an impairment in temporal coordination in nonfluent aphasic subjects [2,3]. This deficit seems to be even more apparent at increased rates of speech.

The results for the fluent aphasics presented a mixed picture. First, as expected, they maintained a bimodal distribution of VOT values [2,3]. However, in terms of the pattern of mean VOT values across changes in speaking rate, the fluent patients differed more from the normals than did the nonfluent patients. These subjects did not show any differences in the magnitude of VOT change between voiceless and voiced consonants or across places of articulation, suggesting a possible deficit in the implementation of rate changes at the segmental level in fluent aphasia. The findings must be interpreted cautiously, though, because there were only 7 subjects in the fluent aphasic group and there was a great deal of individual variability within the group.

The results of the current experiment indicate that both nonfluent and fluent aphasic patients demonstrate impairments in phonetic implementation or execution [1,7]. Such findings suggest that the neural representation of speech motor control involves both anterior and posterior neural structures and is more complex than earlier hypothesized [10,1,20]. Although not tested directly in the present investigation, it seems unlikely that reductions in speaking rate (e.g., to a slow/normal rate) would significantly improve speech production in aphasic patients. Further research is warranted on this topic to clarify the precise nature of the temporal control deficits found.

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