

PLATELET SURVIVAL MEASUREMENT - CLINICAL RESULTS. E.Genton. Faculty of Health Sciences McMaster University, Hamilton, Ontario, Canada.

Platelet survival (PS) time has been studied in patients in a variety of clinical circumstances associated with abnormal numbers of platelets, suspected thrombotic process, or after platelet suppressing drug administration.

In conditions with thrombocytopenia, PS may differentiate decreased platelet production (ineffective thrombopoiesis or megakaryocyte hypoplasia) where PS is normal from increased destruction of platelets where PS is shortened. Increased destruction may arise from: extrinsic mechanisms, e.g. immunologic (ITP-SLE-drug reactions) or abnormal surface (diseased endothelium or foreign surface) are associated with short PS, often to extreme degrees; intrinsic platelet defect (Wiskott-Aldrich Syndrome), where autologous PS is shortened and isologous PS is normal.

In thrombotic disorders, PS is shortened during active thrombosis and may be chronically shortened in conditions with recurrent thrombosis (homocystinemia, atherosclerosis, valvular heart disease). The test may prove useful in prognosis (e.g. valvular heart disease patients with shortened platelet survival may have higher risk of embolic events than with normal PS).

Only a few platelet suppressing drugs (including sulfinpyrazone and dipyridamole) affect a shortened platelet survival time. These reduce thrombosis in patients with prosthetic heart valves and silastic AV shunts, suggesting PS may identify useful drugs.

Thus, PS may differentiate disease conditions associated with abnormal platelets, predict the course of patients at high risk of thrombotic complications, and identify clinically useful platelet suppressing drugs, or serve as a monitor for measuring effects of drug treatment.

THE CLINICAL INTERPRETATION OF PLATELET SURVIVAL TIME IN THROMBOTIC PLATELETS. J.J.C.Jonker, L.H.M.van Riel, W.Schopman, G.J.H.den Ottolander. Municipal Hospital Beragweg, Rotterdam, Holland.

We obtained experience in 800 determinations of platelet survival time (PST) with a modified method according to Aster and Jandl. In 20 normal volunteers we obtained a mean $T_{1/2}$ of 99 hours with a S.D. of 13.4 hours. The reproducibility of the method was obtained from two PST measurements with an interval of one week. The S.D. of replication was 13.4 percent. The reproducibility of PST over a long period of time (18-30 months) was determined from patients of the placebo group of a trial on patients with angina pectoris. Compared with the PST at entry in 72 patients the mean standard deviation in at least three determinations was 14.5%. Each single PST has a range of 90% reliability ($T_{1/2} \pm 2 \times \text{S.D.}$) depending on the correlation factor between disappearance in comparison with time. Our mean correlation factor (R) is 0.92 with a range of 0.85-1.00. A single PST with a R of 0.92 has an S.D. of 15%, meaning that there is a significant difference between two PST determinations in a single individual when there is more than 30% difference. At entry 30% of angina patients has a shortened PST. After six months there was an increase of the PST in the clofibrate treated group. After 18 months however there was also an increase in the placebo group. This means that no trial of drug effects on platelet survival can be accepted which are lacking a double blind setting and a control group.

HAEMOSTATIC FINDINGS IN MEN WITH AND WITHOUT ISCHAEMIC HEART DISEASE. T.W.Meade, R. Chakrabarti, A.P. Haines, W.R.S. North and Y. Stirling. MRC-DHSS Epidemiology and Medical Care Unit, Northwick Park Hospital, Harrow, U.K.

The main purposes of the prospective Northwick Park Heart Study (NPHS) are to improve the prediction of arterial disease, especially ischaemic heart disease (IHD), and to elucidate its pathogenesis. Some 1800 men and 800 women have so far been recruited. Measures of haemostatic function are made along with those of variables (E.G. cholesterol) already known to be associated with IHD; all participants are followed up. Of the first 873 white men aged 40-64 at entry, 18 had already had a "definite" myocardial infarction (MI) at least 2 years previously, and 18 had "definite" angina pectoris (AP). Recruitment values for factors II, V, VII, VIII and X, fibrinogen, antithrombin III, α_2 -macroglobulin, fibrinolytic activity, platelet count and platelet adhesiveness are compared with similar values in those free of clinically manifest IHD and with normal electrocardiograms (ECGs). Those with MI had significantly higher levels of fibrinogen, and platelet adhesiveness, and significantly lower fibrinolytic activity than those without IHD and normal ECGs; their factor VII and VIII levels were also higher. Changes in those with AP were similar, though not as great as in those with MI. There were no differences in those without clinical manifestations of IHD but with ischaemic ECGs. IHD prevalence data, based on results after the clinical event, generally give less striking contrasts than incidence data (which NPHS will eventually provide), in which recruitment findings are related to the subsequent onset of disease. Nevertheless, the prevalence findings are compatible with the concept of a "hypercoagulable state" in which thrombosis is the main complication of atheroma, and responsible for many of the clinical manifestations of IHD.