961 NITRO-BLUE TETRAZOLIUM (NBT) TEST IN BACTEREMIC NEONATES. B.D. Chandler, N. Kapoor, B. Barker, R.Boyle, and W. Oh. Brown Univ. Program in Med., Women & Infants Hosp. of R.I., Dept. of Pediatrics, Providence, R.I.

The NBT test is used as supporting evidence for the diagnosis of sepsis in children and adults. In neonates the previously observed high values in normal subjects preclude such use. Our preliminary observations on 15 healthy term newborns showed that high heparin doses gave falsely elevated NBT values; when heparin dosage was controlled (1 unit/ml NBT solution) consistent values of <20% were obtained in 68 normal infants of 25-43 wks of gestation. Since respiratory distress is a common presenting sign for both hyaline membrane disease (HMD) and neonatal sepsis, we have performed the NBT test in 25 infants with such signs to evaluate the NBT test in the differential diagnosis of HMD vs. sepsis. Endotoxin-stimulated NBT preparations were also performed to insure phagocytic inducibility of the neutrophils. NBT slides were read without knowledge of each infant's clinical diagnosis. HMD was confirmed by classical chest roengenograms and a negative rapid surfactant test in gastric aspirate, and sepsis by positive blood (4 cases) or CSF (1 case) cultures. The results were as follows:

	Normals (n=68)	H.M.D. (n=20)	Sepsis (n=5)
%NBT (M ±SD)	11.3 ±7.5	36.7 ±21.8	73.0 ±9.3
p values	<0.001	<0.001	

Three additional bacteremic infants had no neutrophils on the NBT smears. The NBT test appears to be a reliable indicator of neonatal sepsis and may prove beneficial in the early differentiation of HMD from bacterial infection.

962 EVIDENCE THAT ENDORPHIN (S) MODIFY THE RESPIRATORY RESPONSE TO NEONATAL ASPHYXIA. Victor Chernick, Deborah L. Madansky, and Edward E. Lawson. Dept. of Pediatrics, Harvard Medical School, Boston, MA.

Endorphins are endogenous polypeptides with morphine-like activity which are widely distributed in the central nervous system and bind to opiate receptors. They presumably function as inhibitory neurotransmitters in pain pathways. We tested the hypothesis that endorphins modified the respiratory response to asphyxia in newborn rabbit pups (3-5 days of age). Pups from the same litter were injected I.P. with either 1 ml saline or 1 ml (0.4mg) naloxone, a specific endorphin antagonist. Five minutes later asphyxia was produced by tracheal occlusion and maintained until gasping resumed after primary apnea. Four occlusions were done on each pup and 3 minutes allowed for recovery between occlusions. The time to primary apnea increased by 20% from 44.2±1.5 (S.E.) sec in saline pups to 52.9±1.9 sec in naloxone pups ($p<.001$) while the duration of primary apnea decreased by 60% (45.5±11.1 vs 18.3±2.0 sec) ($p=.01$). The tracheal pressure achieved during the first gasp following primary apnea was identical in saline and naloxone pups (54.2±2.7 vs 54.3±2.5 cm H_2O). Naloxone acts by competitive blockade of opiate (endorphin) receptors. These data therefore provide evidence that endorphins modify the respiratory response to asphyxia by decreasing the frequency of respiratory center discharge but do not appear to decrease the amplitude of the respiratory center output at the time of the first gasp.

963 THE NEED FOR FOLLOWUP OF FAMILIES WHO EXPERIENCE A PERINATAL DEATH. Ronald Clyman, Jane Rowe, Charlotte Green, Cynthia Mikkelsen, Jeannette Haight, Linda Ataide. (Spon. by Roderic Phibbs) Dept. of Pediatrics, University of California, San Francisco.

We conducted a retrospective study by telephone interview (12-24 months later) of 26 families who had experienced perinatal death (7 stillborn, 19 neonatal). 14/26 had had subsequent pregnancies. 6/26 had a prolonged grief reaction (12-20 months). Those mothers with a surviving twin or subsequent pregnancy <5 months following the death were at higher risk for a prolonged grieving period than were those without subsequent pregnancy or one >6 months later ($p<.01$). 12/26 families obtained information about the cause of death and risk of recurrence only during hospitalization; subsequent contact, weeks to months later, provided additional information in 14/26 (by phone in 5, in person in 9). 22/26 mothers met predetermined criteria for having adequate understanding of cause of death and/or risk of recurrence; 4/26 knew neither. Understanding was significantly related to followup contact by phone or in person. 15/22 with adequate understanding were partially or totally dissatisfied with the information they received or the way they received it. Families who received no in person followup contact were more likely to be dissatisfied ($p<.05$). In summary, the presence of a surviving twin or early subsequent pregnancy may be associated with a prolonged maternal grief reaction following perinatal death. Followup by phone or in person resulted in better understanding of the cause of death and risk of recurrence but only in person followup was associated with satisfaction with the information.

964 INFLUENCE OF HEME ON THE KINETICS OF BILIRUBIN BINDING. A.H. Dawodu, J.M. Sutherland, L.E. Becker and W. J. Keenan, Univ. of Cinti., Col. of Med., Dept of Ped. Cincinnati.

Non-bilirubin products of hemolysis have been suggested to reduce the bilirubin binding capacity of serum albumin. If these products interfere with the albumin binding of bilirubin a child with hemolytic disease would be at a higher risk for bilirubin encephalopathy at a given level of serum bilirubin than a child without hemolytic disease. Experiments were conducted to test the hypothesis that heme interferes with the albumin binding of bilirubin. Aliquots of pooled cord sera with increasing concentrations of heme and unconjugated bilirubin were compared to control aliquots without heme. Unbound, unconjugated bilirubin was measured by enzymatic oxidation. Heme concentrations of greater than 7.5μM resulted in significantly higher levels of unbound bilirubin (paired t, $p<0.001$) over a wide range of total bilirubin concentrations. The dissociation constant of the primary binding site for control and heme containing aliquots (Heme=15μM) were 3.9×10^{-8} and 8.3×10^{-8} respectively. The second class of binding sites appeared to be similarly affected. As one of the non-bilirubin products of hemolysis, heme appears to interfere with both the primary and secondary binding sites for unconjugated bilirubin.

965 DEVELOPMENT OF THE NEWBORN'S SUCKING RESPONSE: RELATIONSHIP TO MATERNAL, OBSTETRIC, AND INFANT VARIABLES. Sheryl Ellison, Gene C. Anderson, & Dharmapuri Vidyasagar, Coll. Nrsg., Dept. Pediat., ALSM Univ. of Ill., Chicago.

The sucking response was quantitatively studied in 13 low-birth-weight newborns (LBWN) and 17 normal-birth-weight newborns (NBWN). An electronic suckometer measured maximum intensity pressure for suction and expression components of the sucking response for four consecutive 15-second intervals at 5,15,30,45,and 60 min. of life. At these times LBWN exerted mean suction scores of 5.8, 8.7,3.1,7.8,and 9.3 mmHg and mean expression scores of 14.0,11.9, 13.0,8.5,and 5.0 mmHg. NBWN exerted mean suction scores of 2.1, 25.0,34.0,69.2,and 93.7 mmHg and mean expression scores of 4.6, 5.6,7.3,8.3,and 7.0 mmHg. Vigorous crying in LBWN at 30 min. of life during possibly life-saving treatments (e.g., I.V.'s) and in NBWN at 5 min. of life may explain low suction scores at these times. Positive correlations were found for both LBWN and NBWN between maximum suction score and birth weight ($r=0.62;p<.01$) and maximum suction score and gestational age ($r=0.60;p<.01$) for NBWN between maternal parity and mean suction score at 5 min. ($r=0.58$; $p<.02$) and for LBWN between second stage of labor and mean expression score at 5 min. ($r=0.60;p<.05$). No significant differences were found in sucking scores based on race, sex, maternal medication, or the occurrence of prenatal, intrapartal, or neonatal complications. This investigation shows the sucking response is present even in most low-birth-weight infants during the first hour of life. Giving sucking experience to the transitional newborn may allow earlier safe feedings. The portable suckometer offers new methodology for quantifying sucking and feeding behavior.

966 HEPATIC BILIRUBIN GLUCURONIDATION IN THE HUMAN FETUS. Bertram F. Felsher, Jack E. Maidman, Noemi M. Carpio, and Kenneth VanCouvering. (Spon. by Harry Wright). University of California, Irvine, School of Medicine; V.A. Hospital, Department of Medicine, Long Beach; Charles Drew Postgraduate Medical School, Department of Obstetrics-Gynecology, Los Angeles.

Direct information concerning the fetal development of bilirubin glucuronidation in man has been lacking. Both the activity of the conjugating enzyme, bilirubin UDP-glucuronyltransferase (UDPGT) and the availability of the substrate, UDP-glucuronic acid (UDPGA) may affect hepatic bilirubin glucuronidation. Assays of UDPGT and UDP-glucose dehydrogenase (UDPGD) were performed in the livers of dead aborted human fetuses and the results were compared with values obtained in normal adults. Hepatic UDPGT activity (unit = μg bilirubin conjugated/gm liver/hr in 2 fetuses, aged 17 and 22 weeks was 140 and 220 units, respectively and in 7 fetuses, aged 11-19 weeks was less than 100 units (normal adult = 600-2,000 units). Hepatic UDPGD activity (unit = ηmoles UDPGA formed/100gm liver/min.) in 10 fetuses, aged 10-18 weeks, ranged from 7.7 - 15.0 units (mean ± SEM = 11.7 ± 8.0) and in 8 normal adults ranged from 26.3 - 49.2 units (mean ± SEM = 38.1 ± 3.0) ($p<0.001$). These data show that the in vitro hepatic formation of bilirubin glucuronide and UDPGA is markedly reduced in the human fetus in the first half of gestation compared to normal adults. The relation of these findings to in vivo hepatic bilirubin metabolism and glucuronide conjugation in general in the human fetus and newborn remains to be determined.