1165 NEONATAL VENTILATORY THERAPY: COMPARISON OF FOLLOW-UP RESULTS OF INFANTS WITH BIRTH WEIGHT (B.WT.) <1500 G. AND >1500 G. Annabel Teberg, Paul Y.K. Wu, and Joan E. Hodgman. Univ. of So. Calif. Sch. of Med., LAC-USC Medical Center, Dept. of Pediatrics, Los Angeles.

Several recent follow-up reports on infants surviving neonatal ventilatory therapy showed an incidence of major neurologic and intellectual defects to be only in the range of 10% to 20% for infants of B. Wt. >1500 g, but were as high as 50% for infants of B.Wt. <1500 g. The physical (Phy.), neurologic (Neuro.), developmental (Dev.), audiology (Audio.) and ophthalmologic (Ophthal.) outcome of 42 infants (16 B.Wt. <1500 g, and 26 B.Wt. >1500 g) who received ventilatory therapy at our center were evaluated at ages ranging from one to three years. Incidence of abnormal findings are listed below:

B.Wt.(g)	Phy. No. %	Neuro. No. %	Dev. No. %	Audio. No. %	Ophthal. No. %
<1500 (16)	3 19	3 19	4 25	1 6	2 13
>1500 (26)	4 15	6 23	6 23	3 12	2 8

Although there is a substantial incidence of abnormal findings in both groups of infants, there were no significant differences in the follow-up results of infants of B. Wt. <1500 g and >1500 g, who received ventilatory therapy in the neonatal period.

1166 CLINICAL SIGNIFICANCE OF INTRACRANIAL PRESSURE (ICP) MONITORING IN ACUTELY ILL NEONATES: D. Vidyasagar, T.N.K. Raju, (By invitation) and J. Chiang (By invitation) Dept of Ped. ALSM., Univ. of Ill., Chicago, Illinois.

ICP monitoring was performed by a previously described non-invasive technique using a fiber optic transducer over the anterior fontanel, a Ladd ICP monitor and a pen recorder (Ped. Res. 10, 452, 1976). Anterior fontanel pressure (AFP) was recorded in 113 infants: normal (grp. I) 39, hyaline membrane disease (HMD grp. II) 44, birth asphyxia (grp. III) 5, hydrocephalus (grp. IV) 8, and miscellaneous (grp. V) 17. At the time of recording 7/44 HMD infants had recovered, 37 were on the respirator, 7 of whom were diagnosed to have intracranial hemorrhage (ICH) within 24 hours. The mean AFP $\pm$ SE in cm. of H₂O in the infants studied were as follows:

Group I	Group II on resp. s	Group III on resp. c	Group IV off resp.	Group V
AFP $\pm$ SE: 9.7 $\pm$ 0.4	13.3 $\pm$ 0.6	18.5 $\pm$ 0.4	10.6 $\pm$ 0.6	21.8 $\pm$ 0.4
(n) (39)	(30)	(7)	(7)	(5)

The data showed that ICP was significantly higher in infants with HMD on the respirator and those with ICH ($p < 0.001$). Group III infants also had significantly higher pressure ($p < 0.001$) than grp. I suggesting cerebral edema. All in grp. IV had very high pressure ranging from 15-54 cm. H₂O, and in 4 of these infants a dramatic fall in ICP was documented following shunt surgery. Repeated ICP recordings were also useful in diagnosis of blockade of shunt. We feel that ICP monitoring greatly helped in quantifying pressure changes, diagnosis and therapy of certain acute neonatal disorders.

1167 PARASAGITTAL CEREBRAL INJURY IN NEONATAL HYPOXIC-ISCHEMIC ENCEPHALOPATHY: CLINICAL AND NEURORADIOLOGIC FEATURES. Joseph J. Volpe and Joseph F. Pasternak, Wash. Univ. Sch. Med., St. Louis Children's Hosp., Dept. Ped. & Neurol., St. Louis.

The clinical and pathologic features of hypoxic-ischemic encephalopathy secondary to intrauterine asphyxia has been especially difficult to define in the newborn period. The objectives of this presentation are: 1) to define in the living infant an important pathological feature of neonatal hypoxic-ischemic encephalopathy, and 2) to correlate this pathological abnormality with an observable neurological deficit.

Three infants exhibited the characteristic features of neonatal hypoxic-ischemic encephalopathy as well as a distinctive pattern of weakness of proximal limbs, greater in upper than lower extremities, and a characteristically abnormal technetium brain scan. The abnormality was compatible with ischemic injury to cerebrum, specifically the parasagittal regions and most marked in the posterior aspects. The areas of injury involved those regions that subserve function of proximal extremities, upper more than lower. Thus, a clear clinical-pathological correlation was established. The cerebral lesions observed in our cases are essentially identical to those observed in fetal monkeys subjected to intrauterine asphyxia and define for the first time the human correlate of those experimental lesions. The etiology of the reduction in cerebral blood flow that leads to the injury is unknown. Our demonstration that affected patients may be identified in the newborn period by characteristic clinical and neuroradiologic criteria should facilitate further study of this important problem.

PULMONOLOGY

1168 EFFECT OF SURFACTANT THERAPY ON RESPIRATORY DISTRESS OF PREMATURE LAMBS. Forrest H. Adams, Bernard Towers, Alan Osher, Machiko Ikegami, Tetsuro Fujiwara and Masahiko Nozaki UCLA School Medicine, Department of Pediatrics.

This study was designed to see if the endotracheal delivery of natural surfactant (NS) prior to the first breath produces a favorable effect on the lungs of prematurely delivered lambs with clinical respiratory distress. Ewes with known breeding dates and twins were delivered by C-section between 120-122 days of gestation, a time when lung surfactant is not normally present and respiratory distress develops. Each twin fetus was ventilated for 2 hours in room air with a volume respirator at 3 cm H₂O PEEP. On an alternate basis, one twin received NS and the other saline prior to the first breath. NS was isolated from 2 day old newborn lambs. The quantity of NS instilled was estimated from its saturated lecithin content and was 4 fold the theoretical amount required to cover the total pulmonary alveolar space with one lipid-protein duplex. Studies of excised surfactant-deficient immature lamb lungs have shown that this amount of NS is capable of improving the pressure-volume characteristics at 37°C. Six lambs received surfactant. In comparison with twin controls the following results were observed: better blood gases; improved effective lung compliance; longer survival; better gross appearance of lungs; better pressure-volume curves; normal surface tension of lung extracts; and better histologic appearance of the lungs. These results support the view that the endotracheal delivery of natural surfactant may be a valuable tool in the treatment of RDS.

1169 ALPHA 1 ANTITRYPSIN DEFICIENCY IN THE PATHOGENESIS OF BRONCHOPULMONARY DYSPLASIA, Jim G. Adams, R. Chandra, Spon. by Joseph A. Bellanti, Georgetown University and Children's Hospital of D.C., Dept. of Neonatology, Washington, D.C..

Bronchopulmonary Dysplasia (BPD) is a major cause of neonatal respiratory morbidity and mortality. The pathophysiology of BPD may be related to an absence of the antiprotease alpha 1 antitrypsin (α 1at) in pulmonary tissue. Post-mortem pulmonary tissue was examined from 28 infants, histologically and by direct immunofluorescence. Histopathology documented 13 with BPD (Stage II-IV), 7 with Idiopathic Respiratory Distress Syndrome (IRDS), 5 with non-IRDS pulmonary injury, and 3 controls dying of non-respiratory causes. Direct immunofluorescent staining with fluorescein tagged antiserum specific for fibrinogen, IgG, α 1at, and α 2 macroglobulin and albumin revealed a striking difference in fluorescence between infants dying with IRDS and BPD. α 1at fluorescence was virtually absent in BPD (2/13) while infants with IRDS fluoresced uniformly (7/7). Infants in the non-IRDS pulmonary injured population demonstrated erratic fluorescence varying with the etiology of the pulmonary insult. Failures to fluoresce with α 1at were seen in pulmonary edema, atelectasis with hyaline membranes and pulmonary hemorrhage, suggesting a specific type of pulmonary insult is required for the diffusion of α 1at, i.e. IRDS. The relative absence of α 1at may be significant in the development of progressive BPD. In response to oxygen, pressure and ventilation alveolar macrophages and migrating leukocytes, relatively uninhibited by α 1at, may elaborate proteolytic enzymes that destroy alveolar microarchitecture leading to the clinical, pathological, and radiographic findings consistent with BPD.

1170 COMPARISON OF AIR AND HELIUM-OXYGEN FLOW VOLUME CURVES IN TWO-DAY OLD INFANTS Saul M. Adler & Alfonso Vargas (Spon. by Lewis A. Barnes) University of South

Florida College of Medicine, Department of Pediatrics, Tampa, FL To determine if the large or small airway is the major site of limitation to air flow in the newborn infant, we obtained partial maximal expiratory flow volume (MEFV) curves by passively increasing transpulmonary pressure in infants while they were breathing air and 5 minutes after they breathed an 80% helium, 20% O₂ mixture (He+O₂). Parental permission was obtained and 19 clinically normal term newborn infants, 3.18 $\pm$ 0.42 kg (mean $\pm$ 1 SD), were studied between the second and third day of life. The maximal expired volume (mev) below functional residual capacity (FRC) was determined. Maximal expiratory flow rates at FRC ($\dot{V}_{max}$ FRC) and at a pulmonary volume below FRC equivalent to 50% of the maximal expired volume ($\dot{V}_{max}$ mev 50) were measured.

	$\dot{V}_{max}$ FRC (ml/sec $\pm$ 1 SD)	$\dot{V}_{max}$ mev 50 (ml/sec $\pm$ 1 SD)
air	171 $\pm$ 67	104 $\pm$ 38
He+O ₂	197 $\pm$ 88	94 $\pm$ 48

There were no significant differences ($p > .05$) in mean flow rate. Only 6 of 19 subjects had $\dot{V}_{max}$ FRC in helium greater by 30% or more than in air, and only 2 of these 6 had a $\dot{V}_{max}$ mev 50 in helium that was greater than in air. Failure of flow rates to increase in the He+O₂ mixture at these pulmonary volumes suggests that in healthy newborn infants, the small airway is the major site of resistance to flow limitation. Thus conditions causing airway irritation that result in even minor narrowing of the small airways could have serious consequences to newborn infants.