and early intervention with ganciclovir for treatment of sensorineural deafness. (Kimberlin DW et al. **J Pediatr** 2003;143:16-25). This report shows that genetic and immunologic variability affects CMV virulence and may be used as prognostic factors to determine severity of infection and outcome.

Normal psychomotor development in infants with CMV infection treated early with intravenous ganciclovir and antiepileptic drugs. (Dunin-Wasowicz D et al. **Epilepsia** July 2010;51:1212-1218). Onset of seizures was generally in the first 6 months of life, most frequently in the second and fourth months. Seizures were controlled in 19 infants (59.4%), and treatment was withdrawn successfully in 11 (34.4%) children after 30-36 months. At a median follow-up of 7 years, psychomotor development was normal in 15 (46.9%), including the 11 patients withdrawn from AEDs. Cerebral palsy was diagnosed in 17 (53.1%).

SEIZURE DISORDERS

EEG EPILEPTIFORM DISCHARGES IN "HEALTHY" CHILDREN

Researchers from Helios Klinikum Wuppertal, Germany, analyzed the prevalence of epileptiform discharges in digitally recorded EEG (DEEG) of 382 healthy children (226 male, 156 female) ages 6-13 years, and compared the data to those of previously published paper analog recordings. The patients referred for EEG had suffered minor head trauma without impaired consciousness or amnesia; they had no focal neurological deficits. Recordings were a minimum of 20 min and included hyperventilation and photic stimulation. Epileptiform discharges were recorded in 25 (6.5%) children; 4 had generalized or bifrontal spikes, 12 had constant localized focal discharges, and 9 showed multifocal discharges. Epileptiform discharges were "rolandic" in 16 children (4.2%). Afebrile seizures, 1 or more, occurred in 3 (12%) of the 25 children with epileptiform discharges during a median follow-up period of 4.2 years (range 1.2-7.1 years).

Comparing prevalences of epileptiform EEG discharges in healthy children, those reported in 3 previous studies were lower than the present cohort (3.5%, 3.5%, and 5%) and significantly lower in one report ($p<0.005$). Subgroup analysis according to age and sex also showed differences, with a higher prevalence in a male subgroup and younger age group of 6-9-year old children in the present cohort. Comparison of prevalence data in selected previous reports of children with ADHD found no significant differences with the present data obtained from healthy children. The findings indicate the need for further research, using digitally recorded EEG, but the significance of the comparative data is limited by the small number of children examined. (Borusiak P, Zilbauer M, Jenke ACW. Prevalence of epileptiform discharges in healthy children - new data from a prospective study using digital EEG. **Epilepsia** July 2010;51:1185-1188). (Respond: Dr Peter Borusiak, Wuppertal, Germany. E-mail: peter.borusiak@helios-kliniken.de).

COMMENT. In this study using digital EEG, the prevalence of epileptiform discharges recorded in "healthy" children is 6.5%, and higher than that reported in three previous studies. The authors also claim that their findings in "healthy" children are not different from those reported in studies of the EEG in children with ADHD. Several

questions limit the significance of the findings: 1) Should the children be accepted as "healthy"? They were referred for EEG because of minor head trauma, and the sequelae of mild traumatic brain injuries are controversial and sometimes serious (Lee LK. **Pediatr Emerg Care** 2007;23:580-583); Hamilton M et al. Incidence of delayed intracranial hemorrhage in children after uncomplicated minor head injuries. **Pediatrics** July 2010;126:e33-e36); 2) A proportion of the patients with epileptiform EEG discharges developed afebrile seizures and some had seizure recurrences and epilepsy. These should have been excluded from the data; 3) Of 7 previous published reports of EEG epileptiform discharges in nonepileptic children with ADHD the authors selected only 2, both with the lowest prevalence of abnormalities; 5 previous reports with significantly higher prevalences were omitted from their discussion.

In a recent study of 612 children evaluated for symptoms of ADHD complicated by episodic altered awareness, the frequency of epileptiform abnormalities in the sleep-deprived EEG was 25.7% and similar to the mean frequency for 7 published studies (Millichap JJ, Stack CV, Millichap JG. **J Child Neurol** 2010; in press). Our findings and previous reports are not in agreement with the current German study conclusions. The weight of evidence favors an increased prevalence of epileptiform EEGs in non-epileptic children with ADHD.

When stimulant medication is prescribed in ADHD children with abnormal EEG, a conservative dose schedule may be advisable. In a study of children with ADHD complicated by EEG rolandic spikes, seizures occurred in 16.7% following methylphenidate compared with 0.6% in a group of ADHD children with normal EEGs. (Hemmer SA et al. **Pediatr Neurol** 2001;24:99-102). The safety of high-dose stimulants in children with ADHD and epileptiform EEGs untreated with antiepileptic medication is unclear. An EEG may allow the physician and parent to make an informed decision regarding risks and benefits of high-dose stimulant versus nonstimulant selection and immediate-release MPH vs extended-release formulations.

OROS-MPH (Concerta) treatment of ADHD with epilepsy. A current report of a randomized control trial of OROS-methylphenidate, 18-54 mg daily for weekly periods, in 33 patients, 6-18 years of age, taking antiepileptic drugs, found an increased daily risk of seizures with increasing dose of OROS-MPH, an extended-release medication. Potential safety concerns require further study. (Gonzalez-Heydrich J, Biederman J et al. **Epilepsy Behav** July 2010;18:229-237).

ALDH7A1 DEFICIENCY AND PYRIDOXINE-DEPENDENT EPILEPSY

Researchers at University College and Great Ormond Street Hospital for Children, London, and other centers in the UK and Europe investigated the genotypic and phenotypic spectrum of pyridoxine-dependent epilepsy (PDE) by measurement of urinary alpha-aminoadipic semialdehyde (a-AASA) concentration and mutational analysis of the ALDH7A1 gene that encodes antiquitin. Twenty-one new patients with elevated AASA and 37 individuals from 30 families with mutations in ALDH7A1 were identified; 17 of these were novel and all others were published previously. The clinical spectrum of antiquitin deficiency and pyridoxine-dependent epilepsy includes ventriculomegaly,