

Cluster headache is uncommon in childhood. Onset is usually in the second and third decade. A retrospective review of cases attending a pediatric neurology clinic in Bristol, UK, between 2000 and 2005 identified 11 patients (7 male, 4 female) with median age of onset of 8.5 years (range 2-14). Median age at diagnosis was 11.5 years (range 7-17). Eight had episodic and 3 had chronic cluster headache. Most had cranial autonomic activation and agitated movement. (Majumdar A, Ahmed MA, Benton S. **Eur J Paediatr Neurol** 2008; Dec 22. (Epub ahead of print).

Maytal J et al modifications of the IHS criteria for pediatric migraine found that decreasing the length of attacks below 2 to 48 hours would increase the sensitivity of diagnosis, but adding associated autonomic symptoms of facial redness or pallor, while improving sensitivity, also decreased the specificity. The addition of CAS while helpful was not recommended. (**Neurology** 1997;48:602-607). Perhaps more attention to autonomic symptoms and behavior in diagnosis of children with migraine would be warranted.

TOPIRAMATE-INDUCED COUGH IN MIGRAINE PROPHYLAXIS

Three adults who developed intractable cough during topiramate prophylaxis of migraine are reported from the University of Padua and other centers in Italy. Cough developed early during the titration phase at dose levels of 75-100 mg/day, and resolved rapidly after withdrawal. Secondary causes of cough, including GERD, were excluded. The cough was episodic, dry, and very annoying, especially at night. Despite effective prevention of headache with topiramate, treatment was discontinued. Literature review revealed no previous case reports of cough as a side effect of topiramate treatment for migraine. (Maggioni F, Mampreso E, Mainardi F, Lisotto C, Malvindi ML, Zanchin G. Topiramate-induced intractable cough during migraine prophylaxis. **Headache** Oct 2009; on line). (Respond: Dr F Maggioni, Dept Neurosciences, University of Padua, Via Giustiniani 5, Padova, 35128, Italy).

COMMENT. Topiramate is a first-line treatment for migraine prophylaxis in adults. Adverse events in 20-25% of patients may require discontinuation of treatment but are rarely severe. They include weight loss, dizziness, somnolence, paresthesias, impaired concentration and memory, and language difficulties. Cough has not been reported and the mechanism is unexplained. No patient received ACE inhibitors for hypertension, a known cause of dry cough in adults. Pubmed search for cough with topiramate treatment of childhood epilepsy or migraine found no reports.

NEUROBEHAVIORAL DISORDERS

ISOLATED EPILEPTIFORM EEG DISCHARGES AND AUTISM

The relationship between EEG abnormalities and neuropsychiatric disorders, and their possible clinical significance are reviewed by an investigator at Wayne State University, Detroit, MI, with special attention to the EEG and autism. Approximately one third of children with autistic spectrum disorder (ASD) develop epilepsy. Of 46 consecutive children with autism (34 boys, and 12 girls, mean age 7.8 +/- 2.7 years), 35% had epilepsy (Canitano

R et al. **J Child Neurol** 2005;20:27-31). Hughes and Melyn reported 46% with clinical seizures (**Clin EEG Neurosci** 2005;36:15-20), and Tuchman and Rapin, 11% with epilepsy (**Pediatrics** 1997;99:560-566). Contrary to the current view, interictal EEG discharges in the non-epileptic ASD patient are more likely to signal abnormal brain activity than to represent an incidental finding. Deonna and Roulet (**Epilepsia** 2006;47(suppl 2):79-82) suggest a possible role for epilepsy in the causation of autism. Tuchman and Rapin found a correlation between clinical deterioration of autism and the frequency of epileptiform discharges in the EEG of non-epileptic autistic children during sleep. One half of the epileptiform discharges were centrotemporal in location. Further evidence of the importance of spike localization in the EEG of non-epileptic autistic children is reported by Rossi et al (**Brain Dev** 1995;17:169-174) who found that 45% of cases of epileptiform activity was typical of benign childhood partial epilepsy with centrotemporal spikes. Treatment of isolated spikes in children with autism using anticonvulsant drugs, especially valproate, is controversial, despite some reported favorable results (Hollander E et al. **J Clin Psychiatry** 2001;62:530-534). A recommendation against EEG screening of autistic children is considered unwarranted, given the high frequency of epilepsy and isolated EEG abnormalities in this population. That spike foci may create other brain foci has been shown in patients with repeated EEGs, a finding that underlines the nonbenign nature of the isolated epileptiform discharge. The significance of the EEG abnormalities in children with autistic spectrum disorders and other neurobehavioral disorders (eg ADHD) requires further study. (Boutros N. Epileptiform discharges in psychiatric patients: a controversy in need of resurrection. **Clin EEG Neurosci** Oct 2009;40(4):239-244). (Reprint requests: Nash Boutros MD, Wayne State University, Department of Psychiatry and Behavioral Neurosciences, 2751 East Jefferson, Detroit, MI 48207. E-mail: nboutros@med.wayne.edu).

COMMENT. This review draws attention to the need for greater interest among clinicians in the EEG and childhood neurobehavioral disorders. Controlled trials of anticonvulsant drugs in the treatment of ASD and ADHD may be justified, based on the frequency of epileptiform discharges in non-epileptic children with these disorders. Early diagnosis may be important, as indicated by the progression of EEG abnormalities associated with clinical deterioration of ASD, and development of intellectual disability.

EEG and MRI findings and their relation to intellectual disability in PDD are reported in a retrospective study of 81 patients treated at Ankara University, Turkey. (Unal O et al. **World J Pediatr** Aug 2009;5(3):196-200). One fourth of patients with PDD had EEG and/or MRI abnormalities. Twenty seven percent had abnormal EEGs; 12% had abnormal MRIs, mostly mild cortical atrophy and periventricular leukomalacia. Patients with severe intellectual disability (ID) had a higher rate of EEG abnormalities (50%) than PDD patients without ID (8%), $P=0.03$. The severity of ID was not associated with abnormal MRI. One third of EEG abnormalities were localized to the temporal lobe.

Causes and pathogenetic pathways of autism are discussed by Benvenuto A et al, Pediatric Neurology Unit, Tor Vergata University, Rome, Italy (**World J Pediatr** 2009;5:169-176). Genetic syndromes, mutations, and metabolic diseases account for less than 20% of autistic patients. Chromosomal abnormalities and potential candidate genes are implicated in the disruption of neural connections, brain growth and synaptic/dendritic morphology in autism.