and IRVING SCHULMAN. Dept. of Ped., The Abraham Lincoln Sch. of Med., Univ. of Illinois, Chicago, IL.

Platelet aggregation was studied in platelet-rich plasma (PRP) prepared from samples of maternal and cord blood taken immediately after delivery. When mothers had received no drugs for analgesia, adenosine diphosphate- and collagen-induced aggregation curves of platelets from the infants and from the mothers were virtually identical and were within the normal range. When mothers (18) had received drugs (acetylsalicylic acid, promethazine, alphaprodine and meperidine) prior to delivery, 17 of 18 of the cord blood samples showed a marked decrease in collagen-induced platelet aggregation. By contrast, in paired PRP samples from these mothers abnormalities of collagen-induced platelet aggregation were found in only 25%, and these changes were minimal. These findings suggested that the infants' platelets were more susceptible to the drug-induced suppression of collagen aggregation than were platelets of the mothers. Dose response curves to promethazine added *in vitro* demonstrated a markedly increased susceptibility of newborn platelets when compared with those of the mothers. These results indicate that a variety of drugs given to mothers before delivery may alter platelet aggregation of the newborn infants, resulting in significant impairment of the platelet-mediated phase of hemostasis.

34 *Plasma 17-OH Progesterone in Maternal and Umbilical Cord Plasma in Children, and in Congenital Adrenal Hyperplasia (CAH): Application to Neonatal Diagnosis of CAH.* MORRIS R. JENNER, MELVIN M. GRUMBACH and SELNA L. KAPLAN. Univ. of California, San Francisco Med. Center, San Francisco, CA.

The concentration of plasma 17α-OH progesterone (17-OHP) was determined utilizing a ligand binding radioassay at delivery in maternal (8) and umbilical venous (8) blood and in the blood of newborn infants (8), children (12), adult males (13), and pre- and post-treatment in 6 patients with CAH. Adult male values (mean = 0.106 μg/100 ml) are in agreement with the data of STROTT *et al.* [J. clin. Invest. *48:* 930, 1969]. Mean concentration in maternal plasma at delivery was 0.365 μg/100 ml and in cord plasma 1.64 μg/100 ml. Twelve newborns (age 1–7 days) demonstrated a rapid fall in concentration of plasma 17-OHP within the first day of life (range <0.100–0.125 μg/100 ml). Twelve normal children (age 5–11 years) had a mean level of 0.036 μg/100 ml (range 0.016–0.059). In contrast, 5 children (age 4 days–7 years) and an adult (age 26 years) with untreated CAH had a mean value of 12.8 μg/100 ml (range 2.4–33.0 μg/100 ml). A 4-day-old female pseudohermaphrodite, the youngest subject with CAH, had a plasma 17-OHP of 5.7 μg/100 ml. In 1 infant with CAH the concentration of plasma 17-OHP fell from 11.4 μg% to 0.12μg% on cortisone therapy, and in a female, age 2–$^{7}/_{12}$ years, from 33.0 μg% to 0.148 μg/100 ml. In 4 patients with CAH, changes in plasma testosterone paralleled those in 17-OHP. The results suggest that estimation of plasma 17-OHP in a 0.2 ml sample by this method permits rapid identification of the 21 hydroxylase deficiency form of CAH by the 2nd day of life and is useful in assessing the adequacy of glucocorticoid treatment. The high concentration of plasma 17-OHP in cord blood is attributable to the capacity of the placenta to convert maternal and fetal steroid precursors to 17-OHP.

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