

is a risk factor for ADHD that is independent of prenatal exposure to nicotine and other familial risk factors. (Mick E et al, 2002). Genes that influence the risk of alcohol and nicotine use may also influence vulnerability to ADHD. Advice regarding hazards of nicotine and alcohol exposure and monitoring of blood count and thyroid function during pregnancy are particularly important for patients with a family history of ADHD. (Millichap JG. Etiological classification of attention-deficit/hyperactivity disorder. *Pediatrics* 2008;121:e358-e365).

## FUNCTIONAL ANATOMY OF GERSTMANN SYNDROME

Structural and functional neuroimaging was used to examine a common denominator for the clinical triad of Gerstmann syndrome (a selective association of acalculia, finger agnosia, left-right disorientation, and agraphia) in a study at centers in Gif-sur-Yvette and Orsay, France; and University College, London, UK. None of the five neurologically healthy right-handed volunteers (1 female, 4 male, mean age 21 years) showed parietal overlap of cortical activation patterns from the 4 cognitive domains. Instead, these specific parietal activation patterns consistently connected to a small region of subcortical parietal white matter at a location congruent with the lesion in a documented case of Gerstmann's syndrome. Gerstmann's tetrad does not arise from damage to a shared cortical substrate in the left parietal lobe but from intraparietal disconnection after damage to a focal region of subcortical white matter. (Rusconi E, Pinel P, Eger E, et al. A disconnection account of Gerstmann syndrome: functional neuroanatomy evidence. *Ann Neurol* Nov 2009;66:654-662). (Respond: Dr Andreas Kleinschmidt, INSERM Unit 562, F91191 Gif-sur-Yvette, France. E-mail: [kleinschmidt@cea.fr](mailto:kleinschmidt@cea.fr)).

COMMENT. In children with learning disabilities, Gerstmann syndrome is found "forme fruste" more often than a complete *Grundstörung* tetrad, as proposed by Gerstmann (1940). The above disconnection account of Gerstmann syndrome with subcortical white matter pathology might explain the developmental form of the syndrome sometimes encountered in children with learning and attention disorders. A familial form of the syndrome is reported in an 11-year-old boy of normal intelligence who had a profound dyscalculia with lack of cardinal/ordinal skills acquisition, dysgraphia, right-left disorientation, and finger agnosia. Several male family members also had the complete syndrome complicated by dyslexia. At birth, the boy was hypotonic, his motor development was delayed, walking independently at 2 and ½ years. Examination revealed gross and fine motor incoordination and inattention. Ordinal number use was compensated for by visual and verbal memory cues but cardinal number skills did not improve. (Ta'ir J et al. *Brain Cogn* 1997;35:184-206).

## TREATMENT OF ADHD AND EPILEPSY OR ABNORMAL EEG

The comorbidity of ADHD and epilepsy is reviewed by researchers at the Universities of Rome and Chieti, Italy. In ADHD children the prevalence for epileptiform EEG discharges ranges from 5% to 60%; 14% of ADHD children with epileptiform EEG abnormalities are at risk of developing seizures (Richer LP, 2002). In children with