Apraxia of Speech: Impaired Access to Motor Programs or Damaged Motor Programs?

Definitions of apraxia of speech (AOS) describe this motor speech disorder as an impairment in planning and programming of speech movements (Duffy, 2005). The exact underlying mechanism of the impairment, however, is not well understood. The purpose of this investigation is to specify the speech planning impairment in AOS by testing two specific hypotheses framed in the DIVA model (Guenther, 2006).

Previous accounts of AOS

Several studies have investigated the underlying nature of AOS. Whiteside and Varley (1998) claim that the problem involves accessing learned motor programs while Aichert and Ziegler (2004) argue that the programs themselves are damaged. However, both accounts were based on a model that does not address speech motor control in detail (Levelt et al., 1999). Moreover, both accounts were based on data from off-line tasks and did not examine speech motor planning in real time.

Speech motor planning in the DIVA model

The present research was based on the DIVA model (Guenther, 2006), which is a model of speech motor control. In this model, speech motor planning takes place primarily in the acoustic domain. It begins by activating a speech sound map (SSM) cell, which can be activated by production or perception of a speech sound. The SSM cell, in turn, activates its auditory target region (i.e., expected feedback for the motor program to be executed) and the feedforward commands constituting the learned motor routines for speech sounds. Because hearing words can activate the SSM and interfere with the speech planning process we designed an experiment based on the auditory interference paradigm to examine two hypotheses. Hypothesis 1 (Damaged Program Hypothesis; DPH) states that the problem in AOS is damaged speech motor programs, i.e. damage to the feedforward commands from the SSM. Hypothesis 2 (Retrieval Hypothesis; RH) states that the problem is in activating or retrieving otherwise intact motor programs. In this case, there is difficulty activating or selecting the intended SSM cell.

Method

Participants

To date, data have been analyzed for two people with AOS and aphasia, four age-matched control speakers (AMC), and eight young control speakers (YCON) (Table 1).

Task and procedures

Because the problem in AOS is thought to lie in the motor planning level of speech production, we used a modified self-select picture naming paradigm (cf. Maas et al., 2008; see Figure 1) in which we manipulated the phase immediately preceding speech onset by sometimes playing a distracter word over headphones. Participants had an opportunity to prepare the response to reduce confounds due to word-finding problems. The experiment consisted of 14

blocks (12 trials per block). Erroneous responses were rerun at the end of each block. Participants heard distracter words in their own voice (recorded in a previous session).

Materials

Experimental targets included six monosyllabic nouns with a CVC structure (e.g. *bed*), elicited by line-drawn color pictures. There were also six filler CVC targets. The distracter words were of three different categories: (1) *Consonant-Vowel (CV) overlap,* where the target and the distracter shared the first two segments (*bed* and *bell*), (2) *Consonant (C) overlap,* where the first segment of the target and distracter were the same (*bed* and *bill*), and (3) *unrelated* words (*bed* and *car*). A fourth condition was a *baseline* condition, in which no distracter was presented.

Design and Analyses

Dependent variables were median reaction time (RT) of correct responses, measured by voice-key from the go-signal to the onset of the speech signal, and percent error for two main error types: (1) *intrusions*: participant produces all (1a) or part (1b) of the distracter instead of target; (2) *other errors*, broken down into *non-contextual sound errors* (2a: participant substitutes, adds or self-corrects sounds that are not present in the distracter) and *other errors* (2b). RT data for each control group were analyzed separately with a one-way ANOVA. Patient RT data were compared to the age-matched control data using Crawford and Garthwaite's (2007) Bayesian comparison method. Given the small control sample, alpha was set at 0.1.

The DPH predicts that there should be no difference in the speech errors and RT whether a distracter is presented or not, because the feedforward commands are damaged regardless of whether the SSM cell is more activated or not. In contrast, the RH predicts more speech errors and longer RT in trials where a distracter is presented (compared to silence), because the distracter will activate an incorrect SSM cell.

Results

Errors

Speakers from either control group made few if any mistakes (AMCON: 0.7%; YCON: 0.3%) and therefore will not be discussed further. Interestingly, the two patients had very different error patterns (Figure 2). Participant 201 made few intrusion errors (no whole-word intrusions) and his error rate decreased as the overlap between the distracter and the target increased over conditions. Participant 202 made several intrusion errors in the C-overlap and CV-overlap conditions, most of which were whole-word intrusions; his overall error rate was lowest in the baseline condition and increased as the overlap between the distracter and the target increased, which appeared driven primarily by intrusion errors.

Reaction times

Median RTs are depicted in Figure 3. Although both control groups had numerically longer RTs in the baseline condition compared to distracter conditions, these differences were not significant in either group (YCON: $F[3,21]=1.45$, $p=0.2564$; AMCON: $F<1$, n.s.). In

contrast, both speakers with AOS showed *longer* RTs in conditions where a distracter was present compared to the baseline condition. This difference was supported by the Crawford and Garthwaite (2007) analyses (Table 2): both patients differed significantly from age-matched controls in the unrelated and C-overlap conditions but not in the baseline condition.

Discussion

These preliminary data indicate that for control speakers, there is no effect of distracter (presence or type) on errors or RT. This differs from the auditory-picture word interference literature, where related distracters show facilitation compared to unrelated distracters, the so-called phonological priming effect (Meyer & Schriefers, 1991). This discrepancy supports the idea that the distracters in our task do not tap into the phonological planning stage but rather into a later, speech motor planning stage.

In this light, the findings of the patients revealed interesting patterns. With respect to RTs, both patients showed a remarkably similar pattern, namely slower RT when distracters were presented than when no distracter was present. This is consistent with the RH, and suggests that distracter words may activate incorrect SSM representations that cause interference, thereby slowing selecting of the target.

The error patterns revealed differences between the patients, though in both cases there was a notable effect of distracter type. In participant 201, the error rate decreased with greater overlap, suggesting that the distracter facilitated selection of the intended target (even though this process took longer, as evidenced by the RT data). For participant 202, more intrusions were noted when there was overlap suggesting that distracter words caused interference for selecting the intended target. Interestingly, the substantial number of whole-word intrusions in participant 202 but not in participant 201 may reflect differences in the size of planning units in the SSM.

Data from additional participants will be available at the conference. Together these initial findings hold promise for differential diagnosis of AOS and specification of possibly different types of apraxic speech motor planning impairments could inform clinical decisions in therapy.

References

Aichert, I., & Ziegler, W. (2004). Syllable frequency and syllable structure in apraxia of speech. *Brain and Language, 88*, 148–159.

Crawford, J. R., & Garthwaite, P. H. (2007). Comparison of a single case to a control or normative sample in neuropsychology: Development of a Bayesian approach. *Cognitive Neuropsychology, 24*, 343–372.

Duffy, J. R. (2005). *Motor Speech Disorders: Substrates, Differential Diagnosis, and Management (Second Edition)*. St. Louis, MO: Mosby-Year Book, Inc.

Guenther, F.H. (2006). Cortical interactions underlying the production of speech sounds. *Journal of Communication Disorders, 39,* 350–365.

Levelt, W. J. M., Roelofs, A., & Meyer, A. S. (1999). A theory of lexical access in speech production. *Behavioral and Brain Sciences, 22*, 1–38.

Maas, E., Robin, D. A., Wright, D. L., & Ballard, K. J. (2008). Motor programming in apraxia of speech. *Brain and Language, 106*, 107–118.

Meyer, A. S., & Schriefers, H. (1991). Phonological facilitation in picture-word interference experiments: Effects of stimulus-onset asynchrony and types of interfering stimuli. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 17,* 1146–1160.

Whiteside, S.P., & Varley, R.A., (1999). Coarticulation in apraxia of speech: an acoustic study of non-words. *Logopedics Phoniatrics Vocology, 23,* 155–163.

Table 1. Participant information

|	AOS		AMCON	YCON
	201	**202**	**(N=4)**	**(N=8)**
Age	58	60	71 (9)	22.7 (5)
Sex	M	M	2F, 2M	F
WAB-R[a]				
Fluency	8	6	-	-
Comprehension	9.8	9.4	-	-
Repetition	9.4	3.3	-	-
Naming	9.4	5	-	-
Aphasia Quotient	*93.2*	*65.4*	-	-
Aphasia Type	*Anomic*	*Conduction*	-	-
ABA-2[b]				
Diadochokinetic rate	27 (no)	1 (se)	-	-
Increasing word length A	3 (mi)	9 (se)	-	-
Increasing word length B	8 (se)	-	-	-
Limb apraxia	35 (mi)	47 (no)	-	-
Oral apraxia	40 (mi)	40 (mi)	-	-
Utterance time	13 (no)	61 (mo)	-	-
Repeated trials	24 (mi)	4 (se)	-	-

[a] Western Aphasia Battery – Revised (Kertesz, 2006)
[b] Apraxia Battery for Adults – Second Edition (Dabul, 2000)

Figure 1. Self-select picture naming paradigm
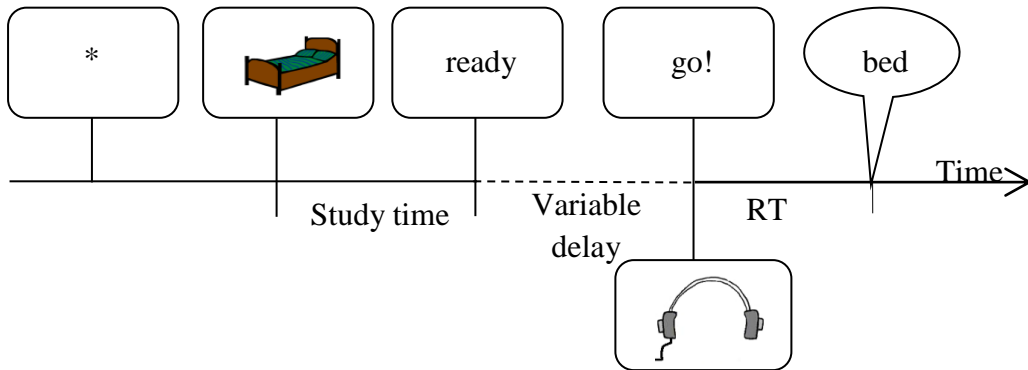
Figure 2. Error data for individuals with AOS. An example of a target and its distracter words are given in parentheses.

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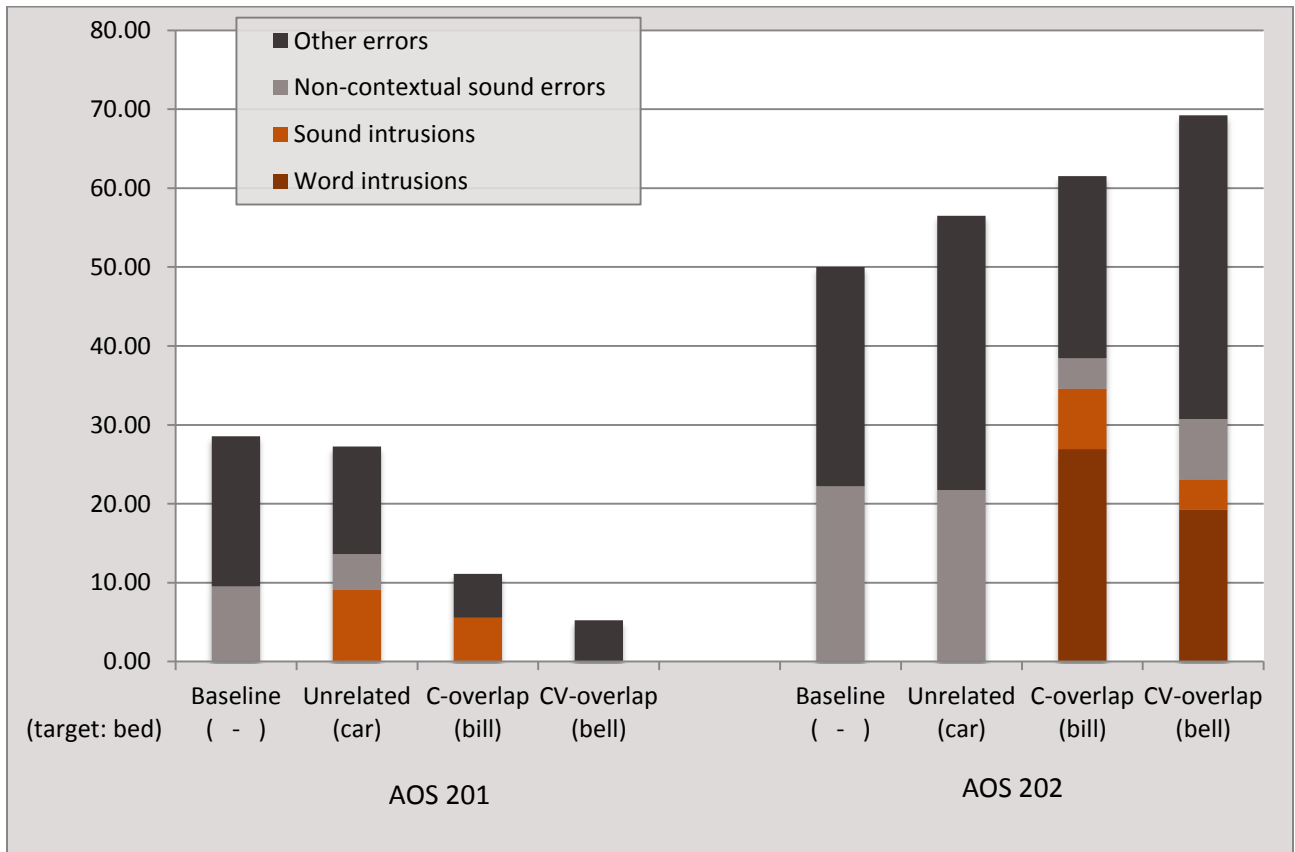
Table 2. RT medians (SD) data with results of individual statistical analyses based on Crawford & Garthwaite (2007): comparisons of participants with AOS to the control group (AMCON) by condition.

Group	Baseline	Unrelated	C-overlap	CV-overlap
AMCON (N=4)	502 (171)	500 (206)	495 (193)	498 (214)
201	745	1050	1031	1051
p-value	*0.29*	*0.09**	*0.08**	*0.10*
Effect size (Z)	*1.42*	*2.67*	*2.77*	*2.58*
202	890	1363	1300	1055
p-value	*0.13*	*0.03***	*0.03***	*0.10*
Effect size (Z)	*2.26*	*4.18*	*4.17*	*2.60*

[*] significant at *p<.1*
[**] significant at *p<.05*

Figure 3. RT data by condition. Error bars represent standard error; asterisks mark a significant difference between the individual participants with AOS and AMCON group by condition (* significant at *p<.1*;   ** significant at *p<.05*).