†1445 AUDITORY NERVE AND BRAINSTEM RESPONSES IN NEWBORN INFANTS WITH HYPERBILIRUBINEMIA.

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To assess early bilirubin toxicity, we have sutdied auditory brainstem responses (ABR) in relation to serum total bilirubin (TB) and unbound bilirubin(UB) levels in 66 non-asphyxiated full term infants at 3 to 5 days of age. Exchange transfusions(ET)were performed in 14 of the 66 infants and the measurements were done pre-ET and on consecutive 3 days of post-ET. ABR was measured with a Teledyne TA-1000 by stimuli of 85 dB and the latencies to wave I and V were averaged over two replications in each ear. TB and UB were determined by the peroxidase method using UB-Analyzer. Wave I latency increased significantly with the elevation of both TB and UB levels. Wave I-V interpeak latencies did not vary with the TB or UB Levels. Prolonged wave I latency pre-ET decreased remarkably at 24 to 48 hours of post-ET. UB levels decreased more rapidly than did the TB levels. These results suggest that the auditory nerve is reversibly damaged in the infants with hyperbilirubinemia.

	no.cases	Latencies(msec); *p<0.05, †p<0.01		
		Wave I	Wave V	Wave I-V
Total bil. (mg/dl) ≦ 14.9	21	1.14± 0.12	6.32± 0.27	5.18± 0.27
15.0-21.9	29	1.24± 0.15 †	6.40± 0.24	5.17± 0.24
≧22.0	16	1.28± 0.23 †	6.45± 0.35	5.17± 0.37
Unbound bil. (ug/dl) ≦ 0.49	26	1.14± 0.13	6.36± 0.26	5.22± 0.25
0.50-0.99	32	1.25± 0.13 * †	6.40± 0.28 †	5.16± 0.25
≧1.00	8	1.38± 0.34 †	6.60± 0.32 †	5.22± 0.34

1446 NEW CLASSIFICATION OF NEONATAL GBS INFECTION — ANALYSIS OF 274 CLINICAL CASES —

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Clinical pictures of 274 group B streptococcus (GBS) neonatal sepsis and meningitis gathered from 50 hospitals in Japan were analysed. Postnatal ages of their clinical onset were distributed more or less equally all through neonatal period except an apparent cluster on the day of birth, which made it difficult to divide these cases into two, early and late onset types. There were three distinct clinical pictures:

AMNIOTIC INFECTION TYPE which manifests symptoms at birth with asphyxia and respiratory distress, and which has history suggesting antenatal infection. RAPID PROGRESS SEPSIS TYPE which presents sudden onset of septic shock and rapid deterioration in an infant who has been clinical well. SLOW PROGRESS MENINGITIS TYPE which has symptoms of fever and/or not-doing well for 24 hours or longer prior to the definite manifestation of meningitis. The latter two types could occur on any postnatal ages.

Vaginal cultures of 2800 pregnant women using Baker's original method revealed 12.1% positive rate for GBS. Serotype Ic which is predominant (45%) in carrier mother is responsible for only 10% of clinical cases, while serotypes III and Ia which are resposible for 73% and 11% of clinical cases are seen only 25% and 0.5% in carrier mother respectively. These findings will suggest the difference of pathogenicity among serotypes and will partly explain their marked different clinical pictures.

†1447 LUNG SURFACTANT REPLACEMENT WITH LIPIDS EXTRACTED FROM BOVINE LUNG LAVAGE: EFFECTS OF DOSE AND DISPERSION TECHNIQUE.

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Extracted bovine lung lipids (CLL) with only 1% protein were instilled intra-tracheally into premature lambs of 18 and 19 weeks gestation to evaluate surfactant replacement therapy for the Respiratory Distress Syndrome. Aqueous dispersions of CLL were prepared by sonication (S) and vortexing (V), prior to pre-ventilatory instillation at low and high doses of 15 mg CLL/kg animal weight and 100 mg CLL/kg. A clear improvement in blood oxygenation and lung compliance was found over controls for lambs instilled with 15 mg/kg and 100 mg/kg CLL(V), and with 100 mg/kg CLL(S). Lambs treated with 15 mg/kg CLL(S) failed to improve over controls, even though they had similar amounts of lavage phospholipids as low dose CLL(V) lambs. Solvent-spread CLL films lowered surface tension to <1 d/cm under dynamic compression in vitro, and both CLL(V) and CLL(S) dispersions also adsorbed to equilibrium surface tensions of 23-25 d/cm in seconds at concentrations greater than 0.25 mg CLL/ml. However, the preparations differed in behavior on an oscillating bubble apparatus which determined a physiologically relevant combination of adsorption and dynamic compression effects at 37°C in 100% humidity; results showed that CLL(V) dispersions retained optimal surface activity to significantly lower concentrations than CLL(S). This biophysical behavior correlated closely with the dose-dependent physiologic efficacy of the dispersions. (HL-00945, HL-22552)

●1448 CLEARANCE OF LARGE AMOUNTS OF NATURAL SURFACTANT AND DIPALMITOYLPHOSPHATIDYLCHOLINE FROM THE LUNGS OF 3-DAY-OLD RABBITS FOLLOWING TRACHEAL INJECTION.

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Eighty-two 3-day-old rabbits were given by intratracheal injection doses of either radiolabeled natural sheep surfactant (NS) or sonicated suspensions of dipalmitoylphosphatidylcholine (DPPC) that contained >4x the endogenous phosphatidylcholine (PC) surfactant pool size. Recoveries of radiolabeled PC, total PC and PC specific activities (cpm/μmol PC) in alveolar washes (AW), lung tissue (L), and the total lung (AW+L) were measured over 72 hrs. More than half of the NS rapidly became L associated, and the labeled PC was linearly and slowly cleared from AW+L (0.28% of the initial injected cpm/hr). The alveolar surfactant pool size continued to increase despite the exogenously administered NS. Sonicated suspensions of DPPC were cleared from the AW+L at 0.71%/hr and only 15% of DPPC became L associated. The administration of large amounts of NS did not decrease the amount of intravenously injected radiolabeled choline, palmitic acid or ^{32}P incorporated into lung PC or the amount of labeled PC secreted to the alveoli. Conclusions: 1) The % of injected PC associated with the L was higher and the rate of clearance from AW+L slower after a large dose of NS compared to an equivalent dose of DPPC. 2) Large doses of natural surfactant did not change endogenous synthetic and secretory rates. 3) Thus, the surfactant pool size apparently was not autoregulated by the animals following the administration of large doses of surfactant.

† 1449 CHANGES IN EPINEPHRINE (E) AND MEAN ARTERIAL BLOOD PRESSURE (MABP) DURING HYPERCARBIA (HC): EFFECTS ON BRAIN BLOOD FLOW (BBF) IN THE NEWBORN PIGLET.

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We have previously shown that HC results in spontaneous elevations of MABP and pressure passive increases in brain blood flow (BBF) in the newborn piglet. We further analyzed the mechanism of this phenomena by correlating these changes with acid base and plasma E data in 20 awake newborn piglets where HC ($PaCO_2$:50-60 mmHg) was induced by breathing 15% CO_2. During HC, E, MABP, and BBF increased significantly ($p<0.05$). The increase in MABP was directly related to E ($r=.71$ $p<0.001$). However, both E and MABP also demonstrated a significant correlation with the decrease in arterial blood pH (pH vs E:$r=-.91$, $p<0.001$; pH vs MABP:$r=-.84$ $p<0.001$) and the increase in base deficit (BD) (BD vs E:$r=0.83$, $p<0.001$), BD vs MABP:$r=0.84$ $p<0.001$). $PaCO_2$ was not related to the E or MABP increase. The increase in total BBF (TBBF) and regional BBF were directly related to the elevations of MABP (TBBF:$r=0.58$ $p<0.01$ cerebrum $r=0.57$ $p<0.01$, cerebellum:$r=0.67$ $p<0.01$, boundary zone $r=0.62$ $p<0.01$ and periventricular area $r=.76$ $p<0.001$). We conclude that HC induced hypertension and brain hyperperfusion is partly due to catecholamine (epineprine) release which in turn is mediated through concurrent metabolic acidosis.

●1450 CEREBRAL HYPERPERFUSION AUGMENTS BRAIN BILIRUBIN DEPOSITION IN PIGLETS.

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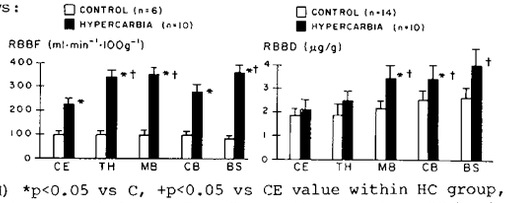
Hypercarbia (HC) induced increases in regional brain blood flow (RBBF) may augment regional brain bilirubin deposition (RBBD) in piglets (P). 10 2-4 day old P were infused with bilirubin (BR) to raise & maintain a serum BR of approximately 8 mg/dl. HC (pCO_2=76mmHg) was induced with 15% CO_2. 14 control (C) P had similar BR levels without HC. Serum free bilirubin was similar in C & HC P. RBBF (by microspheres) & RBBD were as follows:



(M±SEM) *p<0.05 vs C, +p<0.05 vs CE value within HC group, CE-cerebrum, TH-thalamus, MB-midbrain, CB-cerebellum, BS-brainstem

RBBF was elevated in all regions during HC; the increases in the TH, MB & BS, were greater than for the CE. RBBD was increased following HC in MB & CB; and the increases in MB, CB, & BS were higher than for CE. Regional brain albumin deposition (^{125}I-albumin) was similar in all brain regions. We conclude that deposition of unbound bilirubin in some brain regions is blood flow dependent.