cells in each stage I, II and III existed in the parenchyma. But almost all megakaryocytes in stage IV existed as free cells presumably in a sinusoid. It is assumed that, after formation of the demarcation membrane in the whole cytoplasm, megakaryocytes in the parenchyma move into the sinusoids, and then the whole cytoplasm of megakaryocytes is broken explosively into small pieces to be designated as platelets.

M. Cortellaro, E. Pogliani, E. Cofrancesco and E. E. Polli (Medical Clinic 1, University of Milan): **Thrombocytopathy Induced by Acquired Circulating Factor VIII Inhibitor. (495)**

A reduced ADP and ristocetin platelet aggregation by acquired circulating factor VIII inhibitor (2 U/ml) was found in a young woman with S. L. E. Bleeding time was prolonged, factor VIII activity decreased (25%), Willebrand antigen and Willebrand factor were normal. PF_3 assay and PF_4 release were normal. Platelet (^{14}C) – serotonin uptake, but not release, was reduced. Antiplatelet antibody was not detected.

Patient's plasma inhibited the ristocetin and ADP induced platelet aggregation of normal PRP, but not of normal and patient GFP.

After steroid treatment F. VIII inhibitor and thrombocytopathy disappeared.

It is suggested that the circulating inhibitor is able to coat autologous and isologous unwashed platelets, interfering with platelet function.

J. H. Sanderson, I. S. Chart and C. Shingles (Central Toxicology Laboratory, Alderley Park, Nr. Macclesfield, Cheshire): **Sex Hormones and Phytomenadione Evidence for their Relationship in an Induced Coagulation Defect. (496)**

Rats dosed with benzyl 3,5-dichloro-2,6-difluoro-2-pyridyl ether show a coagulation defect which is much more severe in males than females, and dose levels can be chosen at which males only are affected. The defect is produced by a lowering of Factors VII, X and II.

This sex differentiation can be abolished if male rats are castrated or if they are treated with oestrogens, and normal male rats can be protected by treatment with phytomenadione.

It is suggested that the use of this compound may provide a tool for understanding mechanisms of anti-coagulation.

P. M. Blatt, W. J. Yount, H. R. Roberts, N. M. Hadler, P. D. Utsinger and J. H. Korn (University of North Carolina, School of Medicine, Chapel Hill, North Carolina, U.S.A.): **Factor XI Deficiency, Juvenile Rheumatoid Arthritis (JRA), and Systemic Lupus Erythematosus (SLE): Report of the First Case. (497)**

A number of inborn errors have recently been associated with a diathesis for collagen vascular disorders. For example, hereditary deficiencies of Clr or C2 components of complement have been associated with certain features of SLE. We wish to record the syndrome of JRA with evolution to SLE associated with a familial deficiency of Factor XI (DTA). The propositus, a 26 yecr old Sephardic female, presented at age 4 with symmetrical destructive polyarthritis, fever and rash. Progressive renal disease began in her early teens, and she developed multiple diagnostic criteria for SLE beginning at age 25. Three maternal relatives manifest arthritis and two showed mild bleeding after surgery. Physical findings included a deforming arthritis and rash. Low levels of C3 and C4, high titers of antibody to native DNA, positive LE cell preparations and Clq precipitins were present. Skin biopsy showed deposits of IgG, IgM, Clq, C3 and C4 at the dermal-epidermal junction.

Factor XI levels were 5 per cent in the propositus and mother, with normal levels in a sister. There was no evidence for a circulating anticoagulant and other clotting factors were normal.

We believe the present case represents the first association of an inborn error in the clotting system (Factor XI deficiency) and collagen vascular disease with immune complex deposition.