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.

1. Poidvin A, Touzé E, Ecosse E, et al. Growth hormone treatment for childhood short stature and risk of stroke in early adulthood. Neurology 2014;83:780–786.
2. Carel JC, Ecosse E, Landier F, et al. Long-term mortality after recombinant growth hormone treatment for isolated growth hormone deficiency or childhood short stature: preliminary report of the French SAGhE study. J Clin Endocrinol Metab 2012;97:416–425.
3. Marchionni M, Caramel S, Stagnaro S. Inherited real risk of Alzheimer's disease: bedside diagnosis and primary prevention. Front Aging Neurosci Epub 2013 Mar 20.
4. Marchionni M, Caramel S, Stagnaro S. The role of "modified Mediterranean diet" and quantum therapy in Alzheimer's disease primary prevention. J Nutr Health Aging 2014;18:96.
5. Ichord RN. Adult stroke risk after growth hormone treatment in childhood: First do no harm. Neurology 2014;83:776–777.

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.

Author disclosures are available upon request (journal@neurology.org).