the bleeding time and platelet adhesiveness defect found in vWd. (SPR)

53 *The Role of Folate and Vitamin B_{12} in the Etiology of the Thrombocytopenia of Iron Deficiency Anemia.* SANFORD LEIKIN and PARVANEH VOSSOUGH*, Children's Hosp. of the D.C., and George Washington Univ. Sch. of Med., Washington, D.C.

Thrombocytopenia is an unexplained complication of iron deficiency anemia. Studies have suggested that the low levels of platelets may in some instances be related to a folate or vitamin B_{12} deficiency. In order to pursue this possibility, neutrophile lobe counts, bone marrow examinations, serum folate and vitamin B_{12} levels, and formiminoglutamic acid (FIGLU) excretion studies were performed on 55 iron deficient anemic infants and children both with and without thrombocytopenia. Similar studies were performed on normal children. There was an increased incidence of hypersegmentation in the iron deficiency patients and a number of these children exhibited increased lobe averages. Twenty-five of the anemic children showed abnormalities of the bone marrow consistent with megaloblastic dysplasia. The serum vitamin B_{12} levels were within normal limits in all patients but the serum folate level was decreased in two of them, and three children excreted increased amounts of FIGLU.

Although there appeared to be a slightly greater incidence of bone marrow megaloblastoid changes in the thrombocytopenic children no clear separation on a morphologic or biochemical basis could be made between those with and without a platelet deficiency. These findings indicate that although a folate or vitamin B_{12} deficiency at the bone marrow level may be involved in the causation of the thrombocytopenia of iron deficiency anemia, other factors may also be important in the etiology of the complication. (SPR)

54 *Congenital Pernicious Anemia with Coexistent Transitory Intestinal Malabsorption of Vitamin B_{12}.* BEATRICE C. LAMPKIN* and ALVIN M. MAUER, Dept. of Pediat. Univ. of Cincinnati, Ohio.

Pernicious anemia (PA) with coexistent transitory intestinal malabsorption of vitamin B_{12} has been documented in adults. This report is the first description of proven transitory selective intestinal malabsorption of vitamin B_{12} in congenital PA. A vitamin B_{12} responsive anemia was demonstrated in the patient, a 2 ½-year-old girl, by change in the bone marrow from megaloblastic hyperplasia to normoblastic hyperplasia 48 h after 5 μg of vitamin B_{12}. No intrinsic factor (IF) was found in gastric fluid by in vitro assay before and again 4 and 6 weeks after therapy. Congenital PA was established by the age of the patient, absence of IF in gastric fluid, lack of antibodies to IF and parietal cells, presence of HCl in gastric fluid, and normal gastric biopsy. The xylose tolerance test, 72 h fecal fat determination, upper GI series, and biopsy of the jejunum were normal, but the Schilling test was abnormal with hog IF of known potency on two occasions, indicating selective malabsorption of vitamin B_{12}. Six months after therapy the Schilling test was still abnormal without IF, but was normal with both human and hog IF. This normal absorption with IF after therapy is indicative that selective malabsorption was originally present, probably as a consequence of the vitamin B_{12} deficiency resulting from IF lack. In patients with abnormal radioactive vitamin B_{12} absorption tests with administration of IF, coexistent PA must be excluded by demonstration of IF in gastric fluid. (SPR)

55 *Oxymetholone Therapy of Aplastic Anemia.* DONALD M. ALLEN*, MORRIS H. FINE*, THOMAS F. NECHELES* and WILLIAM DAMESHEK*, Tufts University School of Medicine, and N.E. Medical Center Hosps., Boston, Mass. (introduced by S.S. Gellis).

A new oral synthetic androgen, oxymetholone (2-hydroxymethylene-17 methyl-17B hydroxy-3-androstanone), has been remarkably effective in the treatment of 5 consecutive children with aplastic anemia, including 2 unresponsive to oral testosterone. The pattern of response was similar in each patient with diminished purpura and transfusion requirement by 4–6 weeks, a reticulocytosis by 8–10 weeks, and a gradual rise in hemoglobin level to normal by 12–20 weeks. A rise in total white cells with an absolute increase in polymorphonuclear forms developed during the 6th to 12th week of treatment. Platelet levels had improved by 12–16 weeks but have remained subnormal. Three of the children with acquired aplastic anemia received oxymetholone and prednisone as their only treatment and all have maintained their improvement for from 7 to 12 months off all medication. One 16-year-old boy with acquired aplastic anemia of 18 months' duration, and unimproved following a 6-month course of sublingual testosterone propionate and splenectomy, had a characteristic response to oral oxymetholone within 10 weeks. The fifth patient, with congenital aplastic anemia, had failed to improve during a 9-month course of sublingual testosterone propionate. Within 10 weeks on oral oxymetholone he had a reticulocytosis followed by a sustained rise in hemoglobin level. Virilization was noted in the 4 pre-pubertal patients with the dosages used (2.5 to 7.0 mg/kg/day). No advance in bone age or other side effects were observed. Oxymetholone is a useful, easily administered, new androgenic steroid for therapy of aplastic anemia and has proved effective in children unresponsive to testosterone. (SPR)

56 *The Adaptive 'Anemia' of Severe Protein Calorie Malnutrition (Marasmus).* NATHAN J. SMITH and ABRAHAM STEKEL*, King County Hospital, Univ. of Washington, Seattle, Wash.

A reduced hemoglobin concentration with normocytic, normochromic erythrocytes commonly cocurs in severely marasmic infants. Severe marasmus has been produced in 20 piglets by feeding a balanced but restricted diet to stabilize their weight at 5 to 7 kg for up to 9 months. Eight additional piglets served as controls. Clinical, autopsy and biochemical observations show the similarity of this animal model to marasmus previously studied in human infants. Red cell indices, marrow morphology, transferrin iron, marrow iron stores and DFP^{32} erythrocyte life span are all normal and fail to indicate the presence of any specific nutritional deficiency that alters the production or survival of erythrocytes in marasmus. In spite of these findings the animals have a persistent but *not* progressive reduction of hemoglobin concentration (10 to 11 gms % compared to 15 to 16 gms % normal). Cr^{51} studies reveal a red cell mass deficit (16.5 ml/kg in 7 months, 6 kg pigs compared to 25 ml/kg in normal 6 kg pigs). The animals were stressed by controlled phlebotomy. A prompt erythropoietic response occurred with return to the prebleeding hemoglobin level. Small increases of food intake were consistently and rapidly followed by increased erythropoiesis as measured by reticulocyte counts, M:E ratio, and $iron^{59}$ kinetic studies. On the