obsessive compulsive disorder. PANDAS, or pediatric autoimmune neuropsychiatric disorders associated with streptococcal infection, are reviewed by Kurlan R (Neurology June 1998;50:1530-1534), and Ped Neur Briefs July 1998;12;49-50.

Prednisone therapy and chorea duration. In a study of 600 cases of rheumatic fever seen between 1971 and 1999 and evaluated at the Primary Children's Medical Center, Salt Lake City, UT, 142 (24%) had rheumatic chorea, and 69 (49%) received treatment, 57 with prednisone. Median time to 100% recovery was 2.75 weeks (range, 3-24 weeks) for prednisone-treated patients vs 10 weeks (range, 3-24 weeks) for untreated children ($p<0.01$). Hemichorea occurred in 39% of patients, a higher incidence than in the Brazil study. (Thompson JA, Tani LY, Bale Jr JF. Sydenham's chorea: the Utah experience. Ann Neurol Sept 1999;46:523, abstract).

HEADACHE DISORDERS

DURATION OF MIGRAINE HEADACHES IN CHILDREN

Children referred to the Headache Center at Children's Hospital Medical Center, Cincinnati, OH, Sept 1996-June 1997, were evaluated for clinical characteristics of the headaches, using criteria of the International Headache Society (IHS), and older criteria by Vahlquist and Prensky and Sommer for comparison. Questionnaires were completed by 184 patients and their families. The age range was 2-18 years (mean, 11 yrs), and the majority had suffered 5 or more headaches. The headache duration was less than 2 hours in 25 (13.6%), 2-4 hours in 65 (35%) of whom 54 were younger than 15 years, 4-72 hours in 71 (39%), and more than 72 hours in 22 (12%). Only 62 patients (34%) satisfied the IHS diagnostic criteria. By amending these criteria to include a duration range from 2-48 hours for children younger than 15 years, 109 (59%) met the criteria. If headache duration was excluded entirely from the IHS criteria, 147 (80%) qualified for a migraine diagnosis. The modified IHS criteria matched the Prensky and Sommer but not the Vahlquist criteria. The duration factor should be a minor criterion for migraine in children, and only headaches longer than 72 hours should be excluded in children younger than 15 years. (deGrauw TJ, Hershey AD, Powers SW, Bentti A-L. Headache July/Aug 1999;39:481-485). (Respond: Dr Ton J deGrauw, Division of Neurology, Children's Hospital Medical Center, 3333 Burnet Ave, Cincinnati, OH 45229).

COMMENT. The authors suggest that the IHS criteria for pediatric migraine should be revised, and the duration factor removed or made a minor diagnostic criterion. Many children with a shorter duration and a number with a very long duration headache still fit the diagnosis of migraine, using the modified criteria. The majority have bilateral, usually frontal, headache, and unilateral location is uncommon.

Prothrombotic risk factors in coexistent migraine and stroke. In 17 patients with coexistent disease, the prevalence of factor V Leiden (5.8%) and other prothrombotic genotypes was not significantly different from that determined in 107 patients with ischemic cerebrovascular disease without migraine, 106 migraine patients, and 202 control subjects. Prothrombotic tendencies do not increase the risk of stroke in patients with migraine. (Iniesta JA, Corral J, Gonzalez-Conejero R, Rivera J, Vicente V. Prothrombotic genetic risk factors in patients with coexisting migraine and ischemic cerebrovascular

disease. Headache July/Aug 1999;39:486-489).

Migraine precipitating factors. Of 226 children with headaches, 148 (65%) had migraine without aura, 43 (19%) migraine with aura, 25 (11%) daily headache, and 16 (4%) had tension/migraine. Precipitating factors were elicited in 206 children (91%); 141 (62%) had one factor and 53 (29%) had two or more. In decreasing frequency, these included stress (23%), sleep deprivation (16%), hunger (11%), heat (11%), bright light (9%), exercise (8%), foods (7%), motion (6%), and medications (5%); MSG and caffeine, odors, and reading were precipitants in less than 5% each. Analgesic rebound occurred in 36% of the daily headache group compared to 1.5% of other headache patients. Environmental manipulations and avoidance of precipitating factors may obviate the need for daily medications in the treatment of childhood migraine. (Riback PS. Factors precipitating migraine headaches in children. Ann Neurol Sept 1999;46:540, abstract). In my own experience and that of colleagues in the UK, specific foods are more frequent precipitants of headache (see Progress in Pediatric Neurology I and II, PNB Publ, 1991;pp542-3, 1994;pp166-168).

Mast cell activation in migraine. Mean levels of urinary histamine, its metabolite, methylhistamine, and the mast cell enzyme, tryptase, were higher in children than in adults. In 8 of 10 children who successfully practiced relaxation imagery techniques for migraine, urine levels of tryptase were significantly lower than controls. Stress may activate mast cells that could be involved in the pathophysiology of migraine. (Olness K, Hall H, Rozniecki JJ, Schmidt W, Theoharides TC. Mast cell activation in children with migraine before and after training in self-regulation. Headache Feb 1999;39:101-107).

Intranasal sumatriptan for acute migraine. A randomized double-blind placebo-controlled crossover study showed that 12 of 14 patients (6-10 years of age) reported a decrease in pain intensity after sumatriptan versus 6 of 14 after placebo (p=0.031), and complete relief was obtained in 9 of 14 vs 2 of 14 (p=0.016). (Ueberall MA, Wenzel D. Intranasal sumatriptan for the acute treatment of migraine in children. Neurology April 1999;52:1507-1510).

HEREDO-DEGENERATIVE DISORDERS

SCREENING FOR SPHINGOLIPID-STORAGE DISEASES

A simple fluorescence assay was used to detect cells from patients with sphingolipidoses in a masked study at the Mayo Clinic, Rochester, MN. Replicate samples of 25 of 26 unique cell lines representing ten different lipid-storage diseases (Fabry's disease, gangliosidoses (Tay-Sachs and Sandhoff forms), metachromatic leukodystrophy, mucolipidosis type IV, Niemann-Pick disease (types A, B, and C), and sphingolipid-activator-protein-precursor (prosaposin) deficiency), and 18 of 20 unique cell lines representing controls were correctly identified. The sensitivity was 96.2% and the specificity 90%.

The artificial fluorescent lipid, a derivative of the natural sphingolipid, lactosylceramide, accumulated in the lysosomes of cultured fibroblasts from patients with sphingolipidoses. It was mainly confined to the Golgi complex in normal control cells and cells from patients with other types of lysosomal diseases. With increasing concentrations in the membranes of the lysosomes, the emission of the fluorescent lipid changed from green to red wavelengths. The method should be useful as an initial general screen for lipid-storage diseases,