

AN EX VIVO TEST OF PLATELET FUNCTION CORRELATES WITH TEMPLATE BLEEDING TIMES AND IS UNAFFECTED BY SYMPTOMS OF PATIENTS WITH THROMBOCYTOPENIA. M. McGill, L.E. Quiroga and R.A. Sacher. Hoxworth Blood Center and Department of Pathology and Laboratory Medicine, University of Cincinnati, and Division of Hematology, Georgetown University, Washington, D.C., U.S.A.

The template bleeding time is used extensively for measurements of in vivo platelet function in thrombocytopenic patients and for evaluations of platelet transfusions. Interpretation of results is complicated by technical difficulties of the test procedure and by patient symptoms including sepsis, fever, splenomegaly, alloimmunization and drug therapy. In an attempt to find additional tests which predict function of circulating platelets, ex vivo measurements of platelet reactivity to vessel wall subendothelium, and template bleeding time tests, were performed on normal individuals and a series of hospital patients. Platelet reactivity in controls was 7.2 ± 3.3 μ m (n=10) and bleeding times were 3.1 ± 0.3 min. Reactivity decreased to 2.6 ± 0.6 μ m and bleeding times increased to 7.6 ± 3.6 min. in aspirinated controls (n=12). Patients with thrombocytopenia and prolonged bleeding times (n=15) consistently gave reduced reactivity values. Thus, the direct, inverse relationship between in vivo function (bleeding time) and ex vivo function (platelet reactivity) support the idea that the laboratory test is a valid measurement of platelet capabilities for function in vivo. Unlike bleeding time tests, ex vivo measurements of reactivity can be performed apart from patient symptoms which complicate measurement of function in vivo and under controlled conditions.

PLATELET HYPERSENSITIVITY IN ACUTE MALARIA INFECTION (PLASMODIUM FALCIPARUM) IN MAN. E.M. Essien & M.I. Ebhota. Department of Haematology, University College Hospital, Ibadan, Nigeria.

We had earlier reported altered ADP-induced platelet aggregation in man during acute malaria infection. The present study sought to determine (i) whether the changes suggested platelet hypersensitivity to ADP and (ii) whether such changes occurred in vivo or in vitro.

The aggregation response of platelets (as citrated PRP) to addition of ADP from thirty patients with acute malaria infection has been compared with that of 29 control i.e., non-infected subjects. The age range of the subjects in both groups varied from 2 to 70 years. These tests were performed before the patients took any drugs. With addition of 1.0μ M ADP to 1 ml of PRP, the mean aggregation amplitude (as % light transmission) obtained from 8 patients, $39.8 \pm 27.1\%$ was significantly greater than that from 9 control subjects ($5.2 \pm 6.7\%$; $t = 3.51$; $P < 0.005$). With higher ADP concentrations ($2.4 - 5.0 \mu$ M) similar response in 22 subjects (mean $89.1 \pm 14.9\%$) was also significantly greater than that in 20 controls ($77.8 \pm 16.5\%$; $t = 12.45$; $P < 0.02$). These results suggest that during acute malaria infection in man, circulating platelets become hypersensitive to ADP in vitro. No instances of spontaneous aggregation were however observed in the patients.

BTG was determined in 7 patients and 6 controls. The mean plasma BTG in the patients (208.3 ± 15.6 ng/ml) was significantly higher than that in controls (59.2 ± 15.7 ng/ml; $t = 13.44$; $P < 0.001$). These latter results suggest that the platelets were probably activated in vivo to release the BTG. They further suggest that the hypersensitive changes noted earlier also probably occurred in vivo.

It is suggested that acute malaria (*P.falciparum*) infection in man is probably another clinical condition associated with platelet hypersensitivity.