These observations have now been somewhat extended by observing the same two parameters in rabbits soon after their production of blood platelets has been completely stopped by a more heavy dose of whole body irradiation. Abnormal leakage of red cells to lymph (drained from the ears which had been shielded during the irradiation) the often occurred at levels of 100–400,000 platelets per μl, whereas the bleeding times in the same animals were usually not significantly prolonged until the platelet concentration fell below 50,000 per μl blood.

An explanation for the two described phenomena would be that the vascular supportive effect and the haemostatic effect of blood platelets are dependent on two different mechanisms.

A.-L. Bergström and J. Kutti (Sahlgren's Hospital, 41333 Göteborg, Sweden): **Platelet Survival and Platelet Production in Systemic Lupus Erythematosus (SLE). (488)**

In 16 patients (3 males and 13 females) with SLE platelet survival and platelet production were determined. At the time of study 3 patients received no therapy, 10 were treated with corticosteroids, and the remaining 3 received corticosteroids and azathioprin. The control group consists of 21 healthy male volunteers. In all experiments autologous platelets labelled with ^{51}Cr were employed.

The mean peripheral platelet count for the SLE patients was 222,000/μl, range 122,000–347,000/μl. In this group the mean for platelet mean life span (MLS) was 6.8 ± 0.3 (S. E.), range 5.5–9.7 days, and did not differ from the mean for the controls (6.9 ± 0.3 days). In the SLE group the mean platelet turnover was $49{,}000 \pm 8{,}000$/μl/day. The corresponding value for the controls was $43{,}000 \pm 3{,}000$/μl/day. The values for platelet MLS and platelet turnover in SLE patients were not related to given therapy.

Previously it has been suggested that a state of compensated thrombocytolysis is present i SLE. Our results could, however, not confirm this.

S. Mitrakul (Bangkok, Thailand): **Platelet Kinetic in Dengue Hemorrhagic Fever. (489)**

In an attempt to reveal certain aspects of pathogenesis of the bleeding disorder in DHF, the studies were carried out in a total of sixty-one children with this disease. As has been shown by others, thrombocytopenia and hypofibrinogenemia were the two most prominent hemostatic defects constantly discovered. Increased consumption in intravascular clotting seemed to be one responsible factor for the hemostatic defect, though not quite outstanding. This was evident by mildly and variably low factor II, VII, IX, X and XII; and mild to moderate increase of FDP, besides low platelets and fibrinogen. Platelet kinetic study, in eleven cases, revealed increased destruction as a main cause for thrombocytopenia, and most probably was by the underlying immunologic mechanism.

G. Gehrmann (Clinicum Barmen, 56 Wuppertal 2, Germany): **Platelet Kinetics in Chronic Alcoholism. (490)**

The pathogenesis of alcoholic platelet depression was studied in twenty patients (average ethanol consumption about 400 g daily). Using ^{51}Cr-labelled platelets, turnover rate, survival time and splenic storage rate were determined and it was found that chronic alcohol intake has a harmful effect on production, destruction and distribution of platelets. The combination and effectiveness of the three pathological mechanisms differs from case to case. A decreased platelet production was, however, most frequent and also proved to te quantitatively the most important mechanism. It, therefore, would seem that alcoholic depression of platelets is largely due to suppression of megakaryocyte production.

R. Dierichs, M. Marcsek and E. Lindner (Universität Regensburg, 84 Regensburg, Germany): **Surface Structures of Human Platelets after Adhesion and Aggregation. (491)**

Human platelets that have adhered, spread out, and aggregated on the artificial surface of an epoxy resin show an intact cellular membrane. After normal fixation and staining a

filamentous material is seen in the extraneous coat which forms intercellular bridges. Incubation with colloidal iron demonstrates the existence of proteoglycans and unsaturated lipid acids. A distinction of proteinous and lipoid components can be achieved by ruthenium red which binds to proteoglycans and to fibrinogen and iodoplatinate which reacts with quarternary ammonium compounds. The latter method delineates the outer lamella of the unit membrane and liberated phospholipids whose relationship to platelet factor 3 is discussed.

T. Tsukada (Toranomon General Hospital, Minato-ku, Tokyo 107, Japan): **Effect of Splenectomy on Platelet Kinetics in Idiopathic Thrombocytopenic Purpura (ITP). (492)**

^{51}Chromium-labeled platelet survival and platelet turnover were studied in ten patients with ITP before and after splenectomy. Prednisolone was administered during the platelet survival study in all cases. Platelet mean survival time (MST) was moderately reduced before splenectomy. Prolongation of MST accompanied an increase of peripheral platelet count in 7 cases after splenectomy. In two cases MST and platelet count did not change significantly after splenectomy although platelet recovery value increased remarkably. Platelet turnover (effective platelet production) was greatly above normal before splenectomy in all cases and reduced to normal range after splenectomy in only two cases. Highly significant linear correlation was noted between MST and peripheral platelet count. But no linear correlation was found between platelet turnover and platelet count ($r = -0.48$).

It was concluded that an increase of peripheral platelet count after splenectomy was mainly due to the prolongation of MST irrespective of the change of platelet production.

J. J. C. Jonker, W. Schopman, L. H. M. v. Riel, J. C. v. d. Steur, W. Stoets and G. J. H. den Ottolander (Municipal Hospital Bergweg, Department of Internal Medicine, Rotterdam): **Platelet Survival Time in Angina Pectoris Treated with Clofibrate or Placebo. (493)**

With platelet survival time (PST) determinations we observed in a group of 20 normal volunteers a platelet half life time ($T^1/_2$) of 99.05 hours. In a group of 35 patients with angina pectoris (A. P.) we obtained a $T^1/_2$ of 84.6 hours ($p < 0.05$).

In a second trial on the effect of Clofibrate in patients with E. C. G. proven A. P., we obtained in 46 patients a $T^1/_2$ of 84.9 hours.

The difference with the "normal" group was again significant ($p < 0.02$). When looking at patients with A. P. + hyperliporoteinaemie (HLP), we found in comparison with the "normal" group in A. P. + HLP type 11A: acc. to Frederickson: $T^1/_2 = 96{,}7$ (ns); in A. P. + HLP type 11B: $T^1/_2 = 88.3$ ($p < 0.2$), in A. P. + HLP type 1V: $T^1/_2 = 82.2$ ($p < 0.02$).

After 6 months treatment with Clofibrate or Placebo there was in the 27 A. P. treated with Clofebrate a significant increase in $T^1/_2$ of 106.8 hours ($p < 0.01$). In the group A. P. + HLP type 11B $T^1/_2 = 90$ (ns) and in the group A. P. + HLP type 1V $T^1/_2 = 94{,}3$ (ns).

In the 27 Clofibrate treated patients cholesterol level decreased from 257 mg% to 217 mg% ($p < 0.001$), the fibrinogen level from 397 mg% to 247 mg% ($p < 0.001$) and the ESR from 22 mm to 14,7 mm ($p < 0.01$). Results and possible explanations will be discussed.

Goro Kosaki (Osaka University, School of Medicine, Osaka, Japan): **Electronmicroscopic Studies on Human Thrombocytogenesis. (494)**

The mechanism of platelet production and platelet release in human bone marrow tissues was studied electronmicroscopically. Promegakaryocytes and megakaryocytes were classified morphologically into 4 stages. Out of the 126 cells studied, 3.2% were in stage I, which was featured by active synthesis of protein and other cellular constituents; 8.2% were in stage II, in which specific granules were produced; 57.9% were in stage III, in which the demarcation membrane was formed; 21.5% were in stage IV, in which platelets were released. Eleven (8.7%) were unclassified. Seventy-three or 75% of the