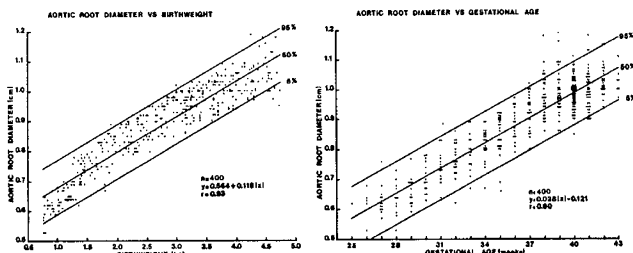


### 151 NORMAL VALUES OF AORTIC ROOT DIAMETER IN NEONATES.

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The combination of pulsed Doppler and M-mode echocardiography can provide estimates of blood flow velocity and diameter of the ascending aorta, from which cardiac output can be calculated. Since the square of the aortic root diameter (ARD) is used in the calculation of cardiac output, precise measurements are vital. This study assessed normal values for ARD, obtained by M-mode echocardiography with leading edge methodology in early diastole. 172 preterm and 228 term newborn infants (BW 750-4,740 g, GA 25-43 w) were studied during the first week of life. ARD increased linearly with advancing birthweight ( $r=0.93$ ,  $p<0.001$ ) and gestational age ( $r=0.90$ ,  $p<0.001$ ).



### 152 BLOOD FLOW IN THE ASCENDING AND THE DESCENDING AORTA IN TERM NEWBORN INFANTS.

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The combination of 2-dimensional real-time and pulsed Doppler echocardiography provides a noninvasive method of measuring blood flow and has been used to study blood flow at the lower thoracic level of the descending aorta (DA) in the human fetus. Using these techniques, we evaluated the proportion of cardiac output distributed through the DA in 13 term newborn infants (mean $\pm$ SEM BW 3551 $\pm$ 95 g, GA 40.1 $\pm$ 0.3 w). Blood flow velocity was measured using a 3.5 MHz, range gated, pulsed Doppler velocity meter with the transducer positioned in the suprasternal notch. Blood flow in the ascending aorta (AA) was measured at the aortic root level and descending flow below the isthmus. The internal diameter of the AA was assessed at the level of the aortic valve using M-mode echocardiography and the diameter of the DA in the posterior atrioventricular groove with the use of the 2-D long axis view.

Blood flow in the AA was 789 $\pm$ 42 ml/min and in the DA 499 $\pm$ 26 ml/min indicating that 63.5% of the left ventricular output is distributed through the DA. The mean blood flow velocity was comparable at both sites, whereas the mean diameter of the DA was 19% smaller than the diameter of the AA.

In healthy term newborn infants approx. 2/3 of cardiac output is directed to the DA and 1/3 to brachial and carotid arteries. The changes in aortic blood flow are correlated with the changes in aortic diameter.

### 153 CARDIAC OUTPUT IN INFANTS OF INSULIN-DEPENDENT DIABETIC MOTHERS.

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Transient hypertrophic subaortic stenosis due to interventricular septal hypertrophy is an important cause of morbidity in the infant of the diabetic mother (IDM). The purpose of this study was to quantitate left ventricular outflow obstruction through estimation of cardiac output (CO) by pulsed Doppler ultrasound. We evaluated 42 IDMs (White class B,C,D) and compared them to 2 control groups, one comparable in BW and the other similar in GA. Septal hypertrophy was found in 18/42 (43%) of the IDMs. Morbidity increased with advancing septal thickness. Hyaline membrane disease was not present in any of the patients.

CLINICAL STATUS	N	SEPTAL HYPERTROPHY
Asymptomatic	29	11
Congestive heart failure	13	7

With increasing septal thickness, CO/kg diminished linearly ( $r=-0.78$ ,  $p<0.001$ ). This was secondary to reduced stroke volumes at comparable heart rates. The IDMs had higher left atrial/aortic ratios and right ventricular systolic time intervals than the control infants.

We conclude that CO is reduced in IDMs with septal hypertrophy. This reduction is secondary to reduced stroke volume due to diminished left ventricular compliance. Pulsed Doppler ultrasound is an important adjunct to the management of symptomatic IDMs.

### 154 CARDIAC OUTPUT IN NEWBORNS WITH TRANSIENT MYOCARDIAL DYSFUNCTION SECONDARY TO PERINATAL ASPHYXIA.

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Transient myocardial dysfunction (TMD) secondary to perinatal asphyxia is an important cause of neonatal morbidity. To determine cardiac output (CO) and stroke volume (SV) in TMD we evaluated 11 newborns with severe perinatal asphyxia: mean ( $\pm$ SD) BW 3.0 $\pm$ 1.2 kg, GA 36.6 $\pm$ 5.3 weeks, initial pH 6.90 $\pm$ 0.08, and initial mean arterial BP 39 $\pm$ 10 mmHg. TMD was diagnosed by M-mode echocardiography in the presence of abnormal left ventricular (LV) shortening fraction (SID), LV systolic time interval and/or left atrial/aortic ratio.

Using pulsed Doppler echocardiography, we found mean CO to be 122 $\pm$ 28 ml/min/kg (normal: 249 $\pm$ 34 ml/min/kg) and SV to be 0.82 $\pm$ 0.21 ml/kg (normal: 1.77 $\pm$ 0.28 ml/kg). Six newborns were treated with dopamine (4-10  $\mu$ g/kg/min). Mean arterial BP increased from 38 $\pm$ 8 to 57 $\pm$ 7 mmHg ( $p<0.001$ ), CO from 114 $\pm$ 26 to 201 $\pm$ 39 ml/min/kg ( $p<0.001$ ), SV from 0.80 $\pm$ 0.19 to 1.26 $\pm$ 0.14 ml/kg ( $p<0.001$ ), and heart rate from 144 $\pm$ 6 to 159 $\pm$ 21 beats/min ( $p<0.05$ ) within one hour. The SID normalized quickly and the other echocardiographic abnormalities resolved over a 24-48 hour period.

We conclude that CO is low in TMD caused by asphyxia, and that dopamine treatment rapidly improves CO by increasing SV. Serial measurements of CO can be useful for monitoring dopamine treatment in newborns with cardiovascular compromise.

### 155 THE ROLE OF LACTATE IN MEETING THE ENERGY REQUIREMENTS OF THE LATE FETAL HEART.

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An isolated perfused working heart preparation was used to investigate the role of lactate in the energy metabolism of the fetal heart. Hearts were perfused under working conditions with Krebs-Henseleit buffer containing either glucose 5mM or glucose 5mM and lactate 5mM combined. Insulin 100  $\mu$ U/ml was present in all perfusions. Pressure development in both great vessels was maintained at 55 mmHg and mean atrial pressure was adjusted to 6 mmHg. Glucose uptake was measured by production of  $^3$ H<sub>2</sub>O from  $^2$ H glucose and lactate uptake by measuring changes in buffer concentration. With glucose as the sole exogenous substrate its utilization (3.48  $\pm$  .29  $\mu$ mol/min/g dry), corrected for lactate production by the heart (3.06  $\pm$   $\mu$ mol/min/g dry) could account for only 56  $\pm$  9% of the total oxygen consumption of the heart (20.7  $\pm$   $\mu$ mol/min/g dry). When both glucose and lactate were present in the buffer, glucose uptake was inhibited (0.71  $\mu$ mol/min/g dry) and lactate uptake (6.55  $\mu$ mol/min/g dry) accounted for approximately 91  $\pm$  12% of the total oxygen consumption (22.3  $\pm$  1.8  $\mu$ mol/min/g dry). These findings suggest that lactate oxidation is the primary source of metabolic energy for the fetal heart in late gestation, since glucose alone does not satisfy the oxidative needs of the fetal heart and since the presence of lactate profoundly suppresses glucose uptake. Lactate is the primary metabolic substrate for the fetal heart when present in concentrations similar to those observed in vivo, much as lipids are the primary metabolic substrate in more mature hearts.

### 156 DEVELOPMENTAL ALTERATIONS IN CARDIOVASCULAR FUNCTION DURING EARLY DEVELOPMENT.

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Cardiovascular parameters were evaluated in the extrauterine preterm lamb (EPL) (GR I:107.27 $\pm$ .51 SE dys gestation, 1.56 $\pm$ .47 SE kg BW, n=11; GR II:121.40 $\pm$ .53 SE dys gestation, 2.54 $\pm$ .16 SE kg BW, n=9) at different levels of arterial oxygenation during early development. Utilizing liquid ventilation with an oxygenated and warmed fluorocarbon (RIMAR 101) effective pulmonary gas exchange (PaO<sub>2</sub> =24-289, PaCO<sub>2</sub> =30-45, pH=7.25-7.45) and thermal stability were achieved. Mean arterial pressure (MAP), heart rate (HR), arterial gas tensions, blood chemistry and core temperature were monitored throughout the experiments. HR and MAP in conjunction with previously reported fetal data were statistically analyzed as a function of arterial oxygen content (CaO<sub>2</sub>=4-25 vol %) and age. Neither group demonstrated a significant change in HR or MAP until CaO<sub>2</sub> fell below 13.30 vol %. Below this level, HR increased ( $p<0.01$ ) from 201 to 250 b/min @ CaO<sub>2</sub>=6.0 vol % and little change in MAP (38 mm Hg) was seen in GR I. In contrast, GR II showed a significant decrease in HR ( $p<0.01$ ) from 195 to 140 b/min @ CaO<sub>2</sub> =5.0 vol % and increase in MAP ( $p<0.01$ ) from 41 to 56 mm Hg @ CaO<sub>2</sub> =6.0 vol %. These findings expand our understanding of age-related cardiovascular compensation over a range of arterial oxygenation previously unobtainable at this early stage of development. (Supported by NIH Grant HL/HD 30525).