majority of brain atrophy will have taken place, and the measurement of improvement following immunosuppressive treatment will not be possible. Estimation of hemispheric volume loss may be used as a parameter of the destructive disease process during the acute phase. An autoimmune response mediated by cytotoxic T lymphocytes is proposed as a key mechanism in the etiology of Rassmussen's encephalitis (Bauer J, Bien CG, Lassmann H. see Ped Neur Briefs April 2002;16:27-28).

ANTI-BASAL GANGLIA ANTIBODIES IN SYDENHAM'S CHOREA

The sensitivity and specificity of methods to detect anti-basal ganglia antibodies (ABGA) in Sydenham's chorea were determined in a study at the Institute of Neurology, Queen Square; Great Ormond Street Hospital for Children; Royal Free and University College Hospital Medical School, London, UK; and Federal University of Minas Gerais, Brazil. Samples from 20 patients with acute SC, 16 with persistent SC, control samples from 16 with rheumatic fever (RF), and 11 healthy pediatric volunteers were tested with ELISA and Western immunoblotting (WB) methods to detect ABGA and compared these assays to immunofluorescent (IF) methods. In acute SC, a sensitivity of 95% and specificity of 93% were obtained with ABGA ELISA , while WB and IF had a sensitivity of 100% and specificity of 93%. In persistent SC, ABGA sensitivity was 69% using WB and 63% with IF. WB identified 3 common basal ganglia antigens (40 kDa, 45 kDa, and 60 kDa) in both acute and persistent SC cases. Antibody reactivity to cerebellum, cerebral cortex, or myelin antigen was absent in all groups. WB and IF are recommended for detecting ABGA in acute and persistent cases of SC. The results support an autoantibody-mediated mechanism in SC. (Church AJ, Cardoso F, Dale RC et al. Anti-basal ganglia antibodies in acute and persistent Sydenham's chorea. Neurology July (2 of 2) 2002;59:227-231). (Reprints: Mr Andrew Church, Neuroimaging Unit, Room 917, Institute of Neurology, Queen Square, London WC1N 3BG, UK).

COMMENT. Anti-basal ganglia antibodies are reported in Sydenham's chorea and pediatric autoimmune neuropsychiatric disorder associated with streptococcus (PANDAS). Pathological studies in SC show basal ganglia involvement. An antibody-mediated mechanism is suggested by the latency of the basal ganglia syndromes that may develop after streptococcal infection. WB and indirect IF are the best methods for detecting ABGA, whereas ELISA may be used to monitor ABGA levels over time and to assess treatment response.

DEVELOPMENTAL DISORDERS

SIMPLIFIED CLASSIFICATION OF FOCAL CORTICAL DYSPLASIA

Sections of cortex from 52 of 224 (23%) patients with cortical dysplasia, operated on for drug-resistant partial epilepsy, were retrospectively re-examined histologically at Niguarda Hospital, and Istituto Nazionale Neurologico 'C. Besta', Milan, Italy. Three subgroups were identified as follows: 1) architectural dysplasia (31 patients) with abnormal cortical lamination and ectopic neurons in white matter; 2) cytoarchitectural dysplasia (6 patients) with altered cortical lamination and giant neurofilament-enriched neurons; and 3) Taylor-type cortical dysplasia (15 patients) with cortical laminar disruption and giant dysmorphic neurons and balloon cells. Group 1 architectural dysplasia cases had a temporal lobe location for the epileptogenic zone, with focal hypoplasia, and a significantly lower seizure frequency than groups 2 and 3. Group 3 Taylor-type dysplasia patients had an extratemporal epileptogenic zone, interictal distinctive stereo-EEG with high