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## Intensity dynamics as evidence of nasal compensation for consonant voicing in Spanish apraxic speakers

Anna Marczyk<sup>1,2</sup>, Yohann Meynadier<sup>1,2</sup>, Thierry Legou<sup>1,2</sup> & Maria-Josep Solé<sup>3</sup>

<sup>1</sup>*Aix Marseille Univ, CNRS, LPL, Aix-en-Provence, France*

<sup>2</sup>*Brain & Language Research Institute, France*     <sup>3</sup>*Universitat Autònoma de Barcelona, Spain*

This paper explores the timing deficits and compensatory mechanisms in apraxia of speech (AOS). Specifically, it examines the phonetic realization of the voicing contrast by Spanish speakers with surgery-related apraxia of speech and matched control speakers. Speakers with AOS have been reported to have a deficit in laryngeal control [1], and show frequent devoicing errors across languages. By contrast, their phonetic realization of nasal consonants is relatively unimpaired [2], suggesting undisturbed velopharyngeal control. This study examines whether speakers with AOS use nasal leak as a compensatory mechanism aimed at facilitating the initiation of voicing in utterance-initial stops. Indeed nasal leak is a common facilitatory mechanism used in ‘true voicing languages’, such as Spanish and French [3].

While nasal leak may prove useful to initiate voicing in stop consonants, apraxic speakers must finely control the timing of velopharyngeal closure and avoid productions that fall into the ‘nasal’ category. The differences between nasal consonants and prenasalized stops are captured by acoustic metrics of duration and amplitude [4, 5], with the latter being more robust. In this paper we propose a method to characterize prenasalization in voiced stops in speakers with AOS on the basis of intensity patterns modelled with 3-term polynomials.

Acoustic data for two Spanish speakers diagnosed with AOS and two control subjects were obtained for isolated words. The consonant productions were transcribed and classified as on-target nasals, voiceless stops, voiced stops, or errors (i.e. ‘voiced stops’ identified as ‘voiceless’). Voiced stops were also categorized as ‘prenasalized’ if they exhibited nasal murmur, a weak formant structure, and increased amplitude of voicing on the acoustic records. Visual inspection showed that data points constituting the intensity trajectories tended to display a curvilinear shape. A curve fitting procedure using a cubic polynomial equation, see (1), was used to reduce data to a set of coefficients ( $a$ ,  $b$ ,  $c$  and  $d$ ). The roots and vertices of the derivative of the cubic expression were used to examine the increase and decrease in amplitude and the timing of these modulations during consonant closure for the different segment types.

The results in Figure 1 and Table 1 show the intensity contour profiles for the different segments and groups, obtained by averaging the function’s coefficients. The linear regression analyses of *intensity values* at consonant onset showed that nasals show significantly higher intensity than voiced stops for both the control and the apraxic group, though control speakers exhibit overall higher intensity values than apraxics. The second model tested the differences in *intensity dynamics* across phonemic categories (nasals vs voiced stops) and groups (apraxic vs control), specifically the falling and raising intensity patterns in voiced stops with prenasalization, subsequent passive devoicing and burst release. The covariation patterns between the coefficients  $a$  and  $b$  varied significantly between groups (apraxic vs control) for voiced stops, but they did not display significantly different slopes for nasal stops. These differences may reflect different patterns of prenasalization and devoicing in apraxic and control speakers. Finally, the analyses of the *roots* and *vertices* for the apraxic group show that voiced stops show a marked drop in intensity (2-4dB) early during the closure— compared to the slighter (1dB) and later drop for nasals. Control speakers, by contrast, show no change in the slope direction for nasals or voiced stops.

The analysis technique proved useful to examine the patterns of compensatory prenasalization. The results provide support for the claim that the apraxic impairment is selective. Differences in the intensity patterns of nasal and voiced stops seem to indicate that apraxic speakers control the velopharyngeal gesture as a function of phonological contrast. The results also suggest that while apraxic speakers may exhibit prenasalization, they also exhibit disturbed temporal coordination between glottal and supraglottal gestures, as evidenced both by the differences in slopes of the intensity contour and temporal measurements.

$$(1) \quad y(x)=ax^3+bx^2+cx+d$$

cubic polynomial equation used to reduce data to a set of coefficients that served as input for statistical testing. The roots and vertices of the derivative of the cubic expression were also calculated.

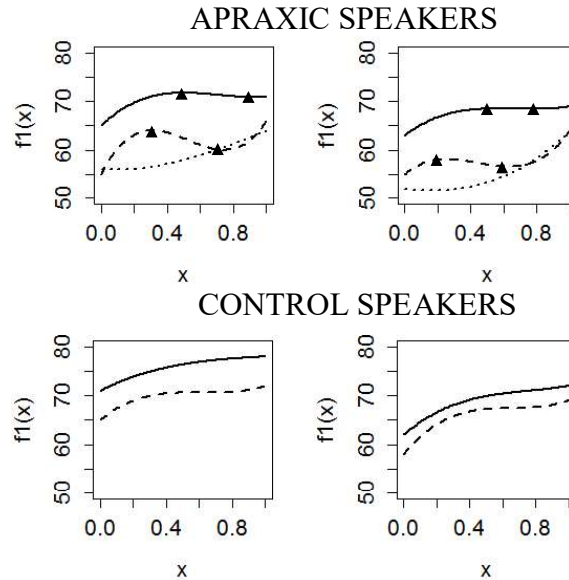


Figure 1. Intensity profiles fitted with 3-terms polynomial equation for two apraxic (top) and two control (bottom) speakers. Solid lines: nasals; dashed: voiced stops; dotted (for apraxic subjects only): voiced stops identified as voiceless. Triangles represent vertices/roots.

	phonemic category	$x^1$	$x^2$	diff(dB)
AOS speaker 1	nasal	0.5	0.9	72-71
	voiced	0.3	0.7	64-60
AOS speaker 2	nasal	0.5	0.8	69-68
	voiced	0.2	0.6	58-56
*no multiple roots for control speakers				

Table 1. Summary of the results for intensity oscillation analyses,  $x^1$  and  $x^2$  represent roots of the function, diff(dB) represents the intensity values at  $x^1$  and  $x^2$  (i.e. vertices of the function).

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