CHANGES Brought ABOUT IN BLOOD COAGULATION IN THE NEONATE BY ADMINISTRATION OF VITAMIN K3.
S. SUGUI, K. ISHINO (Department of Obstetrics & Gynecology, University of Hokkaido, School of
Medicine, Sapporo, JAPAN)

the blood coagulation factor, especially of vitamin K-dependent factors is a
major disturbance of extraneous life in the neonatal period. It is well known that vitamin
K3 is converted to vitamin K1 in the human body. In order to compare the effectiveness of
vitamin K3 and that of vitamin K1 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 K3 or vitamin K1 was injected intramuscularly into the thigh of the
newborn, and the effects were observed by thromboplast and Dornotest 3, 15, and 24 hrs after
injection. In this experiment, the infants treated with vitamin K3 showed higher values in the
thromboplast than those treated with vitamin K1 after 3 and 15 hrs. After 24 hrs however,
there was no longer any significant difference between the K3 and K1 groups.

As a clinical application of vitamin K3, gastrointestinal bleeding was studied polargraphi-
cally with the aim of meconium or stool. Since the origin of vitamin K3 is the
gastrointestinal bacterial flora, changes in the occult blood were studied in one group given
2 mg of vitamin K3 and mixed milk (18 cases), a second group given 2 mg of vitamin K3 and
mixed milk (22 cases), and a control group (24 cases). Hemoglobin percentage of newborns
are greatly diminished by the oral administration of vitamin K3. Both results may indicate rapid
action of vitamin K3.

ABNORMAL PLATELETS IN FULMINANT HEPATIC FAILURE. Weston, H.J. 1, 2, Rubin, M.H. 1, Bullock, G.,
Roberts, J., Lasonoy, P.C., White, R., Williams, R. 1, Liver Unit, King's College Hospital,

Because bleeding occurs in fulminant hepatic failure, platelet function was investigated. In
12 patients, platelet counts were slightly lower than in controls (187 and 285 x 10^9/l, p<0.05),
platelet volumes were smaller (5.80 and 6.60 fL, p<0.001) and bleeding times were double (15.9
and 7.4 minutes, p<0.001). Patients' platelets required ten times more ADP than controls to
reach second phase aggregation (21.4 and 2.2 x 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 structure are presented in an accompanying abstract.

ANTI-HEPARIN AND PLATELET AGGREGATION ACTIVITIES OF POLYAMINO ACIDS. Javed Farooq, Barry H. 
Meerson, John U. Balie and Rosario Menchola, Loyola University Medical Center, Maywood,
ILLINOIS, U.S.A.

An earlier report from this laboratory has described the antagonism of the anticoagulant
effects 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 mg/ml.
Addition of these two polyamino acids in quantities up to 50 mg/ml citrated plasma, significant-
ly 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 pentamethyldisulfone 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-ornithine was found to induce aggregation of platelets.
Polyornithine in the amount of 50, 50.0 and 12.5 µg/ml to platelet rich plasma caused a 65,
45 and 30% change in transmittance, respectively. The polyornithine induced aggregation (PPA)
of platelets was only partially blocked by acetylsalicylic acid. Contrast media (6.0 mg/ml) used
in diagnostic radiology and magnesium (5 mg/ml) totally blocked the PDA. The PPA 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 overaggregation. In addition, polyornithine may prove useful in the diagnosis
of platelet function defects.