175 FACTORS INFLUENCING DIAGNOSIS (DX) OF VENTRICULAR SEPTAL DEFECT (VSD) BY PULSED DOPPLER ECHOCARDIOGRAPHY (PDE). James G. Stevenson, Isamu Kawabori, and Warren G. Guntheroth. Department of Pediatrics, University of Washington School of Medicine, Seattle.

The DX of VSD by PDE can be made by following the harsh VSD jet through the ventricular septum (IVS). The presence of a discrete jet in the right ventricular (RV) apex is presumptive evidence of a low or muscular VSD. 31 youngsters with catheterization proven left to right shunt through isolated VSD were examined by PDE to determine factors influencing ability to DX VSD by PDE. In 24/31, the turbulent VSD jet could be followed through the IVS. In 7/31, harsh systolic flow was found on the RV side of IVS but could not be followed through the IVS. Two of these were apical RV jets, and had a small apical VSD at cath, and 1/7 was a VSD obscured by the tricuspid valve. Four of the 7 were in the usual location, but had higher pulmonary resistance*, pulmonary to systemic resistance ratios** (560+258 dynes-sec-cm⁻⁵, 1/7+1.5**) and subjectively less pulmonary artery turbulence by PDE than those whose VSD jets could be followed across the IVS (247+227 dynes-sec-cm⁻⁵, 1/14+5.8**), p=.05*, p=.01**. The DX of VSD by PDE was made in 26/31 youngsters with known VSD, 84%. The DX could not be made in one whose VSD was under the tricuspid valve, and in 4 whose VSD's were high, but were associated with high resistance. The turbulence of the VSD jet is a useful guide in following VSD flow across the IVS; diminution of turbulence, as may be expected with high pulmonary resistance, may compromise the ability to follow the VSD jet across the IVS.

176 PULSED DOPPLER ECHOCARDIOGRAPHIC (PDE) EVALUATION OF PATENT DUCTUS ARTERIOSUS (PDA) IN PREMATURE INFANTS. James G. Stevenson, Isamu Kawabori, and Warren G. Guntheroth. Department of Pediatrics, University of Washington School of Medicine, Seattle.

Twenty-eight premature infants, mean weight 1590 grams, were examined by PDE because of concern of PDA. PDE, by supplementing M-mode echo with Doppler flow detection, allows determination of the site of turbulent blood flow. All 14/28 infants with continuous murmurs were found by PDE to have continuous turbulent pulmonary artery flow. 8/28 had only a systolic murmur, but PDE detection of continuous turbulent pulmonary artery flow confirmed PDA in all. 6/28 had no murmur, were examined by PDE because of increasing respiratory requirements, and 4/6 had PDE findings of PDA. 3/6 with no murmur, and 6/8 with only a systolic murmur had abnormally large left atrial dimensions, a significant left to right shunt, and in each the site of shunt could be localized by PDE. The results indicate that the continuous turbulent pulmonary artery flow present in premature infants with PDA can be detected by PDE, that continuous turbulent pulmonary artery flow and a significant shunt may be present with only a systolic murmur or no murmur at all, and that the site of shunt can be detected by PDE. PDE appears to have significant clinical utility in the evaluation of PDA in premature infants, in whom invasive studies carry substantial risk of mortality and morbidity, and may be especially useful when clinical findings and murmurs are not diagnostic of PDA.

177 MEDICAL MANAGEMENT OF PATENT DUCTUS ARTERIOSUS IN HYALINE MEMBRANE DISEASE Andrew R. Stewart, Robert Moriarty, Orest O. Ulan & Neil N. Finer (Spons. by D. Schiff) University of Alberta, Royal Alexandra Hospital, Dept. of Pediatrics, Edmonton, Alberta

From April 1975 to October 1976, 19 out of 141 infants (13.5%) with severe hyaline membrane disease (HMD) developed patent ductus arteriosus (PDA) and were treated by aggressive medical management. Birth weight ranged from 925-2480 gm and gestation from 28-36 wk. Evidence for the presence of PDA included increased pulses by palpation, widening of pulse pressure on direct intra-aortic monitoring, hyperactive precordium, systolic or continuous murmur, and x-ray evidence of pulmonary plethora. The HMD was severe enough to require ventilation in 13 of the 19 patients, or 20% of all patients with HMD requiring ventilation. Treatment included the use of digoxin in 17 patients (89.5%) and intermittent diuretic (furosemide) in 12 patients (63%). In addition, distending pressure by nasal prongs, face mask or endotracheal tube was utilized in 17 patients (89.5%). Pressure used varied from 4-6 cm H₂O for a mean duration of 422 hr and a maximal duration of 54.5 days. There were three deaths, all before the 5th day with intracranial hemorrhages. No patient required surgical ligation in the newborn period although one patient had a PDA ligated at 5 months of age. There were 10 additional cases of PDA without HMD seen in the same period and treated as above. None required surgical correction. It is suggested that surgical ligation of PDA in the neonatal period be reserved for those cases where aggressive medical therapy, including distending pressure has failed.

178 THE HEART IN THALASSEMIA MAJOR: AN ECHOCARDIOGRAPHIC ASSESSMENT. Masato Takahashi, Carol B. Hyman, Jorge Ortega, Alan B. Lewis, and Paul R. Lurie.

University of Southern California School of Medicine and Childrens Hospital of Los Angeles, Dept. of Ped., Los Angeles 14 patients (pts.), 3 to 27 years of age, were studied with M-mode echocardiography. 6 years ago transfusions (tx) were increased to maintain the pre-tx Hgb level above 10.5 g% with total iron loads (TIL) via tx of 35 to 508 g. The pts. were divided into Group A (7 pts. with TIL < 120 g) and Group B (7 pts. with TIL > 120 g). Two pts. had arrhythmias, and 3 had congestive failure with 2 deaths. The remainder had no cardiac symptoms. Positive echo findings are summarized below:

	Group A (7 pts.)	Group B (7 pts.)
Dilated right ventricle (RV)	6	5
Dilated left atrium (LA)	7	7
Dilated left ventricle (LV)	1	5
Hypertrophy of LV	5	6
Decreased fiber shortening fraction (D)	0	2
Decreased mean shortening velocity (Vcf)	1	5
Pericardial effusion (≥ 7 mm)	2	3

Mean Vcf was 1.35±.14(SD) in Group A and 1.10±.26 in Group B (p<.025). 2 pts are alive 8 and 24 months after discovery of marked LV dysfunction. 1 pt. with normal LV function deteriorated rapidly and died 6 months later. In conclusion, RV and LA dilatation and LV hypertrophy occur commonly in Thalassemia pts. LV dilatation and dysfunction occur usually but unpredictably in pts. with TIL over 120 g. LV function indices do not seem predictive of survival time.

179 LUNG FUNCTION AND STATUS OF DUCTUS ARTERIOSUS (DA) IN PRETERM LAMBS TREATED WITH INTRAVENOUS STEROIDS IN UTERO. Donald W. Thibeault, George C. Emmanouilides, and Michael E. Dodge. UCLA School of Medicine, Harbor General Hospital, Department of Pediatrics, Torrance, California.

Nineteen control fetuses and 12 chronic preparations (111-130 days' GA) were delivered by C-section and ventilated for 3 hours. The 12 chronically catheterized fetuses (jugular vein) received hydrocortisone q.8.h. (30 to 180 mgm/day) for up to 10 days. The GA in steroid and control lambs were not different (119 ± 2.2 and 119.5 ± 1.5). Aortic (AOP) and pulmonary artery (PAP) pressures and blood gases were monitored. Ductal shunts were assessed by cineangiograms. Pressure-volume (PV) curves were done on the excised lungs and expressed as V10 (lung volume/gm. of wet lung tissue at a deflation pressure of 10 cm. H₂O).

Arterial PO₂ (100% O₂) in steroid vs control lambs were not different (83 ± 17 vs 79 ± 14 mmHg). Mean PAP and AOP in the steroid-treated lambs were 43 ± 4 and 61 ± 4 and were not different from controls (43 ± 4 and 54 ± 3). The DA in the control lambs <120 days' GA was widely open, but 3 of the steroid-treated lambs had "closed" DA. The DA was "closed" or constricted in all lambs >120 days'. Lambs with open DA had large L+R shunts. The V10 was not different in steroid-treated lambs vs. controls (.36 ± .12 ml vs .18 ± .05 p>.05). Also, no differences in the V10 was seen in 6 twins, in which one twin was a control and the other given steroids. The results indicate that lung gas exchange and P-V curves were not altered by the fetal IV steroid therapy. In lambs with open DA at 1.5 hours, there was a large L+R shunt. The PAP was always less than systemic in spite of non-compliant lungs.

180 PROSTAGLANDIN E-1 (PGE-1) & PULMONARY VASCULAR RESISTANCE IN NEONATAL LAMBS. M.E. Tripp, M.A. Heymann & A.M. Rudolph. Cardiovascular Research Institute, Univ. of California, San Francisco.

PGE-1, a known pulmonary vasodilator in isolated lungs, has been proposed as a mediator of the rapid fall in pulmonary vascular resistance (PVR) in neonates. A previous study found that in fetal animals a 50% drop in PVR was accompanied by severe systemic effects including a 44% drop in placental flow. To exclude such effects we studied chronic PGE-1 infusion directly into the pulmonary circulation of 7 neonatal lambs. Animals were instrumented in utero under maternal anesthesia. Peripheral arterial and venous catheters were placed. Thoracotomy was performed with placement of a pulmonary arterial catheter and a pulmonary trunk electromagnetic flow transducer. The ductus was ligated, the animal delivered, and mechanical ventilation instituted. Systemic and pulmonary arterial pressures, pulmonary arterial flows, heart rate, and arterial blood gases were monitored. PVR was calculated before and during infusion under normoxemic and hypoxemic conditions. In 9 infusions in 7 animals, basal PVR was 0.225 mm/Hg/ml/min and rose to 0.521 with hypoxia. Basal systemic vascular resistance (SVR) was 0.534, and rose to 0.568 with hypoxia. With PGE-1 infusion at a mean dose of 1.6 mcg/kg/min PVR fell 27% to 0.185 in room air while SVR fell 25% to 0.400. Under hypoxic conditions PVR fell 49% to 0.264 while SVR fell 44% to 0.319. We conclude that PGE-1 is a nonspecific vasodilator which during infusion is not degraded in a single pass through the lungs. It decreases PVR and SVR equally during both normoxemia and hypoxemia.