ROLE OF THE LYSOLECITHIN PATHWAY IN THE FORMATION OF DISATURATED LECITHIN IN DEVELOPING LUNG. M. Hallman and K. Raivio, Univ. Helsinki, Children's Hosp., Dept. Ped., Helsinki, Finland, and Univ. of Calif., San Diego, Sch. of Med., Div. Ped., Div. Perinatal Med. (MH). (Intr. L. Gluck).

Lung lecithin once formed *de novo* may be affected by deacylation-reacylation loop, which transforms unsaturated to disaturated lecithin, with superior surfactant qualities. This hypothesis was studied in rabbit lung slices. 1-Acyl-2-lysolecithin-palmitate-14C (Ly), liver lecithin-palmitate-14C (Le), CDP-choline-methyl-14C (Col), and S-adenosyl methionine-methyl-3H (Met), were the precursors. Ly was effectively converted to lecithin in all age groups studied. The distribution of the radioactivity in disaturated versus total lecithin was as follows:

	Time min	Fetus		1 day	Adult
		24-26 days	28-30 days		
Ly	60	0.16	0.31	0.30	0.33
Le	150	0.14	0.21	0.18	0.22
Col	10	0.11	0.13	0.10	0.14
Col	150	0.14	0.22	0.19	0.24
Met	60	0.02	0.03	0.03	0.02
Cons*		0.21	0.33	0.29	0.31

*Ratio of the contents of disaturated to total lecithins.

The results suggest that in the lung the lysolecithin pathway has an important role in providing surfactant lecithin. Further evidence using primates is needed to prove the clinical significance of this assumption.

NASAL END-EXPIRATORY PRESSURE (NEEP) IN THE MANAGEMENT OF HYALINE MEMBRANE DISEASE (HMD) H Harris*, Y Brans*, S Wilson, and G Cassidy. University of Alabama in Birmingham

End-expiratory pressure utilizing nasal prongs was used in 43 babies, 30 with clinical HMD, during a 7 month period. In the HMD group, $\bar{x}$ birth weight was 2020g (8<1500g) and $\bar{x}$ gestation was 33½ wks. PaO_2 was <60mmHg in $\geq 60\% \text{FiO}_2$ in all HMD prior to NEEP. Treatment was begun at $\bar{x}$ 23½ hrs. after birth and $\bar{x}$ duration of treatment was 43 hrs. Pressures employed ranged from 6-15cmH₂O, the mouth not taped shut, and gastric decompression was routine. Within $\bar{x}$ 36 minutes of treatment, $\bar{x}$ PaO_2 nearly doubled in stable FiO_2 ($\bar{x}$ PaO_2 47 $\pm$ 17.5 before and 80 $\pm$ 36.2 after; $\bar{x}$ FiO_2 72% before and 73% after). PaCO_2 and pH were unchanged. In 18 in whom NEEP was begun within 24 hrs. of birth, $\bar{x}$ ΔPaO_2 of 116% was significantly higher than the 35% of those 12 treated later ($p < .025$). The ΔPaO_2 exceeded 50% in 12/18 early treated and in only 3/12 late treated ($p < .01$). Prognosis was not predicted by initial PaO_2 response: $\bar{x}$ ΔPaO_2 was 79% in survivors and 96% in those who died. Of the 25 survivors, only 1 required further ventilatory support, while in none of the dying babies was NEEP failure corrected by subsequent endotracheal CPAP and/or mechanical ventilation. FiO_2 of $\bar{x}$ 73% was decreased to <60% in $\bar{x}$ 15 hrs. (range ½-86). In 13 babies with other causes for respiratory difficulty, NEEP failed to significantly affect PaO_2 ($\bar{x}$ 49 before; 69 after). These data suggest that NEEP is a simple, non-invasive, effective technique in the management of HMD. Our only complication has been pneumothorax, occurring in 3 infants who survived.

TRACHEAL ASPIRATE (TA) LECITHIN SPHINGOMYELIN RATIO (L/S) AND RECOVERY FROM RDS. William P. Kanto, Jr., Robert C. Borer, Jr., Mason Barr, Jr. and Dietrich W. Roloff (Intr. by William J. Oliver), Univ. of Michigan Med. Ctr., Dept. of Ped., Ann Arbor.

Previously we have demonstrated that changing TA L/S were associated with survival from RDS. In this current report L/S of serial TA obtained during the first 120 hrs. of life were examined in 30 infants with severe RDS requiring endotracheal intubation. The infants were divided into two groups by survival: survivors (S), n=19; non-survivors (NS), n=11. The means of all L/S values per day in infants with at least three samples per 24 hr. period were calculated. In S 10/14 (71%), 10/12 (83%) and 9/9 (100%) infants had mean L/S > 2.5 on days 3, 4 and 5 respectively. In NS 1/5 (20%), 1/6 (17%) and 1/7 (14%) infants had mean L/S > 2.5 on days 3, 4 and 5 respectively. This difference was significant by day 4 ($p < 0.05$). Both S and NS were similar in their requirement for ventilatory support at 48 and 72 hrs. of life. At 96 and 120 hrs. there were significantly fewer S requiring mechanical ventilation. Thus, the attainment of a TA L/S > 2.5 by day 4 appears to be a chemical predictor of survival in infants with severe RDS.

A significant observation in this study was that the attainment of a TA L/S > 2.5 on day 4 was independent of gestational age. This observation suggests that TA L/S is related to postnatal factors.

MIDDLE EAR FUNCTION OF NEONATES. Robert W. Keith, University of Cincinnati College of Medicine, Department of Otolaryngology, Cincinnati, Ohio (Intr. by Irwin Light)

Results of impedance audiometry can determine middle ear pressure, eustachian tube function, tympanic membrane mobility, and presence of fluid in the middle ear cavity. The present study utilized an impedance audiometer to measure middle ear function of neonates.

Subjects included 20 infants with a mean age of 7.4 hrs. Their average weight was 3120 grams. All were healthy with no obvious congenital defects. Both ears were tested on every baby. The vernix caseosa was removed from the external auditory meatus prior to taking impedance measurements.

Results indicated that all of the infants had normal tympanic membrane mobility and normal middle ear pressure. The data show 33 ears with Type A₁ tympanograms and 7 Type W tympanograms. The average middle ear pressure was 6.75 mm. (H₂O). The average compliance at the tympanic membrane was 1.113 cu. cm.

Many pediatric textbooks state that the middle ear of neonates is filled with a mucoid material which is present for the first few days of life and is a factor in the ability of newborns to respond to sound. The finding of normal middle ear function in neonates would suggest that mucous is not commonly present and therefore not a factor in infant's response to sound.

ANTEPARTUM BETAMETHASONE IN THE PREVENTION OF RESPIRATORY DISTRESS SYNDROME. Joseph L. Kennedy Jr. (Intr. by S.S. Gellis) Tufts Univ. Sch. of Med. & St. Margaret's Hosp., Dept. of Ped., Boston

Thirty-nine infants were born to women receiving betamethasone for threatened premature (6-13 wks) delivery using the regime of Liggins. Infants over 27 wks and under 6 lbs whose mothers had been treated 24 or more hrs prior to delivery were matched with infants of similar weight, gestational age (G.A.) and status of membrane rupture. There were no differences between control and study patients in sex ratio, clinic vs. private status, or antepartum bleeding. 11 treated and 5 control infants were delivered by section. 3 treated and no control mothers received alcohol. 25 in each group had ruptured membranes for median times of 75 hrs (treated) and 48 hrs (controls). Median time of drug administration was 70 hrs prior to delivery. Results were as follows:

Group	n	WT:lb-oz	GA:SE (WKS)	RDS	ASST VENT	DIED
<4 lbs.-Rx	13	3-2:2	31.2:5	0	0	0
" Control	13	3-2:3	30.7:5	7*	6	5#
3-4 lbs.-Rx	12	4-7:1	34.2:3	1	1	0
" Control	12	4-7:2	34.5:3	6	3	0
5-6 lbs.-Rx	14	5-9:1	34.9:3	0	0	0
" Control	14	5-5:2	35.6:4	0	0	0
Total Rx	39			1**	1	0
" Control	39			13**	9**	5#

* $p < 0.01$
$p = 0.02$
** $p < 0.001$

Betamethasone at least 24 hrs prior to delivery appears effective in preventing RDS in infants under 5 lbs. & 34 weeks.

EFFECTS OF INTRA-UTERINE ASPHYXIA ON NEONATAL BLOOD VOLUME. Joseph A. Kitterman and Mureen A. Schlueter (Intr. by R.H. Phibbs). NHLI Sp. Ctr. of Res.-Pulm. Dis., CVRI and Dept. of Ped., Univ. Calif., San Francisco, California

We studied effects of intra-uterine asphyxia on neonatal blood volume in 11 fetal lambs (gestational age 130-147 days, birth weight 2.65-4.71 kg) with the ewes under spinal anesthesia. Fetal and maternal blood gases, pH and blood pressures were measured. We determined feto-placental red blood cell volumes (RBCV) with Cr⁵¹ tagged red cells and plasma volumes (PV) with I¹²⁵ albumin. In 6 animals varying degrees of asphyxia were produced by maternal hypotension caused by hemorrhage; in 5 no attempt was made to cause fetal asphyxia. The lambs were delivered; umbilical cords were clamped before the first breath. Ranges of fetal hind limb arterial blood gases and pH at birth were oxygen tension (Po_2) 9-24 mmHg, pH 6.98-7.41, carbon dioxide tension (Pco_2) 34-70 mmHg. After resuscitation, neonatal RBCV and PV were measured with a second injection of the same labels. We expressed neonatal blood volumes both as ml/kg and % of feto-placental volumes (%FP). Neonatal RBCV and PV (ml/kg) correlated closely with birth weight ($p < 0.01$). Neither RBCV nor PV (ml/kg or %FP) correlated with Po_2 , pH or Pco_2 at birth. However, neonatal RBCV (%FP) had an inverse correlation with the fall in maternal mean arterial pressure before birth ($p < 0.05$). These data indicate intra-uterine asphyxia has little effect on neonatal blood volumes, but acute maternal hypotension immediately before birth may lead to neonatal hypovolemia.