Neurologic sequelae occurred in 33.8% of survivors: 54% had mental retardation, 50% epilepsy, 33% paralysis, 16% sensorineural hearing loss and 4% blindness. Other abnormalities included growth retardation, hyperactivity and irritability. Dexamethasone reduced the incidence of hearing loss.

Cranial CT conducted in 199 children with tuberculous meningitis in Cape Town, Republic of South Africa, showed basal ganglia infarcts in 30%, which corresponded with hemiplegia (Schoeman JF et al. Pediatr Neurol Sept/Oct 1992; 8:355 (abstract)).

**SEIZURE DISORDERS**

**BENIGN NEONATAL SLEEP MYOCLONUUS**

Benign neonatal sleep myoclonus was diagnosed in 10 infants at the Children's Hospital, Winnipeg, Manitoba. The neonates were referred for assessment of seizures. The myoclonus increased with gentle restraint and in 2 patients it worsened in spite of anticonvulsant therapy. None of the clinical events was associated with EEG seizure phenomena. All infants were neurodevelopmentally normal between the ages of 4 months and 5 years. The myoclonus remitted by age 6 months in all but 2 infants (Daoust-Roy J, Seshia SS. Benign neonatal sleep myoclonus. A differential diagnosis of neonatal seizures. AJDC Oct 1992; 146:1236-1241). (Reprints: Dr. Seshia, Section of Pediatric Neurosciences, Children's Hospital, AE208, 840 Sherbrook St., Winnipeg, Manitoba, Canada R3A 1S1.)

**COMMENT.** Benign neonatal sleep myoclonus has the following characteristics: 1) neonatal onset, 2) myoclonic jerks only during sleep, 3) abrupt and consistent cessation with arousal, 4) absence of EEG seizure activity and 5) good prognosis. This entity must be distinguished from benign familial neonatal seizures and anticonvulsants should be withheld. The condition should also be differentiated from neonatal hyperekplexia which is characterized by massive jerks in response to sudden tactile and acoustic stimuli and long lasting myoclonic jerks closely resembling epileptic seizures (Pascotto A, Coppola G. Neonatal hyperekplexia: a case report. Epilepsia Sept/Oct 1992; 33:817-820).

Benign myoclonic epilepsy of early childhood reported in 20 children from Riyadh, Saudi Arabia (Gascon GG et al. Pediatr Neurol Sept/Oct 1992; 8:348 (abstract)) was considered a phenotype differing only in the age of onset, but sharing the same genotype as benign infantile myoclonic epilepsy and juvenile myoclonic epilepsy.

Benign myoclonus of early infancy was reported in 6 cases from Milan, Italy (Nardoccin et al. Pediatr Neurol Sept/Oct 1992; 8:404
(abstract). The sudden onset of monoclonic jerks mainly involving the trunk and upper limbs, normal developmental milestones, absence of EEG abnormalities and self limited course were consistent with the diagnosis as described initially by Lombroso and Fejerman.

AMANTADINE IN ABSENCE EPILEPSY
Refractory absence epilepsy in 4 children was treated with amantadine as an add-on drug at the Tel Aviv University Sackler School of Medicine, Israel. The patients responded within 1 week and remained free of symptoms for 27-36 months without adverse effects. The dosage was 5-7 mg/kg per day given twice a day up to a maximum of 200 mg daily (Shahar EM, Brand N. Effect of add-on amantadine therapy for refractory absence epilepsy. J Pediatr of Neurol Nov 1992; 121:819-821). (Reprints: Eli M. Shahar, M.D., Child Neurology Unit, Chaim Sheba Medical Center, Tel Hashomer 52621, Israel.)

COMMENT. Amantadine was initially used by Shields et al. in the treatment of 10 children with minor motor seizures refractory to medications. Myoclonic seizures and atypical absence seizures responded well (Neurology 1985; 35:579).

VALPROATE AND EEG ABNORMALITIES
The effect of valproate on EEG epileptic abnormalities was investigated at the Institute of Neurophysiology, Genoa, Italy. Sixteen patients aged 14 to 44 years were administered a single (14-37 mg/kg) oral dose of magnesium valproate. At peak serum concentrations of 65-139 ug/ml of valproate, there were increases in spike wave frequency at 2, 3, 4 and 5 hours. The peak concentrations of ammonia ranged from 15-72 ug/l, with values exceeding normal in 13 determinations. The VPA and ammonia concentrations varied independently. The frequency of EEG epileptic abnormalities was not correlated with either the ammonemia level or the amount of variation in ammonemia concentration (Sannita WG. Valproate acute administration, EEG epileptic abnormalities, and ammonemia. Neurology Oct 1992; 42:2003-2005). (Reprints: Professor Walter G. Sannita, Centro Farmaci Neuroattivi, Dipartimento di Scienze Motorie, Universita, Ospedale San Martino, viale Benedetto XV, 1-16132 Genova, Italy.)

COMMENT. A direct CNS drug action has been proposed to explain the quantitative EEG abnormalities in response to acute VPA administration and the stupor states in some patients. The stupor was not necessarily related to the increased ammonia concentration in the blood.

BRAIN NEOPLASMS
BRAIN RADIOTHERAPY AND COGNITION
Cognitive function and school achievement were studied prospectively over 3 to 4 years in 19 children treated for brain tumors with whole-brain