VIRAL INFECTIONS AND FEBRILE SEIZURES

Among abstracts of the 19th annual conference on febrile convulsions, Tokyo, Japan, Dec 14, 1996, and reviewed by Dr Yukio Fukuyama (<u>Brain Dev</u> July 1997;19:369-374), four referred to the association with viral infection.

Abe T, Teikyo University School of Medicine, Tokyo, studied the mechanism of convulsions associated with rotavirus (RV) infection. Three percent of children with RV diarrhea develop convulsions compared to 0.3% with non-RV diarrheal infections. The mechanism may involve electrolyte imbalance or the direct invasion of neurons by RV virus or enterotoxin.

Asano Y et al, Fujita School of Medicine, Toyoahe, Aichi, report on human herpesvirus 6 (HHV-6B) as a cause of febrile seizures in 8 - 36% of children with exanthem subitum. The viral DNA is found in CSF samples within 4 days after onset of infection. Afebrile seizures may also occur with HHV-6 infection and exanthem subitum and without evidence of invasion of the CNS by the virus. A vasculitis is proposed as the mechanism of afebrile seizures with HHV-6 infection. (Ojima K et al).

Kajitani T et al, Kawasaki Hospital, Okayama, reviewed the causes of fever in 197 children admitted with febrile seizures. Viral infections accounted for 82%, of which 3% were exanthem subitum.

COMMENT. Seizures with fever, HHV-6 and exanthem subitum are frequently prolonged, recurrent, and complex, and sometimes a manifestation of encephalitis or encephalopathy. HHV-6 infection and febrile seizures are reviewed in <u>Progress in Pediatric Neurology III</u>, 1997, pp 24-28; VOL II, pp 27-28.

ATTENTION DEFICIT AND LEARNING DISORDERS

LONG-TERM STIMULANT THERAPY AND ADHD OUTCOME

The long-term effects of amphetamine sulfate (5 mg AM and lunchtime, increasing to a mean dose of 17 mg/d, 0.52 mg/kg body weight, and a maximum of 45 mg/d) were evaluated in 62 children, aged 6 to 11 years, with ADHD treated for 15 months in a randomized, double-blind, placebo-controlled study at the Child and Adolescent Psychiatry University departments in Goteborg. Uppsala, Malmo-Lund, and Umea, Sweden. Twent six (42%) had comorbid diagnoses, including pervasive developmental disorders, mild retardation, and oppositional defiant disorder. Treatment was stopped or changed to open treatment, usually within 3 months, in 71% of the placebo group and 29% of those receiving amphetamine. Amphetamine was superior to placebo in effects on inattention, hyperactivity, and disruptive behaviors, measured by Conners parent and teacher scores, and on scores on the WISC-R. The mean change in IO from 0 to >9 months was +4.5 with amphetamine and +0.7 for placebo for 6 months or more. Decreased appetite occurred in 56% of the amphetamine group. Abdominal pain recorded in 32% was no more frequent during amphetamine treatment than placebo. Tics diagnosed before baseline in 4 children were exacerbated during double-blind amphetamine (15 mg/d)in one; 18 developed tics during the study but placebo and amphetamine were equally represented. Visual hallucinations occurred in 3 during amphetamine and required reduction in dose or drug withdrawal. Amphetamine inhibited weight gain but had no significant effect on growth. (Gillberg C, Melander H. von Knorring A-L et al. Long-term stimulant treatment of children with