hyperactivity, 55 were included in a 6-week open trial of the Feingold diet, 26 (47%) showed a placebo response, and 14 were identified as likely reactors. Of 8 who subsequently completed a double-blind crossover study (utilizing each child as his own control), 2 demonstrated a significant dependent relationship between the challenge and ingestion of azo dye colorings (tartrazine and carmoisine 50 mg) and behavioral change. Extreme irritability, restlessness and sleep disturbance rather than attention deficit were the common behavioral patterns associated with the ingestion of food colorings, as described by the parents in this study. The authors conclude that the inclusion of children in trials on the basis of attention deficit alone may miss some reactors, and there is little place for use of a coloring-free diet in children with ADD unless the other behavioral features of irritability, restlessness and sleep disturbance are present. (Rowe KS. Synthetic food colourings and 'hyperactivity': A double-blind crossover study. Aust Pediatr J. April 1988;24:143-147).

> **COMMENT.** The phoenix of the Feingold diet rises again with the suggestion that the treatment has been erroneously discarded because of inappropriate behavioral rating instruments and failure to identify specific reactors to food additives. In England, where the avoidance of all foods containing additives is widespread, the major problem is the level of public misinformation, occasionally leading to handicapping dietary restriction. (David TJ. Arch Dis Child 1988;63:582).

BIOTINIDASE DEFICIENCY AND SEIZURES

Preliminary experiences with screening of 24,300 newborns detected 1 infant with biotinidase deficiency at the Depts of Paediatrics, Univ of Verona, Policlinic Borgo Roma, Verona, Italy, and the Hosp for Sick Children, Toronto, Canada. The patient was a full-term baby girl with uncomplicated delivery and a positive family history for seizures in an aunt who had died at 8 months of age. At 2 months of life, the infant developed dermatitis and sparse scalp hair followed by multifocal motor seizures resistant to anticonvulsant drugs. Neurological exam showed hypertonia and hyperreflexia, the EEG revealed increased slow wave activity, and the CT finding was a mild cortical atrophy. Large amounts of 2-oxoglutarate and small amounts of 3-hydroxyisovalerate were found on chromatographic examination of the urine. Treatment with 10 mg/biotin daily resulted in complete recovery within 2-3 days. (Burlina AB et al. Neonatal screening for biotinidase deficiency in north eastern Italy. Eur J Pediatr April 1988;147:317-318).

> COMMENT. The authors consider that biotinidase deficiency is as common as other well-known metabolic disorders and satisfies all criteria for inclusion into neonatal mass screening programs for inborn errors of metabolism. The absence of the expected organic acidopathy noted in the present case-report confirms the need for biotinidase enzyme estimations in diagnosis.