Finland, and the Regional Pediatric Habilitation Center, Gothenburg, Sweden. Slit ventricles are caused by overdrainage of the cerebrospinal fluid and collapse of the ventricles following shunting of hydrocephalus. The incidence was 56% in this group of patients followed for a mean of 8.9 years. In patients who developed SLV the age at initial shunting was significantly lower (1.2 years) than for those who did not (2.7 years). Spike and sharp wave activity in the EEG developed more frequently in patients with SLV (81%) than in those without (54%). The severe generalized spike wave activity disappeared from the EEG after treatment of the slit ventricles. Epileptic seizures appeared after initial shunting in 44% of patients who developed SLV but in only 6% of the non-SLV group. Treatment of the SLV's reduced the frequency of epilepsy to the level corresponding with the non-SLV group. (Saukkonen A et al. Electroencephalographic findings and epilepsy in the slit ventricle syndrome of shunt treated hydrocephalic children. Child's Nerv Syst Dec 1988; 4:344-347).

COMMENT. This study demonstrates the value of repeated EEGs in shunt treated patients. If EEG abnormality appears after the initial shunting and especially severe spike wave activity, a shunt malfunction and overdrainage of the CSF should be suspected. The slit ventricle syndrome should be prevented or at least treated early to avoid permanent brain damage and long-term psychomotor retardation. Epileptic seizures have been reported in 10-40% of shunted hydrocephalic children. The position of the shunt, the frequency of the shunt revisions and epileptic seizures have been correlated in the present study. The ventricular size is also correlated with the frequency of epileptic seizures. Six patients suffering from West and Lennox syndromes associated with slit ventricle syndrome showed dramatic improvement and became asymptomatic after treatment for the slit ventricle syndrome. Anticonvulsant prophylactic therapy is warranted for at least a year after shunting and particularly in patients who develop slit ventricles. Raimondi AJ provides an editorial comment on shunts, indications, problems, and characteristics (Child's Nerv Syst Dec 1988; 4:321).

CNS MALFORMATIONS

CEREBELLAR HYPOPLASIA AND AUTISM

The size of the cerebellar hemisphere and vermal lobules was measured in ten autistic and eight normal control subjects at the Neuropsychology Research Laboratory, Children's Hospital Research Center, and the Departments of Neurosciences and Radiology, School of Medicine, University of California at San Diego, LaJolla. On sagittal MRI's the cerebellar hemispheres of the autistic subjects showed hypoplasia and a near total absence of the cerebellar tonsils in one. In contrast, a comparison of the average cerebellar width measured on axial images revealed no significant difference between the autistic group and the

normal control group. The mean area of the superior-posterior vermis in the autistic subject group was 20% smaller than in the normal control group, while there was no significant difference between the mean anterior vermis areas of the two groups. The results indicated that the decreased size of the cerebellar hemispheres and the vermal lobules VI through VII was associated with autism. (Murakami JW et al. Reduced cerebellar hemisphere size and its relationship to vermal hypoplasia in autism. Arch Neurol June 1989; 46:689-694).

COMMENT. The results of this study confirm those of a previous study by the same authors that showed that hypoplasia of the superior-posterior vermis (lobules VI and VII) is frequently observed in autistic individuals. The nature of the link between cerebellar dysgenesis and autistic symptoms has not been determined. The authors refer to clinical and research observations indicating that the cerebellum also plays a role in a variety of cognitive functions, such as language, learning and memory, emotional behavior, and complex motivated behaviors. They believe that the hypoplasia of cerebellar hemispheres and vermis observed in many autistic individuals is linked with behavioral and cognitive symptoms.

MUSCLE DISORDERS

SELENIUM AND MUSCULAR DYSTROPHY

Selenium metabolism and supplementation in patients with Duchenne muscular dystrophy was studied at the Muscle Research Center, Department of Medicine, University of Liverpool, and the Universitat Klinik Mainz, Mainz, FRG. Plasma selenium concentrations measured in seven Duchenne muscular dystrophy patients and in 11 age matched normal boys showed no significant difference after two months of sodium selenite supplementation (1 mg selenium daily). All patients demonstrated a rise in plasma selenium concentration as did all but one of the normal subjects. The studies did not confirm any abnormality of selenium metabolism in patients with muscular dystrophies, and there was no evidence that high dose selenium supplementation influenced the activity of the selenium dependent enzyme glutathione peroxidase in skeletal muscle. An elevation of thiobarbituric acid-reacting substances in the muscle of patients with Duchenne muscular dystrophy was unaffected by selenium supplementation (Jackson MJ et al. Selenium metabolism and supplementation in patients with muscular dystrophy. Neurology May 1989; 39:655-659).

COMMENT. The present finding of normal plasma selenium concentrations in Duchenne muscular dystrophy patients differs from reports from Finland where selenium in soils and indigenous food stuffs is naturally low in concentration. The increase in thiobarbituric acid-reacting substances in dystrophic muscle confirms previous reports but the elevated levels in patients with Duchenne muscular dystrophy contrasted with normal levels in