Feedback and Feedforward Control in Speech Production in Apraxia of Speech and Aphasia

Background

Apraxia of speech (AOS) is considered to be a speech motor planning impairment (e.g., McNeil et al., 2009), but the nature of this impairment remains poorly understood. The present study was designed to test two hypotheses about the nature of AOS, framed in the DIVA model (Guenther et al., 2006). The DIVA model assumes that speech targets are regions in auditory space, and combines two control mechanisms to reach those targets: feedback control and feedforward control. The feedback mechanism generates corrective motor commands when the actual speech sound deviates from the intended speech sound. The feedforward mechanism generates predictive motor commands based on past experiences with the speech target.

In the context of the DIVA model, we developed two hypotheses about possible underlying deficits in AOS. The Feedforward Control Impairment (FF) hypothesis states that feedforward control is impaired in AOS, with consequently a greater reliance on feedback control (Jacks, 2008). The Feedback Control Impairment (FB) hypothesis states that feedback control is impaired in AOS; concurrent feedback may be disruptive (cf. Ballard & Robin, 2007).

We tested these hypotheses by measuring acoustic vowel contrast in two conditions: normal listening and auditory feedback masking. Under masking conditions, unimpaired speakers maintain segmental contrast (suggesting adequate feedforward commands to support speech without auditory feedback) even though contrast is somewhat reduced (suggesting on-line use of auditory feedback) (Perkell et al., 2007). The FF hypothesis predicts a greater reduction of segmental contrast with feedback masking in speakers with AOS than in controls, because effective removal of the auditory feedback control strategy will reveal the impaired feedforward commands. The FB hypothesis, in contrast, predicts increased segmental contrast with feedback masking, because removal of auditory feedback will allow the intact feedforward commands to produce adequate contrasts. One previous study that used feedback masking in AOS examined vowel duration and found longer vowels with masking in AOS and controls (Rogers et al., 1996); the present study also examined vowel duration.

Methods

Participants

Participants included six participants with AOS and varying degrees of aphasia, two speakers with aphasia without AOS, and nine age-matched control speakers (Table 1). AOS was diagnosed by three independent raters (one clinical researcher, two SLPs) based on presence of slow speech with longer segment and intersegment durations, dysprosody, distortions and distorted substitutions, and segmental errors that were relatively consistent in type (distortions) and location within the utterance. Normal rate and prosody were exclusionary criteria for the diagnosis of AOS (Wambaugh et al., 2006).
Materials & Procedures

Targets were five vowels /i ɛ æ ʌ u/ in /bVt/ words in a carrier phrase (“a ____ again”). Participants saw the phrase and associated picture and waited for a go-signal to say the phrase (Figure 1). In the masking condition, speech-shaped noise was presented over headphones at 95 dB-SPL (Perkell et al., 2007) during production, effectively removing self-produced auditory feedback. A sound level meter was used to maintain comparable loudness between conditions. Phrases were presented in random order within each block, and the 16 blocks alternated between normal (Silence) and masking (Noise) blocks.

Analyses

Formant frequencies (F1 and F2) for perceptually correct tokens were extracted at three points in the vowel (20%, 50%, 80%; Jacks et al., 2010) using Praat (Boersma & Weenink, 2008), converted into Mel-space (Perkell et al., 2007), and used to derive the primary dependent measure Average Vowel Spacing (AVS: mean of Euclidian distances between all vowel pairs; Perkell et al., 2007). At the group level (AOS group and controls), AVS was analyzed with a 2 (Group) x 2 (Condition) x 3 (TimePoint) ANOVA, and vowel duration with a 2 (Group) x 2 (Condition) x 5 (Vowel) ANOVA, with Tukey posthoc tests. Each individual speaker (including speakers with aphasia without AOS) was further compared to controls using Crawford and Garthwaite’s (2007) single-case methods for main effects and interactions. Alpha level was 0.05 for all analyses.

Results

Average Vowel Spacing (AVS)

AVS data are presented in Figure 2. There was no main effect of Group (F<1), but there was a significant effect of TimePoint (F[2,26]=18.55, p<0.001) indicating lower AVS at 20% (326 Mels) than at 50% (358 Mels) and 80% (361 Mels). A significant effect of Condition (F[1,13]=14.68, p=0.002) indicated greater AVS in the Silence condition (357 Mels) than in the Noise condition (340 Mels). Critically, the Group x Condition interaction was significant (F[1,13]=11.52, p=0.005), reflecting a contrast reduction for the AOS group (Silence 363 Mels vs. Noise 323 Mels) but not for controls (354 vs. 351 Mels). The absence of interactions with TimePoint (all Fs<1.3, ps>0.28) indicated that the Condition effect for the AOS group and the Group x Condition interaction were present at all time points.

All speakers with AOS demonstrated the group pattern (greater contrast reduction than in controls) numerically; for three participants this pattern reached the level of a trend or significance. Neither speaker with aphasia without AOS demonstrated this pattern.

Vowel Duration
Vowel duration data are presented in Figure 3. There was a significant main effect of Group ($F[1,13]=11.66, p=0.005$), indicating longer vowel duration for the AOS group (182 ms) than in the control group (142 ms). There was also a main effect of Condition ($F[1,13]=24.77, p<0.001$); vowels were longer in the Noise condition (168 ms) than in the Silence condition (148 ms). A main effect of Vowel ($F[4,52]=25.91, p<0.001$) reflected longer duration for /æ/ than for all other vowels, and the Condition x Vowel interaction ($F[4,52]=4.38, p=0.004$) indicated a difference between /ʌ/ and /u/ in the Noise condition but not in Silence, and between /ɪ/ and /ʌ/ in Silence but not in Noise. All vowels showed the Condition effect (Noise > Silence). There were no interactions with Group ($Fs<1.7, ps>0.16$).

Four speakers with AOS showed significantly longer vowels than controls in Silence and three in Noise. None of the speakers with AOS showed disproportionate Condition effects on vowel duration, except AOS_205 who showed a reverse pattern (longer vowel duration in the Silence condition). While Aph_304 showed a disproportionate Condition effect, neither patient with aphasia without AOS demonstrated longer duration than older controls.

**Discussion**

Compared with age-matched controls, speakers with AOS showed (1) comparable overall acoustic vowel contrast but a disproportionate reduction with noise masking, (2) longer overall vowel duration but a comparable lengthening effect under masking. Increased vowel duration was expected given previous literature (e.g., Kent & Rosenbek, 1983; Rogers et al., 1996).

The disproportionate reduction in contrast in the AOS group compared to controls was predicted by the FF hypothesis, and is inconsistent with the FB hypothesis. Thus, the present findings delineate the nature of AOS and suggest that feedforward control is impaired (cf. Jacks, 2008). Speakers with AOS appear to rely to a greater extent than controls on auditory feedback to achieve and maintain adequate segmental contrasts. Neither of the two speakers with aphasia without AOS demonstrated this pattern, suggesting that this disproportionate contrast reduction may be specific to AOS and not attributable to aphasia.

These findings highlight the importance of auditory feedback in AOS, and may enable development of more objective and specific diagnostic tools to identify underlying impairments in speakers with AOS, with possible implications for treatment candidacy considerations. Finally, this study supports the utility of current detailed models of speech motor control in understanding neurological communication disorders.
REFERENCES


Table 1. Patient information.

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Hand</th>
<th>Education (yrs)</th>
<th>Language</th>
<th>Hearing</th>
<th>Time post (y;m)</th>
<th>Etiology</th>
<th>Lesion</th>
<th>Aphasia Type</th>
<th>AOS severity</th>
<th>AOS rating</th>
<th>Dysarthria</th>
<th>Oral apraxia</th>
<th>Limb apraxia</th>
</tr>
</thead>
<tbody>
<tr>
<td>58</td>
<td>M</td>
<td>R</td>
<td>12</td>
<td>AE</td>
<td>pass</td>
<td>4:6</td>
<td>LH-CVA</td>
<td>B; I, PreC</td>
<td>WNL</td>
<td>mild-moder.</td>
<td>2.7</td>
<td>none</td>
<td>mild</td>
<td>none</td>
</tr>
<tr>
<td>68</td>
<td>M</td>
<td>R</td>
<td>22</td>
<td>AE</td>
<td>pass</td>
<td>6:9</td>
<td>LH-CVA</td>
<td>I; PF; P</td>
<td>Anomic</td>
<td>mild-moder.</td>
<td>3.0</td>
<td>none</td>
<td>mild</td>
<td>none</td>
</tr>
<tr>
<td>67</td>
<td>M</td>
<td>R</td>
<td>12</td>
<td>SE</td>
<td>pass</td>
<td>2:7</td>
<td>LH-CVA</td>
<td>FT</td>
<td>Broca’s</td>
<td>severe</td>
<td>3.0</td>
<td>none</td>
<td>mild</td>
<td>mild</td>
</tr>
<tr>
<td>56</td>
<td>F</td>
<td>R</td>
<td>12</td>
<td>AE</td>
<td>pass</td>
<td>5:6</td>
<td>Tumor &amp;</td>
<td>n/a</td>
<td>Anomic</td>
<td>mild</td>
<td>2.7</td>
<td>mild</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>59</td>
<td>F</td>
<td>R</td>
<td>13</td>
<td>SE</td>
<td>pass</td>
<td>5:1</td>
<td>LH-CVA</td>
<td>PT; P; IO</td>
<td>Wernicke</td>
<td>mild</td>
<td>2.3</td>
<td>mild</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>72</td>
<td>M</td>
<td>R</td>
<td>16</td>
<td>AE</td>
<td>pass</td>
<td>7:6</td>
<td>LH&amp;CVA</td>
<td>B; FT; P</td>
<td>Conduction</td>
<td>mild</td>
<td>2.0</td>
<td>none</td>
<td>mild</td>
<td>none</td>
</tr>
<tr>
<td>68</td>
<td>F</td>
<td>R</td>
<td>15</td>
<td>AE</td>
<td>pass</td>
<td>7:10</td>
<td>LH-CVA</td>
<td>T; O; PF</td>
<td>none</td>
<td>none</td>
<td>1.0</td>
<td>none</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>64</td>
<td>M</td>
<td>R</td>
<td>15</td>
<td>AE</td>
<td>pass</td>
<td>2:10</td>
<td>LH CVA</td>
<td>none</td>
<td>none</td>
<td>none</td>
<td>1.3</td>
<td>none</td>
<td>none</td>
<td>none</td>
</tr>
</tbody>
</table>

AE = American English; SE = Spanish-English bilingual; BE = British English; LH = Left hemisphere; RH = Right hemisphere; CVA = cerebro-vascular accident; n/a = not available; B = Broca’s area; I = Insula; PreC = Precentral gyrus; FP = Frontoparietal; PL = Parietal; FT = Fronto-temporal; PT = Posterior & middle Temporal lobe; IO = Inferior Occipital lobe; T = Temporal lobe; PF= Posterior Frontal lobe; O = Occipital lobe.

1 Pure tone hearing screening at 500, 1000, 2000, and 4000 Hz; pass at 45 dB level for better ear.

2 Based on WAB-R (Kertesz, 2006), except for Aph_304 (based on BDAE-3; Goodglass et al., 2000)

3 Based on ABA-2 (Dabul, 2000)

4 Mean rating across three diagnosticians (1 = no AOS, 2 = possible AOS, 3 = AOS) (cf. Haley et al., 2012).

5 Dysarthrias were diagnosed perceptually based on a motor speech exam (Duffy, 2005) and were all of the unilateral upper motor neuron type.
Figure 1. Experimental task overview.
Figure 2. Average Vowel Spacing (AVS; in Mels) across time points for age-matched controls (AMC) vs. AOS group (top), and individual patients vs. AMC group. Circles represent age-matched control group data in all figures. Note different AVS range for Aph_304.
Figure 3. Vowel duration by condition, collapsed across vowels, for the age-matched control group, the AOS group, and individual patients. Error bars represent standard error.