

(range 5 days to 14 years). The 61 school-age children had lower performance and full-scale IQs than their randomly selected, age- and sex-matched controls. Visual acuity was more often reduced and EEG and ENG abnormalities were more frequent. A poor prognosis was infrequent, the incidence being 3.5 per million children at risk annually. Dysarthria was the most frequent sequel of HSV encephalitis. Ataxia, seen most commonly with varicella encephalitis in the acute phase, persisted in only 1 of 17 children at follow-up examination. The prognosis for childhood encephalitis in this study is much better than anticipated on the basis of earlier follow-up studies that included a greater number of HSV cases (Rantala H et al. Outcome after childhood encephalitis. Dev Med Child Neurol Oct 1991; 33:858-867).

**COMMENT.** A case of severe macrocephaly and brain damage is reported in association with second trimester congenital varicella infection from the Departments of Neurology and Clinical Genetics, The Hospital for Sick Children, Great Ormond Street, London, England (Scheffer IE, Baraitser M, Brett EM. Dev Med Child Neurol Oct 1991; 33:916-920).

## **SEIZURE DISORDERS**

### **VALPROIC ACID AND CARNITINE METABOLISM**

Plasma total, free, and acyl carnitine levels were determined in children treated with valproic acid at the Valley Children's Hospital and University of California, San Francisco, Fresno, CA. The mean total carnitine level was significantly lower in patients given valproic acid polytherapy compared with normal subjects and with those receiving valproic acid monotherapy. The levels in both the valproic acid monotherapy and polytherapy groups were significantly lower than those treated with other antiepileptic drugs. The mean free carnitine level was significantly lower in both the valproic acid monotherapy and polytherapy groups compared to normal subjects and the other antiepileptic drug group. Acylcarnitine levels were also lower in the polytherapy valproic acid group compared to the monotherapy valproic acid group and those receiving other antiepileptic drugs. The study indicates that a general decrease in the carnitine pool should be anticipated in patients taking valproic acid polytherapy and to a lesser degree, monotherapy. (Opala G, et al. The effect of valproic acid on plasma carnitine levels. AJDC Sept 1991; 145:999-1001).

**COMMENT.** Plasma and erythrocyte carnitine was significantly lower in 37 children on sodium valproate alone or in combination with other drugs compared to levels in 22 children on drugs other than sodium valproate in a study from the Royal Aberdeen Children's Hospital, University of Aberdeen, Scotland (Thom H et al. Ammonia and carnitine concentrations in children treated with sodium valproate compared with other anticonvulsant drugs. Dev Med Child Neurol Sept 1991; 33:795-802). Plasma ammonia was also elevated in the valproate

group. Urinary free carnitine was reduced in valproate treated children with a significant increase in the ratio of bound to free carnitine. Carnitine supplementation is recommended.

Alterations of renal carnitine metabolism by valproic acid were studied in mice at the University of Santiago de Compostela, Spain (Camina MF et al. Neurology Sept 1991; 41:1444-1448). Valproic acid induced a significant increase in renal clearance of acylcarnitine without affecting that of free carnitine, whereas other anticonvulsants increased clearance of free carnitine but not that of acylcarnitine.

**Erratum.** We thank Dr. J.M. Prats, Hospital de Cruces, Bilbao, Spain, for the correction of an error in the article concerning sodium valproate treatment of infantile spasms reviewed in Ped Neur Briefs Aug 1991; 5:63. The sentence should read as follows: "The hypsarhythmia EEG pattern was controlled after two weeks treatment with VPA 100 to 300 mg/Kg/daily in 80% of patients."

## **EPILEPSY AND MENTAL RETARDATION**

The cumulative risk of seizures and epilepsy was studied in a prospectively identified cohort of 221 children with mental retardation born between 1951 and 1955 in Aberdeen, Scotland and reported from the Montefiore-Einstein Epilepsy Management Center, Albert Einstein College of Medicine, Bronx, NY. Epilepsy had developed in 15% by 22 years of age. The cumulative risk of developing epilepsy varied from 5% in children with mental retardation to 75% in those with mental retardation associated with a postnatal brain injury. An apparent increased risk of epilepsy associated with severe mental retardation was attributed to the higher proportion of postnatal brain injury among this group. The cumulative risk of epilepsy at 22 years was 38% in children with mental retardation and cerebral palsy and 66% in those with postnatal injury. Of 20 children whose initial seizure was a febrile convulsion, 35% went on to develop epilepsy. The authors conclude that in the absence of associated disability or postnatal injury, the risk of epilepsy in the retarded population is low. (Goulden KJ, Shinnar S et al. Epilepsy in children with mental retardation: a cohort study. Epilepsia Sept/Oct 1991; 32:690-697).

**COMMENT.** This study is important in counseling and management of the child with mental retardation. The incidence of various seizure patterns known to be associated with mental retardation, particularly infantile spasms and Lennox-Gastaut, in the subgroups of retarded children would be of interest. West and Lennox-Gastaut syndromes are reviewed from the Children's Hospital, Ohio State University, Columbus, OH (Donat JF, Wright FS. Epilepsia July/Aug 1991; 32:504-509).