1290 HEMODYNAMIC EFFECTS OF HIGH FREQUENCY OSCILLATORY VENTILATION (HFOV) IN PRETERM LAMBS. Kouki Oguchi, Barry G. Baylen, Machiko Ikegami, Harris Jacobs, David Berry, Alan Jobe, George C. Emmanouilides.

The effects of (HFOV) on the preterm cardiovascular system have not been evaluated. We studied 14 surfactant-treated pre-term twin lambs (120 days gest. age) after occlusion of the ductus arteriosus with a balloon catheter. Left ventricular (LV) performance was evaluated using cineangiography and radio-nuclide labeled microspheres. Studies were obtained after 2 and 3 hrs of conventional ventilation [CV] (Grp A, n=7) or HFOV (Grp B, n=7). HFOV was fixed at 15 Hz (900/min) and settings were adjusted to maintain similar blood gases in both groups. The following measurements were obtained: cardiac output (CO) ml·min⁻¹·kg⁻¹, organ flows (% distribution,); LV ejection fraction (EF %), end diastolic volume (EDV); aortic:pulmonary mean (AO/PA) and mean airway pressures (MAP); ratio PaO₂/F_iO₂.

m(±SE)	MAP	PaO ₂ /F _i O ₂	pH	HR	CO	EDV	EF	AO/PA	Heart(%)	GI(%)
A(2hr)	13	90±12	7.31	185	124±14	2.1	63	1.3	11.1	7.0
B "	12	62±7	7.32	175	97±12	1.7	53	1.5	11.9	9.3
A(3hr)	13	125±24	7.27	163	168±37	2.0	66	1.1	9.7	8.8
B "	11	83±20	7.27	176	119±25	1.9	61	1.3	14.6	9.0

At similar end diastolic pressures and systemic resistances, and at comparable MAP and blood gases, there were no significant differences of LV performance or blood flow distribution with HFOV or CV (Wilcoxon test). Oxygenation (PaO₂/F_iO₂) was similar in the groups. HFOV is an equally effective mode of ventilation and has no apparent deleterious effects upon the preterm cardiovascular system.

1291 TRANSIENT PULMONARY VASCULAR LABILITY (TPVL)- IMPORTANCE OF RECOGNITION AMONG LGA INFANTS AT RISK FOR PERSISTENT FETAL CIRCULATION (PFC). Bedford W. Bonta, CAPT MC USN (Spon. by F. Stanley Porter). Dept. of Pediatrics, United States Naval Hospital, Portsmouth, Virginia.

Infants at risk for development of PFC may require hyperventilation (HV) & muscle relaxation (MR) to improve lung compliance and oxygenation. During the past 4 months (6-10/83), we have identified a separate population of infants who may present initially with symptoms indistinguishable from those infants who develop PFC but who respond to hyperoxia (FIO₂ = 1.00) and can be successfully managed with oxygen without the need for HV and MR. We studied 12 infants (BW @ 3997.5, GA @ 40.1), 8 with evidence of perinatal aspiration, and compared their initial response to oxygen with 16 infants (BW @ 3102.5 gm, GA @ 38 wk) who required HV & MR for therapy of PFC this past year. No significant differences were noted in PaO₂ response in FIO₂ = 0.21 or 0.40 among TPVL or PFC infants. A significant rise in PaO₂ was observed among TPVL infants, but not PFC infants, when exposed to FIO₂ = 1.00 (250.7 torr vs 86.0 torr, p<.0001). No significant difference in pH or PaCO₂ were observed. Avg. hours of oxygen (48.8 vs 273.7, p<.0001) and average hours of increased pulmonary vascular lability (LI) 5:1 (12.3 vs 87.4 hrs, p<.0001) were significantly different between groups. All infants survived. TPVL appears to present among term or post-term LGA infants who have experienced perinatal factors known to be associated with an increased risk of PFC. Among these infants, endotracheal intubation may be avoided, substantially reducing the risk of morbidity associated with HV & MR usually required among infants with PFC.

1292 VOLUME REPLACEMENT IN INFANTS WITH PERSISTENT FETAL CIRCULATION (PFC) TREATED WITH HYPERVENTILATION (HV) & PAVULON (P). Bedford W. Bonta, CAPT MC USN & Mark E. Swanson, LT MC USN (Spon. by E. Stanley Porter). Dept. of Pediatrics, United States Naval Hospital, Portsmouth, Virginia.

Effective mechanical ventilation of infants with PFC often requires simultaneous administration of P to improve lung compliance and decrease the risk of air leaks. During the past year, we have studied 18 infants (Pr) (BW @ 3222.8 gm; GA @ 38.4 wks) using CVP catheters and volume replacement (VOL), either as colloid or PRBCs, to maintain CVP>5 torr, U₀>2 cc/kg/hr and U_{sg}<1.010 while requiring HV & P for PFC. The controls (C) consisted of 16 infants studied retrospectively (BW @ 3018.8 gm; GA @ 39.3 wks) who required HV & P for PFC. 16/18 Pr survived (88.9%) while only 8/16 C survived (50%, p<.01). Among the C group, survivors (CS) and non-survivors (CNS) were compared separately to Pr with respect to avg. MAP, FIO₂, PaCO₂, U₀, U_{sg}, CVP, and VOL during each 8 hour interval for 160 hours. Hours of O₂, HV and P were similar among groups. Avg. MAP, PaCO₂ and U_{sg} measurements were significantly lower among Pr compared to CNS (p<.005, <.005, <.0001), while avg. PaO₂ & U₀ were significantly higher among Pr compared to CNS (p<.0001, <.0001). Significant differences in VOL were observed, especially during the first 72 hours of P. During the entire study period, CS & CNS received <35% VOL compared to Pr (38.7; 59.7 vs 169.0 cc/kg; p<.005, <.05). Tolazoline was ineffective in both C & Pr except when PaCO₂ <25 torr (3/22, 13.6% treated) and did not improve survival. Use of VOL to compensate for peripheral loss of circulating blood volume during P appears to improve survival among infants with PFC.

1293 NEUROPATHOLOGIC ASSESSMENT AND CLINICAL CORRELATES OF CHOROID PLEXUS HEMORRHAGE (CPH) IN A NEONATAL AUTOPSY POPULATION. Marylou Behnke, Emmalee S. Setzer, William E. Ballinger (Spon by Willa H. Drummond), Depts of Peds and Path, U. of FL, Gainesville and U. of Miami, Miami, FL

To determine the incidence of neuropathologically-proven CPH and its correlation to birthweight (BW), gestational age (GA) and intrauterine growth, we reviewed the clinical courses and cranial postmortems of 81 consecutive neonates >500g BW who died between 6/1/80-5/31/82. Study cases represented 63% of all NICU deaths with specific exclusions for CNS malformations, severe brain autolysis and lack of parental permission for cranial post-mortem. All post-mortem specimens were reviewed by a neuropathologist to determine intracranial hemorrhage (ICH) sites and extension.

No hemorrhages were detected in 26 infants. Fifty-five of 81 infants had ICH (68%). Sites of ICH included periventricular (PVH;n=43) intraventricular (IVH;n=38), cerebellar (CBH;n=4), subarachnoid (SAH;n=49), and choroid plexus (CPH;n=9) areas. CPH accounted for 16% of the infants with ICH. Five of the 9 infants with CPH were <1500g BW and appropriate for GA (AGA). Two infants were between 1501-2500g BW (one AGA; one small for GA (SGA)). Of the 2 infants >2500g BW, 1 was AGA and 1 was large for GA (LGA). Isolated CPH occurred in only 1 infant (Term,LGA). CPH was the only site of initiation of IVH in 4 infants (1 Term AGA; 1 Term SGA; 2 Pre-Term AGA). The 4 remaining infants with CPH also had PVH with IV extension (4<1500g, AGA). This study defines the incidence of CPH in a neonatal autopsy population and shows that CPH occurs not only in gestationally mature infants but frequently occurs in pre-term AGA infants as well. It also shows that since CPH rarely occurs as an isolated phenomenon in those babies who die, the pre-mortem diagnosis of CPH may be obscured by the existence of other sites of ICH.

†1294 PERIVENTRICULAR NECROSIS IN PRETERM INFANTS: SONOGRAPHIC OBSERVATIONS. Raul Bejar, Ronald Coen, T.A. Merritt, Jill Trice, Sami Ikonen, Mary J. Felice.

Univ. of Calif. San Diego, Dept. of Ped. & Neurol. La Jolla, CA
 Periventricular cerebral white matter necrosis (PVN), common in preterm infants dying in the 1st week, is associated with motor abnormalities in survivors & is characterized by necrosis, vascular congestion & hemorrhage. Cavitation develops in the necrotic area. The clinical aspects of PVN are unknown since the diagnosis is done at autopsy. With ultrasound (US) we studied this lesion in 10 preterm infants <2000 gm finding 2 distinct phases. Initially, there are very echogenic zones adjacent to atria and anterior horns. Later, cystic lesions develop within these regions. Echogenic areas were found as early as 1 hr after birth & cysts were found on day 1 or between 4 & 23 days. PVN was associated with asphyxia, hypoxia, hypotension &/or apnea. Cysts were present in 2 infants without clinical problems. Post-mortem examinations of the brains in 4 infants found congestion, petechial hemorrhages, perivascular bleeding &/or necrosis in the echogenic zones. Computerized tomograph (CT) showed periventricular areas of low attenuation & did not display cysts, congestion or small hemorrhages. Two infants with large cysts in US had "normal" CT studies by the 5th month. By the 2nd yr these 2 infants had spastic quadriplegia. Four infants are <6 mos. old & are being followed. Two have abnormal neurologic exams. PVN can be diagnosed & followed with US. The presence of cysts on day one indicate that in some patients PVN may develop in utero.

†1295 HIGH FREQUENCY VENTILATION IN THE MANAGEMENT OF HYALINE MEMBRANE DISEASE. R Bell, T Kuehl, M Escobedo, D Null, J Coalson, N Ackerman, R deLemos. Departments of Pediatrics and Pathology, Wilford Hall USAF Med Cen, University of Texas Health Sciences Center, Southwest Foundation for Research & Education, San Antonio, TX. Spon by: Robert Kotas

Controlled 24 and 96 hour experiments compared the efficacy of HFV and an intermittent "sigh" to PPV/PEEP in the management of premature baboons with HMD. Twenty-five animals were delivered by hysterotomy at 140+/-3 days, resuscitated and managed with conventional ICU techniques. Ten of the animals were placed on either HFV or PPV and sacrificed after 24 hours. The a/A ratio was maximized by adjustment of the MAP. Frequency was kept at 10Hz in the HFV animals and pCO₂ regulated by adjustment of V_T. The a/A ratio was higher and mean airway pressure (MAP) lower in the HFV animals during the 24 hr period. Lungs of the HFV baboons were more evenly aerated at necropsy. Fifteen additional premature baboons were ventilated for 96 hours using an oscillator (HFO) or flow interrupter (HFFI) at 10Hz or PPV/PEEP. Clinical management was as in the first experiment. a/A ratios were higher and MAP lower in the high frequency groups in the first 48 hours; these differences were not present by 96 hours. In both experiments, HFFI resulted in better oxygenation at lower pressures than did either HFO or PPV, but led to more pressure-related lung injury. Animals ventilated with HFO had an abundance of active type II pneumocytes with a stratified surface layer. Hepatic derangement was also seen in this group. These studies suggest that HFV with an appropriate strategy decreases both oxygen and pressure exposure early in the course of HMD.