before the 1 mg challenge. Platelet survival in rabbits was also shown to be significantly lengthened when sulphinpyrazone treatment (50 mg/kg/day) was commenced on the same day as the animals received ^{51}Cr-labelled platelets. Both these results suggest that sulphinpyrazone can act rapidly on platelets *in vivo*.

E. H. Mürer, K. Davenport and H. J. Day (SCOR Center for Thrombosis Research, Temple UHSC, Philadelphia, PA 19140): **Platelet Secretion Induced by Calcium Ionophores: Effect of Anti-Inflammatory Drugs. (169)**

Washed human platelets prelabeled with 3 H-serotonin and 14 C-adenine were incubated at 37° C with ionophores A23187 and X537A. 0.1 μM A23187 released the serotonin store of preincubated platelets after 1 min at 37° C, but was less effective when added in the cold. An increase in incubation time at 37° C did not result in increased release. Platelets preincubated with indomethacin showed reduction of up to 85% in released serotonin, while the metabolic parameters 14 C-ATP and 14 C-IMP were not significantly altered. The platelets from some donors did not show reduced release after treatment with indomethacin. This may indicate a variation in sensitivity to the release inducer similar to that described for Sr^{++}-induced release (Biochim. Biophys. Acta *53–59*, 362, 1974), or to the effect of indomethacin. 1 μM X537A caused a time-dependent serotonin release which increased from 2% at 1 min to 58% at 10 min incubation at 37° C. There was little change in 14 C-ATP following release and none in intra- or extracellular 14 C-IMP. 10 μM X537A caused release of 75–80% of the platelet serotonin after 1 min incubation. Longer incubation resulted in 14 C-IMP accumulation and leakage of 14 C-IMP to the surrounding medium. The results do not support the view that X537A and A23187 cause release from platelets by different mechanisms.

(Supported by grant HL 14217 from NHLI. A23187 was a gift from Dr. R. L. Hamill, Eli Lilly and Co., Indianapolis, Ind., and X537A from Drs. Zane Gaut and W. E. Scott, Hoffmann-LaRoche, Inc., Nutley, N. J.)

B. Broussolle, J. F. Stoltz, M. Verry and R. Hyacinthe (C. E. R. B. Toulon, Nancy, Paris, France): **Blood Platelets and Decompression Accidents in the Rat. Therapeutic Trials. (170)**

The pathogenesis of decompression accidents involves particularly disseminated vascular microthrombi composed of platelets clumps, lipids and red blood cells grouped around air microbubbles. In rat, the role of platelets, after rapid decompression (1 bar/20 sec. after being kept at 8 ATA for one hour), is investigated by means of filtration pressure measurements and PRP platelet counts. With decompression accidents, a rise in filtration pressure is observed but there is, above all, a 51% decrease in platelet counts due to disseminated platelet aggregates formed. After slow decompression, there is also a moderate depression (15%) of platelet levels, these not rising until the third day. In order to inhibit platelet aggregation, we used injectable anti-aggregating agents: Aspirin (20 mg/kg) Diamicron (50 mg/kg), S 2574 (100 mg/kg) before and after decompression and for the following three days. The best results were had with S 2574 which prevented fall in platelet levels. Parallely, mortality is reduced if the injection is given before rapid decompression.

These results confirm the importance of platelets in decompression accidents. These therapeutic trials allow to consider the use of these products in human therapy.

P. Barth, R. Zimmermann, J. Zieboll and D. Lange (Med. Univ. Klinik, Germany-W., D-69 Heidelberg, Bergheimerstr. 58): **The Antithrombotic Effect of Acetylsalicylic Acid, Heparin and Phenprocoumon in Experimental Thrombosis. (171)**

The effect of acetylsalicylic acid (ASS) was studied in 450 experiments using 250 rabbits in which thrombus formation by endothelial lesions, hypercoagulability and stasis was standardized and induced to various degrees of severity. The effects of ASS were compared with those of heparin and phenprocoumon singly and in combination with ASS. In the *first series* of experiments in 120 rabbits stasis thrombi were induced according to Wessler