●211 MYOCARDIAL BLOOD FLOW (q) AND OXYGEN CONSUMPTION ($\dot{V}_{O2}$) IN CHRONICALLY INSTRUMENTED LAMBS WITH AN AORTO-PULMONARY SHUNT (S). Paul G. Toorop, Rudy Hardjowijono, Jan H. Koers, Michiel Dalinghaus, Jaap R.G. Kuipers. Depts of Pediatrics and Thoracic Surgery, University Hospital, Groningen, The Netherlands (Spon. by A.M. Rudolph).

We showed that heart rate (HR) in lambs with a S is higher than in those without a S. This increase will affect myocardial $\dot{V}_{O2}$. We studied myocardial q and left ventricular (LV) $\dot{V}_{O2}$ in 7 lambs with a S (aged 7±1 wks, mean±SEM). 4-14 days prior to the studies we inserted catheters into aorta (Ao), coronary sinus (CS) and left atrium, and a Goretex® graft between descending Ao and pulmonary artery (PA). Electromagnetic flow probes (EFP) were placed around Ao and PA to measure pulmonary (q_p) and systemic (q_s) blood flows, respectively. As controls another 8 lambs (aged 7±1 wks) were instrumented in the same way except for the Goretex® graft and PA EFP. Myocardial q of LV was calculated by the radioactive microsphere method. LV $\dot{V}_{O2}$ was calculated from myocardial q of LV and the difference in O_2 content between Ao and CS blood. The q_p:q_s ratio of the S lambs was 2.3. The S lambs had a higher HR (151±6 vs 110±7, $p<0.001$), myocardial q of LV (241±26 vs 136±17 ml/min/100 gLV, $p<0.005$), LV $\dot{V}_{O2}$ (757±74 vs 405±35 µmol/min/100 gLV, $p<0.001$), and a higher total heart weight (108.2±12.0 vs 77.3±6.9 gr, $p<0.05$) than the control lambs. Peak Ao pressure and q_s were not significantly different in the two groups. These findings suggest that the increase in HR in the S lambs may be the main reason for the increase in LV $\dot{V}_{O2}$.

214 PERINATAL FACTORS LEADING TO LEFT VENTRICULAR DYSFUNCTION IN THE NEONATE. Lota Viray, Bijan Siassi, Paul Y.K. Wu, Nam Dong. USC Sch. of Med., LAC-USC Med. Ctr., Dept of Ped., Los Angeles.

Severe perinatal asphyxia is associated with myocardial dysfunction in the neonate, however, the contributory roles of hypoglycemia and hypocalcemia is less clear. Serum glucose, total and ionized Ca++ and pH were measured at 0,1,4,24 and 48 hours in 10 term asphyxiated infants (BW=3381±772g, GA=40±1.5 wks). M-mode echocardiograms and serum total creatine phosphokinase (CPK) and CPK isoenzymes were measured during the first 8,24, and 48 hours of life. All neonates had fetal distress as evidenced by severe variable or late decelerations in their fetal heart rate tracing. Eight infants required emergency C-section. One minute Apgar scores in all infants were ≤3. Significant left ventricular (LV) dysfunction was diagnosed in the presence of abnormalities in at least 2 of the following 3 echocardiographic findings: left atrial/aortic ratio >1.3, LV pre-ejection period/LV ejection time ratio >0.42 and the LV shortening fraction < 0.28. Four neonates had evidence of LV dysfunction. The MB fraction of CPK isoenzymes was elevated to a greater extent in these 4 infants. Three of the 4 infants with and only one of the 6 without LV dysfunction had hypoglycemia. Serum ionized Ca++ remained normal in all 10 infants. Conclusion: 1) Hypoglycemia significantly contributes to asphyxial myocardial dysfunction; 2) Hypocalcemia is not a significant contributing factor; 3) Serial measurement of M-mode and MB fraction of CPK isoenzymes identifies asphyxiated neonates with significant myocardial dysfunction.

212 ECHOCARDIOGRAPHIC ABNORMALITIES IN OSTEOGENESIS IMPERFECTA (OI). Petros Tsipouras, Jay Shapiro, John Hortop, Barry Maron, William Bossert. Rutgers Medical School, Piscataway, NJ; McGill University, Montreal; Children's Hospital; Clinical Center and NHLBI, NIH, Bethesda; Harvard University, Cambridge, MA: (sponsored by A.Sadeghi-Nejad)

The prevalence of abnormalities of the heart valves, aorta, and myocardium was studied in 112 subjects with OI at two centers (MCH and NIH). The groups differed in the numbers of families included: MCH 42/65 subjects from 12 families, NIH 22/47 subjects from 9 families. Results are reported as % of predicted in a normal population, except for % fractional shortening(fs); (Henry, W. et al, Circulation 62: 1054, 1980).

Group	MCH				NIH			MCH	NIH
Observation	$\bar{X}$	S.E.	N	A	$\bar{X}$	S.E.	N	B	B
Aorta	114.6	1.87	65	P=<.01	107.3	1.64	47	P=<.01	P=<.01
LA diameter	89.4	1.61	53	-	90.7	1.76	47	<.01	<.01
LV diameter	104.4	1.66	28	-	104.5	1.63	41	<.05	<.05
V_{PW}	89.1	2.54	24	<.01	97.8	2.61	43	<.01	NS
Septum	87.0	3.12	30	<.05	94.5	2.37	43	<.01	<.01
%fs(.28-.40)	0.17	0.03	30	<.01	0.29	0.02	43	<.01	<.01

Mitral valve prolapse(MVP) occurred in 4/64 MCH series, 1/47 NIH series (5% total) LA and LV diameters were virtually identical in the two groups, yet all other measurements were significantly different between them(column A). Except for V_{PW}, NIH, both groups differed significantly from the expected norm(column B). Aortic dilatation was more frequent with families than expected due to chance(P<.01). Thus, decreased wall thickness and aortic dilatation are common in OI, the latter in certain kindreds. MVP is not increased above the expected norm.

●215 EFFECT OF ADENOSINE ON CEREBRAL METABOLIC RATE FOR O_2 ($CMRO_2$) AND CEREBRAL BLOOD FLOW (CBF) IN NEWBORN LAMBS. L. Craig Wagerle,* Jaques Belik,* and Maria Delivoria-Papadopoulos. University of Pennsylvania, Depts. of Physiolology and Pediatrics, Phila., PA.

Previous studies suggest that adenosine released by tissue may mediate local vasodilation and thus couple local flow to the O_2 requirements of the tissue. The present experiments examine the relationship between $CMRO_2$ and CBF in newborn lambs during infusion of adenosine. A total of 17 lambs were divided into two groups. In Group I (n=7) intracarotid infusion of adenosine concentrations of 0.06, 0.12, and 0.24 mg/kg/min increased CBF by 35, 114, and 102%, respectively, suggesting that maximal adenosine effects were attained with 0.12 and 0.24 mg/kg/min. In Group II (n=10) CBF and $CMRO_2$ were determined during infusion 0.24 mg/kg/min adenosine. Of these, 5 served as controls, studied unanesthetized ($CMRO_2$=5.02 ml/min/100g), and 5 were studied anesthetized (alpha-chloralose) to reduce $CMRO_2$ to 2.20 ml/min/100g. In the anesthetized group CBF was decreased to 37±3 ml/min/100g compared to the control group (CBF=87±7 ml/min/100g). Adenosine increased CBF by 113% and 80% in the control and anesthetized lambs respectively. Cerebrovascular resistance was 0.84±0.08 and 2.48±0.25 mmHg/ml/min/100g in the control and anesthetized groups respectively and decreased by 56% and 58% during adenosine infusion. In spite of the concentration of adenosine infused, CBF was less in the anesthetized lambs as a function of the level of $CMRO_2$. Since adenosine action on smooth muscle is receptor mediated, these data would suggest that the relationship between CBF and $CMRO_2$ is intact when adenosine receptors may be saturated.

213 EVALUATION FOR PDA IN PRETERM INFANTS UTILIZING 7.5 MEGAHERTZ 2-D ECHOCARDIOGRAPHY

G. Wesley Vick, III, James C. Huhta. Spon. by Paul C. Gillette. Baylor College of Medicine and Texas Children's Hospital, The Lillie Frank Abercrombie Section of Cardiology, Dept. of Pediatrics, Houston, TX.

Evaluation for patent ductus arteriosus (PDA) by both Doppler examination and direct two-dimensional (2-D) echocardiographic visualization has been reported in term infants and children. However, visualization of a PDA in preterm infants with lung disease has been more difficult. Utilizing a recently developed 7.5 MHz mechanical scanner with interfaced 2-D directed pulsed Doppler we evaluated our initial experience in 14 premature infants with lung disease. Doppler sampling was performed at the pulmonary end of the ductus arteriosus from a suprasternal approach. Patients ranged in age from 1 day to 30 days (mean 11 days) and from 510 to 2300 grams in weight (mean 1015 grams). Imaging of the ductus arteriosus was successful in 13/14 (93%). Doppler evaluation of flow in the main pulmonary artery and descending aorta was successful in all. In 8 infants the DA was closed by both 2-D echo and Doppler exam (excluded by retrograde aortography in 2). In 5 infants a PDA was present by both methods. In 1 infant the DA was patent by Doppler exam but could not be imaged adequately by 2-D echo. This markedly improved imaging capability should allow longitudinal noninvasive studies of PDA morphology in premature infants with lung disease.

216 MYOCARDIAL GLYCOGEN METABOLISM IS MODIFIED DURING CHRONIC HYPERGLYCEMIA AND HYPERINSULINEMIA IN THE FETAL LAMB. David Warburton. (Spon. by T.G. Keens) Neonatal-Respiratory Disease Division, Childrens Hospital of Los Angeles, Dept. Pediatrics, University of Southern California School of Medicine, Los Angeles.

I studied the developmental profile of glycogen, glycogen synthase and phosphorylase in myocardia of control fetal lambs at 123, 131, and 142d gestation (term 150d) and in the myocardia of their twins treated with intravenous glucose (16±2 mg/kg/min, M±SE) from 112d onwards. Serum glucose (34±2 mg/dl) and insulin 47±11 µU/ml) were higher in the glucose treated fetuses than serum glucose (19±3 mg/dl, P <0.01) and insulin (14±2 µU/ml, P <0.01) in the controls.

	123d	131d	142d	
Glycogen content	117	60	74	Control
µg/mg wet wt	138	319	78	Glucose
Synthase a	1.0	2.5	1.6	Control
Nmole/min/mg prot	1.3	2.1	2.7	Glucose
Synthase a + b	6.7	9.2	6.6	Control
Nmole/min/mg prot	7.4	12.1	7.9	Glucose
Phosphorylase a	2.0	1.1	0.7	Control
Nmole/min/mg prot	0.9	1.1	0.6	Glucose
Phosphorylase a + b	13.6	15.5	12.1	Control
Nmole/min/mg prot	10.6	17.3	10.4	Glucose

Myocardial glycogen was increased 1.2 fold at 123d gestation and 5.0 fold at 131d gestation in the glucose treated fetuses. Enzyme activities were also modified by chronic hyperglycemia.