COMMENT. Vitamin D insufficiency is prevalent among almost all children with epilepsy, and the risk is significantly increased for female patients and in patients with increased body mass index. Specific antiepileptic drug usage, comorbid cerebral palsy or intellectual retardation, and seizure control, potential risk factors for vitamin D insufficiency, did not contribute to the risk in this patient cohort. Patients with partial seizures were at increased risk, but the significance of this observation was confounded by the associated elevated body mass index. Prevalence of hypovitaminosis D is high in the general pediatric population, but patients with epilepsy are at additional risk for bone injury and other complications. Increased attention to vitamin D levels and more extensive use of vitamin D supplementation are warranted in children with epilepsy, especially girls and those with elevated body mass index.

In a review article on vitamin D and bone health in children with epilepsy (Shellhaas RA, Joshi SM. **Pediatr Neurol** 2010;42:385-393), the authors suggest methods for vitamin D supplementation based on 25-hydroxyvitamin D levels (ng/ml), pending official guidelines. They advocate screening for vitamin D insufficiency and supplementation with cholecalciferol to maintain optimal vitamin D levels.

In an editorial, Wirrell E, Mayo Clinic, Rochester, MN, (**Pediatr Neurol** 2010;42:394-395) agrees with Shellhaas and Joshi that all children with epilepsy should receive 400 IU of vitamin D and an optimal calcium intake and diet. Further, a dose of vitamin D at 1000 IU per day should be considered for children with symptomatic generalized epilepsy, those with impaired mobility, and those treated with polytherapy. Screening for vitamin D levels should be considered for all children with epilepsy. Bone mineral density screening without pathologic fracture requires further study. Vitamin D insufficiency in pediatric epilepsy appears to be neglected, probably as a result of conflicting study findings.

DEMYELINATING DISORDERS

VITAMIN D STATUS AND MULTIPLE SCLEROSIS RELAPSE

Researchers at University of California, San Francisco, and State University of New York at Stony Brook, NY consecutively recruited patients with pediatric-onset multiple sclerosis into a prospective cohort to determine if vitamin D status is associated with the rate of subsequent clinical relapses. Among 110 patients followed, the mean serum 25-hydroxyvitamin D level was 22 +/- 9 ng/ml, and only 16 (15%) had normal unadjusted levels (> 30 ng/ml). After adjustment for age, gender, race, ethnicity, disease duration, therapy, and length of follow-up, every 10 ng/ml increase in adjusted 25-hydroxyvitamin D level was associated with a 34% decrease in the rate of subsequent relapses (p=0.024). Only Hispanic ethnicity was independently associated with an increased risk of relapse. The finding suggests that supplementation of vitamin D stores may reduce risk of multiple sclerosis relapse and warrants investigation. (Mowry EM, Krupp LB, Milazzo M et al. Vitamin D status is associated with relapse rate in pediatric-onset multiple sclerosis. **Ann Neurol** May 2010;67:618-624). (Respond: Dr Mowry, Department of Neurology, Multiple Sclerosis Center, University of California, San

Francisco, 350 Parnassus Avenue, Suite 908, San Francisco, CA 94117; E-mail: ellen.mowry@ucsf.edu).

COMMENT. Low vitamin D levels are associated with an increased relapse rate in pediatric-onset multiple sclerosis. Relapse rate in pediatric-onset is significantly higher than that in adult-onset cases (Gorman MP et al. **Arch Neurol** 2009;66:54-59). Mean age of the patients in the above study at time of blood collection was 15 years +/- SD3.

VASCULAR DISORDERS

ANTICOAGULANTS IN PEDIATRIC SINOVENOUS THROMBOSIS

Safety and outcome of anticoagulant therapy in neonates and children with cerebral sinovenous thrombosis (CSVT) were determined in a study at the Hospital for Sick Children and Toronto Western Hospital, Ontario, Canada. Neonates presented with seizures and encephalopathy, children had headache and raised intracranial pressure. Prothrombotic abnormalities occurred in 76%. Among 162 pediatric patients, 85 received anticogulants (standard/low molecular weight heparin, warfarin), including 29/83 (35%) neonates and 56/79 (71%) children. Mean interval from diagnosis to anticoagulant initiation was 4 days. Major hemorrhage occurred in 6% (6/99) of treated patients; they were all nonfatal and clinical outcome was favorable in 50%. Follow-up imaging showed thrombus propagation in 11/57 neonates (10/35 [28%] without and 1/22 [4%] with anticoagulant therapy (p=0.037)) and in 10/63 children (7/19 [37%] without and 3/44 [7%] with anticoagulant [p=0.006]). Propagation was associated with new venous infarcts in 10% neonates and 40% children and worse clinical outcome in children (p=0.053). Recanalization occurred earlier and more completely in neonates (p=0.002). Clinical outcome was unfavorable in 47%. (Moharir MD, Shroff M, Stephens D et al. Anticoagulants in pediatric sinovenous thrombosis: a safety and outcome study. **Ann Neurol** May 2010;67:590-599). (Respond: Dr G deVeber, Division of Neurology, the Hospital for Sick Children, 555 University Ave, Toronto, Ontario M5G 1X8 Canada. E-mail: Gabrielle.deveber@sickkids.ca).

COMMENT. In this large single-center cohort study of anticoagulant safety in pediatric CSVT, treatment-related hemorrhage was infrequent whereas in untreated patients, thrombus propagation was frequent, occurring in one-third patients. The authors conclude that anticoagulants deserve strong consideration in pediatric CSVT.

TRIAL OF INSULIN FOR POSTSTROKE HYPERGLYCEMIA

The effect of glucose potassium insulin (GKI) infusion (compared to saline as control) within 24 hours of ischemic stroke was studied in patients (> 18 years old) with blood glucose >126mg/dl (7mmol/l) admitted to Southern General Hospital, Glasgow, Scotland. Infarct growth on MRI between baseline and day 7 was the primary endpoint. Brain lactate concentrations were measured with MR spectroscopy. Forty patients were randomized, 15 to saline and 25 to GKI infusions. From 6 to 12 hours after infusion,