few days after onset of measles infection are reported from the University of Catania, Italy. The cases occurred during measles epidemics in Italy (1988 and 1992) and the UK (1988). Spontaneous burning pain, increased by stimulation, or loss of pain sensation, over one side of the body was associated with mild ipsilateral paralysis. MRI showed bilateral swelling of thalamic areas and deep cerebral vein thrombosis. Symptoms remitted and MRI abnormalities resolved over a period of weeks or months. (Ruggieri M, Polizzi A, Pavone L, Musumeci S. Thalamic syndrome in children with measles infection and selective, reversible thalamic involvement. Pediatrics Jan 1998;101:112-119). (Respond: Martino Ruggieri MD, Division of Pediatric Neurology, Pediatric Clinic, University of Catania, 95125 Catania, Italy).

COMMENT. Acute measles infection may be complicated by deep cerebral venous thrombosis and symptoms of the thalamic syndrome. The symptoms can resolve without serious sequelae.

HIV-ASSOCIATED COGNITIVE DISORDERS AND CSF VIRAL RNA

The relationship of CSF levels of HIV type 1 RNA to neuropsychological (NP) test performance in 97 prospectively enrolled, HIV-infected subjects (mean age, 31 yrs) was studied at the University of California, San Diego. In patients with AIDS, dementia and minor cognitive-motor disorders were associated with higher CSF RNA levels, whereas those without AIDS showed no relation between CSF RNA and NP impairment. In AIDS, CD4+ lymphocyte cell depletion (<200) correlates with CNS HIV infection and dementia. (Ellis RJ, Hsia K, Spector SA et al, and the HIV Neurobehavioral Research Center Group. Cerebrospinal fluid human immunodeficiency virus type 1 RNA levels are elevated in neurocognitively impaired individuals with acquired immunodeficiency syndrome. Ann Neurol Nov 1997;42:679-688). (Respond: Dr Ellis, University of California, HIV Neurobehavioral Research Center, 2760 Fifth Ave, San Diego, CA 92103).

COMMENT. CSF viral studies link AIDS with cognitive impairments. HIV-1 RNA should be monitored in the CSF as well as plasma during antiretroviral therapy.

LYME DISEASE AND FACIAL PALSY REVISITED

The frequency of abnormal CSF findings in 40 children, ages 3-19 years, with new onset facial palsy and Lyme disease was determined at the State University of New York at Stony Brook. CSF white cell count, protein, or both were elevated in 68% of cases. The majority (89%) had markers of *B burgdorferi* CNS invasion or immune involvement. Lyme disease-associated facial nerve palsy was related to CNS involvement and occult meningitis. Of 22 with CSF pleocytosis, none had meningeal signs and only one third had headache. (Belman AL, Reynolds L, Preston T et al. Cerebrospinal fluid findings in children with Lyme disease-associated facial nerve palsy. Arch Pediatr Adolesc Med Dec 1997;151:1224-1228). (Reprints: Anita L Belman MD, Department of Neurology, HCS T-12-020, State University of New York at Stony Brook, Stony Brook, NY 11794).

COMMENT. The diagnostic value of CSF examination in children with peripheral facial palsy and suspected Lyme borreliosis was reported from Zurich, Switzerland, in Neurology Sept 1997;49:817-824 (see Ped Neur Briefs Oct 1997). Analysis of CSF for specific antibodies to *B burgdorferi* in children with acute facial palsy may facilitate eary diagnosis and prompt antibiotic treatment.

There are **more questions than answers**, according to an editorial comment by Shapiro ED of Yale University, New Haven, CT, and Gerber MA, University of Connecticut, Farmington. They consider that the clinical significance of CSF abnormalities in Lyme disease-associated facial palsy has not been established, and routine spinal tap and *parenteral* antibiotic therapy, recommended by some authorities in cases with CSF pleocytosis, is unnecessary and not justified.

Facial palsy in Kawasaki syndrome is reported from Children's Hospital, Boston, MA. (Bushara K, Wison A, Rust RS. Pediatr Neurol Nov 1997;17:362-364). A 12-week-old-boy with coronary artery aneurysms and Kawasaki syndrome (KS) developed a facial palsy which resolved after treatment with IV immunoglobulin. KS is a vascular inflammatory disease of young children of unknown cause, presenting with unexplained fever, conjunctivitis, red lips, tongue and pharynx, skin rash, cervical adenopathy, erythematous hands and feet, and 25% have coronary aneurysms. Encephalopathy, seizures, stroke, ataxia, myositis, and facial palsy are rare neurological complications.

VASCULAR DISORDERS

SUBTYPES OF ISCHEMIC STROKE: CHILDREN CF YOUNG ADULTS

Children aged 1 to 18 years with acute ischemic stroke, seen at Indiana University, and young adults aged >18 to 45 years, identified from the Indiana University and Northwestern University Young Adults Stroke Registries, were classified in subtypes as atherothrombotic (AT), cardioembolic (CE), small-vessel (SV), or other determined or unknown causes. The percentages of these stroke subtypes in children cf young adults were as follows: AT 0/16, CE 15/14, SV 0/3, other 49/44, and unknown 36/23. Prothrombotic causes (sickle cell disease) were more common in children (25/14%), and dissections more common in young adults (3/15%). Causes of stroke in the 15 to 18 year group of children were more similar to the young adults. (Williams LS, Garg BP, Cohen M, Fleck JD, Biller J. Subtypes of ischemic stroke in children and young adults. Neurology Dec 1997;49:1541-1545). (Reprints: Dr Linda S Williams, Department of Neurology, IUMC, 541 Clinical Drive, CL 365, Indianapolis, IN 46202).

COMMENT. Children with cardioembolic stroke have cyanotic heart disease, and sickle cell disease and moyamoya are the most common causes of the "other determined" subtype in this age group. Young adults with CE stroke have right-to-left atrial shunts due to patent foramen ovale, and arterial dissection and antiphospholipid antibodies are the most common "other determined" causes. Age 15 years is the most appropriate dividing line for subtyping ischemic stroke in children and young adults.

Protein C and S deficiency are risk factors for stroke, according to a study at the Childrens Hospital, Los Angeles, CA. (Koh S, Chen LS. Pediatr Neurol Nov 1997;17:319-321). Among 37 children with ischemic stroke, protein C and S deficiencies were the only identified risk factors in 2 (5.4%) and 5 (14%) patients, respectively.

Transcranial doppler screening for long-term stroke risk in chidren with sickle cell disease was studied at the Medical College of Georgia, Augusta, GA. Elevated TCD velocities, 200 cm/sec or greater, predict increased stroke risk. (Adams RJ, McKie VC, Carl EM et al. Ann Neurol Nov 1997;42:699-704).

Language delay in children with sickle cell disease and stroke is