respiratory distress of unclear cause. Consanguineous parents of a child with SIDS should be checked for IGHMBP2 mutations.

NCS/EMG and muscle biopsy together are of diagnostic value in infants with arthrogryposis multiplex congenital (AMC), when the history, examination, and genetic evaluation are unrevealing. (Kang PB et al. Ann Neurol 2003;54:790-795). Analysis of 38 patients with AMC seen over a 23-year period at Children's Hospital, Boston, showed that the disorder was neurogenic in 8, myopathic in 10, other in 12, and unknown in 8. Neither EMG nor biopsy alone had consistently high sensitivities or specificities, but when concordant for neurogenic or myopathic findings, they were more accurate than either test alone.

INFECTIOUS DISORDERS

HUMAN HERPESVIRUS-6 IN MESIAL TEMPORAL LOBE EPILEPSY

Brain samples obtained from surgical resections in 8 patients with mesial temporal lobe epilepsy (MTLE) and 7 patients with neocortical epilepsy (NE) were quantitatively analyzed for HHV-6 in a study at the National Institute of Neurological Disorders and Stroke, Bethesda, MD. DNA obtained from 4 patients with MTLE had significantly elevated levels of HHV-6 determined by real-time PCR assay, but HHV-6 was not amplified in specimens from NE patients. Hippocampal sections from MTLE cases had the highest levels of HHV-6, subtype HHV-6B. Expression of HHV-6 was localized to astrocytes and confirmed by western blot analysis and immunohistochemistry. (Donati D, Akhyani N, Fogdell-Hahn A, et al. Detection of human herpesvirus-6 in mesial temporal lobe epilepsy surgical brain resections. Neurology November (2 of 2) 2003;61:1405-1411). (Reprints: Dr S Jacobson, Neuroimmunology Branch, National Institute of Neurological Disorders and Stroke, NIH, Bethesda, MD 20892).

COMMENT. The detection of HHV-6 in hippocampal and temporal lobe astrocytes of patients with MTLE suggests a possible role for reactivation of the virus in infected astrocytes as a cause of MTLE. The specificity of the finding requires additional studies in patients without seizures. HHV-6 and roseola infantum are also associated with febrile convulsions, a proposed antecedent to mesial temporal sclerosis and MTLE. (see Progress in Pediatric Neurology III, PNB Publishers, 1997;pp24-28).

RISK OF HEARING LOSS AFTER BACTERIAL MENINGITIS

Presence of sensorineural hearing loss (>25 dB) was determined in 628 school-aged children born between 1986 and 1994 and survived non-*Hemophilus influenzae* type B (HiB) bacterial meningitis between 1990 and 1995 in a study at VU Medical Center, Amsterdam; and University Medical Center and Wilhelmina Children's Hospital, Utrecht, the Netherlands. The incidence of hearing loss was 7%; unilateral in 20 (3%) and bilateral in 23 (4%). Hearing loss was severe (71-90 dB) or profound (>90 dB), and 5 received

cochlear implants. Three quarters of the cases were detected at routine follow-up after meningitis. Risk factors for hearing loss were as follows: symptoms of meningitis for >2 days before admission, absence of petechiae, CSF glucose <0.6 mmol/L, *Streptococcus pneumoniae*, and ataxia. Based on this prediction rule of 5 factors, 62% of postmeningitic children were considered at risk. All cases of hearing loss were in this at-risk group. (Koomen I, Grobbee DE, Roord JJ, et al. Hearing loss at school age in survivors of bacterial meningitis: Assessment, incidence, and prediction. Pediatrics November 2003;112:1049-1053). (Reprints: AM van Furth MD, PhD, VU Medical Center, Department of Pediatrics, Box 7057, 1007 MB Amsterdam, the Netherlands).

COMMENT. The inclusion of hearing evaluation in the routine follow-up of children after bacterial meningitis should prevent the missed diagnoses of hearing loss. The occurrence of prediction factors should mandate repeated hearing tests to rule out fluctuating or late onset hearing loss. Vaccination against *S pneumoniae* in children aged 2 years or older with certain risk factors (AAP Red Book 2000) may prevent some cases and may alter the prediction rule established in this study.

The omission of some important references from the current report is unfortunate. A seminal study and prospective evaluation of hearing impairment as a sequel of acute bacterial meningitis was reported by Dodge PR and colleagues (N Engl J Med 1984;311:869).. Post-meningitic sensorineural hearing loss pre-1980 occurred in up to 30% of patients with pneumococcal meningitis, 10% of meningococcal, and 5-20% of H Influenzae type B meningitis. Regardless of the bacteria, type of antibiotic therapy, or use of dextromethasone, audiological assessment was recommended in all meningitis patients before or soon after discharge from hospital.

Another report emphasized psychological and educational adverse outcomes as well as deafness in a group of 130 children examined at a mean age of 8 years, and 6 years after meningitis (Grimwood K, et al. Pediatrics 1995;95:646-656; see Ped Neur Briefs June 1995). In this Australian study, even with optimal therapy, one in 4 children who recovered from meningitis had severe or functionally significant disabilities that affected school performance.

ABSCESS OF CAVUM SEPTUM PELLUCIDUM COMPLICATING PNEUMOCOCCAL MENINGITIS

A 7-year-old boy with pneumococcal meningitis complicated by abscesses involving the cavum septum pellucidum (CSP) and cavum vergae (CV) is reported from the Children's Hospital and University of California San Diego, CA. He was admitted with fever, headache, vomiting, and nuchal rigidity. Symptoms of rhinorrhea and cough had developed 9 days before admission. CT scan demonstrated sinusitis, left frontal subdural empyema, and abscesses of the CSP and CV. *Streptococcus pneumoniae* was isolated from the CSF and was susceptible to penicillin and ceftriaxone. Antibiotic and dexamethasone therapy resulted in resolution of fever, headache, and nuchal rigidity. Relapse occurred 3 days after discharge, and CT showed an interval increase in size of CSP and CV. Vancomycin was added to the ceftriaxone and the CSF culture was sterile. A repeat CT 5 days later showed further increase in size of CSP and CV, and hydrocephalus. CT-guided drainage of the CSP released 10 ml of purulent material with negative culture. Following 6