MICHELIN TIRE BABY SYNDROME VARIANT

A syndrome of multiple congenital anomalies, mental retardation, and symmetrical circumferential skin creases of arms and legs in a 4.5-year-old male is reported from the Montreal Children's Hospital, Quebec, Canada. Craniofacial anomalies included a high forehead, microphthalmia, optic nerve hypoplasia, telecanthus, and micrognathia. CT showed dilated lateral ventricles. Chromosomes were normal. (Elliott AM, Ludman M, Teebi AS. New syndrome?: MCA/MR syndrome with multiple circumferential skin creases. Am J Med Genet 1996;62:23-25). (Reprints: Dr AS Teebi, Division of Medical Genetics, Montreal Children's Hospital, 2300 Tupper Street, Montreal, Quebec, Canada H3H 1P3).

COMMENT. This mental retardation syndrome resembles the "Michelin tire baby syndrome" but has some additional unique anomalies.

NONSPECIFIC BRAIN ANOMALIES IN AUTISM

Measurements of the cerebellar vermis in 125 normal individuals and 102 patients with a variety of neurogenetic abnormalities were compared, using quantitative MRI analysis in a study at the Universities of Nebraska, Omaha; Oklahoma, Norman; West Virginia, Morgantown; and Texas, San Antonio. The average size of cerebellar vermal lobules (CBL) VI and VII in patients with infantile autism was not significantly different from that in age-matched normal subjects. Relative CBL VI-VII hypoplasia occurred in patients with Rett syndrome and Sotos' syndrome, both having autistic behaviors, but the same was true for conditions without autistic behaviors. CBL VI-VII hypoplasia is not limited to disorders with autistic behavior and is not a specific neuroanatomical marker for autism. (Schaefer GB, Thompson JN Jr, Bodensteiner JB et al. Hypoplasia of the cerebellar vermis in neurogenetic syndromes. Ann Neurol March 1996;39:382-385). (Respond: Dr Schaefer, University of Nebraska Medical Center and Meyer Rehabilitation Institute, 600 S 42nd Street, Omaha, NE 68198).

COMMENT. Several neurogenetic disorders have relative CBL VI-VII hypoplasia, and cerebellar vermal hypoplasia is not specific for autism.

Temporal lobe morphology in childhood-onset schizophrenia was studied by MRI in 21 patients examined at the NIMH, the University of Maryland School of Medicine, Baltimore, and Northwestern University School of Medicine, Chicago. (Jacobsen LK et al. Am J Psychiatry March 1996;153:355-361). These schizophrenic patients had smaller cerebral volumes, but larger volume of the superior temporal gyrus. They lacked the normal (right-greater-than-left) hippocampal asymmetry. Early onset schizophrenia was not associated with a severe medial temporal lobe lesion in these patients.

LEARNING AND BEHAVIOR DISORDERS

CLONIDINE FOR SLEEP DISORDERS WITH ADHD

A retrospective analysis of 62 children and adolescents treated with clonidine for sleep disturbances associated with ADHD is reported from the outpatient Pediatric Psychopharmacology unit, Massachusetts General Hospital, Boston. Using the National Institute of Mental Health global assessment of sleep improvement, 85% of patients treated with nighttime clonidine (50-800 mcg, mean 157) for 35 months were much to very much improved. Concurrent pharmacotherapy and comorbidity showed no relation

to response. Mild adverse effects in 31% included morning sedation and fatigue. (Prince JB, Wilens TE, Biederman J et al. Clonidine for sleep disturbances associated with attention-deficit hyperactivity disorder: a systematic chart review of 62 cases. J An Acad Child Adolesc Psychiatry May 1996;35:599-605). (Reprints: Dr Wilens, ACC 725, Massachusetts General Hospital, Boston, MA 02114).

COMMENT. Two thirds of the patients had medication-induced ADHD-associated sleep disturbance, mainly stimulants. The addition of clonidine in combination with stimulants such as methylphenidate in this study appeared to be safe and effective in correcting sleep disturbances. Fatalities have been reported using clonidine and methylphenidate together, and this combination therapy is being discouraged. The authors recommend further systematic assessment in large groups of children to clarify this issue. Clonidine is indicated in the treatment of ADHD, tic disorders, and comorbid ADHD and tic disorders. Somnolence, the most common side-effect of clonidine, often reduces its usefulness.

Pharmacotherapy of ADHD is reviewed in a special article from the Massachusetts General Hospital. (Spencer T, Biederman J, Wilens T et al. J Am Acad Child Adolesc Psychiatry April 1996;35:409-432). The efficacy of stimulants in 70% of subjects has been documented in 155 controlled studies of 5,768 children, adolescents, and adults reported in the literature. Stimulants improve abnormal behaviors in ADHD, self-esteem, cognition, and social and family function. Response varied with age and comorbid conditions. The efficacy of tricyclic antidepressants in ADHD is also documented in more than 1000 subjects. Increased interest in comorbidity in ADHD has not been followed by related therapeutic advances. Data are limited on the response of medications in comorbid ADHD, and on the effects and safety of combined pharmacotherapy.

One of my readers has reminded me of studies carried out in the 1970s regarding the **effect of pyridoxine hydrochloride on hyperactive children** having low levels of whole blood serotonin. Colleagues interested in revisiting the use of vitamin B6 in ADHD are referred to studies by Coleman M, Coursin DB et al. Pediatrics 1975;55; Monogr neural Sci 1976;3:133-136; Biol Psychiatry 1979;14:741. Pyridoxine-induced sensory neuropathy has occurred with doses as low as 50 mg/day when continued for months or years, and megadoses for the treatment of ADHD must be employed with caution. (see Ped Neur Briefs Feb 1995;9:14).

LEAD INTOXICATION IN CHILDREN WITH AUTISM

The incidence of reexposure to lead poisoning in 17 children with pervasive developmental disorders (PDD), including autism, compared to a randomly selected group of 30 children without PDD who were treated for plumbism over the same six year period, was evaluated by a retrospective chart review at the lead treatment program, Children's Hospital, Harvard Medical School, Massachusetts Poison Control System, Boston, MA. Despite close monitoring, inspection and lead hazard reduction or alternative housing, 75% of children with PDD were reexposed to lead compared to 23% without PDD. Those with PDD were older at diagnosis (46 vs 30 months) and had a longer period of elevated lead (39 vs 14 months) during management. (Shannon M, Graef JW. Lead intoxication in children with pervasive developmental disorders. Clin Toxicology March 1996;34:177-181). (Respond: Dr Michael Shannon, Children's Hospital, 300 Longwood Ave, Boston, MA 02115).