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0316 DECREASED α -ADRENERGIC RECEPTORS IN NEWBORN PLATELETS: CAUSE OF ABNORMAL RESPONSE TO EPINEPHRINE?

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Platelets of newborn infants (NBP) fail to aggregate or release adenosine diphosphate in response to epinephrine (E). Because E-induced aggregation is an α -adrenergic event, we considered the possibility that NBP possess fewer E receptors than do those of adults. Therefore we compared the specific binding of the α -adrenergic antagonist, ^3H -Dihydroergocryptine (DHE), in intact washed platelets prepared from paired samples of maternal and cord platelet rich plasma. NBP demonstrated normal kinetics of ^3H -DHE binding and normal affinity for ^3H -DHE. Scatchard analysis of ^3H -DHE binding indicated a single class of binding sites that exhibited a high affinity for the radioligand ($K_d = 7.5\text{nM}$). Maternal platelets (MP) were found to bind approximately 2-fold more DHE than NBP (3.70 ± 0.28 vs. 1.74 ± 0.17 fmol/ 10^7 platelets) at saturation. This corresponds to 223 ± 17 vs. 105 ± 11 binding sites per platelet ($p < 0.001$). Repeat washing of NBP did not yield increased DHE binding suggesting the binding sites had not previously been masked by elevated circulating levels of E and/or nor-E in venous cord blood. When control platelets were incubated with concentrations of ^3H -DHE that half-saturated the α -adrenergic receptors, diminution of platelet function comparable to that seen in NBP was observed. Since NBP and MP are similar size, it appears that a deficiency of α -adrenergic receptors may account for the diminished response of NBP to epinephrine.

P5-036 0317 IMMUNOLOGICAL STUDIES OF PROTHROMBIN IN NEWBORNS

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Prothrombin of newborns was studied by means of the quantitative immunoelectrophoresis(QIE) and the two dimensional immunoelectrophoresis(TDIE). In the TDIE the prothrombin showed one precipitate in alpha-2-beta-globulin position when a buffer containing calcium was used. Another precipitate in alpha-1-globulin position was frequently found in preterm and term newborns. After administering vitamin K to the newborns the precipitate in alpha-1-globulin position was no longer demonstrable. When a buffer without calcium was used, in all newborns only one precipitate in the alpha-1-globulin position was found. The QIE and the activity assay gave equal results in those infants who showed only one precipitate in the TDIE. In those newborns who showed two precipitates, the QIE gave higher results than measurement of the activity.

From this study no evidence of differences between the prothrombin of newborns and that of adults could be derived. Vitamin K deficiency as judged from the appearance of a second peak in alpha-1-globulin position in the TDIE was a frequent finding in newborns, even on the first day of life.