†1362 POLYSOMNOGRAPHIC FINDINGS AT DISCHARGE IN INFANTS BORN AT LESS THAN 32 WEEKS OF GESTATIONAL AGE (GA). Julio Perez Fontan, Samuel Hawgood, Gregory P. Heldt, William H. Tooley. Cardiovascular Research Institute, University of California, San Francisco, 94143.

Premature infants, particularly survivors of respiratory distress syndrome (RDS), have an increased incidence of sudden infant death syndrome (SIDS). Also, infants who had an episode of aborted SIDS often have abnormal polysomnographic recordings. To determine whether these abnormalities are present in premature infants at discharge from hospital, we recorded chest and abdominal circumference, respired CO_2, skin surface PO_2 (P_sO_2) and PCO_2 (P_sCO_2), ECG, and esophageal pressure (P_{es}) in 25 infants born at less than 32 weeks of GA. Fourteen infants had RDS defined by clinical and radiological criteria [GA=28 ± 2, birth weight (BW)=1033 ± 265gr, age= 10.8 ± 6 weeks], and 11 had no RDS (GA=30.8 ± 1.3, BW=1250 ± 128, age=6.7 ± 2.4 weeks). Our results show an increased frequency of obstructive apnea (OA), defined as absence of respired CO_2 signal in the presence of respiratory effort; and either a >10 torr decrease in P_sO_2, a heart rate <120, or a P_{es} <-30 torr; in the RDS group (4.9 vs. 1.9 OA/h, P<0.05). Also, more OA episodes were associated with bradycardia in this group (1.9 vs. 0.4 episodes/h, P<0.10). Both central apnea (CA), defined as absence of both respired CO_2 and respiratory effort for more than 10 s, and periodic breathing (PB) occurred with a similar frequency in both groups (0.2 vs. 0.4 CA/h, 9.8 vs. 16.9% PB, in the RDS and NO RDS groups, respectively), in spite of a lower baseline P_sO_2 in the RDS group (67.7 ± 9.7 vs. 59.2 ± 7.9 torr, P<0.05). These findings suggest that infants born at less than 32 weeks of GA have significant sleep abnormalities which may only be detectable by means of polygraphic recordings. OA is particularly frequent in this group, and its association with serious apnea at home needs to be established.

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†1363 CONTINUOUS, DIRECT MEASUREMENT OF ICP BY A SUBARACHANOID BOLT IN ASPHXYIATED TERM NEONATES. R.C. Clancy, R.W. Newell, D.A. Bruce, J. Goplerud, W.W. Fox. Dept. of Peds. & Neurosurgery, Children's Hospital of Philadelphia and Univ. of Pa. Sch.of Med., Phila., PA.

A newly developed infant subarachnoid bolt was used to continuously and directly monitor intracranial pressure (ICP) in four severely asphyxiated (Apgars 1^1 and 2^5) full-term neonates. The plastic bolt was inserted through a dural puncture and secured to the margins of a right frontal bone craniotomy. No hemorrhage, CSF leak, infection or mechanical dislodgement occurred. The bolt was successfully used until the patients' death at postnatal ages 30, 42, 71 and 84 hours. Direct (bolt) ICP values were polygraphically recorded and correlated with simultaneous indirect (transfontanelle) ICP values, mean arterial blood pressure, clinical status and seizures confirmed by EEG. After the transfontanelle pressure transducer was applied with sufficient external tension to produce indirect ICP values that matched direct values, subsequent indirect ICP measurements correlated well from 4 to 13 torr (direct/indirect ratio: M = 1.07; range = 0.8 to 1.5). Indirect ICP measurements consistently and significantly underestimated true ICP from 14 to 35 torr (direct/indirect ratio; M = 2.3; range 2.1 to 3.2). Clinical neurological and/or EEG abnormalities preceded (by age 28 hrs) peak ICP (peak ICP = 18 torr) in all infants. A modest (4 torr) ICP elevation accompanied only 2 of 128 brief (duration = 39 sec; range = 12 to 180 sec), focal electrographic seizures. We conclude that direct ICP measurements by a subarachnoid bolt in appropriately selected, critically ill neonates can be safely achieved and may be more reliable than transfontanelle measurements.

1364 TRACHEAL SECRETION INPACTION DURING HYPERVENTILATION FOR PERSISTENT PULMONARY HYPERTENSION OF THE NEONATE. W.W. Fox, A.R. Spitzer, D. Smith, M. Musci, J.R. Beatty, D.Z. Myerberg. Div. of Neonatology & Respiratory Therapy, Children's Hospital of Phila., Dept. of Peds., Univ. of Pa. Sch.of Med., Philadelphia, PA. & W. VA. Univ. Sch. of Med., Morgantown, WV.

We have recently observed at two institutions the occurrence of tracheal secretion casts or extensive mucous plugging in five neonates hyperventilated for Persistent Pulmonary Hypertension of the Neonate. This finding represents a significant clinical problem that can be recognized by pneumothoraces or CO_2 retention often in the face of adequate oxygenation. Tracheal suctioning or bronchoscopy may be necessary to remove the thick secretions. Five neonates were reviewed with mean BWt 3.51 kg, mean GA 39.6 wks. All infants were on the Bournes Bear Cub infant ventilator and all required max. insp. O_2 concentration of 100%. Ventilation was initiated at a mean of 11 hrs and these infants had been ventilated a mean of 191.4 hrs when the secretions were removed. At the time of recognition of tracheal plugs, mean ventilator settings were: inspired O_2 concentration 92%, rate 80 breaths/min, peak inspiratory pressure 55 cmH_2O, PEEP 4.4 cmH_2O, and mean arterial blood gas values were: PO_2 68, PCO_2 133, pH 7.25. All patients were on pancuronium and all had pneumothoraces associated with tracheal plugging. Large tracheal plugs were removed by suctioning in 2 infants, bronchoscopy in 2 infants and following jet ventilation in 1 infant. An increase in PCO2 during hyperventilation with high rate and pressures and adequate PO2 may signal tracheal plugs. Aggressive suctioning or bronchoscopy may be necessary to remove these plugs. Effective airway humidification systems are necessary for hyperventilation.

1365 INTESTINAL PERFORATIONS SECONDARY TO MECHANICAL VENTILATION WITH NASAL PRONGS (NP) OR FACE MASK (FM) Jeff Garland, David Nelson, Thomas Rice, Josef Neu (Spon. by. C.L. Kien) Medical College of Wisconsin, Department of Pediatrics, Milwaukee Children's Hospital, Milwaukee, WI.

To identify factors contributing to neonatal intestinal perforation (IP), all cases of IP not associated with necrotizing enterocolitis (NEC) or bowel obstruction referred between 1975-1982 were reviewed. Eighteen babies met these criteria. Three met classic criteria for spontaneous perforation. Fifteen were receiving cycled mechanical ventilation by either NP or FM at the time of perforation. Perforations were localized to the stomach in 7, duodenum in 1, jejunum in 1 and ileum in 6. Gestational ages of mechanically ventilated infants ranged from 26-40 weeks (mean=30), weights from 890-2810 grams (mean=1500) and 5 minute Apgar from 1-10 (mean=6). The most common primary diagnosis was RDS. Twelve infants were males and two were enterally fed prior to perforation. Two babies died. All the cases and 57% of the controls were ventilated by NP or FM. To determine if the type of ventilation contributed to perforations, a matched case-control analysis was performed. Each NP or FM ventilated case was matched with four controls for gestation, age at perforation and Apgar scores. Using the Mantel-Haenzel estimate of odds ratio and test for significance for matched pairs, children with intestinal perforation were much more likely than controls to have been ventilated by either NP or FM; odds ratio =23.8 (χ^2_{MH}=35.6, p<0.001). We conclude that mechanical ventilation with NP or FM is associated with an unnecessary risk to babies when compared to ventilation with ET tube.

†1366 DOCUMENTATION OF PRENATAL BRAIN INJURY. Boyd W. Goetzman, Jeffrey A. Lindenberg, William Ellis. School of Medicine, Department of Pediatrics and Pathology, University of California, Davis, California.

The timing of brain injury which leads to neurologic handicap in infants is difficult to establish. However, in infants who die it is possible to estimate the duration of injury by neuropathologic assessment of the state of necrosis, gliosis, alteration of extravascular red cells and calcification.

We reviewed the neuropathologic findings, birth history, and clinical course of neonates autopsied at our center during 1982. Five of the 6 term infants and 10 of the 25 premature infants who died at less than 7 days of age were shown to have brain lesions which predated their time of delivery. Two of the 5 term infants and 7 of the 10 preterm infants had Apgar scores of <3 at 1 min and <5 at 5 min of age. Fetal distress was usually unrecognized and only 1 term and 3 preterm infants were delivered by C-section. Clinical characteristics observed did not suggest a recognizable syndrome of prenatal brain injury. However, the gut and lungs had also been affected prenatally in several cases. Respiratory failure was the usual cause of death.

We conclude that a number of infants dying at less than one week of age have evidence of prenatal brain injury. Birth asphyxia is frequently associated with this finding and this suggests that such infants do not tolerate labor. The probability of prenatal brain injury in surviving neurologically damaged infants with similar birth histories seems high. The medicolegal implications are important and we recommend careful neuropathological evaluation of all such infants who die.

●1367 THE EFFECTS OF LEUKOTRIENE ANTAGONIST FPL 57231 ON HYPOXIC PULMONARY HYPERTENSION (HPH) IN PIGLETS. Ronald N. Goldberg, Tahir Ahmed, Cleide Suguihara, Belsy de Cudemus, Patricia Barrios, Emmalee S. Setzer, Eduardo Bancalari. Univ. of Miami, J.M.H., Dept. of Ped. Miami, Florida.

Leukotrienes (L) have been implicated in the pathogenesis of HPH in adult animals. Elevated (L) levels have also been noted in neonates with persistent pulmonary hypertension. We studied the hemodynamic effects of FPL 57231 in HPH induced in 9 piglets by ventilation with 12% 02. Cardiac index (CI), pulmonary artery (Ppa), systemic arterial (AoP), pulmonary wedge and right atrial pressures were measured, and pulmonary (PVR) and systemic vascular (SVR) resistances and their ratio (PVR/SVR) calculated prior to and after hypoxia (H). These parameters were compared during continued hypoxia between a control group (C) (n,4;wt,3596±1284g; age 17±10 days) and a treatment group (T) (n,5;wt,2958±1130g;age 14±5 days) which received FPL 57231 (2 mg/kg/min x 10 min). Patterns of response were compared by ANOVA.

		BASAL-H	5'	10'	15'	25'	P
$\bar{P}\bar{p}\bar{a}$(mmHg)	C	26± 7	26± 5	26± 6	26± 5	26± 5	
	T	33± 3	27± 4	22± 5	21± 4	23± 4	<.02
PVR (mmHg/	C	92± 39	83± 24	82± 30	81± 24	91± 31	
L/min/kg)	T	127± 26	80± 15	64± 16	57± 12	71± 16	<.008
PVR/SVR	C	.22±.09	.20±.05	.20±.06	.20±.05	.20±.05	
	T	.27±.02	.21±.02	.17±.01	.16±.03	.17±.02	<.02

$\bar{AoP}$ was similar between groups. The decrease in PVR and $\bar{P}\bar{p}\bar{a}$ after FPL 57231 suggest that (L) may in part mediate HPH in piglets. In addition, these effects can be ameliorated by FPL 57231 without significant vascular effects.