VARIABILITY OF PLATELET FUNCTION RESPONSE TO ASPIRIN. M. Aris, M.D. Drew Postgraduate Medical School, University of California, Los Angeles, California, U.S.A.

To evaluate the effect of aspirin on platelet function, three groups of subjects were studied. Platelet adhesiveness (pl. adh.) was determined by the Hemm glas test retension method, and platelet aggregation (pl. agg.) by the Born turbidimetric method using standardized collagen solution. In 8 healthy volunteers, the mean pl. adh. decreased insignificantly to 15.2 ± 2.4 after 12 ± 2 days of 300 mg/day of aspirin. They showed slight to moderate impairment of platelet aggregation. In 16 patients with massive gastrointestinal bleeding, following ingestion of aspirin, the mean pl. adh. was 22 ± 5 which is significantly lower (P<0.01) than for the normal individuals. These patients showed poor to no platelet aggregation. By contrast in 14 patients with rheumatoid arthritis, despite being on large doses of aspirin, the mean pl. adh. was 61 ± 15 which is significantly higher (P<0.05) than for the non-arthritis. In 8 of these patients pl. agg. was also measured. They showed no or slight impairment. These observations provide a plausible explanation for the conflicting reports regarding efficacy and complications of aspirin and emphasize the need to differentiate between its anti-inflammatory and antithrombotic effects by actual determination of platelet function in patients treated with aspirin.

INHIBITORY EFFECT OF DIAMINES AND POLYAMINES ON HUMAN PLATELET AGGREGATION AND [14C]-SEROTONIN RELEASE REACTION. K. Subbarao and P. Forsenier. Temple University School of Medicine, Philadelphia, Pa., U.S.A. and Hôpital Notre-Dame de Bon Secours, Paris, France.

Physiological diamines and polyamines occur in high concentrations in various parts of animal tissues. These amines are known to interact with and stabilize nucleic acids, membranes and ribosomes (Tabor and Tabor, Pharmac. Rev., 16, 265). The effect of putrescine, cadaverine, spermidine and spermine on platelet function is not yet fully explored. We studied the effect of these substances on in vitro aggregation of human platelet rich plasma (PRP) induced by the addition of ADP, thrombin, collagen and serotonin. Cadaverine, spermidine and spermine at concentrations from 5-50 μM strongly inhibited the aggregation of platelets and the [14C]-serotonin release reaction induced by ADP and thrombin in a concentration dependent manner, but did not show any effect on aggregation induced by other agonists. Putrescine, on the other hand, failed to produce any effect on the aggregation of platelets and [14C]-serotonin release reaction. Studies on the binding of purified human thrombin created with [14C]-dinitropropyldichloroacetate to washed human platelets indicated that cadaverine (1-5 μM) increased the binding of total [14C]-DPF-thrombin to platelets by 30%. The data suggest that the alteration of platelet function by diamines and polyamines was probably achieved by their binding to platelet membranes.

TWO DIMENSIONAL IMMUNOELECTROPHORETIC PATTERNS OF ALPHA2 MACROGLOBULIN IN SUBJECTS WITH AND WITHOUT THROMBOTIC TENDENCIES. Mabel M. Stevenson, Ann G. Davidson, and Thomas J. Tallman.

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In an attempt to determine whether compensatory changes in alpha2 macroglobulin occur in patients with thrombotic tendencies, studies were performed by the two dimensional immunoelectrophoretic technique of (ancone) on the plasma of healthy laboratory workers and patients with possible thrombotic tendencies. A single, broad, asymmetrical peak was noted in 16 of 19 laboratory workers. A slower moving derivative second spike of lesser magnitude was found in 2 of the 16. A patient with two peaks appeared in 11 of 14 patients on warfarin therapy for recurrent thrombophlebitis (8 of 8), prophylaxis after artificial heart valve replacement (2 of 3), and coronary revascularisation (1 of 3). Antithrombin III patterns studied according to Sni were normal in all but 2 of these patients. A volunteer ingesting warfarin was normal. A single patient with liver disease showed two peaks.

Preliminary in vitro incubation of plasma with thrombin, plasmin, or streptokinase failed to create more than one peak.

These preliminary data suggest changes in alpha2 macroglobulin may represent in vivo coagulation and might provide another means to identify thrombogenic prone individuals.