chronic granulocytic leukemia [Lancet *ii:* 1320, 1967]. We have observed similar long-term cycles in 8 children during remission of acute leukemia (myeloblastic or monomyelogenous) in the absence of infections or changes of any chemotherapeutic agents. Likewise, a child with trisomic Down's syndrome and an unusual aplastic anemia showed long-term cycles for 18 months prior to his demise.

A prospective study of neutrophil cycles in 6 prepuberal males was undertaken under controlled conditions for 3 ½ months. Results of this study are as follows:

Type of patient and cycle	Period of cycle	Mean ampl.
Nonmongoloid short cycles	8.6– 15.5 days	1280–2250
Mongoloid short cycles	11.3– 19.0 days	2240–3460
Mongoloid long cycles	59.5–101.5 days	2265–3438
	Max. ampl.	Min. ampl.
Nonmongoloid short cycles	2700–3900	600–1500
Mongoloid short cycles	4000–6900	1000–1200
Mongoloid long cycles	2850–3550	1100–2600

Graphic display of neutrophil counts done twice weekly in 3 nonmongoloid controls shows only short-term cycles. However, the 3 patients with trisomy 21 reveal long-term cycles as well, upon which the short-term cycles are superimposed.

The short-term cycle appears to be an oscillatory feature of normal granulopoiesis, whereas the long-term cycle may denote abnormality of granulopoiesis.

128 *The Contribution of Relative Renal Mass to Erythropoietin Production.* J. A. Soler, W. D. Noyes, H. L. Cromroy and G. A. Richard, Univ. of Florida, Sch. of Med. (introduced by Elia M. Ayoub).

Androgenic hormones have been reported to increase erythropoiesis by direct stimulation of erythroid marrow. Because androgens also possess a renotrophic effect, the contribution of renal mass to erythropoietin production was investigated.

Rats were injected for 6 weeks with depotestosterone (in cotton seed oil), prior to and after nephrectomy. Controls received cotton seed oil or were sham-operated. Endogenous erythropoietin production was suppressed during the experiment by transfusion with packed red blood cells. Erythropoietin production was stimulated by subjecting the animals to hypoxia for 18 h. Kidneys were then removed and blood obtained for erythropoietin bioassy.

Hypoxia resulted in variable increases in erythropoietin levels in all animals. This variation in erythropoietin production was found to correlate closely with the relative renal mass (renal weight:body weight ratio), with erythropoietin levels increasing proportionately to renal mass in animals receiving either testosterone or in controls. Animals not subjected to hypoxia did not show any increase in erythropoietin levels, despite administration of androgens and resultant increase in renal mass.

These studies show that the enhanced production of erythropoietin by androgens during hypoxia is due not only to its direct action on bone marrow, but is also related to the renotrophic effect of the steroid.

129 *Primary Erythrocytosis, Increased Erythropoietin Excretion and an Intrarenal Perfusion Abnormality.* C. Thomas Kisker and Leonard I. Kleinman, Dept. of Ped. Univ. of Cincinnati Coll. of Med. (introduced by Alvin M. Mauer).

A unique etiology for erythrocytosis was found in a 6-year-old boy. He was normotensive with increased erythropoietin and renin levels. His hemoglobin was 24 g% and hematocrit 73%. The red cell mass by ^{51}Cr tagging was greater than 2 standard deviations above expected values. WBC, platelet count, hemoglobin electrophoresis and serum electrolyte values were normal. No tumor was demonstrated and he increased normally his urinary erythropoietin excretion after phlebotomy. Arterial oxygen saturation was 98% and his hemoglobin did not have increased oxygen affinity. Despite normal creatinine clearance, urine concentration to 1,260 milliosmoles/l, and normal renal biopsy, intrarenal narrowing of distal arteries was found on selective renal arteriography. Increased levels of renin (0.105 and 0.133 au/ml) and erythropoietin (2.6 and 2.5% ^{59}Fe incorporation) were found in renal vein blood. During simultaneous steady state infusion with inulin and PAH, renal plasma flow (282 ml/min) and glomerular filtration rate (85 ml/min) were normal. However, inulin extraction was high normal (30%) while PAH extraction was abnormally low (80%). These values are best explained by intrarenal shunting of blood, establishing areas of ischemia resulting in increased erythropoietin and renin excretion and thus causing the clinical and laboratory characteristics of primary erythrocytosis.

130 *Bone Marrow Transplantation for Aplastic Anemia Following Hepatitis.* Margaret W. Hilgartner, Philip Lanzkowsky, Ralph L. Nachman and Mark B. Weksler, The New York Hospital-Cornell Univ. Med. Center, New York, N.Y. (introduced by Carl H. Smith).

The fatal outcome of aplastic anemia following hepatitis is well documented. Therefore bone marrow (BM) transplantation was performed in a 12-year-old girl who developed pancytopenia following mild hepatitis. Repeated BM aspirations revealed absent megakaryocytes, and erythroid and myeloid precursors. Despite prednisolone and oxymethylone therapy the pancytopenia progressed (hg 6.4 g%, WBC 100/mm^3, platelets < 10,000/mm^3). Staphylococcal sepsis following a finger infection and gram-negative sepsis following a perirectal abscess were treated with multiple antibiotics. Thawed frozen red cell and platelet transfusions were given as required. Because the clinical course was one of progressive deterioration without evidence of BM regeneration, BM transplantation was performed. The patient was pretreated with transfused donor buffy coat cells followed by cyclophosphamide 60 mg/kg for 3 days. 505 ml of HLA matched BM from an uncle were given. On the 8–10th day following transplantation reticulocytes were seen and the WBC rose to 2,200/mm^3. BM revealed megakaryocytes and clusters of myeloid and erythroid cells. Chromosomal analysis revealed XY pattern. Concurrently the patient developed periorbital edema and desquamating, erythematous rash, purpura, abdominal pain, gross bleeding from the bowel and low factors II, V, VII and VIII with elevated factor I suggesting a consumption coagulopathy. She died 10 days following BM transplantation. Postmortem revealed regenerating marrow and extensive plasmacytoid cell proliferation