patients with hydrocephalus. Sequelae from the cysts included optic atrophy, stroke, or diplopia in 4 patients. Medical treatment alone can be effective in the treatment of NC with giant subarachnoid cysts. Surgical removal of cysts is reserved for patients with life-threatening intracranial hypertension despite treatment with corticosteroids. (Proano JV, Madrazo I, Avelar F et al. Medical treatment for neurocysticercosis characterized by giant subarachnoid cysts. N Engl J Med September 20, 2001;345:879-885). (Reprints: Dr Proano, Santiago Valverde No 68, Col Presidentes Ejidales, CP 04470, Mexico DF, Mexico).

COMMENT. Patients with neurocysticercosis with giant cysts who respond to corticosteroids or shunting should receive cesticidal drug treatment. The majority will respond to one or more courses of albendazole or praziquantal, and surgical removal of cysts may be necessary in a minority with persistent intracranial hypertension.

Human cysticercosis caused by the larvae of *T solium* is a leading cause of epilepsy in underdeveloped countries and is uncommon in the United States. Diagnosis of taeniasis is by demonstration of ova in feces or perianal swab. Diagnosis of neurocysticercosis is by head CT or MRI. An enzyme immunotransfer blot assay is available to detect antibody to *T solium* in serum or CSF. Treatment is individualized based on the viability of cysts. For patients with calcified nonviable cysts, symptomatic therapy with anticonvulsants and shunting for hydrocephalus are advised. For those with viable parenchymal cysts and inflammation, the role of antiparasitic drugs has not been firmly established. The use of antiparasitic drugs is favored by many clinicians, and the coadministration of corticosteroids for the first few days is recommended to decrease edema and adverse events. (American Academy of Pediatrics Red Book, 25th ed, 2000;pp560-562).

NEUROCUTANEOUS SYNDROMES

AUTISM AND EPILEPSY IN TUBEROUS SCLEROSIS COMPLEX

The relationship between autism and epilepsy and functional brain abnormalities in children with tuberous sclerosis complex (TSC) were examined at the Children's Hospital of Michigan, Wayne State University, Detroit. Based on tests for autism, behavior and intelligence, patients were divided into three groups: autistic (9 cases); mentally-retarded nonautistic (9); and normal intelligence (8). PET studies in the autistic compared to the retarded nonautistic groups showed decreased glucose metabolism in the lateral temporal gyri bilaterally, increased glucose metabolism in the deep cerebellar nuclei bilaterally, and increased a-methyl-tryptophan (AMT) uptake in caudate nuclei. Disturbed communication skills were associated with a history of infantile spasms and glucose hypometabolism in the lateral temporal gyri. Stereotypical behavior and impaired social and communication interaction were correlated with glucose hypermetabolism in deep cerebellar nuclei and increased AMT uptake in caudate nuclei. (Asano E, Chugani DC, Muzik O et al. Autism in tuberous sclerosis complex is related to both cortical and subcortical dysfunction. Neurology October (1 of 2);57:1269-1277). (Reprints: Dr Diane C Chugani, PET Center, Children's Hospital of Michigan, 3901 Beaubien Blvd, Detroit, MI 48201).

COMMENT. In children with TSC, infantile spasms and functional deficits in the temporal cortex are associated with delayed communication skills, and functional imbalance in subcortical circuits are correlated with stereotypical behavior and impaired social interaction.