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