was lower than that used in the above myopathy case report (720 mg/m^2 daily). Moderate elevations in CK levels are to be expected with zidovudine therapy, and muscle biopsy and EMG may be reserved for children with clinical myopathy or markedly elevated CK levels.

GUILLAIN-BARRE SYNDROME: AGE VARIATIONS

The medical records of 83 children with Guillain-Barre Syndrome were reviewed with reference to the age associated changes at the Department of Pediatrics, University of Tokyo, Japan. Children between 3 and 9 years were most susceptible and summer was the most common time of onset. Limb pain was the initial symptom for 53% of all patients and for 24% in the younger age group ($P < 0.05$). Muscle weakness was the most frequent initial symptom in the younger age group. Upper respiratory infections were the most common preceding illnesses; the interval between the previous illness and the onset of disease was significantly shorter for the older age group. The total duration of the illness was shorter in older children. Cranial nerve involvement occurred in 1/3 of children under 5 years of age and in the same proportion of children over 5 years. In the younger age group bulbar nerves were most commonly affected whereas the facial nerve was involved most frequently in the older age group. Patients with cranial nerve involvement suffered more frequently from respiratory insufficiency; the ages of those affected were distributed evenly between the two groups. The differences in initial symptoms and in interval between preceding illnesses and onset of the disease seemed to be related to the maturational processes of peripheral nerve and spinal root ganglia. It is speculated that the stage of the myelinogenic process might be important in the explanation of age associated alterations in symptomatology (Sakakihara Y, Kamoshita S. Age associated changes in the symptomatology of Guillain-Barre syndrome in children. Dev Med Child Neurol July 1991; 33:611-616).

COMMENT. The incidence of Guillain-Barre Syndrome increases with age but 25% of cases occur in childhood and adolescence. Since nerve conduction velocity and the thickness of the myelin sheath in peripheral nerves reaches adult levels at around five years of age, some variation in symptomatology between young and older children might be expected. Electrodiagnostic tests have been shown to corroborate the neuropathologic sequence in the Guillain-Barre Syndrome and these findings may be modified by age and maturation (Ropper AH et al. Arch Neurol 1990; 47:367; McFarland HR Arch Neurol July 1991; 48:678)

DEVELOPMENTAL DISORDERS

LISSENCEPHALY: CLINICAL AND MRI FINDINGS

Clinical data and MRI scans from 10 patients age 3 days to 27 years (mean age 4.6 years) with lissencephaly were reviewed in the Departments of Radiology, Neurology and Pediatrics, University of California, San Francisco,

CA. The youngest patients had the most severe lissencephaly but no correlation was found between the severity of the malformation as graded by MRI and the severity of clinical disease. One child age 2 1/2 with frontal lobes most severely affected was walking, speaking in 3 to 4 word sentences, and following instructions appropriate to her age, and physical examination revealed only very mild hypotonia. The anatomical deformity in the brain was not associated with any delay in myelination in patients in this study. Seizures occurred in all but one of the patients greater than four months of age; they began with infantile spasms or focal motor seizures and tonic seizures commonly occurred by the end of the second year. Hypsarrhythmia was present in two patients (Barkovich AJ et al. The spectrum of lissencephaly: report of ten patients analyzed by magnetic resonance imaging. Ann Neurol August 1991; 30:139-146).

COMMENT. The normal pattern and rate of myelination and the absence of porencephalies or other destructive lesions in 9 of the 10 patients in this report suggests that the arrest of neuronal migration in lissencephaly results from the absence of molecular components of the glial fibers or intracellular matrix. Lissencephaly is described in two types according to clinical, radiological, and pathological characteristics. In **type I lissencephaly**, agyria and pachygyria regions have a molecular layer, an outer cellular layer, a cell-sparse layer, and a deep cellular layer composed of heterotopic incompletely migrated neurons. In **type II lissencephaly**, the thickened cortex is disorganized without evidence of layers, and hydrocephalus and multiple anomalies are usually present.

GENERALIZED CORTICAL DYSPLASIA

The clinical and neuropathological findings in three children with diffuse cortical dysplasia are reported from the Departments of Pathology, Neurology and Pediatrics, University of Rochester Medical Center, Rochester, NY. The children had seizures unresponsive to antiepileptic medication throughout life, they were profoundly mentally retarded, and they died after a progressive neurological deterioration at ages 10 months, 3 years and 7 years. Pathologically, the brains showed generalized cortical dysplasia, without any major malformation of the external gyral pattern. The cortical gray matter was thickened and the demarcation of the gray-white matter junction was indistinct. Microscopically, the cortex contained increased numbers of large neurons with disordered cortical lamination and heterotopic neurons scattered throughout the white matter. The cortical involvement was multifocal involving all lobes of the cerebral hemispheres, and the underlying myelin was pale and rarified. (Kazee AM et al. Generalized cortical dysplasia. Clinical and pathologic aspects. Arch Neurol August 1991; 48:850-853.

COMMENT. The authors report these cases as a newly recognized entity of abnormal neuronal migration. In contrast to classic lissencephaly no distinct lamination of the cortical layers was present