PARADOXICAL FACILITATION: THE RESOLUTION OF FOREIGN ACCENT SYNDROME AFTER CEREBELLAR STROKE

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Clinical/Scientific Notes

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Foreign accent syndrome (FAS) is a rare speech disorder characterized by a change in prosody and other speech variables yielding altered phonetic characteristics that are perceived as a foreign accent. Lesions associated with FAS typically involve left frontoparietal regions. However, 2 reported left-hemispheric stroke patients presenting with FAS also had hypoperfusion of the right cerebellum on ^{99m}Tc ECD SPECT imaging, presumably from diaschisis. In both cases, there was a close parallel between the normalization of the cerebellar perfusion and resolution of the clinical syndrome up to 3 years later despite continued hypoperfusion of left hemispheric structures.[1,2] The authors argued that the temporal association between the clinical improvement and the right cerebellar perfusion suggested a functional role of the cerebellum in this speech disorder. We describe a unique case that provides convergent evidence for a causative role of the cerebellum in FAS.

Case report. A right-handed English-speaking woman was 58 years of age at the time of her left frontoparietal infarct (figure, A), presenting with right upper limb paresis and aphemia. Within hours, the patient's paresis resolved to a slight hand ataxia and her speech sounded like English spoken with an unlearned accent. Workup suggested an embolic event, and she was discharged on warfarin. The foreign accent persisted for approximately 3 years until she had a right inferior cerebellar hemorrhage (figure, B–D) from accidental excessive anticoagulation. Following this second stroke, the patient and her family noted that the foreign accent was no longer perceptible in her speech.

Analysis. Detailed acoustical analysis was performed on speech samples recorded prior to the patient's left frontoparietal infarct (CVA1), 4 and 20 days following this infarct, shortly after her cerebellar hemorrhage (CVA2), and later (BIMDC CVA2). Results indicated that post CVA1, she showed the constellation of impaired and spared speech production characteristics of FAS.[3] She displayed pathologic patterns in prosody, vowel formant frequencies, vowel durations, and increased variability on these vowel measures, and she showed normal patterns in the production of voicing and place of articulation in stop consonants as well as preservation of the distinction between tense and lax vowels. Subsequent to her cerebellar stroke (CVA2), the patient's pathologic speech patterns resolved and she showed normal speech output (appendix e-1 and figures e-1 through e-4 on the *Neurology*® Web site at www.neurology.org).

Discussion. Rather than further impairing her speech, a right cerebellar stroke paradoxically extinguished the FAS. This case demonstrates that abnormal right cerebellar activity can play a causal role in perpetuating the FAS rather than being merely an epiphenomenon of damage to the reciprocally connected left hemisphere. Many of the presenting features of FAS relate to alterations in timing and rhythm of speech,[4] and a functional role of the cerebellum in FAS is consistent with the model that the cerebellum plays a generalized role in processing temporal information.[5] The prosodic disturbances in FAS may reflect deficits in timing resulting in impairments in the control of speech melody, in maintaining the correct vocal posture for the production of vowels, in maintaining the correct durational patterns of speech, and in producing syllabic stress and quantity.

The occurrence of the paradoxical facilitation[6] in this case suggests that normal cerebellar control mechanisms may involve competitive interactions between the 2 cerebellar hemispheres. When the right cerebellum is lesioned, however, the contralateral left cerebellum may be disinhibited and thus assume a functional role, in the current case controlling the rhythmic and prosodic patterns for speech output, resulting in the resolution of the FAS. The idea that damage in one area of the brain may result in a release from inhibition and activation of an alternative network has been described.[7] In that case, fMRI demonstrated a shift of the cortical representation of speech motor control to the right rolandic cortex and left cerebellum during rapid recovery from dysarthria following an infarct in the left internal capsule. In the case of our patient, persistent maladaptive activation in the right cerebellum may have prevented a similar

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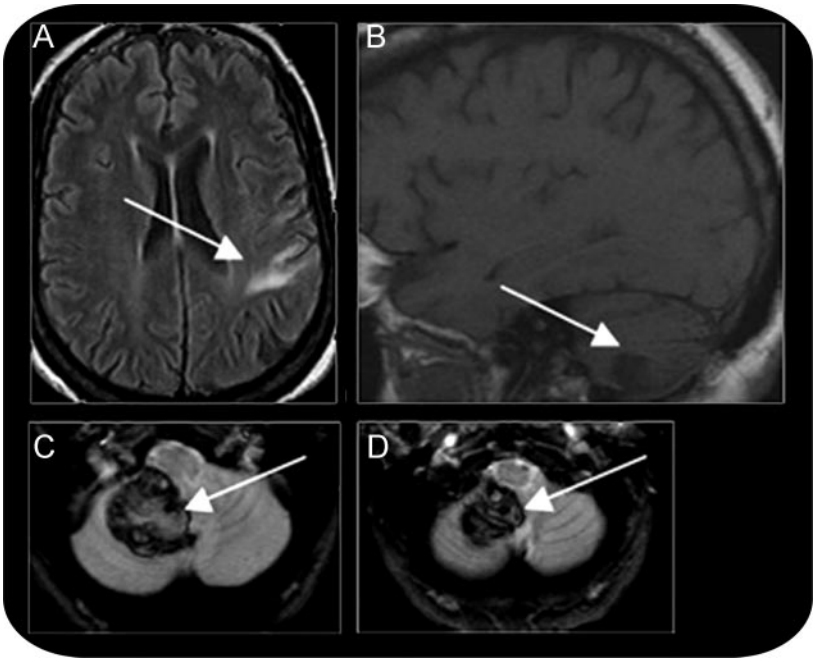
Figure MRI scans of the patient's strokes



The foreign accent syndrome patient's initial embolic middle cerebral artery stroke (A, axial image, fluid-attenuated inversion recovery sequence, lesion appears whiter than surrounding cortex) resulted in a lesion at the frontoparietal junction. The patient's second stroke was a hemorrhagic stroke (3 cm × 4 cm × 1–3 cm hematoma) in the inferior portion of the right posterior lobe of the cerebellum reaching the midline and impinging on the vermis (T1-weighted sagittal image B, lesion appears darker than surrounding cortex, and susceptibility axial images C and D, where blood appears black). All images are presented in radiologic convention with the left hemisphere appearing on the right-hand side of the image. The white arrows indicate the lesioned area.

type of shift and the failure of her speech abnormalities to resolve spontaneously.

Ultimately, the behavioral consequences of a brain insult reflect the capacity of the rest of the brain to cope with the injury. Thus, the findings from our patient reveal that the neural control mechanisms of speech production depend on maintaining a delicate balance between bihemispheric cerebral and cerebellar connections and that changes in these dynamics can have effects on the operation of the entire system. Furthermore, the findings of resolution of FAS following right cerebellar disruption raise the intriguing possibility of a therapeutic potential of neuromodulatory interventions.

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ACUTE HIPPOCAMPAL SCLEROSIS FOLLOWING ECSTASY INGESTION

We report 2 patients presenting with brief tonic-clonic seizures following ecstasy (3,4-methylenedioxymethamphetamine [MDMA]) ingestion who initially showed hippocampal swelling and subsequently atrophy.

Case reports. ***Case 1.*** A 25-year-old right-handed man presented following 2 generalized tonic-clonic seizures (GTCS). The first, lasting 30 seconds, occurred 5 hours after taking 2 ecstasy tablets and 8 units of alcohol. The following day he felt unwell and vomited several times but his partner did not report any other unusual behaviors until the afternoon when he had a further 2-minute GTCS, and presented to hospital.

In the days prior to the ecstasy ingestion, he had been well. There was no past history or family

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