CHANGES BROUGHT ABOUT IN BLOOD COAGULATION IN THE NEWBORN BY ADMINISTRATION OF VITAMIN K₂.
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Deficiency of the blood coagulation factor, especially of vitamin K-dependent factors, is a major disturbance of extrauterine life in the neonatal period. It is well known that vitamin K₁ is converted to vitamin K₂ in the human body. In order to compare the effectiveness of vitamin K₁ and that of vitamin K₂ in the newborn period, changes in coagulability were studied not only at the end of the day after birth but also along the time course. Six hrs after birth, 2 mg of vitamin K₁ or vitamin K₂ was injected intramuscularly into the thigh of the newborn, and the effects were observed by thrombotest and normotest 3, 15, and 24 hrs after injection. In this experiment, the infants treated with vitamin K₂ showed higher values in the thrombotest than those treated with vitamin K₁ after 3 and 15 hrs. After 24hrs however, there was no longer any significant difference between the K₁ and K₂ groups.

As a clinical application of vitamin K₂, gastrointestinal bleeding was studied polarographically with the emulsion of meconium or stool. Since the origin of vitamin K₂ is the gastrointestinal bacterial flora, changes in the occult blood were studied in one group given 2 mg of vitamin K₂ and mixed milk (18 cases), a second group given 2 mg of vitamin K₁ and mixed milk (21 cases), and a control group (24 cases). Haemoglobin percentage of newborns are greatly diminished by a oral administration of vitamin K₂. Both results may indicate rapid action of vitamin K₂.

ABNORMAL PLATELETS IN FULMINANT HEPATIC FAILURE. Weston, M.J.⁺, Rubin, M.H.⁺, Bullock, G., Roberts, J., Langley, P.G.⁺, White, Y.⁺, Williams, R.⁺. ⁺Liver Unit, King's College Hospital, London, England and ⁺CIBA Laboratories, Horsham, England.

Because bleeding occurs in fulminant hepatic failure, platelet function was investigated. In 12 patients, platelet counts were slightly lower than in controls (197 and $255 \times 10^9/l$, $p < 0.05$), platelet volumes were smaller (5.89 and $6.68 \mu^3$, $p < 0.001$) and bleeding times were double (15.9 and 7.6 minutes, $p < 0.001$). Patients' platelets required ten times more ADP than controls to reach second phase aggregation (21.4 and $2.2 \times 10^{-6}M$, $p < 0.001$). Abnormalities of aggregation correlated with the prolonged bleeding times. Patient platelet poor plasma (PPP) did not prevent aggregation reaching second phase in controls and PPP from controls did not facilitate second phase aggregation in patients' platelets. These defects of platelet function in fulminant hepatic failure are related to the severity of hepatic necrosis as platelet function in 16 jaundiced but non comatose patients did not differ from controls. Abnormalities of platelet ultrastructure are presented in an accompanying abstract.

ANTI-HEPARIN AND PLATELET AGGREGATION ACTIVITIES OF POLYAMINO ACIDS. Jawad Fareed, Harry L. Messmore, John U. Balis and Rogelio Moncada. Loyola University Medical Center, Maywood, Illinois, U.S.A.

An earlier report from this laboratory has described the antagonism of the anticoagulant effect of heparin by certain basic polyamino acids. Of the numerous polyamino acids tested, only basic polyamino acids such as poly-L-lysine (MW 85,000) and poly-L-ornithine (MW 120,000) effectively neutralized heparinized plasma (1 u/ml) in concentrations less than 10 $\mu g/ml$. Addition of these two polyamino acids in quantities up to 50 $\mu g/ml$ citrated plasma, significantly shortened the thrombin time. Poly-L-proline (MW 19,000), poly-L-histidine (MW 16,000) and poly-L-lysine (MW 85,000) possessed weak anti-heparin action. These polyamino acids also neutralized the anticoagulant activity of hirudin and polyanetholsulfonic acid in varying degrees. The effects of polyamino acids on platelet aggregation was also tested. Of the 15 basic polyamino acids tested, only poly-L- α -ornithine was found to induce aggregation of platelets. Polyornithine in the amounts of 50.0, 25.0 and 12.5 $\mu g/ml$ to platelet rich plasma caused a 65, 45 and 30% change in transmittance, respectively. The polyornithine induced aggregation (PIA) of platelets was only partially blocked by acetylsalicylic acid. Contrast media (6.0 mg/ml) used in diagnostic radiology and meglumine (5 mg/ml) totally blocked the PIA. The PIA of platelets was found to be a biphasic process, an initial lag time of 30 seconds, after which irreversible aggregation was observed. These studies suggest that basic polyamino acids may be used clinically to antagonize overheparinization; in addition, polyornithine may prove useful in the diagnosis of platelet function defects.