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IRON ABSORPTION IN IRON DEFICIENCY STATUS.
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Iron deficiency is caused by the increase of iron loss, decrease of iron absorption, abnormal eating habbit and etc. Iron loss is mainly caused by bleeding and the amount of blood loss is determined by using Cr-51-RBC. Iron absorption is determined by using a whole body counter measuring 14 day whole body retention after oral Fe-59 dose. Iron deficiency status is consist of simple iron deficiency anemia(IDA), iron deficiency with complication(IDA+c), iron deficiency without clinical symptom(ID), and etc. Among these iron deficiency status, iron absorption was increased in IDA, less in IDA+c and it was between IDA and normal in ID. No significant difference was observed between normal male and female in iron absorption, although storage iron was lower in normal female than male.
In general, iron absorption was mainly controlled by the amount of storage iron and effected by hematopoietic activity.
Iron absorption delivers the important informations for the diagnosis of iron deficiency status and for choosing the route ,oral or intravenous, of iron administration in treating the patients with IDA and iron deficiency tissue disorders.

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THROMBOKINETICS COMBINED WITH RES FUNCTION ASSESSMENT IN CHRONIC I T P.
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Thrombokinetics( PlK ) with Cr-51 or In-111 oxine labeled platelets was carried out in 28 chronic ITP patients in association with splenic RES function study using Tc-99m labeled, IgG coated( D-R ) and/or Cr-51 labaled NEM-treated( N-R ) red cells. With the values of effective survival in PlK and extraction ratio( ER ) in D-R and/or N-R clearance, the subjects were classified in four groups.
In group A with remarkably reduced platelet survival( ESv ) and suppressed ER, patients demonstrated poor response to splenectomy(SpX) or high-dose immunoglobulin therapy( IG ).
In group B with remarkably reduced ESv but nearly normal ER, better response to steroids ( STH ) and SpX and much better one to IG developed than those observed in group A.
In group C with slightly reduced Esv and nearly normal ER, the response was fairy well to SPX but relatively poor to STH and IG.
In group D, slightly reduced ESv with remarkably suppressed ER were considered to reflect suppressed state of RES by STH or IG, where post effective SpX cases could be included.
Actual platelets' survival reflects severity of their autosentitization relative to RES' destruction capacity. Thus, thrombokinetics study combined with RES' function assessment especially with D-R for macrophage Fc-receptor mediated extraction was useful for evaluation of the severity of the disease in reference to therapeutic effect

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PLATELET KINETICS BY RADIOISOTOPE LABELED PLATELETS IN PATIENTS WITH SPLENOMEGALY.
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Platelet kinetics by radioisotope labeled platelets were investigated in patients with splenomegaly. Five cases of chronic myelogenous leukemia,2 cases of essential thrombocythemia as a group of myeloproliferative disorders with splenomegaly,2 cases of hereditary spherocytosis as a group of hyperplastic splenomegaly,3 cases of liver cirrhosis, 3 cases of so-called Banti's syndrome as a group of congestive splenomegaly were studied. Platelets were labeled by the methods of International Committee for Standardization in Hematology for Cr-51, Thakur et al for In-111-oxine,Dewanjee et al for In-111-tropolone respectively.Characteristic features of platelet kinetics in all patients with splenomegaly were increased platelet pooling in the spleen with normal platelet life span,very low recovery of labeled platelets,and marked accumulation of labeled platelets in the spleen observed by scintillation camera. Platelet counts and platelet production calculated from platelet kinetic data were different between each group of splenomegaly. Platelet production were markedly accelerated in myeloproliferative disorders,moderately accelerated in hereditary spherocytosis,normal in so-called Banti's syndrome and low in liver cirrhosis.
In conclusion,increased platelet pooling in the spleen were commonly observed in various disorders with splenomegaly.

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EVALUATION OF WARFARIN THERAPY ON THE INTRACARDIAC THROMBOGENECITY BY IN-111 SCINTIGRAPHY.
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Seventeen patients in whom intracardiac thrombi were detected by indium-111 platelet scintigraphy (the first platelet scintigraphy) were prospectively studied to examine the effect of mild-dose warfarin on the intracardiac thrombogenecity. Eleven patients (group I) who received 2-6 mg/day of warfarin and six patients (group II) who did not receive warfarin had the second platelet scintigraphies 14-71 days after the first platelet scintigraphies. In group I, ten platelet scintigraphies became negative and one remained positive for intracardiac thrombi after administration of warfarin, while in group II, five platelet scintigraphies remained positive and only one changed to negative. The incidence of negative image at the second platelet scintigraphy was significantly lower in group II than that in group I.
These results indicated that mild-dose warfarin inhibited the deposition of platelets on the intracardiac thrombi and thrombogenecity in the patients with intracardiac thrombi which were detected by indium-111 platelet scintigraphy.