

lipid kinetic studies were performed serially at intervals post ligation. Osmotic fragility studies show progressively increasing red cell sensitivity to osmotic lysis following ligation. Morphologically, a striking increase in "burr cells" is noted within 24-48 hrs. Elevation of erythrocyte membrane and plasma lecithin levels are noted by 3-4 days post ligation reaching maximal values near 40% of total membrane phospholipid by one week. Injection of P³² at varying intervals from 2-16 days post ligation show the specific activity of phospholipid phosphorus in both red cells and plasma to be higher in the bile duct ligated animals than in controls. Rise in plasma P³² activity precedes that of red cell and is maximal at 24 hrs. Red cell labelling shows a progressive rise over a 3-4 day period. Despite the quantitation difference of specific activity in experimental and control groups, the ratio of red cell specific activity: plasma specific activity is similar in both groups. These results suggest that the qualitative and quantitative alterations of erythrocyte phospholipid are directly related to the plasma phospholipid alterations.

NEONATOLOGY

Factors influencing predisposition to serious illness in low birth weight infants. LEONARD GLASS, NORMA KOLKO, and HUGH E. EVANS (Intr. by Gilbert W. Mellin). *Columbia Univ. Harlem Hosp. Ctr., N. Y., N. Y.*

Both retrospective and prospective studies of low birth weight infants born at Harlem Hospital and discharged to their mothers showed high rates of serious illness requiring rehospitalization during the first 9 months of life to be related to specific socio-medical factors. These factors were utilized in forming the following weighted prognostic index:

Failure of mother to receive prenatal care	2	0
Absence of father from home	1	0
Receipt of public assistance	1	0
Other children in the home	1	0

Thus a maximum score of 5 would indicate an infant at highest risk, and a minimum score of 0 lowest risk. In both series, infants scoring 4 and 5 had three times the rehospitalization rate of those infants who scored 0, 1 and 2 (p < 0.05). Infants scoring 3 occupied an intermediate position. By prospective assignment of a score to each low birth weight infant, those at highest risk of inadequate follow-up care and rehospitalization may be identified prior to discharge from the nursery so that intensive medical, nursing and social services can be directed toward this high-risk group.

Hepatitis-associated antigen: A possible relationship with premature delivery. ELIZABETH M. SMITHWICK, ELEANOR PASCUAL, and SUAT CHENG GO. *Downstate Med. Ctr., Brooklyn, N.Y.*

Preliminary observations in 271 pregnancies indicated a high incidence of hepatitis-associated antigen (HAA) in mothers delivered at a large metropolitan hospital. On analysis, the frequency of HAA appeared to be higher in mothers of premature infants and these infants had a poor survival rate. The present study was designed to check these findings and to determine the incidence of HAA and the outcome of pregnancy in mothers with viral hepatitis. The outcome of pregnancy in apparently healthy HAA+ women was also analyzed. *Mothers of premature and fullterm infants.* Ninety mothers of infants

weighing 2000 gms or less were studied. The controls were 90 mothers of the nextborn fullterm infants. Three of the 90 mothers of prematures were HAA+; 3 of their 4 infants (1 set of twins) died. On the other hand, only 1 of 90 mothers of infants >2000 gms was HAA+; her infant survived. The overall 2.2% incidence is identical to that of the preliminary observation. *Mothers with clinical hepatitis.* Eight pregnant women with hepatitis were studied. Five of them were HAA+. Four of their 5 infants were premature; 3 of the prematures died. The infants (2 fullterm, 1 premature) of the negative mothers survived. *HAA+ mothers, apparently healthy.* A total of 7 mothers, HAA+ at delivery and with no history of hepatitis, were studied. Five of their infants (1 set of twins) were premature; 4 died. The 3 fullterm infants survived.

The data suggest that pregnant women who are HAA+, with or without hepatitis, tend to deliver prematurely and that their infants, if premature, have a high mortality rate.

Identification of the high-risk infant from placental phase microscopy. AVROY FANAROFF, SILVIO ALADJEM, F. LANE FRANCE, and MARSHALL KLAUS. *Case Western Reserve Univ. Sch. of Med., Cleveland, Ohio.*

Fresh placental tissue was studied by phase contrast microscopy following 125 normal and complicated pregnancies. 76 infants were full-term, 29 premature, 11 small for gestational age, and 9 from insulin-dependent diabetics. The fetal outcome was correlated with the placental score determined by grading pathological features in the 1) syncitium (hypoplasia, hyperplasia); 2) stroma (edema and intravillous hemorrhage); and 3) vascularity of the villus (congestion, ischemia and avascularity). A total score of 0 indicated normal features for gestational age. Significant correlation was observed between placental score, fetal mortality and morbidity. The mortality was 52% (11/21) with placental scores 6 or above; whereas only 2 of 104 infants with scores below 6 died (p < .001). The table below shows results in infants below 37 weeks.

N	Survived	Score range	Mean score	Mean gest. age	Mean weight	Weight range (gm)
14	14	0-5.9	2.6	34.1	2029	1550-2820
15	4	6-23	11.1	31.0	1564	880-2180

19 of the 21 infants with scores of 6 or above presented with problems of extra-uterine adaptation including asphyxia, anemia, respiratory problems, in contrast to 12 of 104 with scores below 6 (p < .001). All infants with severe hyaline membrane disease (arterial PO₂ <50 mm. Hg. in 100% O₂) had scores above 6 and demonstrated placental vascular changes with syncytial hypoplasia. Phase microscopy of the placenta is a simple (ten minute) procedure and appears to be helpful in predicting fetal outcome.

Increased skin permeability in preterm infants. RICHARD L. NACHMAN, and NANCY B. ESTERLY. *Univ. of Illinois Coll. of Med., Ohio State Univ. Med. Sch.* (Intr. by Irving Schulman).

Localized cutaneous blanching of preterm neonates following the topical application of a 10% solution of Neo-Synephrine attests to the permeability of immature skin. Skin permeability was evaluated in 18 healthy infants between 30 to 40 weeks of gestational age. The response consisted of speckles or islands of

blanching which gradually coalesced to form a homogeneous pale area. Infants 30–35 weeks had a very rapid response which persisted from 30 minutes to 8 hours. A response was no longer apparent by 21 days of postnatal life. Infants 36–37 weeks had a longer lag period and responded less intensively. Infants 38–42 weeks failed to exhibit blanching in most cases. The degree of permeability correlated inversely with gestational age. Surface temperatures on strongly blanched and contiguous unblanched skin were identical when measured simultaneously. Toxicologic implications are clear. Other considerations include the intra-uterine role of fetal skin as a dialyzing membrane, the relationship of skin structure to barrier function and the potential of further pharmacologic studies on the accessible cutaneous circulation.

Post-natal transient cataracts in pre-term infants. PETER HAHN, ANDREW Q. McCORMICK, and SYDNEY SEGAL. *Univ. of British Columbia, Vancouver, B. C., Canada.*

An additional 15 cases have been added to the 7 cases reported originally in 1968. In contrast to those where the cataracts occurred in infants weighing 1500 gms or less, and where they appeared at 7 to 10 days following birth, the new cases include infants weighing up to 2610 gms, the cataracts appearing as late as 3 weeks of age.

Speculation in the original report is now supported by evidence that these opacities may be the result of a delayed capability to metabolize one of the ingested carbohydrates.

The only cataracts in infancy known to be reversible are those associated with galactosemia. Therefore it seemed reasonable to study the galactose-1-phosphate uridyl transferase in those small infants who developed transient cataracts. In 3 cases examined this enzyme was found to be completely absent and was present at extremely low levels in 2. In a pair of twins, the larger one with normal enzyme levels maintained clear lenses, while its smaller brother who was shown initially to have no measurable level of the enzyme developed transient cataracts.

The possible effects of excess galactose will be discussed. A question to explore is whether infants should be fed a carbohydrate which they are unable to metabolize.

Coagulation studies in the newborn. LILY M. YOUNG and JUDITH G. POOL. *Stanford Univ. Sch. of Med., Stanford, Calif.* (Intr. by Herbert C. Schwartz).

Hemorrhagic tendency has been cited as a serious complication in certain sick newborn infants. A prospective study was undertaken to determine the coagulation status of sick newborn infants with the aim of differentiating conditions such as disseminated intravascular coagulation from the normally low coagulation factors characteristic of the newborn period. Forty-two premature and 15 full-term newborn infants admitted to an intensive care unit for diagnosis and treatment necessitating umbilical vessel catheterization, were studied. Parameters studied were (1) clinical observation of hemorrhage, (2) coagulation factors by Partial Thromboplastin Time (PTT), Prothrombin Time (PT), Thrombin Time (TT), Prothrombin and Proconvertin Time (P & P), Factor V, Factor VIII and Platelet Count, (3) autopsy findings of hemorrhage and microthrombi. Nine premature and 2 full-term infants bled. The results of the coagulation factors for these infants who bled indicated prolonged PTT, normal PT, normal TT, mean values of P & P 20%, Factor V 57%, Factor VIII 81%, Platelet count 147000/

mm³. These values were consistent with values for the newborn period and no significant difference was observed between bleeders and non-bleeders. These results did not provide convincing evidence that disseminated intravascular coagulation was the basis of bleeding in these sick newborn infants.

Disseminated intravascular coagulation (DIC) and hyaline membrane disease (HMD). CARMEL Z. MARGOLIS, MARCELLO M. ORZALESI, ALLEN D. SCHWARTZ, and MICHAEL G. BLACKBURN (Intr. by C. D. Cook). *Yale Univ. Sch. of Med., New Haven, Conn.*

A prospective study was planned to determine whether or not DIC contributes to the pathogenesis of HMD and to ascertain the possible role of DIC in the production of the intra-cranial hemorrhages (ICH) frequently found at autopsy in HMD.

In order to detect DIC, platelet counts, fibrinogen, factors V and VIII levels, ethanol gelation tests and tanned red cell hemoagglutination inhibition immuno-assays were performed on the first or second day of life in 11 premature infants with the typical clinical and radiological findings of severe HMD. In 5 infants the tests were repeated on one or more occasions during the first 10 days of life.

16 normal premature infants of similar gestational and post-natal ages were also studied and served as controls; none of these infants had evidence of DIC and they all survived.

Conclusive evidence of DIC was found only in 2 infants with HMD: one had DIC on day 1 and died; the other developed DIC on day 8 while on the respirator and survived. Four of the remaining 9 infants with HMD died; autopsy was performed in 3 and showed extensive HMD in all and severe ICH in 2.

These data, although preliminary, suggest that DIC does not play a major role in the pathogenesis of HMD and that it is not an important contributing factor in the high mortality and frequent ICH in babies with HMD.

Effect of exchange transfusion (ET) on serum ionized calcium (Ca⁺⁺). M. JEFFREY MAISELS, TING-KAI LI, JOSEPH T. PIECHOCKI, and MILTON W. WERTHMAN (Intr. by Nicholas M. Nelson). *Walter Reed Army Institute of Research, Walter Reed General Hosp., and Washington Hosp. Ctr., Washington, D. C.*

The citrate in ACD blood is known to complex Ca⁺⁺ and calcium gluconate is usually infused during ET to counteract this effect. We have measured Ca⁺⁺ with a Ca⁺⁺ selective electrode during ET with non-buffered ACD and heparinized blood and have studied the effect of administering 0.1g calcium gluconate per 100 ml ACD blood exchanged. Heparinized blood produces negligible changes in Ca⁺⁺, whereas with ACD blood a profound fall in Ca⁺⁺ occurs even when calcium gluconate is given. Values for Ca⁺⁺ during ET and ACD blood and added calcium gluconate are much lower in premature than in full term infants (p < 0.001). Omitting calcium gluconate further lowers Ca⁺⁺ levels in full term infants (p < 0.05). The injection of calcium gluconate produces a transient rise in Ca⁺⁺ which is rapidly neutralized by the infused citrate. Cessation of ET produces a prompt return toward normal values. No relationship was shown between Ca⁺⁺, total Ca and protein. In spite of frankly hypocalcemic levels of Ca⁺⁺ (particularly in prematures) no clinical evidence of tetany was seen. Ca⁺⁺ levels could not be correlated with the infant's clinical state during the ET. We conclude that in full term infants the use of calcium gluconate in ET using ACD blood exerts a small but significant