prognostic value in assessment of neurological outcome. (See **Progress in Pediatric Neurology**, Millichap JG ed, 1991, page 333)

Cerebro spinal fluid examination in symptom-free infants with risk factors for infection was evaluated in 284 newborns at the Department of Pediatrics, Rush Presbyterian-St. Luke's Medical Center, Chicago, IL (Fielkow S et al. J Pediatr Dec 1991; 119:971-973). Positive cultures without pleocytosis in 5 infants (1.8%) were contaminants and none of the symptom-free infants had meningitis. The authors conclude that CSF examination is not indicated in the diagnostic evaluation of symptom-free infants born to mothers with chorioamnionitis or other risk factors for neonatal infection.

NEUROCUTANEOUS SYNDROMES

VISUAL LOSS IN TUBEROUS SCLEROSIS

Visual loss in 4 patients with tuberous sclerosis complicated by subependymal giant-cell astrocytoma is reported from the Departments of Ophthalmology, Neurology, and Radiology, University of Michigan Medical Center, Ann Arbor, MI. The patients presented at 12-20 years of age with obstructive hydrocephalus. Surgery relieved elevated pressure in all cases but 2 patients became blind and 1 has severe visual field loss from the effects of chronic papilledema on the optic nerves. Early surgical decompression prevented visual loss in the fourth patient whose tumor was removed at 12 years of age and who required further resection of a giant-cell astrocytoma at 20 years. (Dotan, SA et al. Visual loss in tuberous sclerosis. Neurology Dec 1991; 41:1915-1917.) (Reprints: Dr. J.D. Trobe, W.K. Kellogg Eye Center, 1000 Wall St., Ann Arbor, MI 48105.)

COMMENT. Periodic opthamologic examination and brain imaging are advisable in tuberous sclerosis patients with subependymal nodules. The timely relief of increased intracranial pressure may arrest or prevent loss of vision.

FAMILIAL SPINAL NEUROFIBROMATOSIS

The clinical features and genetic linkage analysis of two pedigrees with familial spinal neurofibromatosis (NF) are described from the Divisions of Neurology and Medical Genetics, Cedars-Sinai Medical Center, UCLA School of Medicine, Los Angeles, CA; the Neurofibromatosis Institute, Pasadena, CA; and Department of Medical Informatics, University of Utah, Salt Lake City, UT. On clinical grounds, it was difficult to assign the two families to NF1 or NF2 based on the criteria established by the National Institutes of Health. Cutaneous tumors, Lisch nodules or acoustic tumors were absent. Cafe-au-lait spots were present in 1 family and absent in the other. The inheritance pattern was autosomal dominant in both pedigrees. Genetic linkage analysis was performed with markers linked to the NF1 gene on chromosome 17 and markers linked to the NF2 gene on chromosome 22. The