We showed that heart rate (HR) in lambs with a S is higher than in those without a S. This increase will affect myocardial ∇_{02} . We studied myocardial q and left ventricular (LV) ∇_{02} 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 (D) 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_g) blood flows, respectively. As controls another 8 lambs (aged 7±1 wks) were instrumented in the same way except for the Goretex (D) graft and PA EFP. Myocardial q of LV was calculated by the radioactive microsphere method. LV ∇_{02} was calculated from myocardial q of LV and the difference in O₂ content between Ao and CS blood. The q_{p:qs} 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 ∇_{02} (757±74 vs 405±35 µmol/min/100 gLV, p<0.005), the votal heart weight (108.2±12.0 vs 77.3±6.9 gr, p<0.05) than the control lambs. Peak Ao pressure and q_g were not significantly different in the two groups. These findings suggest that the increase in LV ∇_{02} .

212 ECHOCARDIOGRAPHIC ABNORMALITIES IN OSTEOGENESIS IMPERFECTA (OI). <u>Petros Tsipouras, Jay Shapiro, John</u> <u>Hortop, Barry Maron, William Bossert</u>. 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, <u>Circulation</u> 62: 1054, 1980). <u>Broup</u> MCH NIH B B

Group	MC	н			IN .			rion	1111	
Observation	X	S.E.	N	Α	X	S.E.	N	В	В	
Aorta	114.6	1.87	65	P=<.01	107.3	1.64	47	P=4.01	P=<.01	
LA diameter	89.4	1.61				1.76			<.01	
LV diameter		1.66			104.5				<.05	
		2.54		1					NS	
V _{PW}	89.1	3.12				2.37			<.01	
Septum	87.0								<.01	
% fe(2840)) 0.1/	0.03	- 30	<.01	0.29	0.02	40	<u>,,or</u>	~. • • •	

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

G. Wesley Vick, III, James C. Huhta. Spon. by <u>Paul C. Gillette.</u> 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. 214 PERINATAL FACTORS LEADING TO LEFT VENTRICULAR DYSFUNCTION IN THE NEONATE. Lota Viray, <u>Bijan Siassi</u>, <u>Paul Y.K. Wu</u>, <u>Nam Dong</u>. 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 hypo-glycemia 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 asphysiated infants (BW=338147729, 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 minthe 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 < 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.

• 215 EFFECT OF ADENOSINE ON CEREBRAL METABOLIC RATE FOR O2 (CMRO2) AND CEREBRAL BLOOD FLOW (CBF) IN NEWBORN LAMBS. L. Craig Wagerle,* Jaques Belik,* and Maria Delivoria-Papadopoulos. University of Pennsylvania, Dents of Physioklowy and Pediatrics. Phila. PA.

CBF) IN NEWBORN LAMBS. L. Craig wagerie, 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 O2
requirements of the tissue. The present experiments examine the
relationship between CMRO2 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, Il4, 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 CMRO2 were determined
during infusion 0.24 mg/kg/min adenosine. Of these, 5 served as
controls, studied unanesthetized (CMRO2=5.02 ml/min/100g), and 5 were
studied anesthetized (alpha-chloralose) to reduce CMRO2 to 2.20
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
CMRO2. Since adenosine action on smooth muscle is receptor mediated,
these data would suggest that the relationship between CBF and CMRO2
is intact when adenosine receptors may be saturated.

	MYOCARDIAL GLYCOGEN METABOLISM IS MODIFIED DURING
216	CHRONIC HYPERGLYCEMIA AND HYPERINSULINEMIA IN THE
	FETAL LAMB David Warburton (Snon, 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

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 12d onwards. Serum glucose (34±2 mg/dl) and insulin 47±11 μ U/ml) were higher in the glucose treated fetuses than serum glucose (19±3 mg/dl, P <0.01) and insulin (14±2 μ U/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.