Neural bases of the foreign accent syndrome: A functional magnetic resonance imaging case study

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<td>Katz, William; University of Texas at Dallas Garst, Diane; University of Texas at Dallas Briggs, Richard; University of Texas Southwestern Medical Center, Radiology Cheshkov, Sergey; University of Texas Southwestern Medical Center, Radiology Ringe, Wendy; University of Texas Southwestern Medical Center, Psychiatry Gopinath, Kaundinya; University of Texas Southwestern Medical Center, Radiology Goyal, Aman; University of Texas Southwestern Medical Center, Radiology Allen, Greg; University of Texas at Austin, Educational Psychology</td>
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<tr>
<td>Keywords:</td>
<td>Foreign accent syndrome, fMRI, magnetic resonance imaging, speech production, articulatory planning</td>
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</table>
An fMRI Study of the Foreign Accent Syndrome

Neural bases of the foreign accent syndrome: A functional magnetic resonance imaging case study


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Keywords: Foreign accent syndrome, fMRI, magnetic resonance imaging, speech production
Abstract

Foreign accent syndrome (FAS) is a rare disorder characterized by the emergence of a perceived foreign accent following brain damage. Despite decades of study, little is known about the neural substrates involved in this disorder. In this case study, MRI images of the brain were obtained during a speech task for an American English-speaking monolingual female who presented with FAS of unknown etiology and was thought to sound “Swedish” or “Eastern European.” On the basis of MR structural imaging, the patient was noted to have frontal lobe atrophy. An fMRI picture-naming task designed to broadly engage the speech motor network revealed predominantly left-hemisphere involvement, including activation of (1) left superior temporal and medial frontal structures, (2) bilateral subcortical structures and thalamus, and (3) left cerebellum. The results suggest an instance of substantial brain reorganization for speech motor control.
Introduction

Foreign accent syndrome (FAS) is a neurological speech disorder that causes individuals to present with foreign-sounding speech (Whitaker, 1982). FAS most frequently results from cerebrovascular brain damage (CVA, or stroke). However, cases have also been reported following brain injury, episodes of schizophrenia, multiple sclerosis, progressive degenerative brain disease, and unknown etiology (see Katz, Garst, & Levitt, 2008, for review). Functional magnetic resonance imaging (fMRI) studies present an opportunity to investigate the underlying bases of speech processing (and speech breakdown) that contribute to the presentation of an apparent “foreign accent.” In this study, we examine the hypothesis that alterations in the organization of the left perisylvian speech zone correspond with the articulation difficulties of an individual with FAS.

Damage associated with FAS has been reported in the left frontal lobe, parietal lobe, basal ganglia and deep frontal white matter (see Carbary, Patterson, & Snyder, 2000; Scott, Clegg, Rudge, & Burgess, 2006, for reviews). These brain structures are thought to be part of a broad, bilateral network involving cortical and subcortical interactions serving speech production (e.g. Soros et al., 2006). A disruption to this network could conceivably lead to (1) a change in speech sequencing or timing or (2) dysprosody resulting from altered vocal tract settings or from changes in speech musculature. Such neurophysiological changes could plausibly create the impression of a foreign accent. With the exception of three cases involving primary right-hemisphere damage (Critchley, 1970; Berthier, Ruiz, Massone, Starkstein, & Leiguarda, 1991; Dankovicova et al., 2001), most occurrences of lesion-related FAS have involved different parts of the left hemisphere. A recent review has argued that these three right-hemisphere cases
probably do not represent true exceptions to left-hemisphere pathology because these patients appear to have anomalous dominance (Blumstein & Kurowski, 2006).

Dynamic diaschisis, the anatomically remote and context-sensitive effects of focal brain lesions (Price et al., 2001), has also been raised as a possible neural mechanism underlying FAS (Hwang, Lin, & Lin, 2001; Marien et al., 2006; Marien & Verhoeven, 2007, Cohen et al., 2009). For instance, Cohen et al. (2009) note paradoxical facilitation of spoken language in a case of FAS resulting from a left fronto-parietal infarct. This patient had a persisting foreign accent for three years. Following the occurrence of a subsequent right cerebral hemorrhage, the accent was eliminated. The authors speculate that for this patient persistent maladaptive behavior in the right hemisphere occurred as the result of the initial cortical stroke. It was further assumed that the resolution of the FAS stemmed from a disinhibition of the second lesion in the contra-lateral left cerebellum, leaving this region free to functionally control the rhythmic and prosodic patterns.

Evidence for the involvement of the cerebellum in FAS has emerged in two recent SPECT studies (Marien et al., 2006; Marien & Verhoeven, 2007). Following left fronto-parietal stroke, one subject showed right cerebellar hypo-perfusion. Clinical remission and recovery of the hypo-perfusion was noted after three years (Marien et al., 2006). A second subject expressed FAS in the context of a conduction aphasia due to a hemorrhagic left basal ganglia lesion that also affected the pariventricular white matter of the parietal lobe, the posterior insula, and medial temporal lobe. SPECT imaging revealed cerebro-cerebellar diaschisis affecting the right cerebellum. Repeat SPECT performed 6 months post-stroke indicated normalized right cerebellar perfusion, corresponding with improved FAS symptoms (Marien & Verhoeven, 2007).
From these data, it was suggested that FAS may follow disruption of a functional interplay between “the supra- and infra-tentorial motor speech centers.”

*In vivo* studies of the brain function in FAS are few. The majority of these studies have examined resting metabolism, using either SPECT (Moonis et al., 1996; Hwang et al., 2001; Marien et al., 2006; Marien & Verhoeven, 2007; Luzzi et al., 2008) or FDG–PET techniques (Poulin et al., 2007). As shown in Table 1, a variety of subjects, languages, and etiologies have been studied. Overall, the data support the notion of disruption within a broad-based network dedicated to speech production, although a recent cortical stimulation mapping study (Abel et al., 2009) has suggested a role for somatosensory cortex, rather than motor cortex.

--- Insert Table 1 here ---

A potential shortcoming in our knowledge base is that resting (baseline) metabolic scans do not tap behavior specifically related to speech production. Thus, from most of the extant data one cannot determine how individuals with FAS compare to healthy adults in terms of brain activation for speech processing. A notable exception is an fMRI study by Fridricksson and colleagues (2005), who examined a 45-year-old male with FAS during a picture naming task. The subject was a native American English speaker from South Carolina who was thought to sound foreign (usually French, Greek, or British) as the result of a small stroke to the left putamen. Functional MRI images were obtained six weeks post-stroke, using a sparse scanning technique to reduce artifact from displacement of soft tissue within the head. The task involved overt naming of color pictures of common objects. As a baseline, the subject also passively viewed abstract pictures that were not named. The results of the naming vs. passive viewing comparison revealed a broad pattern of activation for the individual with FAS, similar to those
described in previous studies of healthy talkers (Blank et al., 2002; Abrahams et al., 2003). This included activation of the superior temporal lobe, inferior frontal lobe, inferior motor strip (face regions), and lateral occipital lobe (object recognition). When activation for the FAS subject was compared with that of a group of healthy talkers, the subject showed an unusual activation of left central sulcus and ventral angular gyrus, suggesting cortical compensation for speech motor processes lost from subcortical damage.

Given the scarcity of functional brain imaging data for speech tasks in FAS, the present experiment employed an overt picture-naming task with identical lexical items and the same design as the case study by Fridricksson et al. (2005). Because an extensive acoustic phonetic analysis of the present FAS patient has been conducted (Katz, Garst, & Levitt, 2008), these brain imaging results may be interpreted with additional functional specificity.

Method

Subject

The patient is a 46-year-old right-handed Caucasian woman who presented with unusual speech, apparently following an allergic reaction to iodine contrast used in an enhanced chest CT scan. She is a monolingual speaker of American English who lived in upstate New York for most of her life, then moved to central Texas, where she had resided for 17 years prior to testing. Her past medical history included a diagnosis of rheumatoid arthritis, fibromyalgia, and Meniere’s disease. No prior history of speech or language disorders was reported by the patient or her family. Brain CT and MRI (without contrast and with axial FLAIR images) imaging performed immediately following the precipitating incident were unremarkable for any ischemic events; however, the MRI revealed moderate ventriculomegaly and frontal lobe atrophy.
The patient reported being frequently mistaken for Swedish, Russian, or more generically “Eastern European.” She also complained of short-term memory difficulties and problems with tasks requiring sustained attention to competing stimuli. She reported reduced sensation on the right side of the face. Facial affect was relatively diminished and a mild facial asymmetry (with a slight left-sided lip droop) remained.

--- Insert Table 2 about here ---

**Language, speech, and behavioral assessment**

A battery of language, speech, and cognitive tests were administered (Table 2). Percentile scores, where available, were computed from the raw scores using demographically-adjusted neuropsychological norms (Heaton et al., 2004). Subtests of the Boston Diagnostic Aphasia Examination-3 (BDAE-3; Goodglass, Kaplan, & Barresi, 2000), including Oral Sentence Reading, considered together with the patient’s occasional word-finding difficulties in spontaneous and read speech, were consistent with a diagnosis of a mild anomic aphasia. Speech testing indicated high intelligibility but some slight difficulties with rate and sequencing. The results of cognitive testing tend to support the patient’s complaints of attention difficulties. A sample transcription of the patient’s speech reading the Grandfather Passage (Van Riper, 1963) is given in Appendix A.

Katz et al. (2008) report detailed acoustic features of the subject’s speech, including stop consonant voice onset time (VOT), consonant burst spectra and duration, vowel formant frequencies and trajectories, prosodic cues for lexical stress assignment, and sentence-level intonation. Results indicate that the patient’s vowels were centralized and realized with reduced dynamic specifications. There was a strong tendency to realize the English alveolar flap as a full
stop and to produce flaps that had greater-than-normal closure durations. Lexical stress assignment was frequently inaccurate and highly variable, with similar problems noted for non-word stimuli. Overall, the data suggest that stress assignment (prosodic) deficits were the basis for many of her segmental difficulties.

**Experimental Procedure and Material**

During the acquisition of fMRI data, the participant overtly named color line drawings of common objects presented on a back-projection screen located at the participant’s feet and viewed through a mirror on the head coil. These drawings depicted 30 high-frequency nouns (Francis & Kucera, 1982), such as *bear*, *ball*, *doll*, *foot*, and *key*. Pictures were selected from a computer graphics database (*Art Explosion*; Nova Development, 1995–2001). Stimuli were presented randomly for 3.4 s each with pseudorandomized inter-stimulus intervals of 10.2 s, 11.9 s, or 13.6 s. The participant was instructed to name each picture when it appeared on the screen.

To obtain baseline data for the purpose of fMRI data analysis, 30 abstract pictures were randomly distributed among the presentation of the real object pictures. These abstract pictures consisted of different abstract paintings. Following Fridricksson et al. (2005), no response was required during the presentation of the abstract pictures. Examples of the real objects and abstract pictures are shown in Figure 1.

- Insert Figure 1 about here -

Naming attempts were monitored for accuracy via a microphone placed in the scanner room. The experimental paradigm was developed using *Presentation* software (*NeuroBehavioral Systems*, Albany, CA).

**MRI Data Acquisition**
MR images were acquired on a Siemens Trio 3.0 Tesla whole-body MR system using an 8-channel head coil for fast parallel imaging. In order to avoid large head motions, the participant’s head was immobilized with tightly fitting foam padding and a head strap that was fastened across the forehead. A sparse scanning sequence modeled after Fridriksson et al (2005) was used for the acquisition of fMRI data. For this sequence, a time series of single-shot, gradient-recalled, T2*-weighted, echo-planar image (EPI) volumes (GRAPPA acceleration factor = 2 with 24 phase-encode reference lines; TR = 1700 ms; TE = 20 ms; FA = 90 deg; FOV = 210 mm; 34 contiguous 3.5 mm axial slices; in-plane resolution = 3.3 x 3.3 mm) was acquired during performance of the overt naming task. Anatomical coverage began in the superior-most portions of cerebral cortex and extended into the cerebellum, though the inferior-most portions of the cerebellum were excluded. Image data were only acquired during a small portion (1.7 s) of each ISI (10.2 s, 11.9 s, and 13.6 s). The stimulus duration of each picture was 3.4 s (2 TRs), followed by a blank screen for 3.4 s (2 TRs), and image acquisition of a full-brain EPI volume for 1.7 s (1 TR). Thus, no data were acquired during stimulus presentation or overt speech. This approach avoided speech-related motion and susceptibility artifacts while timing the image acquisition to coincide with the peak blood oxygenation level-dependent response.

In order to identify the neuroanatomic location of functional activation sites, high-resolution structural MR images were acquired during the same scan session using a T1-weighted 3D gradient-recalled echo sequence (FOV = 256 mm; TR/TE/FA = 22ms/6ms/30°; 1.0 mm x 1.0 mm x 1.0 mm resolution, bandwidth = 100 Hz/pixel, GRAPPA acceleration factor = 2; 24 phase encode reference lines).

Data Analysis
AFNI software (Cox, 1996) was used for all analyses. EPI data were aligned to the high-resolution structural MRI data using the AFNI script `align_epi_anat.py` (Saad et al., 2009). All MRI data were spatially normalized according to the system of Talairach and Tournoux (1988). Motion correction was conducted in two steps. First, two-dimensional image alignment was performed on a slice-by-slice basis. Second, a three-dimensional volume registration algorithm (Cox & Jesmanowicz, 1999) was applied to the EPI dataset. The linear trend of the time series was next removed from the data using linear regression.

Following the pre-processing steps, a $t$-test was used to identify areas of significant naming-related activation. The $t$-test was used to determine, on a voxel-by-voxel basis, whether the mean signal in response to nameable objects was significantly different from the mean signal in response to abstract pictures. The output from this $t$-test was then thresholded using a voxel-cluster-size method for the rejection of false positive activations. Prior to the cluster-size threshold, all voxels whose $t$ value did not exceed an uncorrected $\alpha$ level of 0.001 were excluded from further analysis. Voxels that survived this threshold and were part of a cluster of at least seven contiguous voxels were considered areas of significant naming-related activation.

**Results**

For this study, the subject was examined at 19 months post-onset. This was at the same time that speech data were collected for analysis (Katz et al., 2008), permitting a comparison between the subject’s pseudo-accented speech characteristics and the current neurological patterns.

The participant performed with high accuracy (99%) in this familiar object naming task. The few naming errors that did occur were mistakes frequently noted in healthy individuals (e.g.,
fly for the target bee). Significant patterns of naming-related activation are summarized in Table 3. The activation data are graphically displayed (on the subject’s structural MRI scans) in Figure 2.

--- Insert Table 3 here ---

The subject showed predominantly left-hemisphere activation in this functional task. Significant patterns of naming-related activation (object naming > baseline) were found for left temporal structures (superior, middle, and transverse temporal gyri; temporal pole), left frontal regions (superior, medial, and inferior frontal gyri; superior and mid orbital gyri; precentral gyrus), left insula, bilateral thalamus, and subcortical structures (left putamen and globus pallidus, right caudate), and the left superior posterior cerebellar hemisphere.

- Insert Figure 2 about here –

**Discussion**

This study reports fMRI data for the speech-related processing of a patient with FAS of a primarily prosodic nature. The patient’s speech characteristics included lengthened vowels, problems with lexical stress assignment, and occasional problems in sentence-level intonation (Katz et al., 2008). The subject did not develop FAS as the result of a frank lesion, but rather from undetermined causes (reportedly from an allergic reaction to iodine). Also, she had substantial frontal lobe atrophy, a pre-existing abnormal condition. Based on previous fMRI studies of healthy and disrupted speech, it was hypothesized that the patient’s constellation of symptoms would correspond to disruption of a broad neural network predominantly centered in the left perisylvian region. Because the subject did not present with FAS as the result of frank
left-hemisphere damage, crossed cerebro-cerebellar diaschisis (e.g. as reported by Marien, Verhoeven, and colleagues) was not considered a likely outcome.

The brain structures observed in the subject’s naming response included a number of left-hemisphere regions described in a recent meta-analysis of studies reporting brain activation during object naming relative to low-level baselines (Price et al., 2005). Specifically, precentral gyrus, inferior frontal gyrus, superior and middle temporal gyri, fusiform gyrus, lingual gyrus, insula, thalamus, and cerebellum showed significant patterns of naming-related activation.

The finding of predominantly left-hemisphere activation, including cortical regions traditionally associated with speech production (Broca’s area, anterior insula) and naming (auditory cortex, Wernicke’s area), does not support our initial hypothesis of FAS resulting from a disrupted left perisylvian speech network. This hypothesis would predict either normal levels of right-hemisphere activation or possibly enhanced right-hemisphere activation (in compensation for disturbed left-hemisphere function). The subject showed the opposite; an overall low level of right-hemisphere activation. The absence of activation of homologous right-hemisphere regions is striking, considering that a number of recent studies have convincingly demonstrated interaction between cortical structures in the left and right hemispheres during speech processing, with the latter believed to be principally responsible for mediating prosody (Meyer et al., 2002; Meyer et al., 2004; Glasser & Rilling, 2008; Zatorre & Gandour, 2008; Sammler et al., 2010). This raises the possibility that the prosodic anomalies underlying this patient’s unusual accent involve either dysfunction in the right hemisphere itself, or in the interaction between right and left hemispheres during speech.
In contrast, some patterns in the data do appear to support the hypothesis of a compensatory reorganization of speech motor and cognitive processing networks. Unlike previous subjects tested under similar task conditions (Fridricksson et al., 2005; 2006), the subject showed activation of bilateral thalamus, subcortical structures (left putamen and globus pallidus and right caudate) and left temporal pole.

The exact brain areas leading to dysprosodic speech under a given condition may be difficult to clearly specify. This is because prosody may play different functional roles (linguistic, affective), is expressed by different acoustic phonetic cues (amplitude, fundamental frequency, duration), and occurs across different-sized processing domains (phonemes, syllables, phrases) (Baum & Pell, 1999). In the present case, the involvement of thalamus and subcortical structures may be particularly informative, as a detailed portrait of the subject’s prosodic deficits has been established in a prior study (Katz et al., 2008). Acoustical analysis of the subject’s speech revealed frequent, local rhythm problems that were the basis for many of her lexical stress assignment problems. There were also occasional global prosody problems affecting sentence-level intonation. A possible explanation for these prosodic difficulties is that heightened activation in the thalamic and subcortical structures resulted from increased “effort” in processing timing/rhythm, and that such processing would ordinarily be more broadly distributed across other brain structures, such as right-hemisphere perisylvian areas and left subcortical structures (Riecker et al., 2002). This type of dynamic diaschisis involving thalamic and subcortical structures has been proposed for at least one other individual with FAS. Based on SPECT data, an individual with closed head injury was noted to have FAS, including dysprosody associated with the thalamus and globus pallidus hypo-perfusion (Moonis et al., 1996).
Two of the affected brain regions, the left superior frontal gyrus and left temporal pole, are structures thought to be involved in various aspects of cognitive processing. The involvement of the frontopolar region has been noted in studies of strategic processing, memory retrieval, and executive function (Petrides, Alivisatos, Meyer, & Evans, 1993; de Boisgueneuc et al., 2006; Dreher, Koechlin, Tierney, & Grafman, 2008).

The anterior and inferior temporal pole has been associated with the overt naming of unique entities in PET studies (Damasio et al., 1996; 2004; Murtha et al., 1999; Grabowski et al., 2001), in a diffusion- and perfusion-weighted fMRI investigation (DeLeon et al., 2007), and in a study of brain metabolites during language tests (Rami et al., 2008). Temporal pole was also a site of activation reported in the meta-analysis of Price et al. (2005), though only for studies comparing naming to high-level baseline tasks that controlled for speech production.

As noted above, the subject had substantial frontal lobe atrophy of unknown etiology. This structural anomaly could account for her showing quite different functional activation patterns from healthy talkers, as well as from the FAS case study described by Fridriksson and colleagues (2005). There are many possible etiologies for frontal lobe atrophy in adults, including infectious, inflammatory, and neurodegenerative disorders (e.g., Geschwind, Yoon, & Goldman, 2006; Scharre, 2006). The fact that she showed frontal lobe activation despite overall atrophy is interesting, and appears consistent with a “retreat” of some frontal lobe functions, while other frontal lobe functions appear preserved. For instance, her poor performance on the Trail Making Test suggests some difficulty with executive functioning. In contrast, she had sufficient strategic processing to perform with a high degree of accuracy in the object-naming task.
In conclusion, an overt naming task designed to engage global speech/language processing has suggested for this FAS patient an atypical pattern of neural activity involving structures relevant to speech motor and cognitive processing. The specific neural structures involved are different than those noted in a previous case study using a similar paradigm (Fridriksson et al., 2005).

The neural activation details may be plausibly related to a number of factors, including marked differences in the patients’ pre-morbid brain status, FAS etiology and time post-onset, and the resultant “foreign” accents and their motoric bases. On the basis of available evidence, it appears that FAS corresponds with unusual brain states for speech production. For the present patient, this included a lack of bilateral activation during the naming task and recruitment of right subcortical structures and bilateral thalamus. An expanded database including more subjects tested on similar tasks will be important to better understand these issues.

Acknowledgments

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Appendix A. Grandfather passage transcription

/ju: 'wi:ft / tu no:l abat mar 'graemfod/ 'wel / hi iz 'nirli / 'naindi_ _@wi / jtorz 'cl / hi*
'd^asis / himself / mon 'emjnt / blaeo fa:r 'ko@/ 'juz1 / mainis soro 'batins / daet hi
stil 'Biiks / az 'swifli / az 'evor / e la^ flowing 'biord kliks /tu hiz 'jfin/ giwin 'oz / hu
ab'o:rn him / e pronans filin af 'admos / ri'spaktn / wen hi 'spiks / hiz 'vakis is zos e bt /
'kw@kt / aend 'kwivos / azn 'twarfof / tways itf de hi 'plez / 'skifoli / end wiiz 'zaest /
@apn 'ar / smol 'orgen / eeksept in de winto wen de 'uz / or 'ns / or ars po:ivens / hi
soli teks e jot 'wok / in di opin e^ itf 'de / e 'wi hai ev nipa / abioz him tu wok 'mor / en
smok 'laes / bot hi 'ovez aensorz / banaen @ul / 'graemfod laiks tu bi 'modorn / in hiz
'laenwadz/

You wished to know all about my grandfather. Well, he is nearly ninety-three years old; he
dresses himself in an ancient black frock coat, usually minus several buttons; yet he still thinks
as swiftly as ever. A long, flowing beard clings to his chin, giving those who observe him a
pronounced feeling of the utmost respect. When he speaks, his voice is just a bit cracked and
quivers a trifle. Twice each day he plays skillfully and with zest upon our small organ. Except in
the winter when the ooze or snow or ice prevents, he slowly takes a short walk in the open air
each day. We have often urged him to walk more and smoke less, but he always answers,
“Banana oil!” Grandfather likes to be modern in his language.
NOTES

1 This step was in keeping with the original goal of Fridricksson and colleagues of “highlighting the entire cortical speech/language and object recognition networks.” However, as pointed out by an anonymous reviewer, it would have been useful to have included a condition in which the patient said something like “junk” during the presentation of the abstract images. This step would potentially have addressed the cortical bases of more complex phonological processing.

References


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<th>Case</th>
<th>References</th>
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<th>FAS Etiology/location</th>
<th>Functional Imaging Type</th>
<th>Imaging task?</th>
<th>Imaging results</th>
<th>Conclusions</th>
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<tbody>
<tr>
<td>1</td>
<td>Moonis et al. (1996)</td>
<td>English → French</td>
<td>Closed head injury</td>
<td>SPECT</td>
<td>Rest</td>
<td>Reduced perfusion of left anterior dorsolateral inferior frontal gyrus; Ipsilateral caudate nucleus</td>
<td>Dysprosody appears to result from damage to the cortico-striato-pallidal-thalamic pathway</td>
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<td>2</td>
<td>Hwang, Lin, &amp; Lin (2001)</td>
<td>Mandarin Chinese → American English</td>
<td>No structural lesion seen on MR; left temporal lobe ischemia suspected based on SPECT</td>
<td>SPECT</td>
<td>Rest</td>
<td>Hypoperfusion defect over left lateral temporal region, with right cerebellar diaschisis</td>
<td>Phonemic tone acutely impaired from ischemia. Accent processing may be related to left lateral temporal region (with diaschisis)</td>
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<td>3</td>
<td>Fridrickssen et al. (2005)</td>
<td>American English → French, Greek, or British English</td>
<td>Small stroke to left putamen</td>
<td>fMRI</td>
<td>Naming common pictures (passive viewing of abstract pictures as control)</td>
<td>Increased activation in left central sulcus and ventral angular gyrus</td>
<td>Compensation for impaired motor speech regulation by other areas of the cortical motor speech network.</td>
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<td>4</td>
<td>Marien et al. (2006)</td>
<td>Dutch → French, German or Russian</td>
<td>Left fronto-parietal stroke</td>
<td>SPECT</td>
<td>Rest</td>
<td>Severe hypo-perfusion in the left frontal motor and parietal cortex, secondary hypoperfusion in the thalamus and striatum of the left hemisphere, relative hyperfusion of the right hemicerebellum</td>
<td>Crossed cerebellar diaschisis; FAS may result from disruption between the supra-and intratentorial centers</td>
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<tr>
<td>Case</td>
<td>Authors and Language(s)</td>
<td>Description of Language and/or Speech Impairment</td>
<td>Imaging and/or Testing Method</td>
<td>State or Condition</td>
<td>Diagnosis or Explanation</td>
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<td>5</td>
<td>Marien &amp; Verhoeven (2007) Patient #2 Dutch → North-African</td>
<td>Left basal ganglia hemorrhage which involved putamen, genu and posterior limb of the internal capsule and extended to the posterior insula, the medial temporal lobe, and the left paraventricular white matter</td>
<td>SPECT</td>
<td>Rest</td>
<td>Crossed cerebro-cerebellar diaschisis affecting right cerebellum, hypoperfusion of left thalamus, left lentiform nucleus, left medial and lateral temporal regions and the left motor cortex</td>
<td></td>
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<td>6</td>
<td>Poulin, Macoir, Paquet, Fossard, &amp; Gagnon (2007) Quebec French → Eastern French Canadian, France, or English</td>
<td>Bipolar disorder with suspected psychotic features, history of epilepsy, abuse</td>
<td>FDG-PET</td>
<td>Rest “Euthymic condition”</td>
<td>Hypometabolism of left insular anterior frontal cortex</td>
<td>Cerebrovascular origin (left insular and anterior temporal cortex) best explanation for observed language deficits</td>
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<td>7</td>
<td>Luzzi et al. (2008) Italian → Spanish</td>
<td>Primary progressive aphasia; mild left perisylvian atrophy</td>
<td>SPECT</td>
<td>Rest</td>
<td>Mild hypoperfusion of the left perisylvian speech area</td>
<td>Perisylvian cortical basis for this patient</td>
<td></td>
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<tr>
<td>8</td>
<td>Abel, Hebb, &amp; Silbergard (2009) American English → Swedish</td>
<td>Metastatic tumor in left anterior parietal lobe</td>
<td>Electro-cortical function stimulation mapping</td>
<td>Naming common objects</td>
<td>Lesion was confined to face somatosensory cortex, not motor</td>
<td>FAS can result from difficulties processing perceptual feedback of speech</td>
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Table 2. Test scores for subject.

<table>
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<tr>
<th>Tests</th>
<th>Subtests</th>
<th>Raw Score</th>
<th>Interpretation</th>
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<td><strong>Language</strong></td>
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<tr>
<td>BDAE-3¹</td>
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<td>Sentence Repetition</td>
<td>9/10</td>
<td>Normal</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>Oral Sentence Reading</td>
<td>9/10</td>
<td>Moderately impaired</td>
<td>*</td>
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<tr>
<td></td>
<td>Comprehension of Orally Read Sentences</td>
<td>5/5</td>
<td>Normal</td>
<td>*</td>
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<td></td>
<td>Reading Comprehension</td>
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<td>Normal</td>
<td>*</td>
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<td>Boston Naming Test</td>
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<tr>
<td><strong>Speech, Praxis</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>AIDS²</td>
<td>Single Word Intelligibility</td>
<td>90% intelligibility</td>
<td>Normal</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>Sentence Intelligibility</td>
<td>90% intelligibility</td>
<td>Normal</td>
<td>*</td>
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<tr>
<td><strong>ABA-2³</strong></td>
<td>Diadochokinetic Rate</td>
<td>13</td>
<td>Mild</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>Increasing Word Length A</td>
<td>4</td>
<td>Mild</td>
<td>*</td>
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<tr>
<td></td>
<td>Increasing Word Length B</td>
<td>6</td>
<td>Moderate</td>
<td>*</td>
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<td></td>
<td>Limb Apraxia</td>
<td>48</td>
<td>Normal</td>
<td>*</td>
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<tr>
<td></td>
<td>Oral Apraxia</td>
<td>44</td>
<td>Normal</td>
<td>*</td>
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<td></td>
<td>Polysyllabic Utterance Time</td>
<td>18</td>
<td>Mild</td>
<td>*</td>
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<tr>
<td></td>
<td>Repeated Trials</td>
<td>25</td>
<td>Mild</td>
<td>*</td>
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<tr>
<td><strong>Cognition</strong></td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>MMSE⁴</td>
<td>n/a</td>
<td>26/30</td>
<td>Normal (intact)</td>
<td>*</td>
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<tr>
<td>Trail Making Test⁵</td>
<td>Part A</td>
<td>61 sec</td>
<td>Moderately Impaired</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Part B</td>
<td>141 sec</td>
<td>Moderately Impaired</td>
<td>1</td>
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</table>

URL: http://mc.manuscriptcentral.com/nncs Email: hbc@mail.med.upenn.edu
D-KEFS Sorting Test 6

<table>
<thead>
<tr>
<th>Description</th>
<th>Score</th>
<th>Average</th>
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<tr>
<td>Free Sorting Correct Sorts</td>
<td>10</td>
<td>Average</td>
</tr>
<tr>
<td>Free Sorting Description</td>
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<td>Average</td>
</tr>
<tr>
<td>Sort Recognition Description</td>
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<td>Average</td>
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<tr>
<td>Combined Description</td>
<td>9</td>
<td>Low Average</td>
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<tr>
<td>Sort Recognition vs. Free Sorting Description</td>
<td>7</td>
<td>Low Average</td>
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NOTES:

1. Boston Diagnostic Aphasia Examination-3 (Goodglass, Kaplan, & Barresi, 2000)
2. Apraxia Battery for Adults (Dabul, 2000)
5. Mini-Mental State Examination (Folstein, Folstein, & McHugh, 1975).
Table 3. Clusters of significant naming-related activation (object naming > baseline)

<table>
<thead>
<tr>
<th>Neuroanatomic Location of Peak</th>
<th>Talairach Coordinates</th>
<th>Peak Cluster Volume</th>
<th>Neuroanatomic Regions Activated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebellar Hemisphere Lobule VI</td>
<td>-22 -65 -13</td>
<td>8.82 452</td>
<td>Posterior quadrangular lobule; Fusiform gyrus; Lingual gyrus</td>
</tr>
<tr>
<td>Superior Temporal Gyrus (BA 22)</td>
<td>-62 -8 7</td>
<td>6.92 301</td>
<td>Superior temporal gyrus; Precentral gyrus; Temporal pole</td>
</tr>
<tr>
<td>Transverse Temporal Gyrus (BA 42)</td>
<td>-61 -17 10</td>
<td>6.58 414</td>
<td>Superior, middle, and transverse temporal gyri</td>
</tr>
<tr>
<td>Superior Temporal Gyrus (BA 38)</td>
<td>-30 22 -32</td>
<td>6.27 1507</td>
<td>Superior, middle, and inferior temporal gyri; Temporal pole</td>
</tr>
<tr>
<td>Inferior Frontal Gyrus (BA 45)</td>
<td>-28 29 2</td>
<td>4.83 264</td>
<td>Inferior frontal gyrus; Insula</td>
</tr>
<tr>
<td>Medial Frontal Gyrus (BA 10)</td>
<td>-7 63 0</td>
<td>4.74 377</td>
<td>Superior and medial frontal gyri; Superior and mid orbital gyri</td>
</tr>
<tr>
<td>Thalamus</td>
<td>0 -7 -2</td>
<td>4.73 339</td>
<td>Thalamus (bilateral)</td>
</tr>
<tr>
<td>Superior Temporal Gyrus (BA 41)</td>
<td>-46 -35 16</td>
<td>4.43 414</td>
<td>Superior, middle, and transverse temporal gyri; Insula</td>
</tr>
<tr>
<td>Globus Pallidus, Lateral</td>
<td>-15 4 -3</td>
<td>4.22 980</td>
<td>Globus pallidus; Putamen; Claustrum; Insula</td>
</tr>
<tr>
<td>Caudate Nucleus</td>
<td>10 -1 12</td>
<td>3.97 264</td>
<td>Caudate; Thalamus</td>
</tr>
</tbody>
</table>

**Abbreviations:** BA = Brodmann Area
Figure Captions

**Figure 1.** The top panels show sample line drawings of high-frequency common objects (*ball, bear, key*) used in the naming task. Examples of abstract art images used in the control condition are shown in the bottom panels.

**Figure 2.** Areas of significant naming-related activation for the patient (picture naming > baseline). The functional activity is displayed on an underlay of the patient’s axial anatomical scans.