Belfast Hospital for Sick Children, Northern Ireland. Theophylline blood level was elevated to 108 mcmol/l (30 mcmol above therapeutic level). Spasms stopped and EEG became normal when nitrazepam was started and theophylline was discontinued. Nitrazepam was withdrawn at 10 months, the sleep EEG was normal at 14 months, and seizures had not recurred at 3 year follow-up. (Shields MD et al. Infantile spasms associated with theophylline toxicity. Acta Paediatr Feb 1995;84:215-217). (Respond: Dr MD Shields, Department of Child Health, Royal Belfast Hospital for Sick Children, 180 Falls Rd, Belfast BT12 6BE, Northern Ireland).

COMMENT. A direct causal relationship was considered probable because of the close temporal association of spasms and a toxic level of theophylline and the complete remission when the drug was discontinued. A dose of 6-8 mg/kg/day theophylline is usually recommended for infants <7 months of age with asthma. The toxic dose in this patient was 16 mg/kg/day.

Infantile spasms in 5 children (4 symptomatic) persisted to 5 to 14 years of age in a report from the Steele Memorial Children's Research Center, University of Arizona, Tucson (Talwar D, Griesemer DA et al. Epilepsia Feb 1995;36:151-155).

MECHANISMS OF ABSENCE SEIZURES

A unifying hypothesis for the pathogenesis of absence seizures, involving the thalamocortical circuitry, is proposed in a neurological progress report from the University of Southern California School of Medicine, Childrens Hospital Los Angeles. Abnormal oscillatory rhythms generated in the circuit involve g-aminobutyric acid (GABA)B-mediated inhibition alternating with glutamate-mediated excitation which triggers a low-threshold calcium current in neurons of the nucleus reticularis thalami. The process is modulated by pathways utilizing various neurotransmitters and projected onto the thalamus and cortex, generating bilaterally synchronous spike wave discharges and absence seizures. Ethosuximide and trimethadione block absence seizures by reducing the low-threshold calcium current via a direct action at the T-type calcium channel. Other anti-absence seizure medications have indirect effects on this calcium current within the thalamus. (Snead OC III. Basic mechanisms of generalized absence seizures. Ann Neurol Feb 1995;37:146-157). (Respond: Dr Snead, Box 82, 4650 Sunset boulevard, Los Angeles, CA 90027).

COMMENT. A knowledge of the mechanisms of absence seizures should facilitate the development of more specific antiepileptic medications and the avoidance of drugs (eg. phenytoin and carbamazepine) that exacerbate absence attacks. For an excellent review of mechanisms of antiepileptic drug action see Talwar D, 1990, and commentary, Progress in Pediatric Neurology I, 1991, pp94-5.

MECHANISM OF OPSOCLONUS-MYOCLONUS SYNDROME

Cerebrospinal fluid measurements of the serotonin metabolite 5-hydroxyindoleacetic acid (5-HIAA) and the dopamine metabolite homovanillic acid (HVA) in samples from 27 children with opsoclonus-myoclonus syndrome and 47 controls are reported from the National Pediatric Myoclonus Center, Children's Research Institute, Washington, DC, and other centers. The mean age at onset was 1.5 years, and patients were symptomatic for 3 years before