and the presence of T2 weighted hyperintensities in the MRI of 52 affected children who had no major neurologic complication or frank retardation. Further studies are needed to define the significance of these hyperintense foci.

SEIZURE DISORDERS

CSF GLUCOSE IN FEBRILE CONVULSIONS

The effects of convulsion and fever on the CSF and blood glucose concentrations in febrile and non-febrile children, with and without convulsions, have been studied at the Department of Paediatrics, Kuopio University Hospital and Department of Pharmacology and Toxicology, University of Kuopia, Kuopio, Finland. The concentration of glucose in the CSF was significantly higher in febrile children with and without convulsions than in non-febrile, non-convulsive children. Both fever and convulsions increased the CSF glucose levels. The body temperature plotted against the CSF glucose showed a linear correlation. Blood glucose parallelled CSF levels in all groups. Hyperglycemia and elevated CSF glucose in febrile convulsions are apparently secondary to both the fever and convulsion, not the convulsion alone. (Kiviranta T et al. The role of fever on cerebrospinal fluid glucose concentration of children with and without convulsions. Acta Paediatr 1995;84:1276-9). (Respond: Dr T Kiviranta, Department of Paediatrics, Kuopio University Hospital, PO Box 1777, FIN-70211, Kuopio, Finland).

COMMENT. Of 110 patients with febrile seizures examined personally, the cerebrospinal fluid was essentially normal in 86 tested. The concentration of sugar was greater than 80 mg/100 ml in 24 patients and 100 mg/100 ml or higher in 11. (Millichap JG et al. 1960). A review of the literature in the 1960s revealed 18 publications between 1934 and 1964, which included the CSF findings of 500 children with febrile convulsions. Elevations of CSF sugar were found in only three reports, in addition to my own study, the first in 1938, and these involved 37 of 68 patients tested. (Millichap JG. Febrile Convulsions, New York, Macmillan, 1968). The present study attempts to elucidate the mechanism of the increased CSF sugar concentration found in some children with febrile convulsions. Both fever and convulsion were found to have a role in elevating the CSF sugar levels.

PAINFUL HAND SEIZURES

A 14-year-old boy with habitual painful seizures of the backs of both hands since age 4 is reported from the Department of Pediatric Neurology, Osaka Medical Center, Japan. He had three febrile convulsions from one to three years of age. Painful hand seizures occurred 5 - 15 times daily, lasting 15 - 60 seconds, and occasionally followed by loss of consciousness and postictal confusion but no secondarily generalized seizures. Seizures were resistant to conventional medications until 13 years of age, when they showed some response to polytherapy with carbamazepine, valproate, and clorazepate. Interictal EEG showed frequent spikes and spike-waves over the right frontopolar area with spread to the left frontal region. Ictal EEG showed right temporal 4-6Hz rhythmic activity after a pain sensation. CT and MRI were normal. SPECT showed right temporal hypoperfusion. These secondary sensory seizures were thought to originate from the S2A area. (Otani K et al. Bilateral painful epileptic seizures of the hands. Dev Med Child Neurol Oct 1995;37:933-

936). (Respond: Dr K Otani, Department of Pediatric Neurology, Osaka Medical Center, 840 Murodo-cho, Izumi, Osaka 590-02, Japan).

COMMENT. The authors cite 4 reports including eight previous patients with secondary sensory seizures published in the last 40 years, the first involving 2 patients of Penfield and Jasper (1954). One study was entitled 'Sensory seizures mimicking a psychogenic seizure.' (Lessor RP et al. Neurology 1983;33:800). It is certainly conceivable that the diagnosis is sometimes overlooked and the symptoms misinterpreted as psychogenic.

EEG MONITORING OF NEONATAL SEIZURES

Sixty-three neonates were investigated using prolonged video/EEG monitoring to identify seizures and determine the diagnostic efficiency of clinical observation and short duration EEGs at the Department of Paediatric Neurology, Prince of Wales Children's Hospital, Sydney, NSW, Australia. Thirty-two patients had seizures confirmed. Clinical observations after anticonvulsant treatment identified seizures in 66%, and a 60 min EEG revealed electrographic seizures in 76%, after phenobarbital treatment, and in 50% after addition of phenytoin. Short duration EEG avoids misdiagnoses in most patients with ambiguous clinical signs and aids substantially in the identification of neonatal seizures. (Bye A, Flanagan D. Electroencephalograms, clinical observations and the monitoring of neonatal seizures. J Paediatr Child Health December 1995;31:503-507). (Respond: Dr A Bye, Prince of Wales Children's Hospital, High St, Randwick, NSW 2031, Australia).

COMMENT. When clinical signs of seizures are controlled by anticonvulsants, a 60 min EEG is required to uncover subclinical neonatal seizures, and in some cases, especially when phenytoin has been given in addition to phenobarbital, prolonged video/EEG monitoring may be necessary in diagnosis. An EEG after infusion of anticonvulsant does not gaurantee seizure identification, but the probability of diagnosis increases in relation to the length of the recording. In a study at the Magee-Womens Hospital, Pittsburgh, PA, more than 50% of 92 neonates with seizures had only electrographic expression of seizures, and 16% exhibited electroclinical dissociation. (see Progress in Pediatric Neurology II, PNB Publishers, 1994, pp 11-16).

DEGENERATIVE AND METABOLIC DISORDERS

CARNITINE DEFICIENCY SYNDROMES

Carnitine deficiency syndromes are reviewed from the Departments of Neurology and Pediatrics, Columbia Presbyterian Medical Center, New York, NY. *Primary carnitine deficiency* is a decrease of intracellular carnitine that impairs fatty acid oxidation and is not associated with another systemic illness. It may be systemic or muscular, presenting as progressive cardiomyopathy, hypoketotic hypoglycemic encephalopathy, or myopathy. Age at onset is 1 month to 7 years, with a mean of 2 years. In encephalopathy, carnitine levels in plasma and tissues are below 10% of normal, and acylcarnitines are proportionately reduced. Acylcarnitine to free carnitine ratio is normal. Treratment is oral carnitine at daily doses of 100 to 200 mg/kg. Intermittent diarrhea and a fishy body odor are described as side effects of carnitine replacement. Muscle carnitine deficiency is characterized by severe reduction