Neurol April 2006;48:294-300). (Respond: Richard W Newton MD FRCPCH, Department of Paediatric Neurology, Royal Manchester Children’s Hospital, Pendlebury, Manchester M27 4HA, UK).

COMMENT. In the majority of young children with acute encephalopathy in this study, the earliest laboratory sign of CNS involvement was an abnormal CSF:serum albumin ratio and disruption of the blood-CSF barrier. This leads to CSF viral invasion, increased production of intrathecal antibodies by activated lymphocytes, and high levels of cytokines such as IFN-a, and CNS autoimmunity. As the authors suggest, early antiviral therapy could result in repair of the blood-brain barrier and attenuation of the immune response. The value of the EEG in predicting outcome of encephalopathy is noteworthy. The EEG has been helpful in assessment of prognosis of complex febrile seizures (FS), sometimes difficult to distinguish from encephalopathy (Millichap JG et al. Neurology 1960;10:643-653). The CSF:serum albumin ratio found abnormal in encephalopathy may prove helpful in the diagnosis and distinction from prolonged, focal or multiple FS. Seizures occurred at onset in the majority of patients in the above study, but the seizure duration and degree of fever are not recorded. Six patients without CSF pleocytosis and 10 who recovered without sequelae might in some circumstances have been classed as complex FS.

CSF CULTURES AND BACTEREMIA IN NEONATAL MENINGITIS

Cerebrospinal (CSF) culture results were compared with results of blood cultures and CSF parameters (WBC, glucose, and protein) in 9111 neonates with culture-proven meningitis and a first lumbar puncture at >34 weeks’ gestational age from 150 NICU’s managed by the Pediatrix Medical Group. The concordance of these values was analyzed by researchers at Duke Clinical Research Institute, Durham, NC. Of 92 (1.0%) neonates with meningitis confirmed by CSF culture, only 62% had a concomitant positive blood culture within 3 days of LP; in 38% the blood culture was negative. In 57 with both positive blood and CSF cultures, the organisms were discordant in 2 (3.5%), the CSF pathogens requiring different antimicrobial therapy than the blood pathogen. In neonates with bacterial meningitis, CSF WBCs ranged from 0-15,900/mm3; 5% had 0-1 and 10% had <3 WBCs. Highest sensitivity (97%) and lowest specificity (11%) for prediction of meningitis was any WBCs in the CSF. When 21 WBCs were used as the upper limit of threshold, the sensitivity and specificity were 79% and 81%, respectively. CSF glucose and protein were variable and not of diagnostic value in the absence of a CSF culture. (Garges HP, Moody MA, Cotton CM, et al. Neonatal meningitis: What is the correlation among cerebrospinal fluid cultures, blood cultures, and cerebrospinal parameters? Pediatrics April 2006;117:1094-1100). (Respond: Daniel K Benjamin Jr MD PhD MPH, Department of Pediatrics, PO Box 17969, Duke Clinical Research Institute, Durham, NC 27715).

COMMENT. A suspected diagnosis of neonatal meningitis should not be dismissed by a negative blood culture or normal CSF cells, glucose or protein. The diagnosis must be established by a timely LP and positive CSF culture. The authors recommend that an LP should be included in the evaluation of sepsis in an infant. For patients pretreated with antibiotics who are asymptomatic and have negative blood and CSF cultures, and elevated
CLINICAL DIAGNOSIS OF SYDENHAM’S CHOREA IN AREA ENDEMIC FOR ACUTE RHEUMATIC FEVER

A retrospective chart review to determine the causes of childhood chorea was conducted in the Division of Allergy, Immunology and Infectious Disease at the Children’s Hospital of Pittsburgh, PA. Of 144 patients with a diagnosis of chorea between 1980 and 2004, 82 had new-onset chorea; 79 (96%) were Sydenham’s chorea (SC), 1 a postoperative cerebral ischemia, and 2 had basal ganglia infarcts. SC patients were female in 71%, the mean age at presentation was 9.8 years (range 5-14 yrs), chorea was unilateral in 30%, and 30% had a family history of acute rheumatic fever (ARF). Symptoms included dysarthria (67%), abnormal gait (51%), behavior change (46%), dysgraphia (29%), and headache (11%). Carditis was present in 44%, arthritis (11%), and erythema marginatum (3%). An elevated antistreptolysin O titer was documented in 99% of 53 tested, anti-deoxyribonuclease B titer in 7, positive streptozyme in 53, and acute throat infection with *Streptococcus pyogenes* in 19 tested. In patients with ARF and SC, brain MRI was abnormal in 8 of 32, and CT in 1 of 20 patients tested; abnormalities were not considered diagnostic of chorea and included petrous bone anomalies, medulloblastoma and macrocephaly, punctate lesions in parietal and frontal lobes, increased signal in right globus pallidus, and Arnold Chiari I. The 3 patients with new-onset chorea without ARF had MRI evidence of basal ganglia ischemic lesions; histories were considered atypical for SC. Neuroimaging should be reserved for patients with atypical presentation, including hemichorea. (Zomorrodi A, Wald ER. Sydenham’s chorea in Western Pennsylvania. *Pediatrics* April 2006;117:675-679). (Respond: Ellen R Wald MD, Division of Allergy, Immunology and Infectious Diseases, Children’s Hospital of Pittsburgh, 3705 Fifth Ave, Pittsburgh, PA 15213).

COMMENT. Although the majority of cases of new-onset chorea in childhood are related to streptococcal infection and are rheumatic in origin, the diagnosis based only on clinical findings is not invariably correct. Typically, Sydenham’s chorea (SC) has an acute or gradual onset, and is a self-limiting disease. In patients with a protracted course, pathology other than ARF should be considered, and a complete neurologic evaluation is essential, including an MRI. A 6-year-old boy who developed hemichorea after scarlet fever and had been treated for a year as SC was subsequently referred and diagnosed with a Grade 3 astrocytoma of the right thalamus (Millichap et al. *JAMA* 1962;179:589-593). Of 300 childhood intracranial tumors treated at the Mayo Clinic 1950-1960, 4% involved the basal ganglia and less than 1% presented with involuntary movements. The differential diagnosis also includes tics, chorea secondary to perinatal anoxia or infarction, drugs, metabolic disorders, paroxysmal choreoathetosis, familial benign choreoathetosis, and Huntington’s chorea. Methylenidate-induced chorea is reported during convalescence (Nausieda PA et al. *Neurology* 1983;33:750).

**EEG abnormalities in SC** are reported in 55%-87% cases (Johnson DA et al. *Arch Neurol* 1964;10:21-27). Although not specific for SC, paroxysmal bursts of 3-5 Hz/sec and 2-3/sec slow waves in the posterior regions following eye closure are characteristic and may be helpful in diagnosis.