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 IA. 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.