THE EARLY USE OF CONSTANT POSITIVE AIRWAY PRESSURE (CPAP) IN HYALINE MEMBRANE DISEASE (HMD). Richard W. Krouskop, Edwin G. Brown, and Avron Y. Sweet. Case Western Reserve Univ. at Cleveland Metropolitan Gen. Hosp., Dept. of Ped., Cleveland.

CPAP improves PaO_2 in HMD, but usually is not applied until 70-80% O_2 is required. Fourteen prematures met strict x-ray and clinical criteria for HMD and were paired by weight and severity of disease. Each infant was placed in 40% O_2 until the PaO_2 fell below 60 mmHg, when one of each pair (Group A) was immediately provided nasal CPAP. The other in the pair (Group B) was continued on to 70% O_2 as necessary. CPAP was begun when the PaO_2 again fell below 60 mmHg. All Group B infants qualified for CPAP. The onset of CPAP therapy averaged 7 hrs later in Group B. There was no significant difference between the two groups in morbidity, mortality or duration of O_2 exposure. CPAP caused no rise in $PaCO_2$ in either group. The average duration of CPAP for Group A was 58 hrs and 73 hrs for Group B ($P > .05$). Group A never required greater than 50% O_2 during CPAP. Group B infants generally required 80% O_2 at some time and remained over 70% an average of 33 hrs. Infants who cried during nasal CPAP exhibited dramatic transient falls in PaO_2 and $PaCO_2$ as well as wide fluctuations in pressure. By 5 hrs of age, 7 of 8 cases under 2000 gms. had a $PaO_2 < 60$ mmHg in 40% O_2, and 7 had x-rays characterized by reticulogranularity, air bronchograms and indistinct heart borders.

Early use of CPAP in this study was not associated with an increased incidence of complications, did lessen the severity but not the duration of the disease, and avoided the necessity of exposure to high levels of environmental oxygen.

CONTINUOUS NEGATIVE PRESSURE IN HYALINE MEMBRANE DISEASE: EARLY VERSUS LATE ONSET. Lee Mockrin and Eduardo Bancalari (Intr. by W. W. Cleveland) Dept. of Ped., Univ. of Miami, Miami, Florida

Nineteen infants with hyaline membrane disease (HMD) were studied to determine if the time of initiation of continuous negative pressure (CNP) affected the course of the disease. All infants had an initial PaO_2 between 50-100mmHg on 0.7FiO_2. They were then randomly assigned to one of two groups: the early-onset group (n=9, mean B.W. 1626 Grams) started on CNP while the PaO_2 was between 50-100mmHg on 0.7FiO_2; the late-onset group (n=10, mean B.W. 1866 Grams) started on CNP after the PaO_2 fell below 50mmHg on 0.7FiO_2. Results are as follows:

	CNP ONSET (Hours)	ΔPaO_2 (mmHg)	VENT. ASSIST. (Hours)	$\geq 0.7FiO_2$ (Hours)	$\geq 0.4FiO_2$ (Hours)
Early: $\bar{x}$	9.9	97.6	38.0	7.1	53.5
Range	2.4-18.8	26-212	13.4-54.7	0.7-39.1	6.8-116.8
Late: $\bar{x}$	15.8	47.0	80.9	32.9	110.1
Range	5.2-24.5	9- 79	31.2-184	4-51.8	37.7-282.5

The early-onset group responded better to CNP in terms of the initial increase in PaO_2 (ΔPaO_2), required a shorter period of oxygen therapy, and needed a shorter period of ventilatory assistance which consisted only of CNP. Three of the late-onset group required mechanical ventilation and four had either pneumomediastinum, pneumothorax, patent ductus arteriosus requiring ligation, or right diaphragmatic paralysis. There were no complications in the early-onset group. This study indicates that it is desirable to begin CNP as soon as possible in infants with HMD.

PROLONGED "GESTATION" FOLLOWING FETECTOMY IN MONKEYS. Jonathan T. Lanman, Rosemarie B. Thau, S. Kalyan Sundaram, Ashley O. Brinson. The Population Council, Rockefeller Univ., New York.

A fetal influence over gestation time is recognized in several mammals; clarification of the mechanisms involved might suggest approaches to the prevention of prematurity. Fetal pituitary or adrenal defects lead to prolonged gestation, and ACTH or glucocorticoid administration can induce premature labor. We have attempted to distinguish fetal and placental mechanisms by performing fetectomy in macaques. Fetuses were removed from 10 monkeys at gestation ages from 65 to 143 days; placentas were left in situ. Of the 10 placentas, 7 were carried past normal term (155-176 days), and were delivered at 181 to 243 days. Four were delivered spontaneously and 2 after giving dexamethasone; 1 was removed surgically. At fetectomy, plasma progesterone values were highest in the umbilical vein, lower in fetal heart and uterine vein, and lowest in maternal peripheral vein. Following fetectomy, maternal progesterone values remained at or above normal pregnancy levels until placental expulsion or shortly before. Following expulsion, values fell promptly to non-pregnancy levels. Estradiol values (5 animals) fell within 7 to 19 days after fetectomy to 1/3 of pre-fetectomy levels and the rise normally seen before delivery did not occur prior to expulsion. These results indicate that in the absence of the fetus, delivery of the placenta is usually delayed, but that the ability of a glucocorticoid to precipitate expulsion remains. Maternal plasma progesterone levels are maintained, but estradiol levels fall.

PROPHYLAXIS AGAINST BICARBONATE INDUCED HYPOCALCEMIA IN INFANTS WITH RDS. Cecilia T. Nervez, Roger J. Shott, William H. Bergstrom and Margaret L. Williams. State University of New York, Upstate Medical Center, Syracuse, New York.

Infants with the respiratory distress syndrome (RDS) often become hypocalcemic after $NaHCO_3$ therapy. This report concerns the effectiveness of prophylactic calcium infusion (CaIV) in prevention of this complication. Forty-eight infants with birth weight less than 2000 gm (mean 1498), gestational age 28-36 wks., and varying degrees of RDS were studied within 48 hrs. of birth. Serum Ca was measured at the beginning and end of a 24 hr. study period during which all urine was collected. Twenty-three subjects received CaIV at 1 mg/kg/hr for 24 hrs. In 4 groups of infants, arranged by treatment, the outcome was as follows:

Group	#	$NaHCO_3$	Ca Infusion	Serum Ca Initial	24 Hr.	P
A	16	-	-	8.6	8.0	.05
B	7	-	+	8.1	8.4	.6
C	8	+	-	8.3	6.7	.001
D	16	+	+	8.1	8.1	.9

The data show that CaIV prevented hypocalcemia without inducing hypercalcemia. Urine Ca was below 3.2 mg/24 hr. in all groups. Ca leaving the plasma during the advent of hypocalcemia, as well as the much larger amounts retained during CaIV, probably formed bone mineral, as urinary Ca did not account for the Ca shifts implicit in the data.

RETINAL OXYGEN TOXICITY. Andrew Q. McCormick*, Gordon E. Pirie*, and Sydney Segal. Univ. of British Columbia Faculty of Med. and Vancouver Gen. Hosp. Vancouver. Depts. of Ped. and Ophthalmology, Vancouver, B.C.

A study has been carried out in 1246 high risk newborn infants to determine the frequency, evolution, variations and severity of retinal vascular changes secondary to oxygen therapy administered in the neonatal period. They ranged from 480 to 5,640 gm in birthweight and 26 to 44 weeks in gestational age. Retinopathy of prematurity (retrolental fibroplasia) was diagnosed in 40, of whom 2 are blind from end-stage cicatricial disease. Changes in the others varied from minor neovascularization of the temporal peripheral retina to more extensive vessel proliferation and from peripheral retinal scarring to traction and distortion of the retina at the posterior pole of the eye. Correlations between severity of the disorder and birthweight and gestational age will be presented as a basis for a new and more useful classification of the disease.

POST MORTEM PULMONARY ARTERIAL INJECTION STUDIES IN HYALINE MEMBRANE DISEASE. J. F. O'Connor, David Ingall and Salvadore Castro. Department of Pediatrics and Radiology, Boston University School of Medicine, Boston City Hospital, Boston, Mass. (Intr. by Robert Klein)

Immediate postmortem injection of radiopaque modified Schlesinger mass into the pulmonary arteries of infants dying of hyaline membrane disease and a control group dying of other causes revealed marked discrepancies between the degrees of small vessel filling. The lungs in hyaline membrane disease revealed the typical muscular arterial tortuosity and pruned tree appearance of pulmonary hypertension as manifest on in vivo pulmonary angiography. The lungs of the control group revealed perfusion of all arterial and arteriolar branches as seen in normal pulmonary arteriograms suggesting that pulmonary hypoperfusion plays a role in the causation or may be the result of hyaline membrane disease. These findings support previous clinical demonstrations of pulmonary hypertension and pulmonary hypo-perfusion in this disease.