HAEMOSTATIC MECHANISM IN MATERNAL UMBILICAL VEIN AND UMBILICAL ARTERY AT THE TIME OF DELIVERY IN NORMAL AND ABNORMAL PREGNANCIES

by

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Bonnar *et al* (1970 and 1971) demonstrated that in normal pregnancy activation of coagulation and fibrinolytic enzyme system has been found in blood draining uterus at placental separation. They also reported decrease in clotting factors and increase in fibrinolytic activity in umbilical vein blood immediately after delivery. Also the coagulation mechanism is altered in cord blood at delivery and may reflect levels of factors in newborn.

The umbilical vein platelets have decreased function as compared to maternal platelets (Hathaway, 1975) umbilical artery blood is more likely than umbilical vein blood to reflect the haemostatic state of baby in utero during labour and in early neonatal period as umbilical artery blood is less influenced by placental separation.

The decrease in potential haemostatic activity thus observed in foetus seems to have beneficial effect on the well being of the foetus in utero, thereby any alteration may be an effect on foetal growth and development. The maintainance of patient's arterial and venous system is essential for placental perfusion and its attraction to speculate the increased fibrinolytic activity in foetus may be an important contributory factor.

Study of haemostatic mechanism was also carried out in pregnancy complicated with toxaemia to delineate the evolution of changes in components of haemostatic mechanism at birth, to determine whether those who developed pre-eclampsia and eclampsia could be differentiated from those with uncomplicated pregnancy by measurable alteration in haemostatic mechanism.

Keeping all these facts in mind, it was decided to study some of the components of haemostatic mechanism in maternal, peripheral, umbilical vein and umbilical artery blood at the time of delivery to define more clearly the changes in normal pregnancy and those complicated with pre-eclampsia and eclampsia.

Materials and Methods

Eighty-one cases were studied for the components of haemostatic mechanism during pregnancy and labour. The cases were grouped as follows:

1. Normal pregnancy 50 cases.
2. Pregnancy complicated with pre-eclampsia 20 cases.
3. Pregnancy complicated with eclampsia 11 cases.

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Collection of Samples: 5 CC of blood was drawn from anticubital vein from maternal side within 30 minutes of delivery and mixed with anti-coagulant contained in a sterilized test tube. Cord blood was collected between delivery of baby and placental expulsion. The samples were collected from the umbilical arteries and vein and mixed with anti-coagulant contained in a test tube. The tests were carried out within 30 minutes of sample collection.

After taking the blood for total platelets count the rest of the blood was centrifuged without delay at 1200-1500 g. for 5 minutes. The supernatant plasma was then removed in clear glass test tube and kept at 40° till the test was performed.

Methods of Estimating Haemostatic Components

(i) Total platelets count—by basic methods using formal citrate red cells diluent.

(ii) Clotting time—by capillary tube method.

(iii) Plasma recalcification time—by calcium time method.

(iv) Plasma fibrinogen—by Turbidmetric method modified by Ellis and Stransky.

(v) Plasma fibrinolytic activity—by Euglobin Clot lysis time.

Platelets count and fibrinolytic activity are lowered in maternal vein in pre-eclampsia, when compared with normal pregnancy otherwise no difference was found. Pregnancy complicating eclampsia shows marked lowering in components of haemostatic mechanism.

All the component of haemostatic mechanism in umbilical vein blood in pre-eclampsia and eclampsia shows lowered value in comparison to normal pregnancy,

TABLE I

Comparative Study of the Components at Birth in Maternal Vein in Normal Pregnancy and Pregnancy Complicated with Pre-eclampsia and Eclampsia

Components	Normal Pregnancy (mean value)	Pre-eclampsia (mean value)	Eclampsia (mean value)
Platelete count per cumm of blood	155240	131200	106272.72
Clotting time	3'38".12	3'32".15	3'32".9
Recalcification time	91.44	88.25	100
Plasma fibrinogen (mg/100 Ml. of blood)	533.68	527.65	488.54
Euglobin lysis time in min.	122.82	99.7	99.9

TABLE II

Comparative Study of the Companent at Birth in Umbilical Vein in Normal Pregnancy and Pregnancy Complicated with Pre-eclampsia and Eclampsia

Components	Normal Pregnancy	Pre-eclampsia	Eclampsia
Platelet count per cumm of blood	227400	223150	217181.81
Clotting time	4'37".5	4'28".2	4'20".63
Recalcification time	182.46	163.95	170.18
Plasma fibrinogen (mg/100 Ml. of blood)	320.96	317.25	298.45
Euglobin lysis time in min.	219.62	138.25	132

TABLE III

Comparative Study of the Components at Birth in Umbilical Artery in Normal Pregnancy and Pregnancy Complicated with Pre-eclampsia and Eclampsia

Components	Normal pregnancy	Pre-eclampsia	Eclampsia
Platelet count per cumm of blood	204200	203250	199181.81
Clotting time	4'05".7	4'01".2	3'58".9
Recalcification time	275.68	2185	208.18
Plasma fibrinogen (mg/100 Ml. of blood)	294.04	292.5	287.36
Euglobin lysis time in min.	299.48	204.65	208.72

except plasma fibrinogen in pre-eclampsia shows not much difference than normal pregnancy.

All the components of haemostatic mechanism in umbilical artery blood in pre-eclampsia and eclampsia at birth shows not much difference when compared to normal pregnancy except recalcification time and euglobin lysis time are shortened than in normal pregnancy.

Comments

Our observation shows mean maternal platelets level in pre-eclampsia are reduced, being more reduced in patients with eclampsia. Cord blood mean platelet count in both the groups of patients shows higher levels than found in maternal vein. But when mean platelete count in maternal umbilical vein and umbilical artery in pre-eclampsia and eclampsia are compared with normal pregnancy level they are found to be low and markedly lower in cases of eclampsia.

Dube *et al* (1975) in their study of coagulation studies in Indian patients with pre-eclampsia, eclampsia and normal pregnancy in third trimester found systematic bleeding diathesis in 2 patients with eclampsia but none in pre-eclampsia. However, excessive uterine haemorrhage was found in 2 patients with pre-eclampsia. They demonstrated significant thrombocytopenia in patients with eclampsia and in 2 with severe pre-eclampsia.

Our observation shows clotting time in pre-eclampsia and eclampsia to be much prolonged in cord blood when compared with maternal vein blood. But we find maternal vein clotting time in pre-eclampsia and eclampsia to be within the range of normal pregnancy. Bonnar *et al* (1971) reported clotting time to be prolonged in umbilical cord blood when compared to maternal blood at birth in cases of pre-eclampsia.

In the present study, cord blood shows increased recalcification time both in cases of pre-eclampsia when compared to maternal side.

Cord blood plasma fibrinogen levels in pre-eclampsia and eclampsia are lower than in maternal blood. Umbilical artery plasma fibrinogen level is found to show lower level than in umbilical vein blood in both the groups of patients. Fibrinolytic activity in euglobin lysis test in cord blood in pre-eclampsia and eclampsia shows increased activity than on maternal side. The difference is more marked when maternal vein is compared with umbilical artery which shows higher level of fibrinolytic activity in ulbilical artery blood.

Bonnar *et al* (1971) reported depressed fibrinolytic activity in patients with pre-

eclampsia and eclampsia when compared with normal pregnancy. This was explained by them that lowered euglobin lysis time reflects reduced levels of circulating plasminogen activator which results from a reduced production of activators in pre-eclampsia from the absorption of circulating activators into intravascular fibrin. When pre-eclampsia and eclampsia were compared no difference in euglobin lysis time was found in maternal, umbilical vein and umbilical artery blood of the two groups.

Study confirms that cord blood is influenced by placental separation and it is possible that placenta influences foetal haemostasis in utero.

With regard to umbilical vein blood finding the presence of "Placental barrier" in normal pregnancy and pre-eclampsia has been confirmed. Also, since local activation of the coagulation mechanism is known to occur during parturition when the foetal circulation is in a state of stasis (Bonnar *et al* 1971) increased cord blood fibrinolytic activity in normal pregnancy could reflect a protective response against these adverse conditions. Cord blood vein fibrinolytic inhibitor levels had been expected to be much higher in blood draining the placenta which is known to be rich source of antiplasmin (Kawano *et al* 1968).

In umbilical vein depressed fibrinolysis could alter the haemostatic balance at birth and predisposes to intravascular coagulation.

In mild pre-eclampsia with uterine growth retardation Bonnar (1973) has shown apparent improvement in foetal growth with anticoagulant therapy. Anticoagulant therapy might perhaps be more effective in patients with recurrent toxaemia or recurrent intrauterine growth retardation, if treatment were recommended before the onset of clinical signs of the disorder. The data permit to speculation that anticoagulant might reduce the adverse effect of placental ischaemia and infarction on foetal growth and also possible effect of placental damage on foetal circulation and placental perfusion by foetus.

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