aggregating agents. One had a latent von Willebrand disease. The mechanism of the diet-induced bruising may be complex, involving interaction between the diet and individual platelet dysfunction. A possible bleeding tendency should be evaluated in patients on the ketogenic diet who are candidates for surgery or anticoagulant therapy. (Berry-Kravis E, Booth G, Taylor A, Valentino LA. Bruising and the ketogenic diet: evidence for diet-induced changes in platelet function. Ann Neurol January 2001;49:98-103). (Respond: Dr Berry-Kravis, RUSH-Presbyterian-St Luke's Medical Center, 1725 West Harrison Street, Suite 718, Chicago, IL 60612).

COMMENT. Despite the absence of serious bleeding in this series of patients treated with the ketogenic diet, a 30% incidence of diet-induced bruising deserves further study and evaluation. A possible interaction with lamotrigine is suggested in some patients receiving concurrent drug and diet.

This is not the first observation of platelet dysfunction and anemia as a complication of the ketogenic diet. Complications in 10% of 52 children treated by Ballaban-Gil et al, 1998 (see Ped Neur Briefs August 1998;12:60) included thrombocytopenia and hemolytic anemia. Valproate interaction could not be excluded in 29 (56%). The proportion of ketogenic/antiketogenic foods was 4:1 in this study but was not specified in the above Pres St Luke's study. This report of diet-induced bruising is another reason to endorse the Mayo Clinic method of slow initiation of the diet with lower ratios, in place of the Hopkins recommended ratio of 4:1. Using the Mayo Clinic method, I have not encountered this or other serious complication as reported with the Hopkins regimen (Ped Neur Briefs 1998;12:61).

A fat-overload syndrome with neurologic complications is reported in 2 children receiving fat emulsion therapy. Both patients died and autopsy showed cerebral intravascular lipid deposition and areas of necrosis and hemorrhage. (Schulz PE et al, 1994; Progress in Pediatric Neurology III, PNB Publ, 1997;p98). A rapid rise in triglyceride levels was invoked as a factor in this complication.

FAMILIAL MESIAL TEMPORAL LOBE EPILEPSY

Clinical, genetic, and MR characteristics of 68 patients with familial mesial temporal lobe epilepsy (MTLE) were analysed at the University of Campinas-UNICAMP, Brazil. Hippocampal atrophy (HA) was identified by MRI in 48 (57%) of 84 patients examined. HA was present in 46% of 13 patients with seizure remission, in 51% of 16 patients whose seizures were well controlled by AEDs, and in all 16 patients with refractory MTLE. HA was also found in some patients without MTLE: in 30% of 10 patients with febrile seizures alone, 60% of 10 with generalized tonic-clonic epilepsy, and in 1 of 4 with a single partial seizure. Familial MTLE is a heterogeneous syndrome with a genetic component in etiology. (Kobayashi E, Lopes-Cendes I, Guerreiro CAM et al. Seizure outcome and hippocampal atrophy in familial mesial temporal lobe epilepsy. Neurology January (2 of 2) 2001;56:166-172). (Reprints: Dr F Cendes, Departamento de Neurologia, Faculdade de Ciencias Medicas-UNICAMP, Caixa Postal 6111, Cidade Universitaria Zeferino Vaz Campinas SP, Brazil, CEP 13083-970).

COMMENT. In this series of patients with familial mesial temporal lobe epilepsy, 57% had MRI evidence of mesial temporal sclerosis. Hippocampal atrophy is found not only in patients with refractory epilepsy but also in patients with a favorable outcome. Genetically determined mechanisms may have a role in hippocampal damage in familial cases of MTLE. In contrast to most patients with temporal lobe epilepsy, a history of febrile seizures is uncommon in patients with familial TLE.