Proceedings of the Institute of Acoustics

REDUCED COARTICULATION IN APRAXIA OF SPEECH: SOME ACOUSTIC EVIDENCE

S. P. Whiteside & R. A. Varley

Department of Human Communication Sciences, University of Sheffield, Sheffield S10 2TA, UK

1. INTRODUCTION

Acquired apraxia of speech (AOS) occurs following damage to anterior portions of the language dominant hemisphere. There are varying accounts on AOS (Lebrun, 1990), but it is most often described as a disorder in the phonetic/motoric implementation of the linguistic message (Kent & McNeil, 1987; McNeil, Liss, Tseng & Kent, 1990; Ryalls, 1987). The main characteristics of AOS include: inconsistent and variable articulatory movements (Hardcastle, 1987; Itoh, Sasanuma & Ushijima, 1979); increased word and vowel duration patterns (Collins, Rosenbek & Wertz, 1983; Hardcastle, 1987; Kent & Rosenbek, 1983; Skenes, 1987); a general slowed rate of speaking with resulting prolongations of transitions, segments and intersyllable pauses (Kent & Rosenbek, 1983; Sasanuma, 1971); a limited variation in relative peak intensity across syllables resulting in abnormal stress and rhythm patterns (Kent & Rosenbek, 1983; Sasanuma, 1971); voicing errors (Freeman, Sands & Harris, 1978; Itoh, Sasanuma, Tatsumi, Murakami, Fukusako & Suzuki, 1982; Kent & Rosenbek, 1983); segmental errors (Kent & Rosenbek, 1983); reduced coarticulation patterns (Zeigler & von Cramon, 1985, 1986) and less coarticulatory cohesion (Chinnery, Docherty & Walshaw, 1995; Weismer & Liss, 1991; Zeigler & von Cramon, 1986). These speech patterns are all symptomatic of a disruption in the phasing of movements between subcomponents in the speech production system (Square-Storer & Roy, 1989, Zeigler & von Cramon, 1986) and in the spatial configurations of articulatory movements (Square-Storer & Roy, 1989).

Coarticulation performs a crucial function in contemporary models of speech production (e.g. Recasens, 1984, 1985, 1987, 1991) and recently, it has attracted interest in an attempt to explain patterns in AOS (Zeigler & von Cramon, 1985, 1986). In the earliest of these studies, Zeigler & von Cramon (1985) found that listeners were able to identify gated vowels in the samples of a speaker with AOS at a much later stage in the vowel relative to those of normal speakers. They interpreted this finding as evidence for reduced anticipatory coarticulation in the speaker with apraxia. They supported this interpretation with an acoustic study of formant frequency (for vowel segments) and linear predictive reflection coefficient analysis (for the burst and aspiration noise), which showed that there was a delay in anticipatory vowel gestures and therefore reduced 'cohesiveness in apraxic speech gestures' (Zeigler & von Cramon, 1986).

Another approach in the investigation of coarticulation in apraxic speech has involved the use of F2 locus equations to describe consonant vowel coarticulation patterns (Chinnery, Docherty & Walshaw, 1995). F2 locus equations were first applied by Lindblom (1963) and have been investigated for normal speech in a number of recent studies (Sussman, McCaffrey & Matthews, 1991; Sussman, Hoemeke & McCaffrey, 1992; Sussman, 1994; Sussman & Shore, 1996). Locus equations are useful phonetic descriptors (Sussman & Shore, 1996) which depict the linear relationship between the F2 midvowel frequencies (plotted along the x-axis) and F2 vowel onset frequencies (plotted along the y-axis) of CVC syllables. Locus equations are expressed by simple regression functions as $F_2onset = k * F_2vowel + c$, where k represents the slope of the function and c, the y-intercept. The slopes of these regression functions vary according to the place of articulation. Generally, the slopes are the greatest for /b/ (with the lowest y-intercept values), and the lowest for /d/ (with the highest y-intercept values), with /g/ having intermediate slope and y-intercept values (Fowler, 1994; Sussman, McCaffrey & Matthews, 1991; Sussman, Hoemeke

& McCaffrey, 1992). The steepness of slopes is an indicator of the extent to which consonant and vowel coarticulate. /b/ therefore has the greatest degree of CV coarticulation, because the lips are not involved in vowel production, which leaves the vowel free to coarticulate with /b/. /d/ in contrast, has the flattest slope because it's production involves the tongue, which means that the vowel is not as free to coarticulate with /d/.

In the present study we present speech data collected from a speaker with AOS and a matched normal subject. We examine a number of acoustic measures acknowledged as being evidence for coarticulation, namely F2 locus equations (Chinnery, Docherty & Walshaw, 1995; Sussman, McCaffrey & Matthews, 1991; Sussman, Hoemeke & McCaffrey, 1992; Sussman, 1994; Sussman & Shore, 1996) and vowel-to-consonant (e. g. Recasens, 1984, 1985, 1987, 1991), consonant-to-vowel (e.g. Recasens, 1985, 1991) and vowel-to-vowel coarticulation patterns (e.g. Recasens, 1987, 1991; Zeigler & von Cramon, 1986). The results are presented and discussed within a new conceptualisation of apraxia of speech (Whiteside & Varley, 1998), by synthesising acoustic evidence from a variety of sources with contemporary psycholinguistic models of phonetic encoding (Levelt & Wheeldon, 1994) and speech motor control (Keller, 1987).

2. METHOD

2.1 Subjects

AD is a 48-year-old retired police officer who had a sub-dural empyema in the left sylvian fissure with an accompanying meningitis four years prior to this study. Subsequent to the evacuation of the empyema, AD experienced aphasic difficulties and a severe motor speech disorder. Following very detailed assessment AD was diagnosed as having a severe AOS. Critical issues in the diagnosis of AOS were: a severe disorder of speech which could not be explained by degree of movement deficit. Although AD showed no restriction in range and power of movement and only a small reduction in speed of movement, a word identification test by a panel of 26 listeners revealed only 4.3% intelligibility. AD also showed inconsistency in error and substitution errors in addition to phonetic distortions. AD was premorbidly right-handed.

In addition to the AOS, AD experience aphasic difficulties. Lexical input processing was relatively intact across spoken and written modalities (PALPA (Kay, Lesser & Coltheart, 1992) spoken word-picture matching 35/40, written word-picture matching 37/40). Lexical output revealed a greater level of impairment. Objective scores for the speech channel were difficult to obtain because of severely compromised intelligibility. Written naming produced a score of 24/60 (PALPA written picture naming). AD had severe difficulties in both input and output grammatical processing. His writing was agrammatic, consisting only of nouns and adjectives. On a test of sentence comprehension, AD performed at chance levels (PALPA auditory 23/60, written 13/30) and he also had verb comprehension difficulties.

RM, who served as the control in this study, is a 48-year-old security officer at a local university. RM has normal hearing, speech and language, and has no history of neurological impairment. Both AD and RM were born and have lived in South Yorkshire.

2.2 Data and recording procedures

Data for AD was collected during two consecutive clinical sessions with a one week interval between them. In the first session, AD repeated a set of 30 monosyllabic CV or CVC real words (15 minimal pairs) after an experimenter. In the second session, the stimulus set was repeated to determine the consistency of productions. All of the speech stimuli were preceded by either 'a' or 'the' and contained voiced/voiceless minimal pairs with the phonemes /p b t d k g/ in initial position. The plosive class of consonants was

chosen because this was the only manner of articulation for which AD had some competence. The same data set was elicited from RM also using the repetition method described above. Speech stimuli from both AD and RM were recorded on a Sony TCD-D3 DAT recorder.

2.3 Procedures

All speech stimuli were digitized using a KAY Computerized Speech Lab (CSL) model 4300. A sampling rate of 10kHz was used. Speech pressure waveforms, wide-band spectrograms (146 Hz bandwidth, with pre-emphasis, Blackman window) and LPC spectral slices and formant tracks (for vowel analysis: 20 ms frame length, 12th order filter, with pre-emphasis, filter response window weighting, autocorrelation method; for burst analysis: 5 ms framelength with other settings as for vowel) were used for the analysis. Measurements were validated using these three methods of analysis. The following acoustic measurements were made for the stimuli.

For all stimuli ('the/a CVC/CV') that were on target:

- total utterance duration (ms);
- formant frequencies (F1 and F2: Hz) of the vowels and diphthongs taken at the temporal midpoint;
- first and second formant frequencies (F1 and F2 - Hz) of the mid point of the schwa preceding the CVC words - F1 and F2 values were later correlated with the vowel midpoints, F2 values were correlated with both second peaks at plosive burst and F2 vowel onset values.

For all stimuli containing bilabial plosives ('a bVC/bV'):

- second formant (F2: Hz) onset values measured at the initial glottal pulse of the vowel;
- the second peak, visible at the burst of the plosive;
- F2 locus equation scatterplots were then generated in line with previous research where F2 vowel midpoints are plotted as function of F2 vowel onset values (Chinnery, Docherty & Walshaw, 1995; Fowler, 1994; Krull, 1989; Lindblom, 1963; Sussman, 1994; Sussman, McCaffrey & Matthews, 1991; Sussman, Hoemeke & McCaffrey, 1992) and second peak at plosive burst values (Sussman & Shore, 1996);
- timelag durations (ms) were recorded as the interval between the F2 onset values (both at the burst onset and the vowel onset) and the vowel midpoint;
- F2 slopes (Hz/ms) were calculated as F2 onset (both at the burst onset and the vowel onset) minus F2 vowel midpoint divided by the timelag duration and

2.4 Detailed phonetic transcription

Two raters using the consensus method, transcribed AD's and RM's utterances using narrow phonetic notation (IPA 1993). These transcriptions were used to determine which data sets to include in some of the acoustic analyses. For example, only data samples where the place of articulation of consonants in the CVC/CV words matched the target were used in the measures for coarticulation.

3. RESULTS AND DISCUSSION

3.1 Utterance duration

Utterance duration values (ms) for both AD and RM indicated that the duration values for AD were significantly longer than those of RM. Wilcoxon tests showed these differences to be significant ($p<0.005$,

one-tailed) for all three places of articulation. When the utterance duration values were pooled for both speakers, AD and RM had mean duration values of 1591.1 ms (s.d. 157.8 ms) and 523.6 ms (s.d. 83.7 ms) respectively. This represents a mean utterance duration ratio value of 1:3 for speaker RM:speaker AD. This observation replicates that reported in previous studies, where apraxic speech patterns have been found to be longer than those of normal speakers (Collins, Rosenbek & Wertz, 1983; Kent & Rosenbek, 1983; McNeil, Liss, Tseng & Kent, 1990; Skenes, 1987).

3.2 F2 Locus Equations

Table 1 gives the data of locus equations for speaker AD (speaker with AOS) and speaker RM (control) for /p/ at burst (@B), /b/ @B, /p/ at vowel onset (@Vo), and /b/@Vo. These data are presented in a style based on Sussman (1994). Using regression analysis, results showed that for speaker AD, statistically significant results occurred for all locus equations except for those describing /b/ @B and /b/ @Vo. Of this data, the order of slope values was /p/@B > /p/@Vo (slopes of 0.595 and 0.529, respectively).

	Speaker AD (AOS)				Speaker RM (control)			
	k	c	R^2	p	k	c	R^2	p
/p/ at burst	0.595	480.35	0.591	0.0154*	0.333	1014.24	0.478	0.0128*
/b/ at burst	0.154	1159.4	0.258	0.1109 (ns)	0.258	1030.41	0.431	0.0204*
/p/ at vowel onset	0.529	480.0	0.559	0.0052**	0.573	643.1	0.737	0.0004***
/b/ at vowel onset	0.128	1127.6	0.127	0.2823 (ns)	0.406	788.9	0.645	0.0017**

Table 1: Locus equation slope (k), y-intercept (c), r^2 and p values for /p b/ for speakers AD (AOS) and RM (control). (ns: not significant, * $p<0.05$, ** $p<0.01$, *** $p<0.001$)

F2 locus equation results for speaker RM (control) were all significant. The order of slope values for the statistically significant data was /p/@Vo > /b/@Vo > /p/@B > /b/@B (slopes of 0.573, 0.406, 0.333 and 0.258, respectively). The order of slope values for RM's data, suggests that there was less coarticulation between the bilabial plosives and the subsequent vowel at the point of the plosive burst, compared to the point of the vowel onset. This pattern is expected given that, by the time voicing has commenced for the vowel, formant frequency values are much closer to those at vowel midpoint (Sussman & Shore, 1996).

3.3 Temporal and spectral aspects of F2

Tables 2, 3 and 4 give the data and results of single factor ANOVA tests for F2 timelag (ms), F2 difference (Hz) and F2 slope (Hz/ms) respectively, for the bilabial data of both speaker AD and speaker RM. The single factor ANOVA results revealed highly significant differences for the timelag values with AD showing much longer duration values. This is in line with the longer utterance duration data presented above. The only significant difference between RM and AD for all F2 differences (Hz) and F2 slope (Hz/ms) values, was found for the /b/@Vo F2 slope values.

	Speaker AD (AOS)		Speaker RM (control)		Results of single-factor ANOVA
Plosive category	Mean F2 timelag (ms)	s.d. (ms)	Mean F2 timelag (ms)	s.d. (ms)	
/p/ at burst	269.8	81.1	92.0	26.6	F(1, 23), F=52.1 p=0.0001***
/b/ at burst	295.9	114.1	63.7	27.5	F(1, 23), F=47.0 p=0.0001***
/p/ at vowel onset	238.2	81.0	52.7	26.3	F(1, 23), F=56.9 p=0.0001***
/b/ at vowel onset	275.1	102.3	51.8	27.2	F(1, 23), F=53.5, p=0.0001***

Table 2: Mean F2 timelag (ms) values for speakers AD (AOS) and RM (control): bilabial data. (***p<0.001).

	Speaker AD (AOS)		Speaker RM (control)		Results of single-factor ANOVA
Plosive category	Mean F2 Difference (Hz)	s.d. (Hz)	Mean F2 Difference (Hz)	s.d. (Hz)	
/p/ at burst	179.9	163.8	78.6	362.3	F(1, 20), F=0.605 p=0.4464 (ns)
/b/ at burst	156.1	275.3	185.3	372.0	F(1, 22), F=0.045 p=0.8337 (ns)
/p/ at vowel onset	239.8	214.1	74.3	263.5	F(1, 23), F=2.854 p=0.1053 (ns)
/b/ at vowel onset	228.6	290.6	183.4	310.0	F(1, 22), F=0.13 p=0.7224 (ns)

Table 3: F2 difference (Hz) values for speakers AD (AOS) and RM (control): bilabial data (ns: not significant).

3.4 Coarticulation-with-schwa

To determine levels of coarticulation with schwa in the CVC words, the F1 and F2 values (Hz) for the schwa were correlated with the F1 and F2 vowel midpoint values for all stimuli. This was done using Pearson's product moment correlation coefficient. These correlations are given in Table 5.

The bilabial data illustrate that for both the normal speaker (RM) and the speaker with AOS (AD), schwa patterns varied according to vowel context. However Pearson *r* values (Table 5) revealed that this covariation was only significant for the F1 patterns of the normal speaker RM. RM's F2 patterns were just below the significance level of $p<0.05$ and none of AD's patterns reached significance. It is suggested that the reason RM's F2 patterns of coarticulation just failed to reach significance can be partly explained by the inclusion of the minimal pairs a pill and a bill which contained the velarised alveolar lateral approximant [ɫ]. Schwa values for F2 suggested patterns of schwa-to-vowel anticipatory coarticulation,

with F2 values being higher for the upcoming front vowel (mean 1571 Hz). However, due to the anticipatory coarticulation of vowel-to-consonant tongue-backing velarisation gestures ([ɪ] to [ɫ]), F2 patterns in the midvowel points resulted in [ɪ] having a mean of 1523 Hz. This compared to a mean of 1837 Hz for the vowel midpoints in a pin and a bin where no tongue-backing velarisation gestures are necessary for the production of [n]. A similar effect of 'coarticulation resistance' for [ɫ] in British English R.P. was reported by Bladon & Al-Bamerni (1976). Recasens (1987) also found that this consonant allowed less V-to-C coarticulation in Catalan and Spanish due to 'a more constricted tongue-dorsum configuration'. Similar effects for [ɫ] were also reported for C-to-V sequences in Catalan (Recasens, 1991).

	Speaker AD (AOS)		Speaker RM (control)		Results of single-factor ANOVA
Plosive category	Mean F2 slope (Hz/ms)	s.d. (Hz/ms)	Mean F2 slope (Hz/ms)	s.d. (Hz/ms)	
/p/ at burst	0.7	0.7	1.6	8.6	F(1, 20), F=0.093 p=0.7632 (ns)
/b/ at burst	0.5	0.8	2.7	4.8	F(1, 22), F=2.385 p=0.1374 (ns)
/p/ at vowel onset	1.0	0.7	0.7	3.5	F(1, 23), F=0.1 p=0.7437 (ns)
/b/ at vowel onset	0.7	0.7	3.1	3.8	F(1, 23), F=4.8, p=0.0387*

Table 4: Mean F2 slope (Hz/ms) values for speakers AD (AOS) and RM (control): bilabial data (ns: not significant, * p<0.05).

		Speaker AD (AOS)		Speaker RM (control)	
Vowel midpoint		Pearson *r*	p value	Pearson *r*	p value
Bilabial data	F1(Hz)	0.128	0.5792 (ns)	0.411	0.0458*
	F2 (Hz)	0.049	0.8323 (ns)	0.398	0.0544 (ns)
Alveolar data	F1(Hz)	0.197	0.3685 (ns)	0.071	0.7423 (ns)
	F2 (Hz)	-0.366	0.1027 (ns)	0.705	0.0001***
Velar Data	F1(Hz)	0.334	0.3158 (ns)	0.494	0.1026 (ns)
	F2 (Hz)	-0.52	0.1012 (ns)	0.674	0.0163**

Table 5: Correlation coefficient (Pearson r) values for: 1) F1 and F2 values in preceding schwa and corresponding vowel midpoints; for speaker AD (AOS) and speaker RM (control): bilabial, alveolar and velar data (nd: lack of sufficient data; ns: not significant; * p<0.05; ** p<0.01; ***p<0.001).

The clearest depiction of anticipatory coarticulation in schwa-to-vowel effects is manifested in the patterns of the normal speaker (RM) for the velar data (Table 5). Here, we again we find a significant correlation for the F2 values of RM. If we concentrate on F2 as being the key indicator of coarticulation effects (Bladon & Al-Bamerni, 1976; Recasens, 1984, 1985, 1987, 1991), the data reported here show significant schwa-to-vowel coarticulation F2 patterns for most of RM's data, where reduced coarticulation patterns were due to coarticulation resistance imposed by articulatory constraints. However, in contrast, none of AD's F2 patterns demonstrated significant schwa-to-vowel coarticulation effects. These results therefore suggest reduced levels of anticipatory coarticulation and hence less articulatory cohesion in the speech patterns of the speaker with apraxia. This replicates studies reported by Mayer (1995) and Zeigler & von Cramon (1985, 1986) who also found reduced coarticulatory patterns in speakers with AOS, and also illustrates the syllabic nature of apraxic speech (Kent & Rosenbek, 1982).

4. GENERAL DISCUSSION

The data reported here on F2 locus equations and coarticulation-with-schwa acoustic patterns suggest that, compared to the control subject RM, the speaker with apraxia demonstrated lower degrees of C-to-V and V-to-V coarticulation. This lack of anticipatory coarticulation and the excessively long duration patterns for AD's speech samples replicate other studies of apraxic speech and suggest a loss of coarticulatory cohesion (Mayer, 1995; Zeigler & von Cramon, 1985, 1986). The data in this study is restricted to a data set from a single individual with left hemisphere damage and apraxia, and on the basis of this data it is not possible to conclude whether reduced coarticulation results specifically from apraxia, or, more generally, a motoric deficit. The results of Zeigler and von Cramon (1985), however, suggest that the critical factor is the apraxic disorder and not general motoric disorder or brain damage. These investigators presented a data set from a dysarthric subject which did not show the reduction in coarticulatory effects. Further research is, however, needed to compare coarticulation across different categories of brain damaged speakers.

Reduced coarticulation, and thus the fragmented nature of apraxic speech, is explained as being the result of disrupted motor control where each syllable is programmed independently (Kent & Rosenbek, 1982). The essence of this explanation is captured by the insight of a subject of Lecours and Lhermitte (1976, p. 93) who writes that, although speech production was once automatic, he has to 'syllabicate'. His articulation is 'no longer automatic and has to be commanded, directed' and 'each vowel, each consonant, in short each syllable' must be articulated with care. This loss of automaticity appears to be a key feature of apraxic speech where, as Lebrun (1990, p. 386) describes it, the memory of 'verbo-motor patterns' is disturbed and therefore, the pronunciation of a speaker with AOS is 'chaotic and dishevelled, and requires constant conscious control and checking'.

There has been some debate as to whether verbo-motor parameters are produced from storage or calculated anew in speech production (Keller, 1987). Keller supports the former view by arguing that the neurological system has the capacity to learn and store articulation parameters for the most frequently used CVC combinations with parameters for the least frequently used CVC sequences being calculated anew as and when necessary. This view is supported by contemporary psycholinguistic models which suggest that there are two possible routes in phonetic encoding (Levelt & Wheeldon, 1994). These models are mixed in that they propose the use of: i) stored syllabic schemas in a phonetic syllabary and ii) the on-line computation of syllables using sub-syllabic units. The first of these routes, which we shall term the 'direct route', contains abstract 'gestural scores' (Browman & Goldstein, 1992) which are then passed on to an 'articulatory network' which is a coordinative motor system that includes feedback mechanisms (Saltzman,

1986). This route is computationally more efficient in that it relies more on storage and less on on-line computation. It is proposed that this route would operate for high frequency words (Levelt & Wheeldon, 1994). In contrast, the second route, which we shall call the 'indirect' route (but described as 'direct' by Levelt & Wheeldon, 1994), relies heavily on on-line computation and is utilised for novel or very low frequency words. We also contend that this route would be used when greater 'care' and hence more conscious control of speech production is required.

We would like to propose that, for AOS, the underlying basis of this disorder, is a failure of direct route encoding and therefore a reliance on indirect mechanisms. We suggest that degrees of coarticulation patterns and syllable durations are critical indices in phonetic encoding routes, with coarticulation patterns increasing and syllable duration decreasing with direct route encoding. The evidence for our alternative conceptualisation of AOS comes not only from our acoustic data which demonstrates reduced coarticulation patterns for the speaker with apraxia (AD), but also from a number of other sources which have documented reduced coarticulation patterns in AOS (Zeigler & von Cramon, 1985, 1986). We propose that these sources of evidence suggest that the speech patterns exhibited by speakers with apraxia are symptomatic of the breakdown in direct phonetic encoding mechanisms and therefore a reliance on indirect phonetic encoding mechanisms. These 'indirect' mechanisms rely on the full on-line computation of all syllables. In normal speakers, with increased usage and practice of new and low frequency words, verbo-motor patterns will become more automatic with less reliance on indirect, on-line routes and therefore a greater reliance on direct, retrieval-from-storage lexical routes. However, because the storage and retrieval facilities of verbo-motor patterns are impaired in speakers with AOS, they are forced to rely on indirect phonetic encoding mechanisms which means computing syllables anew each time. Having to compute syllables on-line anew each time, may explain the *syllable segregation* and therefore the *articulatory prolongation* (Kent and Rosenbek, 1982) exhibited by speakers with apraxia.

This conceptualisation of apraxia of speech is different from a theory of apraxia as a motor programming disorder. This traditional view focuses on the notion of impaired indirect, assembly route mechanisms, with the suggestion that either segment-sized verbo-motor patterns are impaired or their assembly to form syllables. An important, and unresolved issue, is to why speakers with AOS are unable to encode speech by indirect route encoding. There may, however, be a sub-group of apraxic speakers who can compensate with a high degree of efficiency via indirect assembly mechanisms. There is a group of brain-damaged speakers who have speech characteristics that are suggestive of a failure to produce smooth and fully integrated speech movements. This group refers to speakers who are not usually diagnosed as having AOS, but are classified and described as having 'foreign accent syndrome' (FAS). The speech characteristics of these individuals include changes in vowel realisations, increased diphthongization (Graff-Radford, Cooper, Colsher & Damasio, 1986), consonantal pre-voicing and vowel epenthesis (Blumstein, Alexander, Ryalls, Katz & Dworetzky, 1987). It is possible therefore, that FAS is a sub-type of apraxia (Whiteside & Varley, in preparation). The issue of AOS representing a failure of direct route encoding and a reliance on inefficient indirect assembly has important implications for intervention. Usual management programs focus on segmental drills and combining segment to syllables i.e. attempting to tutor the indirect route in order to increase its efficiency. An alternative strategy might be to reaccess and enhance direct route encoding.

5. ACKNOWLEDGEMENTS

We wish to thank AD and RM who made this study possible.

6. REFERENCES

Bladon, R. A. W. & Al-Bamerni, A. 1976. Coarticulation resistance in English. *Journal of Phonetics*, 4, 137-150.

Blumstein, S. E., Alexander, M. P., Ryalls, J. H., Katz, W. and Dworetzky, B. (1987). On the nature of the Foreign Accent Syndrome: a case study. *Brain and Language*, 31, 215-244.

Browman, C. P. & Goldstein, L. 1992. Articulatory phonology: An overview. *Phonetica*, 49, 155-180.

Chinnery, C., Docherty, G. J. & Walshaw, D. 1995 Formant locus equations and coarticulation in dyspraxic speech. *Proceedings of the XIIIth International Congress of Phonetic Sciences*, 1, 90-93.

Collins, M., Rosenbek, J. C. & Wertz, R. R. T. 1983. Spectrographic analysis of vowel and word duration in apraxia of speech. *Journal of Speech and Hearing Research*, 26, 224-230.

Freeman, F. J., Sands, E. S. & Harris, K. S. 1978. Temporal coordination of phonation and articulation in a case of verbal apraxia: A Voice Onset Time Study. *Brain & Language*, 6, 106-111.

Graff-Radford, N. R., Cooper, W. E., Colsher, P. L. and Damasio, A. R. (1986). An unlearned foreign "accent" in a patient with aphasia. *Brain and Language*, 28, 86-94.

Hardcastle, W. J. 1987. Electropalatographic study of articulation disorders in verbal dyspraxia. In J. H. Ryalls (Ed.), *Phonetic approaches to speech production in aphasia and related disorders*. Boston: College-Hill Press.

Itoh, M., Sasanuma, S., Tatsumi, I., Murakami, S., Fukusako, Y. & Suzuki, T. 1982. Voice onset time characteristics in apraxia of speech. *Brain & Language*, 17, 193-210.

Itoh, M., Sasanuma, S & Ushijima, T. 1979. Velar movements during speech in a patient with apraxia of speech. *Brain & Language*, 7, 227-239.

Kay, J., Lesser, R., & Coltheart, M. 1992. *Psycholinguistic Assessment of Language Processing in Aphasia*. Hove: Lawrence Erlbaum.

Keller, E. 1987. The cortical representations of motor processes of speech. In E. Keller & M. Gopnik (Eds.) *Motor and Sensory Processes of Language*. Hilldale New Jersey: Lawrence Erlbaum Associates.

Kent, R. D. & McNeil, M. R. 1987. Relative timing of sentence repetition in apraxia and conduction aphasia. In J. H. Ryalls (Ed.), *Phonetic approaches to speech production in aphasia and related disorders*. Boston: College-Hill Press.

Kent, R. D. & Rosenbek, J. C. 1982. Prosodic and neurologic lesion. *Brain and Language*, 15, 259-291.

Kent, R. D. & Rosenbek, J. C. 1983. Acoustic patterns of apraxia of speech. *Journal of Speech and Hearing Research*, 26, 231-249.

Krull, D. 1989. Second formant locus pattern and consonant-vowel coarticulation in spontaneous speech. *Perlius* , X, 87-108.

Lebrun, Y. 1990. Apraxia of speech: a Critical Review. *Journal of Neurolinguistics*, 5 (4), 379-406.

Lecours, A. R. & Lhermitte, F. 1976. The "pure form" of the phonetic disintegration syndrome (pure anarthria); Anatomo-clinical report of a historical case. *Brain and Language*, 3, 88-113.

Levelt, W. J. M. & Wheeldon, L. 1994. Do speakers have access to a mental syllabary ? *Cognition*, 50, 239-269.

Lindblom, B. 1963. On vowel reduction. Report 29, *The Royal Institute of Technology, Speech Transmission Laboratory*, Stockholm, Sweden.

Mayer, J. A representational account for apraxia of speech. *Proceedings of the XIIIth International Congress of Phonetic Sciences*, 1, 82-85.

McNeil, M. R. & Kent, R. D. 1990. Motoric characteristics of adult aphasic and apraxic speakers. In G. E. Hammond (Ed.) *Cerebral control of speech and limb movements*. North Holland: Elsevier.

McNeil, M. R., Liss, J. M. Tseng, C. H. & Kent, R. D. 1990. Effects of speech rate on the absolute and relative timing of apraxic and conduction aphasic sentence production. *Brain and Language*, 38, 135-158.

Recasens, D. 1984. V-to-C coarticulation in Catalan VCV sequences: an articulatory and acoustic study. *Journal of Phonetics*, 12, 61-73.

Recasens, D. 1985. Coarticulatory patterns and degrees of coarticulation resistance in Catalan CV sequences, *Language and Speech*, 28, 97-114.

Recasens, D. 1987. An acoustic analysis of V-to-C and V-to-V coarticulatory effects in Catalan and Spanish VCV sequences, *Journal of Phonetics*, 15, 299-312.

Recasens, D. 1991. An electropalatographic and acoustic study of consonant-to-vowel coarticulation. *Journal of Phonetics*, 19, 177-196.

Ryalls, J. H. 1987. Vowel production in aphasia: towards and account of the consonant-vowel dissociation. In J. H. Ryalls (Ed.), *Phonetic approaches to speech production in aphasia and related disorders*. Boston: College-Hill Press.

Saltzman, E. 1986. Task dynamic coordination of the speech articulators: a preliminary model. In H. Heuer & C. Fromm (Eds.) *Generation and Modulation of Action Patterns*. Berlin: Springer-Verlag.

Sasanuma. S. 1971. Speech characteristics of a patient with apraxia of speech. *Annual Bulletin Research Institute of Logopedics and Phoniatrics, University of Tokyo*, 5, 85-89.

Skenes, L. L. 1987. Durational changes of apraxic speakers. *Journal of Communication Disorders*, 20, 61-71.

Square-Storer, P. A. & Roy, E. A. 1989. The apraxias: commonalities and distinctions. In P. A. Square-Storer (Ed.), *Acquired Apraxia of Speech in Aphasic Adults*. Hove: Erlbaum.

Sussman. H. M 1994. The phonological reality of locus equations across manner and class distinctions: Preliminary observations. *Phonetica*, 51, 119-131.

Sussman, H. M. Hoemeke, K., & McCaffrey, H. A. 1992. Locus equations as an index of coarticulation for place of articulation distinctions in children. *Journal of Speech and Hearing Research*, 35, 769-781.

Sussman, H. M., McCaffrey, H. A. & Matthews, S. A. 1991. An investigation of locus equations as a source of relational invariance. *Journal of the Acoustical Society of America*, 90, 1309-1325.

Sussman, H. M. & Shore, J. 1996. Locus equations as phonetic descriptors of consonantal place of articulation. *Perception and Psychophysics*, 58 (6), 936-946.

Varley, R. A. & Whiteside, S. P. (1998). Voicing in severe apraxia of speech: perceptual and acoustic analysis of a single case. *Journal of Neurolinguistics*. 11(3), 259-273.

Whiteside, S. P. & Varley, R. A. (1998). A new conceptualisation of apraxia of speech: a synthesis of evidence. *Cortex*, 34, 221-231.

Whiteside, S. P. and Varley, R. A. Foreign accent syndrome: a sub-type of apraxia ?. In preparation.

Weismer, G. & Liss, J. M 1991. Acoustic/ perceptual taxonomies of speech production deficits in motor speech disorders, In C. A. Moore, K. M. Yorkston & D. R. Beukelman (Eds.) *Dysarthria and apraxia of speech: perspectives on management*. Baltimore: P. H. Brookes Publishing Co.

Zeigler, W., & Cramon, D. von 1985. Anticipatory coarticulation in a patient with apraxia of speech. *Brain & Language*, 26, 117-130.

Zeigler, W., & Cramon, D. von 1986. Disturbed coarticulation in apraxia of speech: acoustic evidence. *Brain & Language*, 29, 34-47.