hemorrhage. A mild tentorial laceration was considered likely in the cases reported. An inherited protein C deficiency may be manifested by massive venous thromboses in the newborn (Seligsohn U et al. N Engl J Med 1984;310:559), an etiology to be considered in the absence of a history of brain trauma.

TRANSPOSITION OF GREAT VESSELS AND MOBIUS SYNDROME

The vascular theory of embryopathogenesis for Mobius syndrome is proposed in a case report of a 3-month-old boy from the University of New Mexico School of Medicine, Albuquerque, NM. At birth, he had bilateral facial and abducens palsies, left acheiria (congenital absence of the hand), and transposition of the aorta and pulmonary artery. He was lethargic, cyanotic, and in respiratory distress, and he expired after an arterial switch procedure with closure of a septal defect. The authors cite 2 reports of Mobius syndrome with cardiac anomalies, both presenting with dextrocardia and the Poland anomaly (unilateral hypoplasia or absence of pectoral muscles, nipple, and upper limb). An intrapartum insult during the fourth to seventh week of gestation is consistent with the vascular theory of embryopathogenesis. (Raroque HG, Hershewe GL, Snyder RD. Mobius syndrome and transposition of the great vessels. Neurology Dec 1988;38:1894-5).

COMMENT. Congenital facial diplegia and abducens palsy, Mobius syndrome, has been explained as either a primary hypoplasia of cranial nerve nuclei or a primary deficiency of the muscles derived from the first two branchial arches. A dysgenesis of both neural and muscular tissue has been proposed in some cases. In the above case report, the concomitant occurrence of the vascular anomaly supports the theory of impaired cranial nerve nuclear development due to interruption of vascular supply at or around the sixth intrauterine week.

DEVELOPMENTAL DISORDERS AND LEARNING

VON RECKLINGHAUSEN NEUROFIBROMATOSIS

A population-based study in southeast Wales and reported from the Institute of Medical Genetics and Section of Neurology, University of Wales College of Medicine, Cardiff, has identified 135 patients with neurofibromatosis type 1 (NF-1), a prevalence of approximately 1/5000. The major clinical features were multiple cafe-au-lait spots, dermal neurofibromas, Lisch nodules in the iris (93%), freckling in the axilla (67%) or groin (44%), macrocephaly (45%), and short stature (34%). Complications included plexiform neurofibromas in 40 (30%) patients, mental retardation in 13 (10%) severe in only 1, epilepsy 6 (4%), severe scoliosis 6, visceral and endocrine tumors 6, optic glioma 2, spinal neurofibroma 2, aqueductal stenosis 2, delayed puberty 2, and congenital glaucoma 1. No cases of acoustic neuroma were seen. The frequency of CNS and malignant tumors was 5%. The authors recommend regular biannual examinations during childhood, with particular attention to intellectual

development and appropriate remedial education as necessary. In families of NF-1 patients, at-risk children who have not developed cafe-au-lait spots or Lisch nodules by 5 years of age are virtually certain to have escaped inheritance of the dominant gene. (Huson SM et al. Von Reckinghausen neurofibromatosis. A clinical and population study in southeast Wales. Brain Dec 1988;111:1355-1381).

COMMENT. This article represents a major study and addition to the literature on neurofibromatosis type 1. Items of interest to the pediatric neurologist include the association of macrocephaly, mental retardation, and epilepsy. Hypsarrythmia occurred more frequently than expected with NF-1 (2 cases) and this association has been noted previously (Riccardi VM, Eichner JE (1986) Neurofibromatosis: Phenotype, Natural History, and Pathogenesis. Baltimore, Johns Hopkins University Press). For correspondence, Dr. Huson's present address: Kennedy Galton Centre for Clinical Genetics, Northwick Park Hospital, Watford Road, Harrow, Middlesex HA1 3UJ, UK.

DRUGS IN TREATMENT OF DEVELOPMENTAL DYSLEXIA

An acute 2 day trial of methylphenidate (10 mg) and of meclizine (12.5 mg) and a 6 month crossover placebo-controlled chronic trial of meclizine (12.5 mg) in children with developmental dyslexia are reported from the University of Calgary, Alberta, Canada. Oral reading fluency, coordination, and motor accuracy improved on methylphenidate, and clinical improvements of eye fixation and tracking were found with meclizine, in acute trials. Three of 6 children showing benefit from acute doses of meclizine also showed significant improvements in eye fixation stability after 3 months chronic administration of the drug (12.5 mg BD), but measures of reading skills including comprehension, phonetic analysis, structural analysis, achievement, and oral and silent reading rates, were not benefitted. (Fagan JE et al. The failure of antimotion sickness medication to improve reading in developmental dyslexia: results of a randomized trial. J Dev Behav Pediatr Dec 1988;9:359-367).

COMMENT. Levinson has proposed a theory of cerebellar-vestibular dysfunction as the etiology of dyslexia and has claimed that treatment with antimotion sickness medications may result in improvement in reading in 77% of cases of dyslexia. The present study fails to confirm these results and suggests that antimotion sickness drug treatment of dyslexia is unjustified. The relationship between fixation error and impaired reading is not completely understood, however. Geiger and Lettvin have found that dyslexics have poor foveal vision, but when the target is moved into the peripheral visual field, their ability to identify letters is better than control nondyslexic subjects. Imperfect oculomotor control in the dyslexic may be accompanied by abnormalities of visual processing and cognitive difficulties that impede the acquisition of reading skills. Further work on this aspect of dyslexia seems justified.