Effect of delayed auditory feedback on normal speakers at two speech rates

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This study investigated the effect of short and long auditory feedback delays at two speech rates with normal speakers. Seventeen participants spoke under delayed auditory feedback (DAF) at 0, 25, 50, and 200 ms at normal and fast rates of speech. Significantly two to three times more dysfluencies were displayed at 200 ms ($p<0.05$) relative to no delay or the shorter delays. There were significantly more dysfluencies observed at the fast rate of speech ($p=0.028$). These findings implicate the peripheral feedback system(s) of fluent speakers for the disruptive effects of DAF on normal speech production at long auditory feedback delays. Considering the contrast in fluency/dysfluency exhibited between normal speakers and those who stutter at short and long delays, it appears that speech disruption of normal speakers under DAF is a poor analog of stuttering. [DOI: 10.1121/1.1466868]

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I. INTRODUCTION

The powerful fluency-enhancing effects of delayed auditory feedback (DAF) among individuals who stutter are well documented (Bloodstein, 1995). In contrast, numerous experiments with normal speakers have shown that DAF produces disruptive effects on the speech. Such effects include speech errors (e.g., repetition of phonemes, syllables, or words), changes in speech rate/reading duration, prolonged voicing, increased vocal intensity, and modifications in aerodynamics (Black, 1951; Fukawa *et al.*, 1988; Howell, 1990; Langova *et al.*, 1970; Lee, 1950, 1951; Mackay, 1968; Siegel *et al.*, 1982; Stager *et al.*, 1997; Stager and Ludlow, 1993). Several theorists (Black, 1951; Cherry and Sayers, 1956; Van Riper, 1982; Yates, 1963) have proposed that the speech disruptions of normal speakers under DAF are an analog of stuttering since these disruptions are similar to stuttering. Put simply, normal speakers can be made to ''artificially stutter'' under DAF.

With respect to speech production errors, three problems become evident when reviewing previous research examining the effects of DAF on normal speakers. First, investigators have typically utilized ''long'' delays ranging 100 to 300 ms. While such long delays have induced errors in speech production in normal speakers, there is a paucity of information concerning the effect of shorter delays. Second, to the best of our knowledge, there is only one study investigating the effect of different rates of speaking (e.g., normal versus a fast rate) and DAF on normal speakers. Zanini *et al.* (1999) reported that participants speaking at a normal rate while receiving 200 ms DAF produced significantly more speech errors that those receiving no DAF. With an increased speaking rate, the total number of speech errors increased for those receiving no DAF but remained approximately the same for those receiving DAF. There was no significant difference in speech errors at an increased speaking rate between those receiving DAF and those not. There is no evidence of the effect of speech rate and DAF at shorter delays. Finally, the absence of an operational definition of ''errors in speech production'' or ''dysfluency'' makes interpretation of earlier work problematic and most likely impossible. Specifically, definitions for dysfluency such as ''misarticulations'' (Ham *et al.*, 1984), ''hesitations'' (Stephen and Haggard, 1980), or ''slurred syllables'' (Zalosh and Salzman, 1965) are not consistent with the standard definition of dysfluent behaviors of individuals who stutter (i.e., part word repetitions, prolongations, and postural fixations).

This investigation sought to further explore the effect of DAF on normal speakers. Specifically, the purpose of this study was to investigate the effect of short and long auditory feedback delays at fast and normal rates of speech with normal speakers. In contrast to previous research, a conventional definition of dysfluency, consistent with the operational construct used in the examination of the dysfluency in those that stutter, was adopted. This definition excluded speech errors that are associated with other pathological conditions (i.e., developmental articulation errors).

II. METHOD

A. Participants

Seventeen normal speaking adult males aged 19 to 57 ($M=32.9$ years, s.d.$=12.5$) served as participants. All participants presented with normal middle ear function (American Speech-Language-Hearing Association, 1997) and normal hearing sensitivity defined as having pure-tone thresholds at octave frequencies from 250 to 8000 Hz and speech recognition thresholds of $\leqslant$20 dB HL (ANSI, 1996). All individuals had a negative history of neurological, otological, and psychiatric disorders.

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B. Apparatus and procedure

All testing was conducted in an audiometric test suite. Participants spoke into a microphone (Shure Prologue Model 12L-LC) which the output was fed to an audio mixer (Mackie Micro Series 1202) and routed to a digital signal processor (Yamaha Model DSP-1) and amplifier (Optimus Model STA-3180) before being returned bilaterally through earphones (EAR Tone Model 3A). The digital signal processor introduced feedback delays of 0, 25, 50, or 200 ms to the participants' speech signal. The shorter delays were identical to those utilized by Kalinowski *et al.* (1996) with persons who stutter. The 200-ms delay was chosen to be representative of a long delay that was employed in numerous previous studies with normal speakers. The output to the earphones was calibrated to approximate real ear average conversation sound pressure levels of speech outputs from normal-hearing participants. All speech samples were recorded with a video camera (JVC Model S-62U) and a stereo videocassette recorder (Samsung Model VR 8705).

Participants read passages of 300 syllables with similar theme and syntactic complexity. Passages were read at both normal and fast speech rates under each DAF condition. Participants were instructed to read with normal vocal intensity. For the fast rate condition, participants were instructed to read as fast as possible while maintaining intelligibility. Speech rates were counterbalanced and DAF conditions were randomized across participants.

The number of dysfluent episodes and speech rates were determined for each experimental condition by trained research assistants. A dysfluent episode was defined as a part-word prolongation, part-word repetition, or inaudible postural fixation (i.e., ''silent blocks;'' Stuart *et al.*, 1997). The same research assistant recalculated dysfluencies for 10% of the speech samples chosen at random. Intrajudge syllable-by-syllable agreement was 0.92, as indexed by Cohen's *kappa* (Cohen, 1960). Cohen's *kappa* values above 0.75 represent excellent agreement beyond chance (Fleiss, 1981). A second research assistant independently determined stuttering frequency for 10% of the speech samples chosen at random. Interjudge syllable-by-syllable agreement was 0.89 as indexed by Cohen's *kappa*. Speech rate was calculated by transferring portions of the audio track recordings onto a personal computer's (Apple Power Macintosh 9600/300) hard drive via the videocassette recorder interfaced with an analog to digital input/output board (Digidesign Model Audiomedia NuBus). Sampling frequency and quantization were 22 050 Hz and 16 bit, respectively. Speaking rate was determined from samples of 50 perceptually fluent syllables that were contiguous and separated from dysfluent episodes by at least one syllable. Sample duration represented the time between acoustic onset of the first syllable and the acoustic offset of the last fluent syllable, minus pauses that exceeded 0.1 s. Most pauses were inspiratory gestures with durations of approximately 0.3 to 0.8 s. Speech rate, in syllables/second, was calculated by dividing the number of syllables in the sample by the duration of each fluent speech sample.

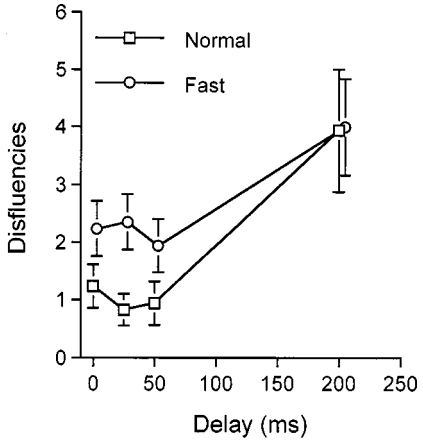

FIG. 1. Mean number of dysfluencies as a function of DAF and speech rate. Error bars represent plus/minus one standard error of the mean.

III. RESULTS

Means and standard deviations for dysfluencies (i.e., number of dysfluent episodes/300 syllables) as a function of DAF and speech rate are shown in Fig. 1. A two-factor analysis of variance with repeated measures was performed to investigate the effect of DAF and speech rate on dysfluencies. Statistically significant main effects of DAF [$F(3,48)=8.73$, Huynh-Felt $p=0.0015$, $\eta^2=0.35$] and speech rate [$F(1,16)=5.88$, Huynh-Felt $p=0.028$, $\eta^2=0.27$] were found. The effect sizes of these significant main effects were large (Cohen, 1988). The interaction of speech rate by DAF was not significant [$F(3,48)=1.10$, Huynh-Felt $p=0.33$, $\eta^2=0.064$, $\phi=0.20$ at $\alpha=0.05$]. *Post-hoc* orthogonal single-*df* contrasts showed that while the mean differences in dysfluencies at 0, 25, and 50 ms were not significantly different from each other ($p>0.05$) they were all significantly less than that at 200 ms ($p<0.05$).

Mean syllable rates and standard deviations as a function of DAF and speech rate are displayed in Fig. 2. A two-factor analysis of variance with repeated measures were performed to investigate the effect of DAF and speaking rate on syllable rate. Statistically significant main effects of DAF [$F(3,48)=39.32$, Huynh-Felt $p<0.0001$, $\eta^2=0.71$] and speaking rate condition [$F(1,16)=31.98$, Huynh-Felt p

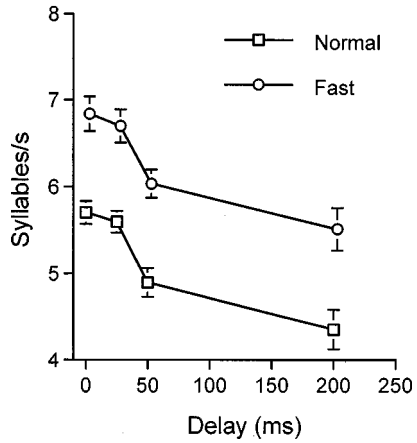

FIG. 2. Mean speech rates (in syllables/s) as a function of DAF and speech rate. Error bars represent plus/minus one standard error of the mean.

<0.0001, $\eta^2=0.66$] were found. The effect sizes of these significant main effects were large (Cohen, 1988). A nonsignificant DAF by speaking rate condition was found [$F(3,48)=0.02$, Huynh-Felt $p=0.99$, $\eta^2=0.001$, $\phi=0.054$ at $\alpha=0.05$]. *Post-hoc* orthogonal single-*df* comparisons revealed that there was no significant difference between syllable rates at 0 and 25 ms ($p>0.05$), they were significantly greater than 50 and 200 ms syllable rates, and the 50 ms was significantly greater than the 200 ms syllable rate ($p<.05$). In other words, participants were able to increase syllable rate when they were asked to speak fast under all DAF conditions. Participants decreased syllable rate at 50 and 200 ms during both speech rates relative to 0 and 25 ms DAF.

IV. DISCUSSION AND CONCLUSIONS

The present findings are threefold: First, DAF induced more significantly more dysfluencies only at the longest delay (i.e., 200 ms). In other words, normal speakers were capable of producing fluent or nearly fluent speech with short auditory feedback delays (i.e., $\leqslant$50 ms) that were equivalent to speech produced with no delay (i.e., 0 ms). Second, more dysfluencies were evident at a fast rate of speech. This finding would be consistent with increased motor load (Abbs and Cole, 1982; Borden, 1979; Borden and Harris, 1984). Finally, consistent with previous research (Black, 1951; Ham *et al.*, 1984; Lee, 1950; Siegel *et al.*, 1982; Stager and Ludlow, 1993), reduced speech rate was evidenced at auditory feedback delays greater than 25 ms with a greater reduction in syllable rate with an increase in DAF (i.e., 200 relative to 50 ms).

These findings suggest that temporal alterations in auditory feedback signal impact the speech-motor control system differentially for people who stutter and those that do not. That is, at delays of $\geqslant$50 ms individuals who stutter experience significant reductions (i.e., approximately 90%) in stuttering frequency (e.g., Kalinowski *et al.*, 1996) while in contrast normal speakers begin to experience dysfluent behavior at delays of $>$50 ms. What remains is a parsimonious explanation for two apparent paradoxical effects in altered auditory feedback.

Models of normal and stuttered speech production/monitoring have generally discounted the role of auditory feedback of having any significant role or any direct impact on central speech production commands since it is too slow (Borden, 1979; Levelt, 1983, 1989). As recognition of running speech is possible only at approximately 200 ms following production (Marslen-Wilson and Tyler, 1981, 1983), one could suggest that it should be of no surprise that the disruption of running speech production does not occur at auditory feedback of delays less than 200 ms in normal speakers. That is, peripheral feedback mechanisms (audition, taction, and/or proprioception) are affecting central speech motor control.

What then is the role of DAF in reducing dysfluency in those who stutter? It was generally posited that the stuttering reducing properties of DAF were due to an altered manner of speaking, specifically syllable prolongation, and not to any antecedent in the auditory system (Costello-Ingham, 1993; Perkins, 1979; Wingate, 1976). However, the role of the auditory system and DAF was revised by Kalinowski *et al.* (1996) who suggested that if a slow speech rate was necessary for stuttering reduction, then the stuttering reducing properties of DAF should not be evident when individuals who stutter speak at a fast speech rate. They had individuals who stutter read passages under conditions of altered auditory feedback including DAF at normal and fast rates of speech. Their results showed that stuttering episodes decreased significantly by approximately 70% under DAF regardless of speaking rate. These findings contradicted the notion regarding the importance of syllable prolongation to fluency induced by DAF. It was not suggested that syllable prolongation is unimportant to stuttering reduction *per se*, but rather, when syllable prolongation is eliminated, such as when speaking at a fast rate, the stuttering reduction properties of DAF are just as robust and can be most likely attributed to their impact on the auditory system.

How then can DAF impact the auditory system of individuals who stutter, particularly at short delays? Recent findings from brain imaging studies provide some answers. Magnetoencephalography (MEG) offers excellent temporal resolution (i.e., ms) in the analysis of cerebral processing in response to auditory stimulation. It has been known for more than a decade that a robust response (M100) is generated in the supratemporal auditory cortex in response to auditory stimuli beginning 20 to 30 ms and peaking approximately 100 ms after stimulus onset (Näätänen and Picton, 1987). More recently it has been demonstrated that an individual's own utterances can reduce the M100 response. Curio *et al.* (2000) examined such during a speech/replay task. In the speech condition participants uttered two vowels in a series while listening to a random series of two tones. In the replay condition the same participants listened to the recorded vowel utterances from the speech condition. The self-produced recorded vowels evoked the M100 response in the replay condition. More interestingly, this response was significantly delayed in both auditory cortices and reduced in amplitude prominently in the left auditory cortex during speech production of the same utterances in the speech condition. Similar findings of inhibition of cortical neurons have been found with primates during phonation (Müller-Preuss *et al.*, 1980; Müller-Preuss and Ploog, 1981). These data have been interpreted to indicate central motor-to-speech priming in the form of inhibition of the auditory cortices during speech production (Curio *et al.*, 2000).

The implications of these findings can lead one to speculate that this motor-to-speech priming may be defective in individuals who stutter. There is evidence to suggest that this is the case: Salmelin *et al.* (1998) reported in another MEG study that the functional organization of the auditory cortex is different in those who stutter relative to normal fluent speakers. MEG was recorded while individuals who stutter and matched controls read silently, read with oral movement but without sound, read aloud, and in chorus with another while listening to tones delivered alternately to the left and right ears. M100 responses were the same in the two silent conditions but delayed and reduced in amplitude during the two spoken conditions. Although the temporal response of the M100 was similar between the two groups, response amplitude was not. An unusual interhemispheric balance was

evident with the participants who stuttered. The authors reported ''rather paradoxically, dysfluency was most likely to occur when the hemispheric balance in stutterers become more like that in normal controls...dysfluent *versus* fluent reading conditions in stutterers were associated with differences specifically in the left auditory cortex...[and] source topography also differed in the left hemisphere'' (p. 2229). It has been suggested that suppression and/or delay of the M100 response during tasks reflects a diminution in the number or synchrony of auditory cortical neurons available for processing auditory input—in the case of speech production and perception (Hari, 1990; Näätänen and Picton, 1987). Salmelin *et al.* (1998) suggested that the interhemispheric balance is less stable in those who stutter and may be more easily unhinged with an increased work load (i.e., speech production). Disturbances may cause transient unpredictable disruptions in auditory perception (i.e., motor-to-speech priming after Curio *et al.*, 2000) that could initiate stuttering. Salmelin *et al.* (1998) pointedly remarked that, during choral reading where all participants who stutter were fluent, left hemispheric sensitivity was restored. This may be the case with all fluency-enhancing conditions of altered auditory feedback including DAF. The left auditory cortex as the locus of discrepancy between fluent speakers and those with stuttering has been implicated in numerous other brain imaging studies (e.g., Braun *et al.*, 1997; De Nil *et al.*, 2000; Fox *et al.*, 2000; Wu *et al.*, 1995). There is also recent converging evidence implicating anomalous anatomy (i.e., planum temporal and posterior superior temporal gyrus) in persons who stutter (Foundas *et al.*, 2001). It remains to be seen if this is a cause or effect of stuttering. Further research is warranted.

Finally, considering the contrast in fluency/dysfluency exhibited between normal speakers and those who stutter and the differences in the functional organization in the brain between individuals who stutter and fluent speakers, it appears that speech disruption of normal speakers under DAF is a poor analog of stuttering. MEG studies have implicated the role of the auditory system on a central level and on a time scale compatible with the behavioral effects of DAF on the overt manifestations of the disorder. The data herein implicate the peripheral feedback system(s) of fluent speakers for the disruptive effects of DAF on normal speech production.

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