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,

capillary blood glucose was lowered significantly compared to the saline group, without alteration of infarct growth. In a secondary analysis, GKI was associated with significantly greater infarct growth in patients with complete intracranial vessel occlusion compared with controls (p=0.011) Brain lactate levels were significantly lower in GKI infused patients, and increased in control subjects. Asymptomatic hypoglycemia occurred in 76% of GKI-treated patients. (McCormick M, Hadley D, McLean JR, Macfarlane JA, Condon B, Muir KW. Randomized, controlled trial of insulin for acute poststroke hyperglycemia. **Ann Neurol** May 2010;67:570-578). (Respond: Prof Muir, Institute of Neurological Sciences, Southern General Hospital, Glasgow G51 4TF, Scotland, UK. E-mail: k.muir@clinmed.gla.ac.uk).

COMMENT. Hyperglycemia in more than 50% of acute stroke patients is an independent risk factor for poor outcome. Guidelines recommend blood glucose monitoring but optimal method of management is not established. In the above trial of insulin infusion within 24 hours of stroke, blood glucose was lowered and an increase in brain lactate was attenuated, but cerebral infarct growth was not affected. In patients with persistent arterial occlusion, GKI infusion was associated with greater infarct growth. The authors conclude that GKI infusion to treat moderate hyperglycemia in acute ischemic stroke requires further study and is not recommended in routine clinical practice.

An editorial (Johnston KC, Parsons M. **Ann Neurol** 2010;67:557-558) questions if the answer is in the imaging, and if intervention should be as early as possible after stroke onset. Studying the natural history of blood glucose after ischemic stroke, Wong AA et al (**Neurology** 2008;70:1036-1041) found that mean glucose levels remain static in patients with ischemic stroke without diabetes until at least 48 hours post-stroke. Levels are higher in patients with more severe stroke. Higher or lower levels regress to the mean over time.

DEGENERATIVE DISEASES

EPIDEMIOLOGY OF PROGRESSIVE NEUROLOGICAL DISEASE

Since 1997, researchers in the UK have searched for variant Creutzfeldt-Jacob and other diseases that cause progressive intellectual and neurological deterioration (PIND) by sending a monthly surveillance card to UK pediatricians. Clinical details are obtained by questionnaire or site visit. In 12 years, 2636 patients <16 years old with suspected PIND were reported, of whom 1114 had a confirmed diagnosis. Of 147 different diseases, the 6 commonest groups were leukoencephalopathies (183 cases), neuronal ceroid lipofuscinoses (141), mitochondrial (122), mucopolysaccharidoses (102), gangliosidoses (100), and perixisomal diseases (69). Districts having high rates of consanguinity reported relatively large numbers of PIND cases. Only 6 children with variant Creutzfeldt-Jacob disease were identified. (Verity C, Winstone AM, Stellitano L et al. The epidemiology of progressive intellectual and neurological deterioration in childhood. **Arch Dis Child** May 2010;95:361-364). (Respond: Dr CM Verity, Child Development Centre, Box 107, Addenbrooke's Hospital, Hills Road, Cambridge CB2 0QQ, UK. E-mail: christopher.verity@addenbrookes.nhs.uk).