

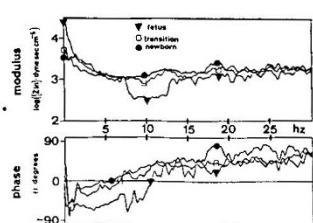
† 139 CONTINUOUS WAVE DOPPLER AND PDA: THE VELOCITY INDEX
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 A ratio of mean aortic systolic and diastolic velocities determined by continuous wave doppler (CW) has been shown to correlate with ductal (PDA) % left-to-right shunt (LRS). A new simpler ratio, velocity index (VI), of peak systolic to diastolic velocities can be determined from peripheral arteries without planimetry. Shown to correlate with clinical estimates of PDA-LRS in 22 infants, VI has not been validated by measured LRS. We examined 8 pigs (mean weight 6.1 kg, mean age 23.6 days). PDA was created by balloon catheter dilation. 4 studies were before PDA (no angiographic shunt), 4 acute PDA, and 7 chronic (mean 15.8 days) PDA. Pigs were sedated with ketamine. Left ventricular output (LVO) and LRS were determined using LV injection of 15µm radioactive spheres. LRS was corrected for systemic shunting (11.3%) of spheres. CW was performed over brachial and femoral arteries using a 7.0 MHz probe. LVO ranged from 89 to 453 ml/min/kg. Results (mean ± SD, *p<0.05 v. closed):

	PDA	LRS	VI	Pulse Pressure	Heart Rate
Closed(A)	0	0	0.11±0.07	32±3 mmHg	172±38/min
Group 1(6)	22.3±9.5	-0.04±0.01		27±6 mmHg	140±26/min
Group 2(5)	50.7±16.4	-0.21±0.05		45±9 mmHg*	150±43/min

There was a statistically significant correlation (r=0.88, p<0.01) of VI v. LRS. Diastolic runoff in PDA produces negative peak diastolic velocity in arteries that increases with larger LRS. Our study confirms that VI reflects the size of LRS in PDA although actual LRS cannot be predicted by VI. However, no pig with VI>-0.01 had LRS>40%, similar to clinical findings in infants.

† 140 HEMODYNAMIC EFFECTS OF INCREASING HEMOGLOBIN(HGB) CONCENTRATION IN CHILDREN WITH A RIGHT-TO-LEFT(R-L) VENTRICULAR SHUNT AND RELATIVE ANEMIA. Robert H. Beekman and Dwight T. Tuuri, (Spon. by Maurice D. Kogut), Wright State Univ., Childrens Medical Center, Dept. of Pediatrics, Dayton, OH.
 The acute effects of increasing Hgb concentration were evaluated at cardiac catheterization in 7 children (age 0.3-7.5 yr) with a R-L ventricular shunt and relative anemia. Diagnoses were tetralogy of Fallot in 6 and L-transposition with VSD and PS in 1. Before and 20 minutes after isovolumic partial exchange transfusion (PET) with 20ml/kg packed red cells the following were measured: Hgb, pO₂, O₂ consumption, O₂ saturation (sat) and pressure in aorta, superior vena cava, right and left atrium. After PET, Hgb increased from 13.7±0.5 to 16.4±0.4gm% (p<.001, mean±SEM). Aortic (Ao) pO₂ increased from 55.0±3.5 to 62.0±4.1 torr (p<.01) and Ao sat from 84.3±2.3 to 90.9±1.3% (p<.002). Effective pulmonary blood flow (Qef) increased by 17% from 2.72±0.10 to 3.17±0.10 L/min/M² (p<.01) while R-L shunt decreased by 59% from 1.44±0.29 to 0.59±0.10 L/min/M² (p<.01). Systemic O₂ transport increased from 658±48 to 738±46 ml/min/M² (p<.002). After PET systemic resistance (Rs) increased from 15.9±1.1 to 20.0±1.4 units (p<.01). A significant relationship was found between % increase in Rs and both % decrease in R-L shunt (y=20.44x^{0.32}, r=.90, p<.01) and % increase in pO₂ (y=.36x+2.88, r=.83, p<.02). Thus, increasing Hgb concentration in 7 children with a R-L ventricular shunt and relative anemia decreased R-L shunt and increased Qef, Ao sat, pO₂, and O₂ transport. These effects appear related to the increase in Rs following PET. Correcting relative anemia in children with a R-L ventricular shunt may have significant hemodynamic benefits.

141 INPUT IMPEDANCE OF FETAL AND NEONATAL LEFT PULMONARY ARTERIAL CIRCULATION. Stephen Bennett, Boyd Goetzman, Jay Milstein, Department of Pediatrics, University of California, Davis, CA.
 Interpretation of the change in pulmonary vascular resistance at birth ignores the hemodynamic consequences of pulsatile pressure and flow. Whereas, impedance analysis can account for such changes. We studied changes of input impedance (Zin) in the left pulmonary arterial circulation (LPAC) before and immediately after birth in four near-term lambs. Epochs of pressure and flow in the LPAC were obtained from a micromanometer and an electromagnetic flowmeter. A computerized spectral analysis program provided Zin in terms of a modulus spectrum [Zin] and phase spectrum (Oin). The [Zin] spectra of the fetus and newborn are qualitatively similar to those obtained by others in adult vascular beds, ie, as a consequence of a pulsatile flow at a given heart rate, the effective circulatory load at the entrance of the LPAC is less than Rp, the resistance of the arteriolar bed ("decoupling" phenomena). We interpret this as the LPAC presenting a similar hydraulic load to pulsatile potential hydraulic power. We conclude that the fetal and neonatal LPAC may not differ markedly in how it accommodates pulsatile hydraulic power at harmonics equal to heart rate.



† 142 Potential Hydraulic Input Power Changes in the Left Pulmonary Arterial Circulation at Birth. Stephen H. Bennett, Jay M. Milstein, Boyd W. Goetzman, Dept. of Pediatrics, University of California, Davis, CA
 We studied hydraulic input power and input impedance changes in the left pulmonary arterial circulation (LPAC) before and after birth because they account for the relative importance of pulsatile and steady power and provide insight into the physiologic mechanisms of accommodating to each. Pulsatile pressure and flow were measured by a Millar micromanometer and electromagnetic flowmeter in 5 near term fetal lambs. Instantaneous power (IP) was calculated as the product of pressure and flow with time. The apparent power was calculated as the root mean square (RMS) of IP after the steady power was subtracted. Input impedance analysis allowed partitioning of pulsatile apparent power into average and reactive (RMS) components. Power values were calculated from 4 epochs of 48 seconds before and after birth in each animal. The calculated Potential Hydraulic Input Power (milliwatts) was:

	Steady	Pulsatile Apparent	Pulsatile Average	Pulsatile Reactive
fetus	10.6 2.9	40.5 5.0	20.0 4.1	3.7 4.2
newborn	31.1 11.9*	40.2 14.0	30.8 14.5*	16.0 10.2* (*p<.01)

Although the steady component of power increased 3 fold after birth, the magnitude of apparent power remained unchanged and was greater than that due to steady power. When apparent power was partitioned, in the fetal state, reactive power was greater than average power while it was less in the newborn. Hemodynamic transmission line theory suggests that average power is the portion of the apparent power dissipated into heat by the viscous properties of the blood and vessel walls, whereas reactive power is given to elastic wall expansion. Since the apparent power associated with pulsatile blood pressure and flow represents a substantial magnitude of the total power delivered to the LPAC, especially in the fetus, how it is partitioned may play a role in normal or abnormal pulmonary vascular development.

● 143 OUAÏBAIN EFFECTS ON OXYGEN TRANSPORT AND CONSUMPTION. William Berman, Jr., Dale C. Alverson and Deborah Christensen, UNM, Ped. Dept., Albuquerque, NM

We studied the effects of anemia and ouabain administration on cardiac function and oxygen (O₂) physiology in 9 newborn lambs. We measured O₂ consumption (V_{O₂}), hematocrit (hct), heart rate, right atrial and aortic blood pressures and oxygen contents, the PEP/LVET ratio and left ventricular (LV)dP/dt; cardiac output (CO), systemic vascular resistance (SVR), systemic O₂ transport (SOT) and the SOT/V_{O₂} ratio were calculated. Following baseline measurements, lambs were made anemic (mean hct=12%) by isovolumic exchange transfusion with Plasmanate. Measurements were repeated prior to and 30 min. after administration of ouabain, 75 µgm/kg bolus followed by 0.05 µgm/kg/min.

Results are shown in the table:

	hct	LVdP/dt	V _{O₂}	CO	SOT	SVR	PEP/LVET	SOT/V _{O₂}
BASELINE	38	4061	9.28	226	32.68	.454	.354	3.66
ANEMIA	12*	5689*	12.86*	398*	19.64*	.231*	.285*	1.57*
ANEMIA + 12*	5711*		8.40	290	13.92*	.300*	.281*	1.69*
OUABAIN	(%)	mmHg/sec		(ml/kg/min)		mmHg		ml/kg/min

* - differs from control, P<0.05, analysis of variance
 Anemia was associated with a heart rate related rise in LVdP/dt, a rise in V_{O₂}, increased CO, decreased SVR and a fall in the SOT/V_{O₂} ratio. Following ouabain administration, V_{O₂}, CO and SOT fell, but LVdP/dt and the SOT/V_{O₂} did not change. In this setting, ouabain effects are manifested primarily by alterations in O₂ physiology variables, rather than by changes in left ventricular contractile function.

● 144 POSTNATAL DEVELOPMENT OF CALCIUM (Ca) RELEASE FROM CARDIAC SARCOPLASMIC RETICULUM(SR). Robert J. Boucek, Jr., Michael Citak, Thomas P. Graham, Michael Artman Vanderbilt University, Nashville, TN.

Potential of contractile force of the first stimulated contraction following a rest interval (rest potentiation; RP) has been attributed to enhanced SR Ca release. Postnatal development of SR Ca release was assessed by evaluating RP in oxygenated left atrial strips isolated from immature (I; 14-21 day old; n=11) and adult (A; n=9) rabbits. Isometric developed tension (DT) and dT/dt (pH=7.4; @30°C) were determined while pacing at 1 Hz (control) and following 2-90 sec. rest intervals (RI). RP was maximal at 60 sec. RI. Ryanodine (10⁻⁸M), a putative blocker of SR Ca release, abolished RP in both age groups. At 2.5mM extracellular Ca (Ca_e), DT was potentiated 176% in I and 287% in A while dT/dt was potentiated 163% in I and 295% in A (>I; p<0.05). However, at 5mM (Ca_e), DT was potentiated 310% in I but only 215% in A (I>A; p<0.05). Mechanisms whereby the increase in (Ca_e) increased RP in I might include either an increase in the action potential-mediated Ca influx that triggers SR Ca release or an increase in SR Ca loading. To evaluate these mechanisms, (Ca_e) was increased from 2.5 to 5mM during the RI. RP was increased comparably in both age-groups, although the increase was small (<25%) when compared with the 176% increase in RP in I when (Ca_e) was increased to 5mM prior to RI. These results indicate that the immature heart requires a higher (Ca_e) to optimize SR Ca loading as compared to the adult, and this difference may be a functional correlate of the recognized morphologic maturation of cardiac SR during postnatal development.