

giography should be considered for patients diagnosed with TA who have severe headache, strokelike episodes, or seizures.

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## A French accent after corpus callosum infarct

D.A. Hall, MD; C.A. Anderson, MD; C.M. Filley, MD; J. Newcombe, MA, CCC-SLP; and R.L. Hughes, MD

An accent is a feature of speech that characterizes certain people or regions. The foreign accent syndrome (FAS) is a rare disorder in which persons develop a speech pattern perceived by listeners to be foreign.<sup>1,2</sup> FAS has typically been observed after anterior cortical or subcortical lesions in the hemisphere dominant for language,<sup>1,2</sup> and a disruption of linguistic prosody has been implicated.<sup>1</sup> We report a patient in whom a French accent appeared after an ischemic infarct in the corpus callosum (CC).

**Case report.** A 53-year-old right-handed (+4 on the modified Annett handedness inventory<sup>3</sup>) English-speaking office worker had the sudden onset of difficulty speaking. Examination on the day of the event showed fluent speech with a staccato quality, and several observers then and later noted that her speech had acquired an accent in English resembling that of a native French speaker. There was a prominent disturbance of melodic line, with altered intonation, stresses, and pauses that imparted the impression of a French accent. Otherwise, her examination revealed normal fluency, repetition, and naming, but auditory comprehension was impaired. No facial or limb apraxia was noted. Reading comprehension was impaired, and there was mild agraphia. The production and understanding of affective prosody was preserved, and there was neither left-hand apraxia nor tactile anomia. The remainder of her neurologic examination was normal. Initial MRI showed an ischemic infarct in the body of the CC; the lesion was primarily in the midline but also extended slightly to the left (figure, A). After discharge, speech therapy produced no substantial improvement. Her speech remained strikingly altered during the ensuing months because of the French accent. On her return to the United States after a visit to Canada, border guards who heard her speak believed she was French Canadian.

MRI performed 6 months after onset showed a small infarct in the body of the CC where the initial lesion had been observed (see figure, B). One year after symptom onset, neurobehavioral deficits persisted with the exception of a diminution in staccato speech. She was unable to work because of the language deficits associated with the foreign accent. She was fluent, had no dysarthria, and had normal repetition. Confrontation naming was also normal: she earned a score of 60/60 on the Boston Naming Test<sup>4</sup> with only mild hesitancy in responding. She had difficulty, however, with commands involving two or more steps and with reading comprehension of complex sentences and paragraphs.<sup>5</sup> In subse-

quent months, many English- and French-speaking listeners who heard a recording of her speech generally concurred that her accent was best characterized as French or French Canadian.

**Discussion.** After an infarct in the body of the CC, this woman developed a French accent. A disturbance of melodic line, with altered intonation, stresses, and pauses, was prominent in her spontaneous speech, suggesting that linguistic a prosody was fundamental to her new accent. She had mild fluent aphasia and agraphia but no deficits involving affective prosody or callosal integration of motor or sensory functions. This patient is unique because the neuropathology was confined to the CC.

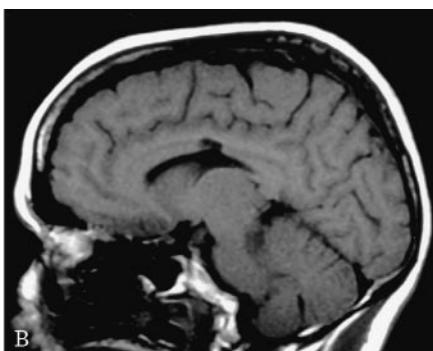
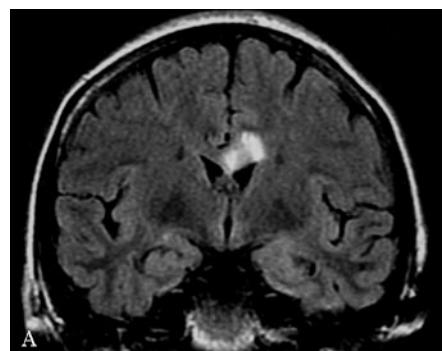
FAS is thought to reflect a disorder of speech production related to damage in motor regions subserving speech.<sup>1,2</sup> Some patients are initially mute, and others have a staccato speech rhythm, as in our patient, or a "sing-song" phrasal intonation.<sup>2</sup> Structural lesions causing FAS typically involve regions of the anterior cerebral cortex, basal ganglia, or white matter in the language-dominant hemisphere. Many authorities assert that deficits in linguistic prosody underlie this syndrome.<sup>1</sup>

The French accent in our patient was apparently caused by the callosal infarct. The unusual lesion location remains puzzling because motor structures subserving speech production were not directly involved, but the localization adds the CC to the cerebral regions implicated in FAS. The CC helps mediate prosodic elements of language; deficits in affective and linguistic prosody have been documented in a patient with damage to the anterior four-fifths of the CC.<sup>6</sup> Because linguistic a prosody also occurs after lesions of the right<sup>7</sup> and left cerebrum,<sup>5</sup> the CC appears to participate in a distributed network for linguistic prosody involving both hemispheres. In our patient, we believe the CC lesion accounted for the FAS by disrupting a bihemispheric distributed network for linguistic prosody. Her speech was characterized by impaired linguistic prosody that produced the French accent. Although further verification is necessary, we conclude that disturbance of interhemispheric connectivity may contribute to the pathogenesis of FAS.

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**Figure.** (A) Fast spin-echo inversion recovery coronal MRI showing an acute ischemic infarct in the corpus callosum (TR, 128; TE, 8002). (B) T1-weighted sagittal MRI showing a discrete area of infarction in the body of the corpus callosum (TR, 400; TE, 14).

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## Acute necrotizing encephalopathy of childhood with radiographic progression over 10 hours

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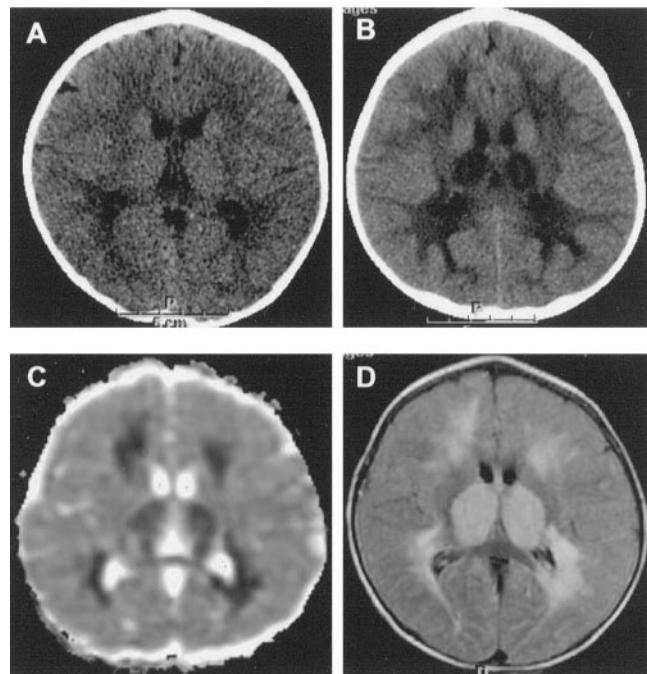
We report a 14-month-old girl who was examined for altered mentation. Although head CT on arrival was normal, CT and MRI 10 hours later demonstrated bilateral thalamic abnormalities. Results of an extensive laboratory evaluation were negative, and the patient was diagnosed with acute necrotizing encephalopathy of childhood (ANEC).

**Case report.** The patient, a 14-month-old girl of western European and African-American ancestry (no known Asian ancestry), with no medical or travel history, was brought to the emergency department after discovered unresponsive. She had been febrile for 2 days, with decreased appetite, but with apparently normal mentation until presentation. She was noted to have episodes of four-extremity extension, prompting loading with phenobarbital. Head CT at that time was normal (figure, A). CSF analysis revealed an erythrocyte count of 3 cells/mL, leukocyte count of 1 cell/mL, protein of 51 mg/dL, and glucose of 64 mg/dL. Urine and serum toxicology screens were negative. Blood gas, routine blood counts, chemistry, hepatic panel, and ammonia were all normal, except for a mild elevation in transaminases, with an aspartate aminotransferase (AST) level of 190 IU/L (normal, 22 to 59 IU/L) and an alanine aminotransferase (ALT) level of 54 IU/L (normal, 3 to 34 IU/L). The patient was admitted for further monitoring.

Nine hours after admission, neurologic consultation was requested because of concern for seizures. Neurologic examination at this time revealed decerebrate posturing, no response to deep

pain, and hyperreflexia throughout, with bilateral extensor plantar responses. The patient was immediately intubated, hyperventilated, and reloaded with phenobarbital. EEG while the patient was posturing showed slowing but no epileptiform discharges, and two follow-up EEGs showed no seizure activity. Head CT nearly 10 hours after the original scan revealed new bilateral thalamic hypodensities (see figure, B). An MRI with MR angiography (MRA) and MR venography (MRV) was performed within 1 hour of the follow-up CT. Neither thrombus nor congenital vascular abnormality was seen with MRA and MRV (data not shown). Apparent diffusion coefficient (ADC) mapping confirmed acute bilateral thalamic involvement and involvement of the periventricular white matter (see figure, C). The extent of the acute insult was well visualized using fluid-attenuated inversion recovery (FLAIR) imaging (see figure, D). Extensive laboratory evaluation for infectious, coagulopathic, vascular, autoimmune, toxic, traumatic, nutritional, and metabolic etiologies was negative (see Discussion). Thus, the patient was diagnosed with acute necrotizing encephalopathy of childhood (ANEC). Two weeks later, the child was blind, responded only to noxious stimuli, and had diffuse spasticity. She was discharged to a rehabilitation facility.

**Discussion.** Our patient is characteristic of the typical description of ANEC, with a mild febrile prodrome, rapid progression to coma, an essentially negative laboratory evaluation, and radiographic evidence of bilateral thalamic involvement.<sup>1</sup> Prognosis is usually poor.<sup>2</sup> Because the etiology and pathogenesis of ANEC are unknown, a wide differential diagnosis must be entertained and eliminated before diagnosing this syndrome.<sup>2</sup> The laboratory evaluation for this patient was unrevealing for infectious agents (negative blood, urine, sputum, and CSF cultures, no bartonella, toxoplasmosis, mycoplasma, herpes viruses, HIV, influenza A and B, respiratory syncytial virus, rotavirus, measles, cytomegalovirus, Epstein-Barr virus, or arboviruses detected),



**Figure.** Radiographic findings. Note that windows are identical for images A and B. (A) Head CT at presentation. (B) Head CT 10 hours after initial CT. Note bilateral thalamic hypodensities. (C) Apparent diffusion coefficient mapping approximately 11 hours after original CT. Note bilateral thalamic and periaqueductal involvement. (D) Fluid-attenuated inversion recovery image approximately 11 hours after original CT clearly delineating extent of acute changes.

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