gabapentin, lamotrogine, and other newer agents during pregnancy is relatively sparse, but teratogenicity has not been reported.

GROWTH SUPPRESSION WITH ACETAZOLAMIDE

The effect of acetazolamide on growth of children with epilepsy was evaluated at Osaka Medical Center, Japan. Standard scores of height and weight were compared in 17 children receiving acetazolamide as an adjunct to AED monotherapy: 1) before AED treatment; 2) during AED monotherapy; 3) during adjunct acetazolamide therapy; and 4) after acetazolamide had been discontinued. Both height and weight were significantly reduced during acetazolamide administration, and growth returned to the original level after acetazolamide was withdrawn. The degree of growth suppression was not related to the age, duration, dosage, or the concomitant AED therapy. Metabolic acidosis induced by acetazolamide was postulated as the cause. (Futagi Y, Otani K, Abe J. Growth suppression in children receiving acetazolamide with antiepileptic drugs. Pediatr Neurol Nov 1996;15:323-326). (Respond: Dr Futagi, Division of Pediatric Neurology, Osaka Medical Center and Research Institute, 840 Murodo-cho, Izumi, Osaka 590-02, Japan).

COMMENT: Anorexia and loss of weight have been reported with acetazolamide therapy for epilepsy (Millichap et al. 1956, 1964), but this seems to be the first report of acetazolamide-induced growth suppression. Height and weight should be monitored carefully during acetazolamide therapy.

Since the metabolic side effects of the ketogenic diet are similar to those of acetazolamide (Millichap JG. Progress in Pediatric Neurology I, PNB Publ, 1991:pp85-88), and loss of weight is a common occurrence with the initiation of the diet, monitoring of height and weight are equally important during ketogenic dietary therapy. Two children under one year of age showed no increase in weight, length or head circumference during a six month period on the diet (Schwartz RH et al, 1989), and the monitoring of acetazolamide or the ketogenic diet in infants and young children should be especially strict.

ATTENTION DEFICIT DISORDERS

ADHD AND PSYCHOACTIVE SUBSTANCE ABUSE

The effect of attention-deficit hyperactivity disorder (ADHD) compared to psychiatric comorbidity, familiality, and adversity, as risk factors for psychoactive substance use disorder (PSUD) was evaluated at the Department of Psychiatry, Massachusetts General Hospital, Boston, MA. Using baseline and 4-year follow-up data from 140 ADHD and 120 normal control subjects, the rates of alcohol or drug abuse or dependence (PSUD) were 15%, with no differences between groups. Conduct and bipolar disorders were predictive of PSUD, but these associations were independent of ADHD. Oppositional defiant disorder, uncomplicated by conduct disorder, did not predict PSUD. Family history of substance dependence and antisocial disorders was associated with PSUD in controls but less so in ADHD probands. Family history of ADHD was not associated with risk of PSUD. (Biederman J, Wilens T, Mick E et al. Is ADHD a risk factor for psychoactive substance use disorders? Findings from a four-year prospective follow-up study. J Am Acad Child Adolesc Psychiatry Jan 1997;36:21-29). (Reprints: Dr Biederman, Pediatric Psychopharmacology Unit (ACC 725), Massachusetts General Hospital, Fruit Street, Boston, MA 02114).