Editors’ Note: Commenting on “A puzzling case of amnesia” by Bereznyakova et al., Harriott and Tatum entertain an alternate diagnosis. Senior author Laforce responds. Stagnaro suggests that results in “Growth hormone treatment for childhood short stature and risk of stroke in early adulthood” should have been more specific in detailing that cardiovascular disease (CVD) was observed only in those children treated with growth hormone and with abnormal biophysical semiotics CVD test. Ichord, the editorialist, responds. Poidvin et al., the article authors, agree that Ichord adequately addressed the raised concerns. —Chafic Karam, MD, and Robert C. Griggs, MD

CLINICAL REASONING: A PUZZLING CASE OF AMNESIA
Andrea M. Harriott, William O. Tatum, Jacksonville, FL: Bereznyakova et al.1 described a woman with focal epilepsy, migraines, and recent coiling of an anterior communicating artery aneurysm. She presented with fever, vomiting, and confusion, forced gaze deviation, and left hemibody jerking followed by an amnestic syndrome, confabulation, and blunted affect. EEG did not detect seizures. Brain MRI revealed restricted diffusion in the anterior fornices and cingulate gyrus. The patient was diagnosed with ischemic stroke related to her recent coil.

Despite the diffusion-weighted abnormalities, post-ictal confusional state with amnesia should be considered as an alternate diagnosis. Sustained versive eye and clonic hemibody movements are important clues that raise suspicion for focal seizure. Confidence in a final diagnosis of stroke should be tempered by the lack of CSF analysis or repeat MRI, and limitations in documenting seizure with interictal EEG. While amnesia can follow stroke involving the thalamus,2 hippocampus, and fornix,3 amnestic syndromes are a common feature of focal epilepsy.4

Furthermore, reversible diffusion abnormalities can follow focal seizures and likely result from network changes in regional cerebral blood flow, oxygen utilization, and electrical stability.5 We enjoyed the article’s discussion yet, in the course of learning neurology, we frequently find that zebras often mingle with horses.

Author Response: Robert Laforce, Jr., Quebec: We thank Drs. Harriott and Tatum for their interest in our report of a puzzling case of amnesia.1 In addition to showing a negative 2-hour EEG monitoring during hospitalization, and absence of seizures over the last 18 months, our patient continues to show the triad of symptoms associated with anterior communicating artery syndrome (ACAS). These symptoms include anterograde amnesia, confabulation, and persistent personality changes. Taken together, we hypothesized that a postictal confusional state with amnesia was unlikely, especially in light of such a prolonged follow-up period without any ictal event. By contrast, an ischemic stroke was clearly documented (both acutely and on follow-up brain imaging) in the anterior fornices and anterior cingulum, both regions well-known to be involved in ACAS. When faced with complex clinical presentations, whether horses or zebras, time remains the neurologist’s best friend.

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GROWTH HORMONE TREATMENT FOR CHILDHOOD SHORT STATURE AND RISK OF STROKE IN EARLY ADULTHOOD

ADULT STROKE RISK AFTER GROWTH HORMONE TREATMENT IN CHILDHOOD: FIRST DO NO HARM
Sergio Stagnaro, Lancenigo, Italy: Poidvin et al.1 found an increased risk of stroke in adults treated with growth hormone (GH) during childhood. Increased mortality rates were also observed by Carel et al.2 in adults treated as children with recombinant GH. We have assessed 95 patients complaining of
aspecific neurologic symptoms using Biophysical Semeiotics Tests (BSTs) with a mean follow-up of 6 years. Out of these patients, 3 developed a stroke.

Interestingly, cardiovascular disease (CVD)—including stroke—was observed only in those patients whose BSTs were already abnormal during the preclinical stage (i.e., when the clinical examination or other instrumental investigations failed to reveal anything suspicious). In our experience, a BST that is abnormal at the preclinical stage is always associated with a congenital risk of developing that particular disorder for which the test is specific: this is called inherited real risk.3,4

Given these premises, the results of Poidvin et al. should have been more specific in detailing that the CVD was observed only in those children treated with GH and with abnormal Biophysical Semeiotics CVD test (CVD inherited real risk).

Editorialist Response: Rebecca N. Ichord, Philadelphia: Stagnaro et al. raised an interesting question regarding the study by Poidvin et al.1 My editorial also expanded on this study’s strengths and limitations.5 Stagnaro et al. considered whether a preexisting condition predisposed those individuals to develop a stroke after GH treatment in childhood. This is certainly possible. However, the comparison of the treated population to an untreated population in this study would have eliminated this effect if this predisposition was randomly distributed in the population.

Stagnaro et al. further suggested that the administration of a BST might disclose a predisposition to adult-onset stroke. While this is an intriguing idea, it is problematic as there is no described BST in this pediatric population, which is proven to be a valid predictor of adult-onset stroke. Moreover, the design of this study involved a retrospective analysis of the association of childhood GH treatment with adult-onset stroke. The suggested approach would have required a prospective design whereby children eligible for GH treatment would be evaluated prospectively for the existence of risk factors for adult-onset stroke. The design of their study precluded this approach. This limitation was acknowledged by the authors. This type of a test would be a welcome addition to the clinical science of childhood precursors of adult-onset cerebrovascular disease.

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CORRECTION
Memory fMRI predicts verbal memory decline after anterior temporal lobe resection

In the article “Memory fMRI predicts verbal memory decline after anterior temporal lobe resection” by M.K. Sidhu et al. (Neurology® 2015;84:1512–1519), originally published ahead of print on March 13, 2015, there is an error in figure 2. The LTLE coronal slice should be on the top row. A corrected version was posted on March 20, 2015. The editorial office regrets the error.