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COMPARISON OF MEAN PLATELET SURVIVAL TIME IN A NON-RADIO-ISOTOPE METHOD CALCULATEND WITH NEW MODEL WITH MEAN SURVIVAL TIME IN CR-51 METHOD. T.Tsukada, T.Tango* Division of Hematologic Research, Toranomon Hospital, Tokyo. *Division of Clinical Epidemiology, Tokyo Metropolitan Institute of Medical Science, Tokyo, Japan.

Platelet survival time using non-radioisotope method measuring malondialdehyde(MDA) generation stimulated by arachidonate prior to and after intake of aspirin(ASA) and Cr-51 method was measured simultaneously in 18 cases with ITP. In cases with normal platelet survival time MDA generation curve showed linear or the letter-S pattern. On the other hand, all cases with shorter survival time had MDA generation curve with letter-S pattern. To describe the S pattern of MDA generation curve following model was devised on the assumption : 1) ASA inhibits MDA generation in megakaryocytes so that newly produced platelets show impaired MDA generation, 2) ability of MDA generation in platelets exposed to ASA recovers during circulating.

$$P(t) = 1 - (\frac{t}{a})^m (1-e^{-\lambda t}) - (\frac{t}{b})^n (e^{-\lambda t})$$

where a is the disappearance time of inhibiting effect of ASA on megakaryocyte MDA generation and b is the time necessary for recovery of MDA generation in platelets exposed to ASA. Mean survival time(MST) is calculated by $1/\lambda$.

The difference of MST between non-radioisotope method and Cr-51 method(gamma model) in 11 cases with MST of less than 4 days in Cr-51 method were 2.9 ± 0.4 days in platelet survival time(PST) at which MDA generation attained to pre-ASA levels, 2.7 ± 0.4 days in MST calculated with linear regression of MDA generation curve and 1.8 ± 0.4(SEM) days in MST calculated by the above model. On the other hand, in cases with MST of more than 4 days in Cr-51 method the difference of MST in both methods were 1.4 ± 0.3(SEM) days in PST, 0.6 ± 0.2 days in linear regression and 0.6 ± 0.2 days when MST was calculated with the above formula.

These results suggest that the model reported is so far the better fitting model for the MDA generation curve in cases with normal and shortened platelet survival.

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KINETICS, REDISTRIBUTION AND FATE OF INDIUM-111-LABELLED-PLATELETS IN PATIENTS WITH AORTIC ANEURYSMS. A. duP. Heyns, M.G. Lötter, P.N. Badenhorst, F. de Kock, H. Pieters, C. Herbst and C.J.C. Nel. M.R.C. Blood Platelet Research Unit, and Department of Surgery, University of the Orange Free State,Bloemfontein, South Africa.

Platelets of 7 patients with abdominal aortic aneurysms were labelled with In-111-oxine prior to surgery. The platelets were reinjected with the patient positioned under a scintillation camera with a computer assisted imaging system. Images were acquisitioned daily, areas of interest selected with the computer, organ radioactivity-quantitated with a geometrical mean method and expressed as a percentage of whole body radioactivity. Platelet survival (PS) in the circulation was determined, and disappearance curves fitted to a gamma function "multiple hit" model.

Mean PS was shortened to $143,2 \pm 47h$ (normal 232 ± 17); the dissappearance curves were exponential in all but the two patients who had PS within normal limits. Redistribution and sites of destruction of labelled platelets were as follows (normal values in parenthesis):

	Aneurysm	Liver	Spleen	Whole body
Equilibrium	$2,0\pm1,2$	$11,9\pm1,6$	$29,4\pm9,8$	100
	( )	$(13,1\pm1,3)$	$(33,7\pm8,8)$	(100)
Final	$5,1\pm3$	$22,1\pm6,3$	$30,2\pm9,9$	$91,7\pm7$
	( )	$(32,4\pm7,2)$	$(44,5\pm16,4)$	(98 ± 1)

The surgically removed aneurysms were dissected and radioactivity of different layers measured. In-111-activity was confined to the superficial layers of the aneurysm.

These techniques allow quantitative studies of the in vivo distribution of labelled platelets. Platelets are deposited in the aneurysms, this shortens PS, the disappearance curves become exponential, and the major sites of deposition of In-111-activity are in the liver and spleen. This indicates that although platelets are damaged and deposited in the aneurysm, the reticuloendothelial system remains a major site of platelet sequestration.

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CIRCULATING PLATELET FACTORS (BETA THROMBOGLOBULIN, PLATELET FACTOR 4) IN PATIENTS WITH PROSTHETIC HEART VALVES. Andrassy, K., Zebe, H., Koderisch, J., Duczek, A., Ritz, E., Med. Univ.-Klinik Heidelberg (FRG)

Despite anticoagulation, thromboembolic complications are frequent in patients with heart valve prosthesis (HVP). The present study was performed to investigate whether activation of platelets could be demonstrated in such patients. Platelet proteins in the circulation were measured as an index of platelet activation. Pat.: 33 pat. with mitral valve prosthesis (MVP) (19 with Bjǿrk-Shiley (BS) and 14 pat. with Starr Edwards type (SE)) and 54 pat. with aortic valve prosthesis (35 BS; 10 SE; 9 bioprosthesis (BP). All pat. were anticoagulated (Dicumarol). Results: In MVP, a significant difference between BS and SE was observed with respect to LDH ($247^{\pm} 48$ vers. $474^{\pm} 246$) (p< 0.05) but not with regard to Beta-TG and PF 4. There was no difference of platelet protein levels in presence/absence of atrial fibrillation. In 35% Beta TG (> 53 ng/ml) and PF 4 (> 11 ng/ml) were above $\bar{X}^{\pm}2$ SD of CO. ADP and collagen induced aggregation (MA) was unchanged in all pat. (collagen $40^{\pm} 12$; ADP $32^{\pm}11$; CO: collagen $39^{\pm}7$; ADP $32^{\pm}6$). In pat. with demonstrable hemolysis (LDH > 250 IU), a significant correlation was observed between LDH and Beta TG/PF 4 both in MVP and AVP. MVP and AVP differed with respect to LDH (higher in AVP, p< 0.05) and PF 4 (higher in MVP (p<0.01) but not with respect to Beta TG (differences of elimination of TG and PF 4?). Comment: In a high proportion of patients with HVP platelets are activated. The observation of elevated platelet indicator proteins is in agreement with previous findings of decreased platelet survival in HVP (Weily, H., New Engl. J. Med. 290, 534, 1974). Elevated platelet prot.may identify risk of thromboembolism.

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PLATELET SURVIVAL IN PATIENTS WITH AORTIC DACRON PROSTHESES TREATED WITH TICLOPIDINE. J.A. Davies, H. Tindall, R.C. Paton, R.L. Doig, R.C. Kester and G.P. McNicol. University Departments of Medicine and Surgery, Leeds, U.K.

Ticlopidine inhibits platelet function ex-vivo and prolongs the bleeding time. We investigated its anti-thrombotic potential in patients with extensive peripheral vascular disease by measurement of survival of $Na^{51}Cr$-labelled platelets before and following two months treatment with ticlopidine, 250mg twice daily. Platelet survival in six patients with aortic Dacron prostheses and one who had undergone aorto-iliac endarterectomy was 8.04 ± 0.14 days (mean $\pm$ SEM) calculated by linear regression and 7.08 ± 0.71 days by gamma function analysis. This is considerably shorter than the mean normal value of 9.98 ± 0.22 days for platelet life-span established in 40 normal subjects by the aspirin-labelling technique, which correlates very closely with survival time of $Na^{51}Cr$-labelled platelets in patients. During the eighth week of treatment with ticlopidine measurement of survival of Na^{51}-Cr-labelled platelets was repeated. Mean platelet survival was not affected by ticlopidine treatment, with survival times of 8.12 ± 0.24 days by linear regression and 7.0 ± 0.54 days by gamma function analysis. Tablet counts indicated that patients complied with the treatment regime and single random blood sampling indicated that adequate plasma concentrations of ticlopidine were attained. It is concluded that ticlopidine 250mg twice daily does not prolong shortened platelet survival in patients with Dacron prostheses.