Understanding Hemodynamics: Optimizing the Cardiac Catheterization Laboratory in Complex Conditions

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Disclosures

- W.L. Gore & Associates: Consultant and Proctor
- Medtronic, Inc: Consultant and Proctor
- Mezzion Pharma: Consultant and Scientific Advisory Board Member
- PECA Labs: Consultant





Outline

- Ground rules
- Assumed vs. measured data
- Static vs. dynamic assessment
- Measurements we didn't know we could obtain
- Conclusions





A common scenario

- 4 yo M with DCM undergoing pre-OHT evaluation
- Sats: PA 63%, DAO 96%
- Pressures: PA mean 16 mmHg, PCWP mean 6 mmHg
- Hgb 12.3, HR 115
- Assumed VO₂ 165 ml/kg/min

- Qp=Qs=2.94 L/min/m²
- Rp=2.72 U*m²





A common scenario

- 4 yo M with DCM undergoing pre-OHT evaluation
- Sats: PA 63%, DAO 96%
- Pressures: PA mean 16 mmHg, PCWP mean 6 mmHg
- Hgb 12.3, HR 115
- Measured VO₂ 97 ml/kg/min
- Qp=Qs=1.76 L/min/m²
- Rp=5.68 U*m²





Ground Rules

- Catheterization reflects a moment in time
- Measured data are measured whereas assumed data are not
- Hemodynamic data are typically acquired in a sedated/anesthetized, supine, and somewhat volume unloaded state
- Airway management strategy, sedation/anesthetic agent selection, ventilation/acid-base homeostasis, extra-cardiac organ pathologies can all impact measured hemodynamics





Data are data, not truth









Accuracy in the measurement of cardiac output





Determination of Cardiac Output

- Integral to assessment of the critically ill patient
- Necessary for determination of vascular resistance
- Plays a major role in care delivery and decision making
- And yet...thermodilution determination of cardiac output (gold standard) cannot be used in the setting of very low output, shunt lesion(s) or valvar regurgitation
- Thus, we frequently rely on the Fick principle for determination of cardiac output





Determination of Cardiac Output

$$CO = \frac{\sqrt{2}}{(AO\{PA)([Hgb])(1.36)(10)}$$





Oxygen Consumption

- Infrequently measured
- Difficult to assess in smaller/younger children or with GETA
- Historical reliance on formulae from LaFarge
- LaFarge data from 1960's
 - No GETA
 - No single ventricle palliation
 - No percutaneous interventions
 - No neonates, infants and young children
- Recent work has demonstrated LaFarge formulae to be inaccurate





Validation of Cardiac Output Using Real-time Measurement of Oxygen Consumption during Cardiac Catheterization in Children Under 3 Years of Age

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ABSTRACT_

Objective. To validate a method for determination of cardiac index (CI) using real-time measurement of oxygen consumption (VO₂) in young children undergoing cardiac catheterization.

Design. Retrospective review comparing thermodilution cardiac index (TDCI) to CI calculated by the Fick equation using real-time measured VO₂ (RT-VO₂) and VO₂ derived from 2 published predictive equations. Paired *t*-test and Bland-Altman analysis were used to compare TDCI to Fick CI. A survey to ascertain pediatric cardiac catheterization practices regarding VO₂ determination was also conducted.

Setting. Quaternary care children's hospital cardiac catheterization laboratory.

Patients. Children <3 years old with structurally normal hearts undergoing cardiac catheterization under general anesthesia with at least one set of contemporaneous TDCI and RT-VO₂ measurements.

Results. Thirty-six paired measurements of TDCI and RT-VO₂ were made in 27 patients over a 2-year period. Indications for catheterization included congenital diaphragmatic hernia postrepair (n = 13), heart disease post-orthotopic heart transplant (n = 13), and suspected cardiomyopathy (n = 1). Mean age was 21.5 ± 8 months; median weight was 9.9 kg (IQR 8.57, 12.2). RT-VO₂ was higher than VO₂ predicted by the LaFarge equation ($190 \pm 31 \text{ vs.}$ $173.8 \pm 12.8 \text{ mL/min/m}^2$, P < .001), but there was no difference between TDCI and Fick CI calculated using VO₂ from any method. Bland–Altman analysis showed excellent agreement between TDCI and Fick CI using RT-VO₂ and VO₂ predicted by the Lundell equation; Fick CI using VO₂ predicted by the LaFarge equation showed fair agreement with TDCI.

Conclusions. In children <3 years with a structurally normal heart, RT-VO₂ generates highly accurate determinations of Fick CI as compared with TDCI. Additionally, in this population, VO₂ derived from the LaFarge and Lundell equations generates accurate Fick CI compared with TDCI. Future studies are needed to identify factors associated with inaccurate VO₂ generated from these predictive equations.







Factor	Group 1 (n=201)	Group 2 (n=301)	p Value
Age (years)	0.82 (0.35–1.64)	8.78 (4.8–12.26)	<0.001
Weight (kg)	7.5 (5.4–10.05)	27.5 (17–42.25)	<0.001
BSA (kg/m²)	0.36 (0.28-0.46)	0.98 (0.69-1.38)	< 0.001
Female gender	79 (39)	162 (54)	0.001
Heart rate (bpm)	116±21.4	88.5±17.8	< 0.001
Haemoglobin (g/dL)	11.25±2.32	11.3±2.17	0.807
Critically ill status	59 (29)	21 (7)	<0.001
Diagnosis			
Structurally normal	2 (1)	13 (4)	0.034
Pulmonary arterial hypertension	19 (10)	19 (6)	0.192
Cardiomyopathy	2 (1)	13 (4)	0.034
Simple biventricular CHD	24 (12)	53 (18)	0.084
Complex biventricular CHD	43 (21)	54 (18)	0.337
Single ventricle	59 (29)	48 (16)	<0.001
Post Stage 1	42 (21)	0 (0)	< 0.001
Post Stage 2	17 (9)	18 (6)	0.286
Post Stage 3	0 (0)	30 (10)	< 0.001
Post heart transplant	52 (26)	101 (34)	0.067

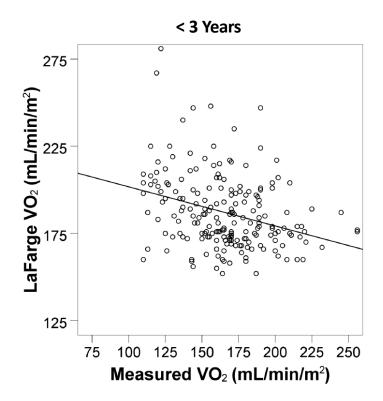
Values are mean±SD, median (IQR) or n (%). Simple biventricular congenital heart disease (CHD) was defined as an isolated patent ductus arteriosus, atrial septal defect, ventricular septal defect, valvar aortic stenosis or valvar pulmonary stenosis. Complex biventricular CHD was defined as tetralogy of Fallot, pulmonary atresia/ventricular septal defect, atrioventricular septal defect or truncus arteriousus. A patent foramen ovale was considered a normal variant.

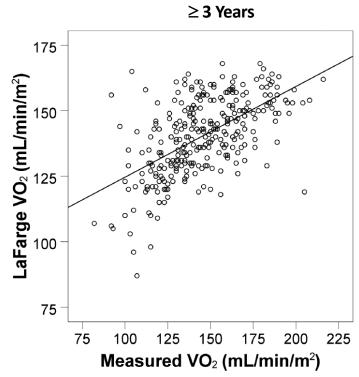
BSA, body surface area





Oxygen Consumption

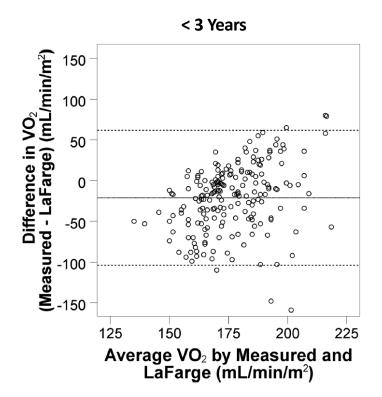








Oxygen Consumption



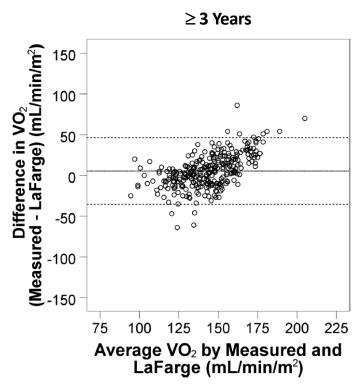






Table 2 Results of simple and multiple logistic regression analyses to identify factors associated with an inaccurate LaFarge VO₂ for each group

	Group 1				Group 2			
Univariate			Multivariable	lultivariable		Univariate		Multivariable
Factor	OR (95% CI)	p Value	OR (95% CI)	p Value	OR (95% CI)	p Value	OR (95% CI)	p Value
Age (years)	0.32 (0.21 to 0.50)	< 0.001	0.41 (0.20 to 0.82)	0.012	0.96 (0.90 to 1.02)	0.149		*
Weight (kg)	0.82 (0.74 to 0.91)	< 0.001	1.04 (0.90 to 1.19)	0.592	0.99 (0.97 to 1.00)	0.114		
BSA (kg/m²)	0.01 (0.00 to 0.04)	< 0.001			0.47 (0.20 to 1.13)	0.093		
Female gender	1.19 (0.67 to 2.10)	0.561			0.94 (0.48 to 1.83)	0.857		
Heart rate (bpm)	1.04 (1.02, 1.06)	< 0.001	1.01 (0.99 to 1.02)	0.330	1.00 (0.99 to 1.02)	0.726		
Haemoglobin (g/dL)	1.16 (1.03 to 1.32)	0.019	0.93 (0.81 to 1.06)	0.265	0.83 (0.70 to 0.99)	0.036	0.84 (0.81 to 0.86)	< 0.001
Critically ill status	3.40 (1.81 to 6.40)	< 0.001	2.00 (0.96 to 4.18)	0.065	2.89 (1.05 to 7.97)	0.040	2.48 (0.90 to 6.82)	0.079
Structurally normal	0 (0)	0.999		(1)	2.04 (0.54 to 7.74)	0.297		
Pulmonary arterial hypertension	0.62 (0.22 to 1.70)	0.347			0.35 (0.05 to 2.67)	0.309		
Cardiomyopathy	1.40 (0.86, 22.7)	0.814			2.04 (0.54 to 7.74)	0.297		
Simple biventricular CHD	0.66 (0.27 to 1.63)	0.373			0.48 (0.16 to 1.41)	0.183		
Complex biventricular CHD	0.78 (0.39, 1.57)	0.493			1.17 (0.51 to 2.70)	0.716		
Single ventricle	3.07 (1.64 to 5.75)	< 0.001			1.14 (0.47 to 2.75)	0.773		
Post Stage 1	5.64 (2.63 to 12.1)	< 0.001	2.98 (1.14 to 7.55)	0.025	-	_		
Post Stage 2	0.55 (0.19 to 1.64)	0.285			1.96 (0.61 to 6.28)	0.257		
Post Stage 3	-	-			0.70 (0.20 to 2.43)	0.578		
Post heart transplant	0.59 (0.31 to 1.15)	0.124			1.08 (0.54 to 2.17)	0.835		

Body surface area (BSA) was not included in the final models as it was found to be collinear with age and weight. Simple biventricular congenital heart disease (CHD) was defined as an isolated patent ductus arteriosus, atrial septal defect, ventricular septal defect, valvar aortic stenosis or valvar pulmonary stenosis. Complex biventricular CHD was defined as tetralogy of Fallot, pulmonary atresia/ventricular septal defect, atrioventricular septal defect or truncus arteriousus.





The Seckeler Equation for VO₂

$$VO_2 = 242.1 + [9.7 \times ln Age] - [34 \times ln Weight]$$

- $[9.6 \times Single Ventricle] - [11.2 \times Critical Illness]$

Predictive equation	All patients (n=100)	<3 years old (n=41)	≥3 years old (n=59)
New	0.53 (0.31 to 0.69)	0.29 (-0.34 to 0.63)	0.61 (0.35 to 0.77)
LaFarge	0.17 (-0.23 to 0.44)	-1.10 (-3.22 to -0.12)	0.58 (0.30 to 0.75)
Lundell	0.45 (0.19 to 0.63)	0.56 (0.16 to 0.76)	0.42 (-0.06 to 0.68)





3 kg 4 kg 5 kg 7 kg 10 kg 15 kg 20 kg 25 kg 30 kg 35 kg 40 kg 45 ka 50 ka 60 ka 70 ka 80 ka 90 ka Table 6 Predicted oxygen consumption (mL/min/m²) by age and weight for not critically ill (top) and critically ill (below) patients with two 0-6 months ventricle anatomy 7-12 months 10 kg 15 kg 20 kg 25 kg 30 kg 35 kg 40 kg 45 kg 50 kg 3 kg 4 kg 5 kg 7 kg 0-6 months 7-12 months 2 years 3 years 4 years 6 years 8 years 10 years 12 years 14 years 16 years 18 years 0-6 months 20 years 25 years 7-12 months 30 years 0-6 months 7-12 months 2 years 3 years 6 years 8 years 10 years 12 years 14 years 16 years 18 years

20 years

25 years

30 years



Table 5 Predicted oxygen consumption (mL/min/m²) by age and weight for not critically ill (top) and critically ill (below) patients with single

ventricle anatomy

2 years

3 years

4 years

6 years

8 years

10 years

12 years

14 years

16 years

18 years

20 years

25 years

30 years

2 years

3 years

4 years

6 years

8 years

10 years

12 years

14 years

16 years

18 years

20 years

25 years

30 years

University of











70 kg 80 kg 90 kg

60 kg

Examples from UPMC Children's in 2023

Diagnosis	Age	Weight	Assumed VO ₂ (LaFarge)	Assumed VO ₂ (Seckeler)	Measured VO ₂	Difference
Truncus (repaired), s/p CVA	19 months	16.2 kg	176 ml/min/m ²	147 ml/min/m ²	84 ml/min/m ²	

Directly measure oxygen consumption whenever possible.





Resting hemodynamics may not be adequate in certain cases or populations





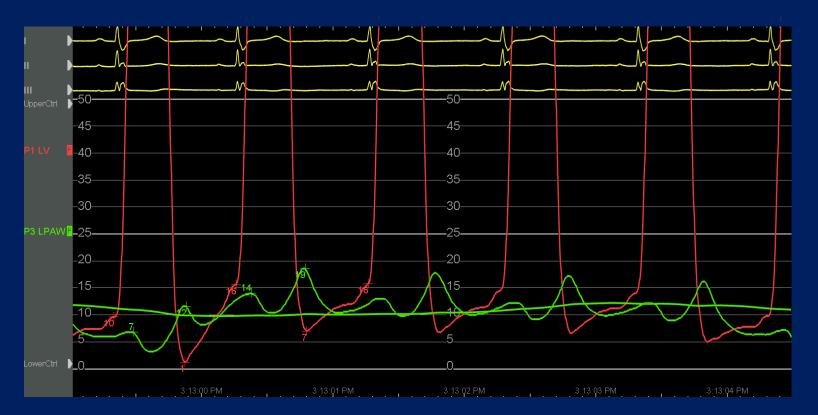
Dynamic Assessment

- Many limitations to hemodynamic assessment in the volume unloaded, anesthetized, supine and paralyzed state
- Stress hemodynamics may unmask underlying pathology that is otherwise concealed
- Exercise hemodynamics well described but frequently unachievable
- Other stressors include inotropic/chronotropic agents or volume loading





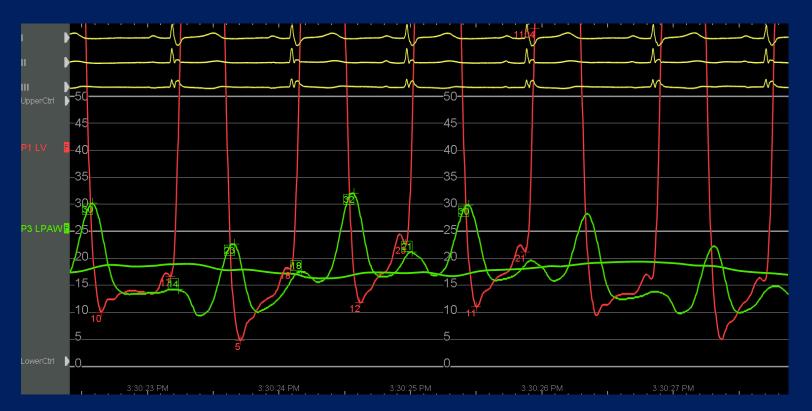
Restrictive Physiology: Classic Example







Restrictive Physiology: Classic Example







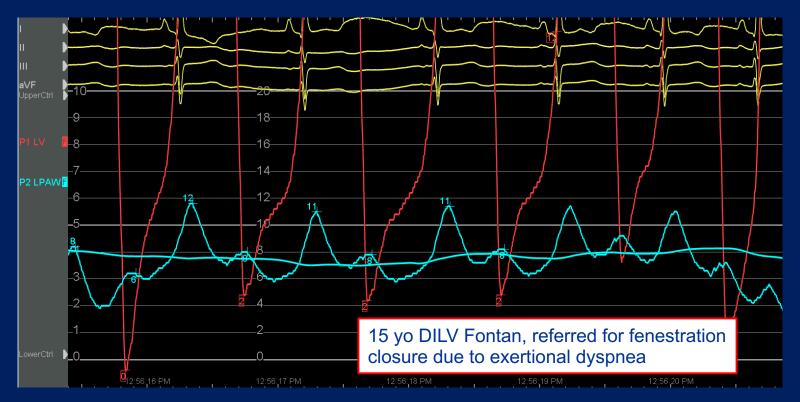
Volume Loading in CHD

- Limited role in hemodynamic assessment
- Fontan circulation offers an intriguing potential case for rapid volume expansion
- Population may be particularly prone to falsely low hemodynamics after NPO
- Patients are often symptomatic despite normal EDP, low PVR and preserved ventricular systolic function
- Only recently has diastolic dysfunction (FHFpEF) become accepted as frequent





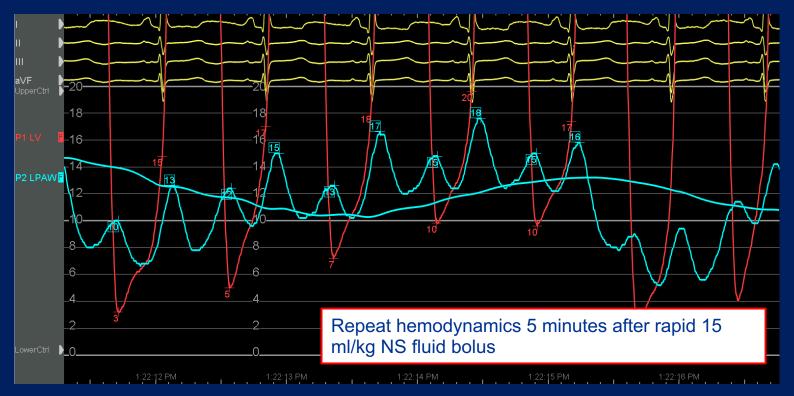
Impact of Volume Loading in Fontan





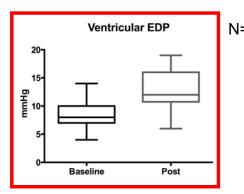


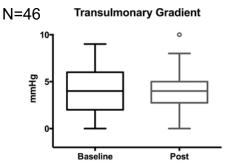
Impact of Volume Loading in Fontan

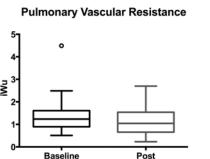


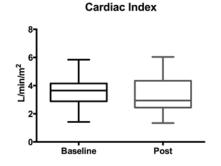


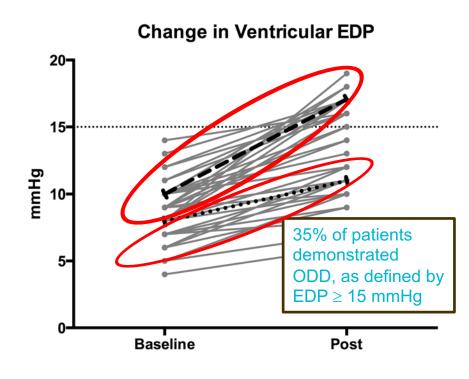
















- In univariate analysis, higher baseline EDP (p<0.001), longer duration of Fontan circulation (p=0.02) and lower baseline CI (p=0.03) were associated with higher fluid challenge EDP
- Longer duration of Fontan circulation (p=0.04) was associated with greater change in EDP
- In multivariable analysis, only higher baseline EDP (p<0.001) was significantly associated with higher fluid challenge EDP





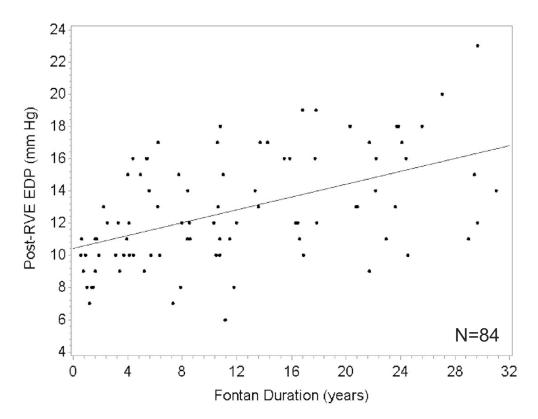




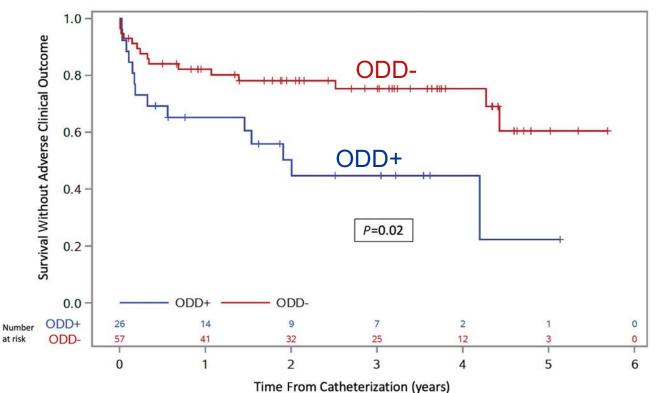


Table 3. Adverse Clinical Outcomes

	AII, N=84	ODD-positive, N=27	ODD-negative, N=57	P value
Composite clinical outcome*	29 (35)	14 (52)	15 (26)	0.03
Death, VAD, or transplant	9 (11)	3 (11)	6 (11)	1
Clinical outcome				
Mortality	4 (5)	1 (4)	3 (5)	1
Heart transplant [†]	5 (6)	2 (7)	3 (5)	0.65
VAD	1 (1)	0	1 (2)	1
Heart failure	10 (12)	5 (19)	5 (9)	0.28
Arrhythmia	10 (12)	4 (15)	6 (11)	0.72
CVA (including TIA)	4 (5)	2 (7)	2 (4)	0.59
Protein-losing enteropathy	2 (2)	2 (7)	0	0.1
Plastic bronchitis	3 (4)	1 (4)	2 (4)	1
Thrombus	1 (1)	0	1 (2)	1
Hemoptysis	2 (2)	1 (4)	1 (2)	0.52
Endocarditis	1 (1)	1 (4)	0	0.32
Seizure	1 (1)	0	1 (2)	1
Follow-up duration, y [‡]	2.9 (1.4–3.8)	2.5 (0.6–3.6)	3.0 (1.5–3.8)	0.44



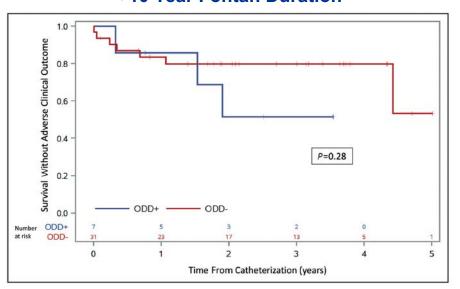




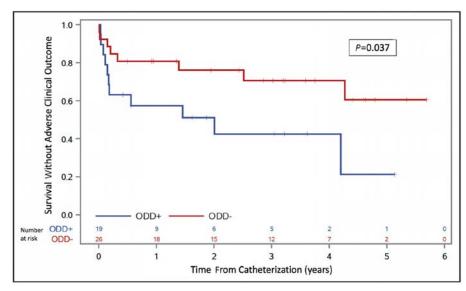




< 10 Year Fontan Duration



≥ 10 Year Fontan Duration







Dynamic Evaluation of Fontan Circulation







Dynamic Evaluation of Fontan Circulation

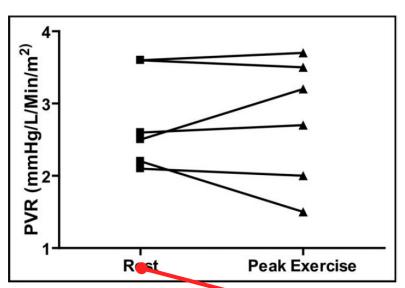
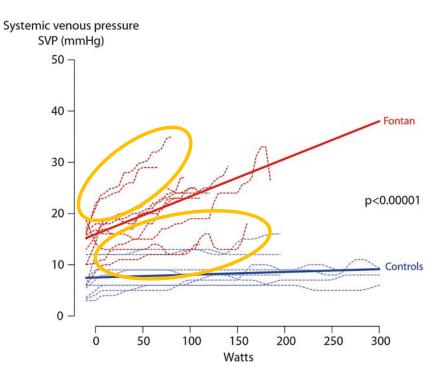


Figure 3. Change in PVR with supine exercise. Individual trends in PVR with supine exercise in 6 Fontan patients as assessed by invasive hemodynamic monitoring and determined using Fick principle.





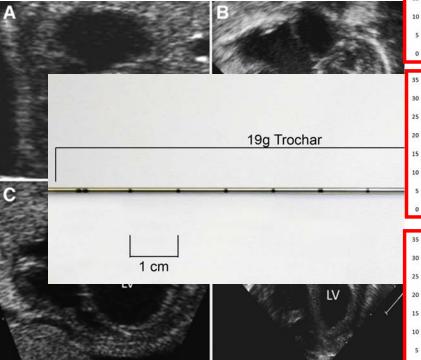


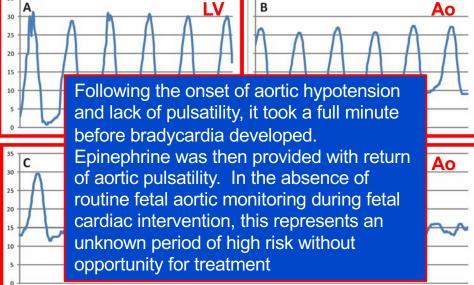
Use of novel technologies to acquire standard data

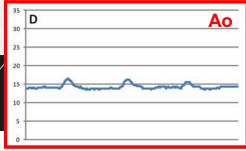


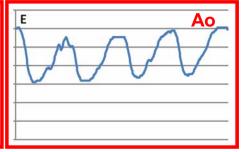


Fetal Critical AS with







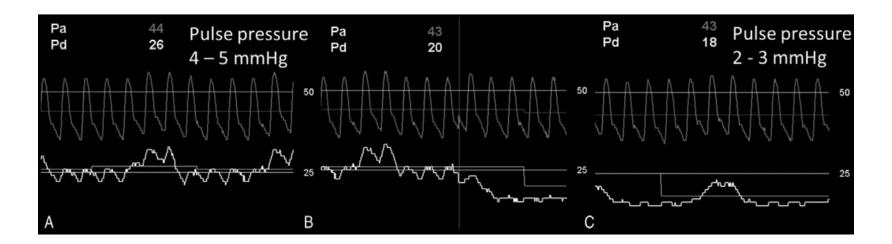






Use of a Pressure Guidewire to Assess Pulmonary Artery Band Adequacy in the Hybrid Stage I Procedure for High-risk Neonates with Hypoplastic Left Heart Syndrome and Variants

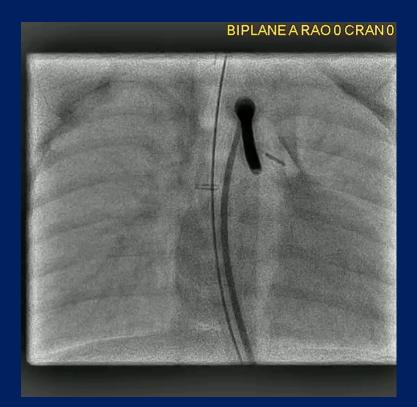
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Hybrid PA Band Placement in HLHS









Hybrid PA Band Placement in HLHS

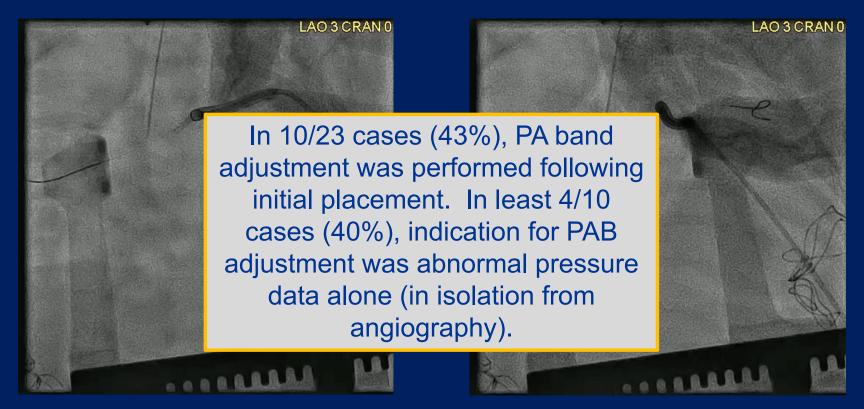








Hybrid PA Band Placement in HLHS







Conclusions

- "There is no magic in magic, it's all in the details"
 -Walt Disney
- Catheterization data are highly dependent upon the circumstances (state) at the time of data acquisition
- Assumed data are just that
- Dynamic testing (volume, exercise, pharmacologic) increases the likelihood of identifying occult pathology – e.g. Fontan ODD
- Even in an era where catheterization-related "press" is dominated by interventional procedures, nuanced hemodynamic interrogations still matter





