The Effects of Utterance Length on Temporal Control in Aphasia

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Abstract: By comparing the same syllables produced in a variety of utterance-length conditions, the present investigation examined the hypothesis that fluent aphasics exhibit a shorter-than-normal speech planning domain, whereas nonfluent aphasic patients exhibit impaired speech timing at all levels. Subjects included nonfluent aphasics, fluent aphasics, right-hemisphere-damaged (RHD) controls, and normal controls. The base stimuli were 10 monosyllabic nouns from which 10 bisyllabic words were derived. Each of the 20 words also appeared medially and in final position in the context of a short and a long sentence. Analyses revealed that normal speakers produced target syllables with shorter durations in 2-syllable relative to 1-syllable words in both long and short utterances. A similar pattern was found for the other speaker groups. In addition, target syllables were longer in final position relative to medial position for both normal speakers and RHD controls. In contrast, both left-hemisphere-damaged (LHD) aphasic groups failed to demonstrate normal phrase-final lengthening effects. Results are discussed in relation to theories of temporal control in brain-damaged patients.

INTRODUCTION

Many investigations have reported impairments in temporal control subsequent to left hemisphere damage (LHD)—particularly damage localized to anterior brain structures (see 4 for review). Early work suggested that such impairments may be limited to the production of speech segments that require the integration of two independent articulators (e.g., 5,6). More recent experiments have focused on larger units of production and the role of syllable structure and speaking rate effects (1,3,7,10,11). These studies have shown deficits in LHD patients that appear to be exacerbated in larger-sized linguistic units (particularly in fluent aphasic patients) (1,8,9,10,11,12); the few studies that have included right-hemisphere-damaged (RHD) patients have reported normal temporal control in these individuals (e.g., 11). Right hemisphere damage is not thought to engender deficits in temporal control due to a hypothesized left hemisphere lateralization of speech timing mechanisms. The goal of the present experiment was to compare specific syllables as they occur in isolation, in multisyllabic words, and in sentences of differing lengths to examine in detail the hypothesis that speech timing breaks down in fluent aphasic patients only in larger speech planning domains. Further, the position of the syllable within the sentences was varied to specifically examine whether the phrase-final lengthening phenomenon (ubiquitous in normal speech production) is robust in both short and long sentences produced by brain-damaged patients.

METHODS

Subjects included 7 LHD nonfluent aphasic patients, 5 LHD fluent aphasic patients, 8 RHD patients, and 10 age-matched normal controls. All subjects were native speakers of English with normal hearing.

The stimuli were derived from a base set of 10 monosyllabic nouns. From these base words, a set of 10 bisyllabic words was derived, all with first syllable stress. Each of the 20 words also appears medially and in final position in the context of a short (4-6 syllable) and a long (8-10 syllable) single-clause sentence frame. Stimuli were printed in orthographic form in large font for presentation to subjects. The isolated words and the sentential stimuli were presented in separate blocks, with the order of presentation counterbalanced across subjects. Within each block, the stimuli were presented two times each in a fixed random order. Productions were recorded in a quiet room using a Sony DAT recorder and high-quality directional microphone.

Stimuli were digitized at a rate of 10k samples/s with a 4.5 kHz low-pass filter and 12-bit quantization via the BLISS speech analysis system. Durations of the entire utterance (monosyllable, bisyllabic word, or sentence) were determined from a waveform display. In addition, durations of the target base monosyllable were computed in each context. Boundaries were determined by visual inspection of the waveform and confirmed by auditory perception. Where appropriate, ratios of base syllable to whole utterance were calculated to adjust for differences in speaking rate across subjects.
RESULTS AND DISCUSSION

Syllable durations were computed as a proportion of sentence durations for each subject. These data were submitted to two separate analyses of variance (ANOVAs), one exploring syllable and sentence length effects and one exploring sentence length and position effects across groups. The group x sentence length x number of syllables ANOVA yielded main effects of group, length, and number of syllables, and a length x number of syllables interaction. However, no interactions with the group variable emerged, indicating that all groups displayed the expected shortening of target syllables as utterance length increased. Not surprisingly, the length differences were somewhat more marked in the short sentences relative to the long sentences. The group x position x length ANOVA revealed main effects of all three variables, as well as multiple interactions including a 3-way interaction \([F(3,26)=11.19, p<.001]\). Post hoc analysis of this interaction using the Newman-Keuls procedure revealed that both normal controls and RHD subjects displayed the expected patterns of shorter syllable durations in medial relative to final position in both long and short sentences, and shorter syllable durations in long relative to short sentences in both utterance positions. In contrast, although both LHD groups produced significantly shorter durations in long relative to short utterances in both positions, neither group showed the normal position effect. In fact, the nonfluent aphasic patients produced significantly longer durations in medial relative to final position, while the fluent aphasic patients produced syllables in both positions with the same duration. Thus, consistent with previous investigations (11,12), the LHD subjects did not produce normal phrase-final lengthening effects and displayed impaired temporal control in multisyllabic utterances (see also 1,8,9,10).

The findings support the long-standing claim that the control of speech timing is mediated by left hemisphere mechanisms. The results are also, at least in part, in keeping with the hypothesis that fluent aphasic patients may have a limited domain of speech planning (1,10). However, these patients did exhibit the normal pattern of syllable shortening in relatively lengthy multisyllabic utterances, suggesting that it is only certain aspects of temporal control that break down in longer utterances produced by these individuals. Moreover, the nonfluent aphasic patients did not display a pervasive deficit in temporal control in the present experiment, although they did demonstrate impairments in the implementation of phrase-final lengthening (see also 1,2). Gandour and colleagues (11) have claimed that the speech timing deficits evidenced by nonfluent and fluent aphasic patients stem from different underlying impairments, even though they may be manifested in similar surface characteristics. Although the present findings can neither unequivocally support nor refute such a claim (particularly given the relatively small subject groups), it seems likely to be the case, given the very different quality of the speech produced by the two patient groups and the relative subtlety of the impairments that have been reported in fluent aphasic patients in previous studies. In sum, the present findings contribute to the mounting body of evidence which indicates that the temporal control of speech is particularly fragile subsequent to LHD; nonetheless, the data are inconclusive with respect to a hypothetical limitation on the domain of speech planning specific to fluent aphasic patients (1,10; but cf. 12).

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REFERENCES