epilepsy the prevalence of ADHD is 30-40% (Dunn DW, 2005), much higher than that in the general pediatric population (Schubert R, 2005). Studies differ in the number of patients, severity and type of epilepsy, and ADHD diagnosis. The types of epilepsy associated with comorbid ADHD include frontal lobe, absence, and Rolandic epilepsies. Prolonged focal epilepsy, as occurring in electrical status epilepticus during slow wave sleep, is associated with attention deficits and hyperactivity (Stores G, 1990).

Treatment of comorbid ADHD and epilepsy is a challenge because some AEDs may cause behavioral abnormalities, and stimulant medications may lower the seizure threshold. Choice of AEDs for seizure control that may improve behavior and attention in children with epilepsy and ADHD include carbamazepine or oxcarbazepine, and valproate, lamotrigine or levetiracetam. AEDs that can exacerbate behavioral disorders and should be avoided include barbiturates and topiramate. For treatment of ADHD in children with epilepsy, studies show that methylphenidate is safe in patients whose epilepsy is controlled with AEDs (Gross-Tsur V et al, 1997). Atomoxetine is recommended as an alternative to methylphenidate in some patients with ADHD and epilepsy or epileptiform discharges in the EEG (Hernandez A et al, 2005; Wernicke JF et al, 2007). Atomoxetine does not interact with AEDs while methylphenidate may increase phenytoin serum concentrations. Methylphenidate serum concentrations are lowered by carbamazepine, leading to a loss of efficacy against ADHD. (Schaller JL et al, 1999). Methylphenidate is considered the most effective and safest treatment for ADHD. (Parisi P, Moavero R, Verrotti A, Curatolo P. Attention deficit hyperactivity disorder in children with epilepsy. **Brain Dev** Jan 2010;32:10-16). (Respond: Dr Paolo Curatolo. E-mail: curatolo@uniroma2.it).

COMMENT. Children with ADHD are at increased risk for seizures, and children with epilepsy are at significant risk for ADHD. Management of the comorbid disorder presents a challenge because treatments for ADHD may lower seizure threshold and AEDs may exacerbate symptoms of ADHD. CNS stimulants should be avoided in children with seizures and used with caution in those with an abnormal EEG (PDR, 2009). Studies show that methylphenidate (MPH) for ADHD is safe provided that comorbid epilepsy is under control with AEDs. In patients with epileptiform EEG untreated with AEDs, the safety of MPH is less well established. Low-dose MPH does not increase the incidence of EEG epileptiform discharges. In a patient who fails to respond to low-dose stimulant, the demonstration of epileptiform discharges by utilization of a sleep-deprived sleep EEG should aid in the choice of alternative non-stimulant medication (Millichap JJ, Stack C. Millichap JG, unpublished observations).

SEIZURE DISORDERS

ACTH-INDUCED CHANGES IN IMMUNITY IN INFANTS TREATED FOR WEST SYNDROME

The timing of vaccinations is delayed after ACTH therapy in Japanese infants, because the immune system may be compromised. To determine the duration of the effect on the immune system, researchers at Kurume University School of Medicine and other centers in Japan examined changes in immunity levels before and after ACTH

therapy by measurement of white blood cell, lymphocyte, T/B cell, CD4 and CD8 T cell counts. The CD 4/8 ratio, lymphocyte blastoid transformation, and levels of IgA, IgM, and IgG were also measured before, immediately after, and at 1, 3, 6, and 12 months after ACTH therapy. Cortrosyn Z, a synthetic analogue of ACTH, was administered at a relatively low dosage between 0.015 mg/kg/day and 0.00625 mg/kg/day. Lymphocyte counts and CD4 T cell levels were significantly decreased immediately after and at 1 and 3 months after therapy, and they returned to normal but not pretreatment levels at 6 and 12 months. Helper T cells were more depressed than cytotoxic T cells, but immunoglobulin levels did not change after ACTH therapy. (Ohya T, Nagai T, Araki Y, et al. A pilot study on the changes in immunity after ACTH therapy in patients with West syndrome. **Brain Dev** Dec 2009;31:739-743). (Respond: Dr T Matsuishi. E-mail: tmatsu@med.kurume-u.ac.jp).

COMMENT. Lymphocyte and T cell counts are significantly decreased immediately after and at 1 and 3 months after ACTH therapy, and then gradually recover. A delay of vaccinations for 6 months after ACTH therapy, as practiced by 55% of pediatricians in Japan, is justified based on the results of this study. The authors point out that cell counts used are not direct measures of the actual immunological response. Rather, they indirectly reflect the ability of the individual patient to produce antibodies with vaccinations.

FOCALITY AND HETEROGENEITY OF FEBRILE SEIZURES

The semiology of febrile seizures (FS), their focality, relation to hippocampal damage, and heterogeneous origin are critically reviewed by researchers at the Institute of Child Health, and Great Ormond Street Hospital, London, UK. Early semiology, the most important clue to focal origin, has been neglected. Aural features have temporal lobe characteristics consisting of fear and lip smacking, and rare motor manifestations. FS arise in the hippocampus with transient and mild temporal lobe features and rapid spread to neocortex. FS include an unknown proportion of non-epileptic reflex and asystolic attacks. The definition of FS with fever not directly involving the nervous system may need to be modified by possible direct cerebral toxin involvement eg Shigella, HHV6 and malaria infections. The early seizure semiology should be used to separate the various seizures included as FS and to determine whether atypical FS such as Dravet syndrome and generalized epilepsy with FS plus have similar focal/temporal lobe features. (Neville BGR, Gindner D. Febrile seizures-semiology in humans and animal models: evidence of focality and heterogeneity. **Brain Dev** Jan 2010;32:33-36). (Respond: Dr Brian GR Neville. E-mail: B.Neville@ich.ucl.ac.uk).

COMMENT. Drs Neville and Ginder emphasize the importance of early seizure semiology in our understanding of the FS origin, and refer to the FS as a "specific epilepsy syndrome." Investigators have debated the definition of FSs and their relation to epilepsy for more than a century. Is a FS a distinct disease entity or a form of epilepsy? The early literature was divided between those who favor the former or the latter definition. WG Lennox (1953) used the terms "mild" and "pure" forms of epilepsy, whereas Livingston (1954) reverted to the older concept of a specific entity, the "simple