unit granulocyte/macrophage (CFU-GM) assays using bone marrow from healthy adult volunteers cultured in the presence of increasing doses of valproic acid (60, 120, and 240 mcg/ml). At a VPA concentration of 120 mcg/ml, similar to that observed in the patient with erythrocyte aplasia, macrocytosis and neutropenia, there was a 67% CFU-GM growth inhibition, and at VPA levels of 240 mcg/ml, 84% of the colony growth was inhibited. The inhibition was specific to VPA and was not related to a change in pH. The addition of the patient's serum to the assays had no significant effect. The results suggested a direct dose dependent suppression of bone marrow neutrophilic progenitors by valproic acid. (Watts RG et al. Valproic acid-induced cytopenias: Evidence for a dose-related suppression of hematopoiesis. J Pediatr Sept 1990; 117:495-499).

COMMENT. This patient had no family history or medical history of anemia, congenital or acquired bone marrow failure, or malignancies. The only recent drug exposure was valproic acid. The authors recommend close hematologic monitoring of patients receiving valproic acid therapy and especially when larger doses are employed.

Of practical importance in the management of a severe overdose of valproic acid, studies of the elimination half life and clearance of valproic acid in a patient with dialysis-induced encephalopathy who was taking divalproic sodium for a seizure disorder showed that hemodialysis and hemoperfusion had little effect on the removal of valproic acid from the body. The equilibrium shifted so that valproic acid redistributed back into the blood from the tissues. (Kandrotas RJ et al. Neurology Sept 1990; 40:1456-1458). Hemodialysis is unlikely to benefit patients with toxic overdose of valproic acid.

FACTOR XIII DEFICIENCY AND INTRACRANIAL HEMORRHAGE

A 38 month old boy with excessive bleeding following circumcision as a newborn and two episodes of intracranial hemorrhage at four months and at 8½ months of age is reported from the Scott and White Clinic, Temple, TX. The diagnosis of Factor XIII deficiency was made in infancy and the bleeding was stopped with cryoprecipitate. Since 11 months of age he was treated with Fibrogammin at four week intervals and no further hemorrhages had occurred. MRI at three years demonstrated a left temporoparietal encephalomalacia and no vascular malformations. (Larsen PD et al. Factor XIII deficiency and intracranial hemorrhages in infancy. Pediatr Neurol July-August 1990; 6:277-278).

COMMENT. Repeated intracranial hemorrhage can occur spontaneously and without evidence of trauma in infants with Factor XIII deficiency. Routine coagulation studies do not detect this factor deficiency and examination for Factor XIII may avoid a misdiagnosis of child abuse. Replacement therapy should be maintained throughout life.