widespread fibrillations, reduced motor and sensory nerve conduction velocities, conduction blocks, and increased F wave latencies, indicative of demyelination and also signs of axonal neuropathy. Following iv immunoglobulin and corticosteroids, dysarthria and dysphagia had resolved and ataxia was mild at 4-week follow-up. (Mewasingh LD, Sekhara T, Dachy B et al. Benign intracranial hypertension: atypical presentation of Miller Fisher syndrome? Pediatr Neurol March 2002:26:228-230). (Respond: Professor Bernard Dan MD, Department of Neurology, Hopital Universitaire des Enfants Reine Fabiola, 15 Avenue JJ Crocq, 1020 Brussels, Belgium).

COMMENT. The syndrome first described by Dr Miller Fisher, neurologist at the Massachusetts General Hospital, is "An unusual variant of acute idiopathic polyneuritis (syndrome of ophthalmoplegia, ataxia and areflexia)." (N Engl J Med 1956;255:57-65). The immune-mediated, acute demyelinating Guillain-Barre variant involves anti-GQ1b antiganglioside antibodies in 90% of cases, and is preceded by a respiratory or gastrointestinal infection in two thirds, *Campylobacter jejuni* being a common isolate in the cases that present with diarrhea. The cases in the present report are particularly rare since they occur in children, and the MF syndrome is preceded by symptoms of pseudotumor cerebri. The co-occurrence of papilledema and Guillain-Barre syndrome is reported in 4% of childhood cases (Peterman AF et al. Neurology 1959;9:533-539). The authors cite only one previous case of MF syndrome, in a 5-year-old male, presenting with pseudotumor (Macaya A et al. Pediatr Neurol 1988;4:120-121). CSF opening pressure and funduscopic examination should be included in the evaluation of patients with Guillain-Barre syndrome.

INFECTIOUS DISORDERS

RISK OF NEUROLOGIC SEQUELAE WITH BACTERIAL MENINGITIS

Predictors of permanent neurologic sequelae or death following bacterial meningitis were determined in a case-controlled study at Sophia Children's Hospital, Rotterdam, The Netherlands. The study population of 93 children presenting with meningeal signs included 23 cases with neurologic sequelae and 70 controls without sequelae (52% boys, median age 2.8 yrs). Of the 23 cases, 2 died (8.7%), 6 were deaf (26.1%), 8 had mild hearing loss (34.7%), 4 were severely retarded (17.4%) and 3 had epilepsy, mild locomotor deficits or mild mental retardation (13%). Significant independent predictors for an adverse outcome were male gender, the occurrence of atypical convulsions before admission (duration >15 min, non-generalized jerks, incomplete recovery, multiple convulsions within 24 hr), low body temperature at admission, and especially, the pathogen type *Streptococcus pneumoniae.* The area under the ROC curve (estimated predictive performance) of this prediction rule was 0.87 (95% confidence interval: 0.78-0.96). A risk score computed for each patient by assigning points for each predictor present was used to classify patients into adverse outcome categories. (Oostenbrink R, Moons KGM, Derksen-Lubsen G, Grobbee DE, Moll HA. Early prediction of neurological sequelae or death after bacterial meningitis. Acta Paediatr 2002;91:391-398). (Respond: Dr R Oostenbrink, Sophia Children's Hospital, Room Sp 1545, Dr Molewaterplein 60, NL-3015 GJ Rotterdam, The Netherlands).

COMMENT. Permanent neurological sequelae or death after bacterial meningitis in childhood may be predicted from the early clinical characteristics.

The pathogen type, *Streptococcus pneumoniae* is the strongest predictor of poor outcome. Additional independent predictors of adverse outcome are male gender, history of atypical convulsions, and low body temperature. Clinical characteristics and laboratory tests during the recovery period are not of value as risk predictors.

SEIZURE DISORDERS

HIGH-DOSE PHENOBARBITAL FOR OHTAHARA SYNDROME

Oral high-dose phenobarbital therapy was effective in the control of tonic spasms in a 1 month-old-infant with early infantile epileptic encephalopathy with suppression bursts (Ohtahara syndrome) treated at Tokyo Metropolitan Hachioji Children's Hospital, Tokyo, Japan. Birth history, CT and MRI and other laboratory tests were normal. Initial treatment with intravenous midazolam (0.5 mg/kg/hour) slightly decreased seizures, but oral vitamin B6, valproic acid, clonazepam, and zonisamide had no effect. On the 38th hospital day, oral phenobarbital beginning at a dose of 15 mg/kg/day decreased seizures from 300 daily to 5-10 times per day, and epileptic discharges on the EEG were markedly decreased. Serum phenobarbital levels ranged between 60 and 100 mg/dL. Sedation and decreased oral feedings but no hypotension were noted. Oxygen was occasionally required because of pneumonia. IV midazolam was gradually withdrawn and discontinued on the 58th hospital day. At 2 years of age, he smiles and takes liquids, but cannot sit or speak. (Ozawa H, Kawada Y, Noma S, Sugai K. Oral high-dose phenobarbital therapy for early infantile epileptic encephalopathy. Pediatr Neurol March 2002;26:222-224). (Respond: Dr Ozawa, Department of Pediatrics, Tokyo Metropolitan Hachioji Children's Hospital, 4-33-13, Daimachi, Hachioji, Tokyo 193-0931, Japan).

COMMENT. High-dose oral phenobarbital may control seizures and epileptiform EEG discharges in Ohtahara's syndrome but the effect on developmental outcome will require further evaluation. Very-high-dose phenobarbital has been used successfully in the treatment of refractory status epilepticus (Crawford TO et al. Neurology 1988;38;1035-1040), neonatal seizures, and in infants with severe perinatal asphyxia (Hall RT et al. J Pediatr 1998;132;345-348). A neuroprotective effect may be induced by reduction of cerebral metabolism and oxygen consumption. Depression of respiratory drive, cardiac suppression and hypotension are serious unfavorable adverse effects.

Effectiveness of phenobarbital in neonatal seizures has been evaluated, using video-EEG telemetry, in 14 babies treated at King's College Hospital;, Denmark Hill, London, UK (Boylan GB, Rennie JM, Pressler RM et al. Arch Dis Cild Fetal Neonatal Ed May 2002;86:F165-F170). Four neonates with normal or moderately abnormal EEG background abnormalities responded to phenobarbital (20-40 mg/kg intravenously over 20 min) and the outcome was good. In 10 with abnormal EEG background activity, electrographic seizures increased after treatment with phenobarbital, whereas electroclinical seizures were reduced. Of 3 treated with second line anticonvulsants, 2 responded. Phenobarbital was ineffective in babies with severe seizures and severely abnormal EEG background activity, even after a second dose up to a maximum of 40 mg/kg. Infants who fail to respond to phenobarbital within 2 hours should receive a second line drug.