

COMMENT. The authors recommend that children who present with ADHD, OCD, or tic disorder of late onset and in the absence of a family history should be investigated with brain MRI. This report emphasizes the importance of a neurological basis for ADHD and tics, and the need to exclude structural lesions in the cerebrum and connections with the basal ganglia. Children with ADHD of a genetic origin alone develop symptoms early, before 7 years of age. Other structural lesions sometimes associated with ADHD include temporal lobe arachnoid cyst (Millichap JG, *Neurology* 1997;48:1435-1439), decreased size of splenium of corpus callosum (Semrud-Clikeman M et al, 1994), and decreased volume of prefrontal cortex, caudate nucleus, and globus pallidum (Castellanos FX et al, 1996).

Two children who presented with chorea, athetosis and dystonia were diagnosed with astrocytoma of the thalamus and a pontine midbrain tumor. (Millichap JG, Miller RH, Backus RE, *JAMA* 1962;179:589-593). Diagnoses of rheumatic chorea, dystonia musculorum deformans, and encephalitis were entertained before fractional pneumoencephalography defined an expanding lesion. Of 300 intracranial tumors in children treated at the Mayo Clinic between 1950 and 1960, 4 per cent involved the basal ganglia, but less than 1 per cent were associated with involuntary movements. A review of the literature found only 9 similar cases between 1923 and 1961.

## **ATTENTION DEFICIT AND LEARNING DISORDERS**

### **TOBACCO AND LEAD EXPOSURES AND ADHD**

The independent and joint effects of prenatal tobacco and childhood lead exposures on ADHD in a national sample of US children were studied by researchers at Cincinnati Children's Hospital, OH; British Columbia Children's Hospital, Vancouver, Canada; and University of North Carolina, Chapel Hill, NC. Using data from a 2001-2004 National Health and Nutrition Examination Survey, a total of 8.7% of children 8 – 15 years of age met criteria for ADHD. Prenatal tobacco exposure and third tertile (1.3-5 mcg/dL) current blood lead concentrations were independently associated with ADHD. (Children with lead levels >5mcg/dL were excluded). Compared with children with neither exposure, children with both exposures had an even greater risk of ADHD than if the independent risks were multiplied ( $p<0.001$ ). Children who attended preschool and boys (vs girls) had increased likelihoods of ADHD. Mexican American and black children had lower risks compared to non-Hispanic white children. (Froehlich TE, Lanphear BP, Auinger P, et al. Association of tobacco and lead exposures with attention-deficit hyperactivity disorder. *Pediatrics* Dec 2009;124:e1054-e1063). (Respond: Tanya Froehlich MD, Cincinnati Children's Hospital Medical Center, 3333 Burnet Ave, Mail Location 4002, Cincinnati, OH 45229. E-mail: [tanya.froehlich@cchmc.org](mailto:tanya.froehlich@cchmc.org)).

COMMENT. The association of prenatal tobacco exposure and ADHD has been demonstrated in previous studies, one involving a national data base population, but this is the first indication of a potentiation of effect when combined with childhood low level lead exposure. A similar association with ADHD is reported with a combination tobacco and alcohol prenatal exposure. Patients with ADHD are 2.5 times more likely to be exposed to alcohol in utero and 2.1 times more likely to be exposed to tobacco. Alcohol

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