junction of gray and white matter, whereas those arising from contiguous sources are usually superficial and close to the infected bone or dura. During the initial cerebritis (septic encephalitis) stage, the clinical picture is nonspecific. A patient with heart disease develops headache, vomiting, seizures, and fever. As the abscess forms, the neurologic signs become more apparent and lateralizing, with hemiparesis, hemianopia, papilledema, and localized percussion tenderness of the skull. (Raimondi et al. 1965). The EEG shows focal slowing, and CT confirms the diagnosis. In the differential diagnosis, thromboses of arteries, veins and dural sinuses are common in cyanotic infants, and symptoms may mimic an abscess, except the onset is more abrupt. Thromboses are rare in infants older than 2 years. Hypoxic attacks occur in 12% to 15% of patients with cyanotic heart disease and are common during the first 2 years of life. Meningitis may also mimic an abscess before symptoms and signs become lateralized. (Menkes JH, 1980). The diagnosis of brain abscess should be considered with new-onset headache and seizure, especially in a child with congenital heart disease or recent sinus or ear infection, commonly *Streptococcus milleri (S intermedius)*, and in an acutely immunosuppressed patient with fungal disease.

MRI/MRS STUDY OF ADEM

Magnetic resonance imaging and H magnetic resonance spectroscopy were used to detect possible structural and neurochemical abnormalities in two children, ages 6-months and 4.5 years, with acute disseminated encephalomyelitis at the State University of New York, Stony Brook, New York. The infant presented with focal seizures and thalamic and cerebral white matter lesions, and the older child with tremor and dystonia with bilateral basal ganglia lesions. Both recovered without the use of steroids or IV immunoglobulin. H MRS of involved areas showed abnormalities in N-acetyl-aspartate, choline, and lactate peaks during the symptomatic phase, and a low N-acetyl-aspartate persisted during recovery. The MRS findings are consistent with neuronal dysfunction, cellular membrane turnover, cellular infiltation, and metabolic stress in the acute phase, and with neuronal loss in the chronic phase. (Gabis LV, Panasci DJ, Andriola MR, Huang W. **Pediatr Neurol** 2004;30:324-329). (Respond: Dr Lidia V Gabis, Pediatric Neurology, Safra Children's Hospital, Sheba Medical Center, Tel Hashomer, Israel 52621).

COMMENT. Metabolic studies using magnetic resonance spectroscopy should aid in diagnosis of ADEM and may prove of value in following the course of the disease and the need for therapy.

MRS METABOLITES IN RASMUSSEN ENCEPHALITIS

The evolution of metabolite changes in an 8-year-old boy with focal Rasmussen encephalitis was studied by MRI and MRS at the Brain Research Institute, University of Melbourne, Australia. Serial structural and metabolite changes during a 9-month period which included an episode of complex partial status epilepticus showed focal swelling and a marked increase in T2-weighted signal intensity in the superior temporal gyrus following status. Follow-up scans showed resolution of the swelling and the development of slight focal atrophy. MRS showed a reduction in N-acetylaspartate, total creatine and trimethylamines after status. These metabolite changes had resolved in subsequent MRS