

Neurology, Children's Hospital of the King's Daughters, Eastern Virginia Medical Neurology School, 850 Southampton Ave, Norfolk, VA 23509, USA).

COMMENT. Reasons for exclusion from the diagnosis of abdominal migraine included irritable bowel syndrome (41%), renal disease (4%), pre-existing neurologic disorder (7%), inflammatory bowel disease (16%), eosinophile esophagitis (4%), and "other" (28%), including food allergies, celiac disease, cystic fibrosis, autism spectrum disorder, and anatomic abnormalities. The differential diagnosis of chronic recurrent abdominal pain in children is broad and may require the expertise of pediatrician, gastroenterologist, psychiatrist, and neurologist. Diagnosis is based on exclusion criteria and positive criteria. Positive ICHD-2 criteria for AM are listed above. Among the neurologic disorders sometimes causative, abdominal epilepsy may be differentiated from migraine by the occurrence of other features of partial seizures, the family history, and an EEG showing interictal epileptiform discharges. Migraine and epilepsy are uncommon causes of recurrent abdominal pain, and investigation of other causes is paramount. My last consultation on a case of recurrent abdominal pain referred for EEG and exclusion of abdominal epilepsy eventually proved to have pancreatitis necessitating surgery.

**Childhood abuse and migraine.** Neurologists at the Universities of Toledo, OH and Johns Hopkins, Baltimore, MD, review the neurobiological effects of abuse on brain function and structure in relation to migraine (Tietjen GE, Peterlin BL. **Headache** June 2011;51:869-879). A possible role of early life stress on the pathogenesis of migraine may impact girls more than boys and may become hard-coded into the genome, leading to migraine at a later age. The emerging field of epigenetics may suggest new treatment strategies such as serotonin-specific reuptake inhibitors that reverse effects of maltreatment and decrease the corticotropin releasing hormone response to stress.

## METABOLIC DISORDERS

### **INFANTILE THIAMINE DEFICIENCY AND LANGUAGE IMPAIRMENT**

Developmental language impairment in 59 children, aged 5-7 years, exposed to a thiamine deficiency in defective milk formula fed during the first year of life is studied by researchers at Tel Aviv University and Sourasky Medical Centre, Israel. Various tests of phrase and sentence comprehension, word retrieval and conceptual abilities were compared in thiamine deficient and normally fed controls. Almost all (57) of the 59 thiamine-deficient children examined had language impairment, compared with 3 of the 35 controls (9%), whereas conceptual and cognitive abilities were spared (only 6 (10%) were conceptually impaired). (Fattal I, Friedmann N, Fattal-Valevski A. The crucial role of thiamine in the development of syntax and lexical retrieval: a study of infantile thiamine deficiency. **Brain** June 2011;134:720-1739). (Response: Prof Naama Friedmann, Language and Brain Lab, School of Education, Tel Aviv University, Tel Aviv 69978, Israel. E-mail: [naamafr@post.tau.ac.il](mailto:naamafr@post.tau.ac.il)).

COMMENT. In 2003, 20 infants were hospitalized in Israel with severe neurological symptoms, including ophthalmoplegia, vomiting, nystagmus, seizures and coma. Brain MRI showed hyperintense signal in the basal ganglia, mamillary bodies and periaqueductal grey matter. Two died of cardiomyopathy and 10 had residual cardiac and brain damage. Wernicke's encephalopathy was suspected, and examination of the infant formula found an absence of vitamin B1. Treatment with supplementary thiamine resulted in improvements in affected infants. The above study was conducted on patients fed the same thiamine-deficient formula but showing no neurological deficits. The research demonstrates the influence of diet deficiencies, specifically thiamine, on the developing brain of infants and the effect on language performance in later childhood.

## **SEIZURE DISORDERS**

### **INTERHEMISPHERIC FUNCTIONAL CONNECTIVITY IN CHILDHOOD ABSENCE EPILEPSY**

Using a blood oxygen level-dependent resting functional connectivity approach to analyze EEG-fMRI data, the properties of bihemispheric brain networks in 16 patients with childhood absence epilepsy (CAE) were investigated during the interictal period, in a study at Yale University School of Medicine, New Haven, CT. Resting functional connectivity between hemispheres was significantly increased in the lateral orbitofrontal cortex of patients with CAE compared to normal matched controls. The abnormal increased connectivity between hemispheres is demonstrated in patients with CAE both during and between seizures. Resting functional connectivity analysis is a promising biomarker for altered brain function in CAE during the interictal period. The findings provide further evidence for focal bilateral network abnormalities in CAE. (Bai X, Guo J, Killory B, et al. Resting functional connectivity between the hemispheres in childhood absence epilepsy. **Neurology** June 7, 2011;76(23):1960-1967). (Response and reprints: Dr Hal Blumenfeld, Dept Neurology, 333 Cedar St, New Haven, CT 06520. E-mail: [hal.blumenfeld@yale.edu](mailto:hal.blumenfeld@yale.edu)).

COMMENT. In an editorial, Zempel JM and Ciliberto M (**Neurology** 2011;76:1952-1953) discuss fluctuating concepts of CAE referring to the recent interest in attention, behavioral, cognitive, and linguistic impairments complicating CAE. The comparative trial of 3 AEDs for absence epilepsy (ethosuximide, valproic acid, and lamotrigine) were all effective for seizure control; ethosuximide had an additional beneficial effect, improved attention. Increased lateral orbitofrontal connectivity demonstrated in the Yale study may be a biomarker of this attention deficit in CAE. Functional connectivity diagrams of brain networks and interictal spike/wave paroxysms may, in the future, predict the degree of cognitive impairment and lack of awareness and attention in a child with CAE.

**Motor system hyperconnectivity in juvenile myoclonic epilepsy.** Vollmar C et al report increased functional connectivity between the motor system and frontoparietal cognitive networks, providing an explanation for the triggering of myoclonic jerks by cognitive effort in JME patients. (**Brain** June 2011;134:1710-1719).