vascular effects in rats. The average hyperglycemic and cardiovascular effects of 8-substituted compounds were lower than those of 6- and 2'0-substituted derivatives despite a high resistance to phosphodiesterase. Four of the tested 8-substituted compounds inhibited platelet aggregation induced in PRP by a standard dose of ADP (3 μM). The most effective was 8-Bromo 3'–5' GMP (50% inhibition of primary aggregation at 0.2–1 mM concentration; N^6, 2'0 Dibutyryl 3'–5' AMP (DB-cAMP): 3–5 mM), which also inhibited ^{14}C serotonin release at lower doses than DB-cAMP. ATP breakdown and hypoxanthine formation induced by exponsure of platelets to ADP was decreased in presence of the compound. Other effective 8-substituted derivatives were 8-mercapto 3'–5' GMP (0.8–1.5 mM), 8-methylmercapto 3'–5' GMP (5–8 mM) and 8-Bromo 3'–5' IMP (5 mM), whereas 8-Bromo 3'–5' AMP and 8-Piperidino 3'–5' AMP were ineffective.

Z. Jerushalmy, J. Pinkhas, M. Krinsky and A. de Vries (Beilinson Medical Center, Petah Tikva, Israel): **Effect of Methotrexate and Vincristine on rat Bone marrow Megakaryocytes and Platelet Function. (67)**

Methotrexate administered intraperitoneally to rats caused a transient disturbance in platelet function manifested by absence of ADP-induced aggregation and serotonin incorporation, and by decreased factor 3 (PF3) availability. These phenomena were noticed when the megakaryocyte count decreased while the platelet count was still within the normal range.

Vincristine injection was not followed by an immediate drop in megakaryocyte and platelet count or PF3 availability, however a transient decrease of ADP-induced platelet aggregation and serotonin incorporation occurred.

K. Zawilska, M. Komarnicki and B. Manka (Ul-Bzierzynskiego 161–61, Poznan, Poland): **Effect of Beta Blockers on Human Blood Plaletets in Vivo. (68)**

ADP and collagen-induced platelet aggregations are diminished one hour after propanolol administration to normal subjects while adrenalin-induced aggregation and platelet factor 3 availability are not influenced. This effect of propanolol in vivo is very different from its in vitro action and is possibly related to the interaction adrenalin-ADP and collagen-ADP. Intermediary products of propanolol metabolism may also be involved in this effect.

The administration of practololol to a second group of normal subjects had no effect on platelet aggregation and on platelet factor 3 availability.

H. Losonczy, I. Nagy, Z. Gregus and I. Szaksz (I. st. Dept. of Int. Med. University Pécs, 7643. Hungary): **The Effect of Benzcyclan and Theophyllin on the ADP Induced Platelet Aggregation in Vitro and in Vivo. (69)**

Autors performed in vitro examinations on platelet-rich plasma of healthy individuals with the methods of Hovig and Born. They found that complete inhibition was caused by Benzcyclan at 6×10^{-4} Mol cc. and by Theophyllin at a cc. of 10^{-3}. Then they studied the combined effect of the two drugs. They found that complete inhibititon occured at a concentration level at which the drugs in question could only cause a slight inhibition when applied separately. When applied in vivo the 100 mg Benzcyclan administered intravenously was found to have an inhibitive effect reaching the maximal effect within 90–120 min. only a slight inhibition was observed when 250 mg Theophyllin was administered intravenously reaching its maximal effect in 30–60 min. At simultaneous administration inhibition was first observed in 15 min. The maximal effect was reached in 60 min., but some effect could still be detected 180 min after administration and the extent of the inhibition was significantly greater than in separate application. Authors discuss this synergy observed at both in vitro and in vivo examinations, its theoretic background and therapeutic significance.