424 THE RELATIONSHIP OF AIRB[illegible] LEAD TO SUDDEN INFANT DEATH AND CORD BLOOD LEAD.

Marilyn M. Erickson and Laura S. Hillman. Dept. Pediatrics, Washington Univ. Sch. Medicine, St. Louis, MO 63178

The epidemiology of Sudden Infant Death Syndrome (SIDS) and toxic metal exposure, especially lead, are similar. We measured tissue lead in lung, liver, kidney & rib from 66 SIDS & 23 age appropriate (4-26 wk) sudden death controls. The SIDS liver and rib specimens contained significantly more lead than non-SIDS tissues (liver, 1.095µg/g vs 0.761µg/g, P<.05; rib, 1.754µg/g vs 0.041µg/g, P<.01, respectively). All 4 SIDS tissues showed significant increases in lead concentrations with age while non-SIDS tissues did not. The slope of the SIDS lung lead regression line was significantly steeper (P<.05) than non-SIDS, suggesting the respiratory route as a major contributor to the SIDS lead burden. Thus, we compared monthly mean air lead levels obtained from St. Louis County Air Pollution Control Office with the monthly incidence of SIDS deaths. We found that the highest air lead levels preceded the peak incidence of SIDS deaths by about 1-2 months. However, we found no difference in the tissue lead levels in those infants who died during the peak SIDS incidence period (Oct-Jan) and those who died during the low incidence period. Subsequently, we have measured whole blood Pb in cord blood from 1518 sequential births in a single hospital. The results show a late summer/fall peak in cord blood Pb with a Sept mean ± S.D. of 15.4±4.5µg/dl with 11.5% ≥20µg/dl. Besides late summer/fall birth, young multigravidity, prematurity and black race are risk factors for SIDS. In July, cord blood Pb was significantly greater than the monthly mean (8.8±3.6µg/dl) for mothers ≤19Y plus ≥2 parity (10.9±4.4µg/dl), infants <38 wk gestation (10.2±4.8µg/dl) and black infants (9.5±3.6 vs 6.8±3.0µg/dl for white infants, P<.001). The significance of these epidemiologic relationships between air Pb, cord blood Pb and tissue Pb in SIDS requires further study.

425 SERUM TOTAL AND UNBOUND BILIRUBIN IN CHINESE NEONATES

Kwang-Jen Hsiao[1,2], Yu-Lin Wong[3], and Wen-Kuo Ting[2]. Depts. of Med. Res.[1], Lab.[2], and Pediatrics[3], Veterans General Hospital; Taipei, Taiwan 112; Rep. of China

The incidence of neonatal jaundice in Chinese is much higher than that in Caucasian population. Since the measurement of serum total bilirubin concentration alone can no longer be considered a sufficient means of assessing hazard to the infant with jaundice. We intended to study bilirubin binding parameters in addition to serum total bilirubin in Chinese neonates.

About 300 neonates delivered in our hospital were randomly selected. For each of them, capillary blood were collected by skin-puncture on the 1st, 3rd and 5th day of age. The total bilirubin concentration was determined spectrophotometrically with within-run and day-to-day precision of 0.5-2.0% and 1.0-1.9% (C.V.) in the working range, respectively. The unbound bilirubin was measured by the "peroxidase" method (Clin. Chem. 20,783-789, 1974) with within-run and day-to-day precision of 0.2-4.0% and 0.4-5.2%, respectively. Both the total bilirubin and unbound bilirubin were peaked at day three ($p<0.001$). Unbound bilirubins were determined to be 0.44±0.50 nM (mean±SD; n=307), 0.73±0.64 nM (n=287), and 0.53±0.58 nM (n=236) with the upper 97.5% limits of 1.65 nM, 2.45 nM and 2.70 nM for day one, three and five, respectively. The total bilirubin of day one, three and five were determined to be 5.9±1.9 mg/dl (n=303), 10.6±2.6 mg/dl (n=286) and 9.6±2.7 mg/dl (n=239), respectively. These data indicated that the mean peak levels of serum total bilirubin in Chinese neonate were twice of those in Caucasian neonates and 30% of Chinese neonates would be considered to have pathological jaundice according to the criteria of Dr. T.A. Blumenfeld. The guideline for the application of these bilirubin indexes to the diagnosis and management of neonatal jaundice in Chinese neonates remain to be established.

426 KODAK EKTACHEM CLINICAL CHEMISTRY SLIDES (NBIL) MINIMIZE INTERFERENCE FROM LIPEMIA AND HEMOLYSIS.

R. Corkey, S. Kahn, F. Kiechle (Hospital, Univ. of Pennsylvania, Phila., PA), L. Connelly, D. Powers, D. Walch, T. Shirey (Eastman Kodak Company, Rochester, NY)

Sera from patients in neonatal intensive care units are frequently lipemic due to administration of hyperalimentation fluids. Neonatal specimens are also often hemolyzed. We, as others, have found these factors to cause large biases on many conventional bilirubin methods.

The NBIL slides for neonatal bilirubin analyses have been designed to minimize interference from these two sources. In the multilayered slides, chylomicrons, lipoproteins, hemoglobin, and other pigmented proteins are retained in the top (spreading) layer, while bilirubin diffuses into a lower (detection) layer. An opaque (screen) layer between the spreading and detection layers shields retained interferents from the reflectometer beam.

To confirm the ability of the screen layer to mask potential interferents, a series of lipemic and hemolyzed samples was prepared by supplementing serum pools (10 mg/dL bilirubin) with human red cell lysates (150 mg Hb/dL) and hyperalimentation fluids (1:10 Liposyn®). Samples were analyzed in replicate by the NBIL slides, as well as by widely used Jendrassik-Grof and spectral bilirubin methods. Results are in percent bias from the control serum pool:

Interferent	EK NBIL	JG1	JG2	Spectral 1	Spectral 2
Hemoglobin	+3%	-26%	+1%	-1%	+1%
Liposyn®	0%	+22%	-54%	+81%	+17%

These data confirm that hemolyzed and lipemic specimens can be analyzed directly by the NBIL slides without specimen pretreatment.

Fluorescent and autoradiographic studies locating hemoglobin and triglyceride in cross section of the NBIL slides are in progress.

427 "Evaluation of Newer Methods For Predicting Respiratory Distress Syndrome of Newborns"

Robert Wenk, Jack Lustgarten, Charles Byrd, Doward Patterson, and Jacob Felix. Dept. of Patho. and Ped.,Sinai Hosp. of Balto. Baltimore, MD 21215

Advantages of amniotic fluid (AF) phospholipid analysis by continous-development thin-layer chromatography (TLC) include ability to rule out respiratory distress syndrome (RDS) with great certainty, to predict RDS in the presence of blood or meconium contamination and to raise suspicion that neonatal respiratory problems are unrelated to presence of immature surfactant. Disadvantages include technically complex, labor-intense methodology. The L/S ratio is falsely increased in the presence of blood or hemolysis (P .05, N=19 paired samples). Phosphatidylglycerol analysis fails to predict RDS with high probability despite high sensitivity, because the AF of many neonates without RDS does not show presence of this lipid (Table 1).

Table 1. Posterior Probability of Amniotic L/S and PG Tests in Predicting RDS.

	Sensitivity	Specificity	Posterior Probability Pos.test	Neg.test
L/S	.714	.914	.385	.977
PG	.857	.828	.273	.987

* Prevalence of RDS = .07, N=100

Comparison of a PG agglutination assays, (Hana Biologics, Inc., Berkley, CA 94710) and foam stability test (Beckman Instruments, Inc. Brea, CA. 92621) for surfactant,, with TLC, is presently in progress.

428 EVALUATION OF A SPECIFIC MINI-COLUMN PROCEDURE FOR HEMOGLOBIN A_{1C} (HbA_{1c})

Patrick K. Jaynes, Marian Willis, and Peter Chou. American Medical Labs., Fairfax, VA 22030; and Steven Anderson and Steven Tanaka. BioRad Labs, Richmond, CA 94804.

We developed and evaluated a specific mini-column procedure for HbA_{1c}. Packed RBCs are lysed in borate buffer containing polyoxyethylene ether. This step liberates RBC Hb and initiates the dissociation of the labile Schiff base (pre-A_{1c}). Following the application of an aliquot of the hemolysate to the BioRex 70 resin a low ionic strength borate/ phosphate buffer is used to elute Hb_{1a} and Hb_{1b} and further eliminate the pre-A_{1c} fraction. A phosphate buffer is used to elute HbA_{1c}. A total Hb tube is prepared by mixing an aliquot of the hemolysate with the second elution buffer. The relative % concentration HbA_{1c} vs total Hb is determined spectrophotometrically at 415 nm.

For 220 nondiabetic controls, the $\bar{x}$ HbA_{1c} value was 4.77% with an S.D. of 0.67%. A second control study using non-hospitalized subjects with normal SMAC and CBC profiles yielded the same result (n=65, $\bar{x}$=4.78%, S.D.=0.55%). Within-run precision at HbA_{1c} levels between 4.0 and 14.0% resulted in CVs between 1.4 and 2.7%. Day to day precision for similar HbA_{1c} levels resulted in CVs between 1.3 and 2.8%. When HbA_{1c} was added to hemolysates between 88 and 92% was recovered. Comparison of this procedure to HbA_{1c} analysis by HPLC demonstrated acceptable correlation (HPLC=1.1 BioRad - 1.6%). We also demonstrated that although total HbA_1 levels show a transient rise when cells are incubated in elevated glucose solutions, the present procedure is unaffected. Studies of possible interferences have shown that HbF and Barts Hb will falsely elevate A_{1c} levels. Studies with a variety of hyperlipidemic plasmas revealed no interference. The use of specific calibrators minimizes the effect of transient temperature fluctuations and negates the need for temperature conversion estimates.

We have described a commercially available mini-column procedure for HbA_{1c} that is precise, cost effective and simple to use.