pronounced when both parents were alcoholic. (Nordberg L et al. Acta Paediatr Nov 1994;Suppl 404:14-18).

MANGANESE SUPPLEMENTS AND DYSTONIA

A 7 month old girl who developed dystonic movements of the arms after a 3 month period of parental nutrition for jejunal atresia and bowel resection is reported from Great Ormond Street Hospital, London, UK. Development and head growth stopped at 12 months. Liver function tests showed cholestatic liver disease, a complication of parenteral nutrition. MRI showed basal ganglia changes in T1 weighted images compatible with trace metal deposition. A high blood manganese of 1740 nmol/L (ref. 73-210 nmol/L) was diagnosed at 17 months. She died 1 month later with neurological deterioration. A subsequent investigation of 53 children who had been on parenteral nutrition for more than 6 weeks showed that all those with cholestatic liver disease (35/53), and consequent impairment of biliary excretion of manganese, had whole blood manganese levels of >360 nmol/L. The parenteral supplement in the UK contained 55 times more manganese than that recommended by the American Society for Clinical Nutrition. This product has now been replaced with one containing 1 mcg/kg manganese, in line with the American guidelines. (Reynolds AP, Kiely E, Meadows N. Manganese in long term paediatric parental nutrition. Arch Dis Child Dec 1994;71:527-528). (Respond: Dr Reynolds, Department of Chemical Pathology, Great Ormond Street Hospital, Great Ormond St, London WC1N 3JH, UK).

COMMENT. Blood manganese should be monitored in patients on parenteral nutrition, especially those who develop cholestatic liver disease. MRI is recommended if blood manganese is >360 nmol/L and/or if patient develops dystonia.

Manganese poisoning with dystonia in an 8 year old girl with Alagille's syndrome (hepatic duct hypoplasia, chronic cholestasis, facial dysmorphism, vertebral malformations, retarded development, and cardiac murmur) responded to treatment with ursodeoxycholic acid (see Progress in Pediatric Neurology II, Chicago, PNB Publ, 1994, pp438-9). Toxicity from dietary sources of manganese appears to require a prolonged period of exposure before neurologic symptoms develop.

THALLIUM POISONING

Four young adults poisoned with thallium contained in maliciously contaminated marzipan ball candy are reported from the New York City Poison Center, and East Carolina University School of Medicine, Greenville, NC. Gastrointestinal symptoms, including diarrhea, vomiting, abdominal cramps, and constipation, and pleuritic chest pains developed on the second day, and painful paresthesiae of hands and feet on the third day. Weight bearing caused pain in the soles of the feet, so that walking was avoided. Stroking the back of the hands elicited severe pain. Radiographs of the candies showed metallic densities, and atomic absorption spectroscopy measurement of thallium content was 4 g/100g candy. Radiographs of the abdomen on the third day were negative for radiopaque thallium. Hypertension and tachycardia developed on day 4 to 8, and alopecia onset began on day 8 to 15. Treatment consisted of prussian blue (2 g 3x/d orally) to bind enteric thallium, activated charcoal orally, potassium chloride infusion, and iv morphine for pain. All patients recovered without sequelae within one month. (Meggs WJ et al.

Thallium poisoning from maliciously contaminated food. Clin Toxicol Nov 1994;32:723-730). (Reprints: Dr William J Meggs, New York City Poison Control Center, 455 First Avenue, Room 123, New York, NY 10016).

COMMENT. Gastrointestinal symptoms followed closely by painful paresthesiae of extremities are the early diagnostic manifestations of thallium poisoning. Alopecia is a late sign. The authors advocate early treatment with prussian blue. Thallium is radiopaque and radiographs of poisoned food may demonstrate metallic densities.

ATTENTION AND LEARNING DISORDERS

TREATMENT OF ADHD IN TOURETTE'S SYNDROME

A double-blind, placebo-controlled study of clonidine (.05 mg 4xd) and desipramine (25 mg 4xd) treatment of attention-deficit hyperactivity disorder (ADHD) behaviors in 34 children with TS + ADHD is reported from the Departments of Neurology and Pediatrics, Johns Hopkins University School of Medicine, Baltimore, MD. Desipramine was superior to clonidine in improving measures of ADHD, including parent-completed global linear analogue rating scale, hyperactivity subscale of the child behavior checklist (CBCL), and teacher CBCL subscales for nervous/overactive, anxious, and unpopular items. More than two thirds of families requested continuation of desipramine at the completion of the study. Neither drug made tics worse. (Singer HS, Denckla MB et al. The treatment of attention-deficit hyperactivity disorder in Tourette's syndrome: A double-blind placebo-controlled study with clonidine and desipramine. Pediatrics January 1995;95:74-81). (Reprints: Dr Harvey S Singer, Department of Neurology, Harvey 811, Johns Hopkins Hospital, 600 North Wolfe Street, Baltimore, MD 21287).

COMMENT. Desipramine is a more effective medication than clonidine for the treatment of ADHD in children with Tourette's syndrome. The authors hesitate to recommend the general use of desipramine. A review of the literature uncovered at least four sudden, unexplained deaths in children receiving desipramine. Careful monitoring, especially of the cardiovascular system, is advised.

Behavioral improvements found with tricyclic antidepressants and the positive effects of stimulant medication on cognitive tasks have prompted combined drug therapy of ADHD. Side effects occurred more frequently when a combination of desipramine and methylphenidate was employed compared to either medication used alone. (see Progress in Pediatric Neurology II, PNB Publ, 1994, pp210-211).

Bilineal transmission (from maternal and paternal sides) of Tourette's syndrome, especially in families in which the proband's symptoms were most severe, was a frequent finding (approx 1/3) in a study at the University of Rochester School of Medicine, NY, and University College London Medical School, London, UK. (Kurlan R et al. Neurology Dec 1994;44:2336-2342).

DYSLEXIA AND SMALL GENU OF CORPUS CALLOSUM

Corpus callosum morphology was studied by MRI in 16 children (mean age, 9.7 yrs) with developmental dyslexia and matched controls at the Center for Clinical and Developmental Neuropsychology, University of Georgia,