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

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 Universitut 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

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

-39-