were characterized by facial grimacing, head twitching, and shoulder shrugging plus eye rolling, facial contorsions, jumping, touching objects and body parts and making obscene gestures. Vocal tics included grunting, sniffing, and snorting sounds. The tics were suppressed after several weeks on Pimozide (Orap 1 mg b.i.d.). (Northam RS, Singer HS. Postencephalitic acquired Tourette-like syndrome in a child. Neurology April 1991; 41:592-593).

COMMENT. The authors refer to reports of acquired Tourette syndrome in adults. Tics followed withdrawal from neuroleptic medications or in association with herpes and other encephalitides, toxic and metabolic encephalopathies, strokes, cerebral tumors, trauma, multiple sclerosis, syphilis, Huntington's, Alzheimer's, and Creutzfeldt-Jakob diseases.

SPINAL CORD SCHISTOSOMIASIS

A seven year old girl with Schistosoma mansoni infection of the spinal cord mimicking cord neoplasm is reported from the Department of Neurology, University of Michigan, Ann Arbor, MI. Exposure to Schistosomal parasites occurred while bathing in a river in Sierra Leone, Southern Africa. The illness presented with back and leg pain and a low-grade fever. Within 24 hours she had weakness of the leg and four days later she was unable to walk, to urinate or defecate. Sensation was diminished below the low thoracic area. CSF showed 2 RBCs, 250 WBCs with 78% lymphocytes, protein 584 mg/dl and glucose 55 mg/dl. Myelography revealed a large intramedullary mass at thoracic levels 11 and 12. MRI with Gadolinium demonstrated enhancement of the cord at T10 level through the conus. Decompressive laminectomy was performed with resection of a yellow gliotic appearing cord of neoplastic appearance. Histologic examination revealed eggs characteristic of S. mansoni. After treatment with praziquantel, lower extremity strength gradually improved and six months later, she was walking well with braces. (Selwa LM, Garofalo EA et al. Spinal cord schistosomiasis: A pediatric case mimicking intrinsic cord neoplasm. Neurology May 1991; 41:755-757).

COMMENT. In patients with acute or subacute paraparesis who have been exposed to endemic areas (South Africa, South America, Caribbean or the Middle East), schistosomiasis must be suspected. Spinal cord involvement can be delayed for up to six years after exposure, and findings on MRI and myelography may mimic spinal cord neoplasms. Patients with mild or early involvement of the spinal cord may be benefitted by antiparasitic medications alone and surgery may be unnecessary.

CNS COMPLICATIONS OF RHEUMATOID ARTHRITIS

Mass lesions in the basal ganglia bilaterally and mainly involving the globus pallidus are reported in a two year old boy with juvenile rheumatoid arthritis from the Department of Pediatrics, Hokushin General Hospital, Nishi, Nagano, Japan. At ten months after the onset of the arthritis he developed a relapse with fever, heart murmur,

vomiting, hypotonia, dystonia, and aphasia. Deep tendon reflexes were exaggerated and plantar responses extensor. The abnormal neurologic signs and basal ganglia lesions gradually improved with corticosteroid treatment and almost disappeared after one year. The right optic fundus showed chorioretinitis. A stereotactic brain biopsy performed to exclude a neoplasm revealed proliferation of astrocytes of undetermined origin, either reactive or low-grade astrocytoma. The dystonia subsided after one year but peculiar mouthing behavior persisted. A CNS primary lymphoma could not be excluded. (Hirabayashi S et al. Basal ganglia mass lesions in juvenile rheumatoid arthritis. Pediatr Neurol March/April 1991; 7:141-3).

COMMENT. A chronic inflammatory process involving cerebral vessels was suspected in this patient but angiography failed to demonstrate a cerebral vasculitis.

Other rheumatic diseases with CNS complications include lupus erythematosus, polyarteritis nodosa and rheumatic fever. Seizures are a common presenting sign of lupus erythematosus.

SEIZURE DISORDERS

BENIGN FAMILIAL NEONATAL CONVULSIONS

Linkage studies with the chromosome 20 markers D20S19 and D20S20 were performed in two families with benign familial neonatal convulsions at the Department of Pediatrics, The University of Texas Health Science Center, San Antonio, TX. In the first family with 14 affected, none had seizures after two months of age. In the second family with 13 affected, seizures did not remit until 6 to 24 months; febrile convulsions occurred in two, and one had refractory epilepsy until late adolescence. In family one, the odds were greater than 20,000:1 against linkage at 10% recombination; whereas the data from family two favored linkage with a maximum odds ratio of 45:1 at 6% recombination. It was concluded that this autosomal dominant primary epilepsy of infancy is clinically and genetically heterogeneous. (Ryan SG et al. Benign familial neonatal convulsions: Evidence for clinical and genetic heterogeneity. Ann Neurol May 1991; 29:469-473).

COMMENT. These data based on large family pedigrees suggest two distinct genetic loci for benign familial neonatal convulsions. The subtype linked to chromosome 20q may be associated with delayed remission and a higher risk for the development of epilepsy. The authors suggest that absence and benign rolandic epilepsy might also show genetic heterogeneity.

EPIDEMIOLOGY OF ABSENCE EPILEPSY

A population based electroencephalographic study of absence epilepsy in 97 children is reported from the Departments of Neurophysiology and Pediatrics, Goteborg University, Sweden. All patients had regular bilaterally synchronous and symmetrical 2-4 Hz spike-and-slow wave discharges and absences with or without generalized tonic-clonic