ACHD with Pulmonary Hypertension in Pregnancy Anesthetic Management

JOSE M. RIVERS M.D
ASSOCIATE PROFESSOR
DEPARTMENT OF ANESTHESIOLOGY
BAYLOR COLLEGE OF MEDICINE
HOUSTON, TEXAS
CASE PRESENTATION

- A 36 year-old, BMI 28, G2 P1 with a 30 week pregnancy
- Had an SVD 13 years ago with NO complications
- Past medical history significant for “hole in her heart”
- BP: 110/50 mmHg, Pulse 96, and oxygen sat 91%
- Mild cyanosis with systolic ejection murmur with loud fixed splitting of S2
- HB 15 g/dL  Hct: 46%
- TTE: Large ASD with bidirectional shunt, with severe dilation of RV, RA and PA. Flattened Septum in Systole and Diastole, moderate to severe TR. Continuous doppler estimate RVSP AND PASP 130mmHg. CI 2.2
- DOE with daily activities
- Referred from OSH for treatment
CASE PRESENTATION

- In MICU: PA Catheter (from OSH) Coiled in RV
- Interdisciplinary plan of care: cardiologist specializing in ACHD, maternal fetal medicine, pulmonary medicine, OB and cardiovascular anesthesiologist, neonatologist and nursing.
- Follow-up every 2 weeks
- Hickman Catheter for Flolan (epoprostenol) infusion
- Sildenafil PO
- Oxygen
- Diuretics
- Lovenox
- Digoxin
- ECHO: 2 Weeks: The reported RVSP is Lower and Cardiac Output has Marginally Increased
THE 9 ACTIONS

1. Recognition of Pulmonary Hypertension (PH)
2. Classification & pathophysicsiology.
3. Assessment of Severity of PH
4. Impact of Pregnancy in PH
5. Perioperative Risk Assessment
6. Preoperative Optimization
7. Choice of Anesthetic Technique
8. Choice of Monitoring
9. Treatment of Decompensated PH
Pulmonary and Systemic Circulation

- **Standard pressures**
  - Mean pulmonary artery pressure, 14 mm Hg
  - PCWP, 8-12 mm Hg
  - LVEDP, 6-12 mm Hg
  - Systemic arterial pressure ≤ 120/80 mm Hg

- **PAH pressures**
  - Mean pulmonary artery pressure > 25 mm Hg
  - PCWP and LVEDP < 15 mm Hg
  - Systemic arterial pressure ≤ 120/80 mm Hg

PCWP = Pulmonary capillary wedge pressure; LVEDP = Left ventricular end-diastolic pressure; © 2007, Gilead Sciences, Inc.
## Hemodynamic Values at Rest

<table>
<thead>
<tr>
<th>Normal Hemodynamic Values at Rest</th>
<th>Diagnostic Criteria for PAH</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>RAP</strong> 1-5 mm Hg</td>
<td>&gt;25 mm Hg</td>
</tr>
<tr>
<td><strong>RVP</strong> 15-30/1-7 mm Hg</td>
<td>≤15 mm Hg</td>
</tr>
<tr>
<td><strong>PAP</strong> 15-30/4-12 mm Hg</td>
<td></td>
</tr>
<tr>
<td><strong>mPAP</strong> 9-19 mm Hg</td>
<td>&gt;25 mm Hg</td>
</tr>
<tr>
<td><strong>PCWP</strong> 4-12 mm Hg</td>
<td>≤15 mm Hg</td>
</tr>
<tr>
<td><strong>CO</strong> 4-7 L/min</td>
<td></td>
</tr>
<tr>
<td><strong>PVR</strong> &lt;3 Wood Units</td>
<td>&gt;3 Wood Units</td>
</tr>
</tbody>
</table>

**RAP**=right atrial pressure; **RVP**=right ventricular pressure; **PAP**=pulmonary arterial pressure; **mPAP**=mean pulmonary arterial pressure; **PCWP**=pulmonary capillary wedge pressure; **CO**=cardiac output; **PVR**=pulmonary vascular resistance.


Resistance = Pressure/Flow

SVR: MAP – CVP = 90-2 = 18 Wood Units
    CO = 5 or 1455 dynes.S.cm^5

PVR: MPAP - (PCWP) (LA) = 15-10 = 1 Wood Units
    CO = 5 or 80 dynes.S.cm^5

MPAP: LAP + (CO x PVR)
Normal MPAP <25 mmHg)
## Diagnosis of PAH*

<table>
<thead>
<tr>
<th>Diagnostic</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>History and physical†</td>
<td>▪ Evaluate signs and symptoms, family history, associated diseases, ANAs</td>
</tr>
<tr>
<td>Chest x-ray†</td>
<td>▪ Assess for RV enlargement, peripheral hypovascularity (pruning), and prominent pulmonary arteries</td>
</tr>
<tr>
<td>Echocardiogram</td>
<td>▪ Assess for RV and RA enlargement, RV dysfunction, TR velocity to measure RVSP</td>
</tr>
<tr>
<td>Electrocardiogram</td>
<td>▪ Evaluate for right heart enlargement and strain, cardiac rhythm</td>
</tr>
<tr>
<td>Cardiac catheterization†</td>
<td>▪ Evaluate for CHD; measure wedge pressure or LVEDP; establish severity and prognosis; test vasodilator therapy</td>
</tr>
<tr>
<td>PFTs</td>
<td>▪ Assess obstructive and restrictive airway disease</td>
</tr>
<tr>
<td>VQ scan</td>
<td>▪ Rule out thromboembolic disease</td>
</tr>
</tbody>
</table>

ANA = antinuclear antibody; CHD = congenital heart disease; LVEDP = left ventricular end-diastolic pressure; PFT = pulmonary function test; RA = right atrial; RV = right ventricular; RVSP = right ventricular systolic pressure; TR = tricuspid regurgitation; VQ = ventilation-perfusion.

*Additional tests may be ordered to rule out possible causes of PAH (pulmonary arteriography, blood tests [HIV, hepatic disease, scleroderma], polysomnography [sleep-disordered breathing]). †Required for referral.
Updated Clinical Classification of Pulmonary Hypertension

1. Pulmonary arterial hypertension (PAH)
   1. idiopathic (“primary pulmonary hypertension”)
   2. secondary to systemic disorders

2. Pulmonary hypertension due to left heart disease (pulmonary venous hypertension)

3. Pulmonary hypertension associated with respiratory disease and/or hypoxia
   1. COPD, Interstitial lung disease, OSA

4. Chronic thromboembolic/embolic pulmonary hypertension

5. Pulmonary hypertension from unclear mechanisms

Journal of the American College of Cardiology
WHO Group 1: Pulmonary Arterial Hypertension

1. Pulmonary arterial hypertension
   1.1 Idiopathic (IPAH)
   1.2 Heritable
      1.2.1 BMPR2
      1.2.2 ALK1, endoglin (with or without hereditary hemorrhagic telangiectasia)
      1.2.3 Unknown
   1.3 Drug- and toxin-induced
   1.4 Associated with:
      1.4.1 Connective tissue diseases
      1.4.2 HIV infection
      1.4.3 Portal hypertension
      1.4.4 Congenital heart diseases
      1.4.5 Schistosomiasis
      1.4.6 Chronic hemolytic anemia
   1.5 Persistent pulmonary hypertension of the newborn

BMPR2 = bone morphogenetic protein receptor type 2; ALK1 = activin receptor-like kinase type 1.
Figure 1. Prevalence of pulmonary arterial hypertension (PAH) in patients with congenital heart disease (CHD) according to defect. The study population includes patients with corrected and uncorrected defects. CHD n=2,839 PAH-CHD: n=248. ASD; atrial septal defect; VSD: ventricular septal defect.
<table>
<thead>
<tr>
<th>1. Type</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Simple pre-tricuspid shunts</td>
<td></td>
</tr>
<tr>
<td>Atrial septal defect (ASD)</td>
<td></td>
</tr>
<tr>
<td>=) ostium secundum</td>
<td></td>
</tr>
<tr>
<td>=) sinus venosus</td>
<td></td>
</tr>
<tr>
<td>Total or partial unobstructed anomalous pulmonary venous return</td>
<td></td>
</tr>
<tr>
<td>Simple post-tricuspid shunts</td>
<td></td>
</tr>
<tr>
<td>Ventricular septal defect (VSD)</td>
<td></td>
</tr>
<tr>
<td>Patent ductus arteriosus</td>
<td></td>
</tr>
<tr>
<td>Combined shunts</td>
<td></td>
</tr>
<tr>
<td>Describe combination and define predominant defect</td>
<td></td>
</tr>
<tr>
<td>Complex congenital heart disease</td>
<td></td>
</tr>
<tr>
<td>Atrioventricular septal defects</td>
<td></td>
</tr>
<tr>
<td>=) partial (Ostium primum ASD)</td>
<td></td>
</tr>
<tr>
<td>=) complete</td>
<td></td>
</tr>
<tr>
<td>Truncus arteriosus</td>
<td></td>
</tr>
<tr>
<td>Single ventricle physiology with unobstructed pulmonary blood flow</td>
<td></td>
</tr>
<tr>
<td>Transposition of the great arteries with VSD (without pulmonary stenosis) and/or patent ductus arteriosus</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td></td>
</tr>
</tbody>
</table>

2. Dimension (specify for each defect if more than one congenital heart defect)


<table>
<thead>
<tr>
<th>Haemodynamic</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Restrictive (pressure gradient across the defect)</td>
<td></td>
</tr>
<tr>
<td>Non-restrictive</td>
<td></td>
</tr>
<tr>
<td>Anatomical</td>
<td></td>
</tr>
<tr>
<td>Small to moderate (ASD ≤ 2 cm and VSD ≤ 1 cm)</td>
<td></td>
</tr>
<tr>
<td>Large (ASD &gt; 2 cm and VSD &gt; 1 cm)</td>
<td></td>
</tr>
</tbody>
</table>

3. Direction of shunt

| Predominantly systemic-to-pulmonary | |
| Predominantly pulmonary-to-systemic | |
| Bidirectional                       | |

4. Associated extracardiac abnormalities

5. Repair status

<table>
<thead>
<tr>
<th>Unoperated</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Palliated (specify type of operation/s, age at surgery)</td>
<td></td>
</tr>
<tr>
<td>Repaired (specify type of operation/s, age at surgery)</td>
<td></td>
</tr>
</tbody>
</table>
Pathogenesis of PAH: Vasoconstriction

Vasodilation

Decreased
NO synthase
Prostacyclin
NO

Vasoconstriction

Increased
Endothelin
Serotonin
Thromboxane
Mechanisms of Pathology for PAH

Endothelin pathway

Preproendothelin → Proendothelin → Endothelial cells

Endothelin-1

Endothelin-receptor A

Endothelin-receptor B

Endothelin-receptor antagonists

Vasoconstriction and proliferation

Nitric oxide pathway

L-arginine

Nitric oxide

NOS

Nitric oxide pathway

Nitric oxide pathway

Vasodilatation and antiproliferation

Phosphodiesterase type 5

Phosphodiesterase type 5 inhibitor

Exogenous nitric oxide

Prostacyclin pathway

Arachidonic acid → Prostaglandin I₂

Prostacyclin

Prostacyclin pathway

Prostacyclin derivates

Vasodilatation and antiproliferation

## Determinants of Disease Severity

<table>
<thead>
<tr>
<th>Determinants of risk</th>
<th>Lower risk</th>
<th>Higher risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical evidence of RV failure</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Progression</td>
<td>Gradual</td>
<td>Rapid</td>
</tr>
<tr>
<td>WHO functional class</td>
<td>II, III</td>
<td>IV</td>
</tr>
<tr>
<td>6MWD</td>
<td>Longer (&gt;400 m)</td>
<td>Shorter (&lt;300 m)</td>
</tr>
<tr>
<td>BNP</td>
<td>Minimally elevated</td>
<td>Very elevated</td>
</tr>
<tr>
<td>Echocardiographic findings</td>
<td>Minimal RV dysfunction</td>
<td>Significant RV dysfunction, pericardial effusion</td>
</tr>
<tr>
<td>Hemodynamics</td>
<td>Normal/near normal RAP and CI</td>
<td>High RAP, low CI</td>
</tr>
</tbody>
</table>

BNP = brain natriuretic peptide; CI = cardiac index; RAP = right artery pressure; RV = right ventricular.

From McLaughlin and McGoon. With permission.

Echocardiographic Indicators of Poor Prognosis in PAH

- **Decrease**
  - Right ventricular function

- **Increase**
  - Right atrial indexed area
  - Diastolic septal shift
  - Doppler right ventricular performance index
  - Pericardial effusion

<table>
<thead>
<tr>
<th>WHO</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I</td>
<td>No limitation of physical activity; no symptoms with ordinary physical activity</td>
</tr>
<tr>
<td>Class II</td>
<td>Slight limitation of physical activity; ordinary physical activity causes PAH symptoms</td>
</tr>
<tr>
<td>Class III</td>
<td>Marked limitation of physical activity; less than ordinary physical activity causes PAH symptoms</td>
</tr>
<tr>
<td>Class IV</td>
<td>PAH symptoms with any physical activity and even at rest; discomfort with any physical activity; signs of right heart failure</td>
</tr>
</tbody>
</table>

Rubin. *Chest*. 2004;126(suppl 1):7S-10S.
New York Heart Association (NYHA) Classification (1979)

- **Class I** – uncompromised
  - No symptoms of cardiac disease

- **Class II** – limited symptomatology
  - These patients are asymptomatic at rest but develop symptoms (chest pain, shortness of breath, fatigue, palpitations) with physical exertion

- **Class III** – marked symptomatology
  - These patients are asymptomatic at rest but become symptomatic with even minimal physical activity

- **Class IV** – symptoms at rest
Correlation of Six-minute-walk Test With Survival in PPH

6-minute-walk distance strongly predictive of survival:
- <332 m: 20% 3-year survival
- >332 m: 92% 3-year survival
## Grading of pulmonary arterial hypertension

<table>
<thead>
<tr>
<th></th>
<th>Systolic</th>
<th>Diastolic</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Grade 1</strong> (Mild)</td>
<td>30-50</td>
<td>20-25</td>
<td>&gt;30</td>
</tr>
<tr>
<td><strong>Grade 2</strong> (Moderate)</td>
<td>50-70</td>
<td>26-35</td>
<td>&gt;40</td>
</tr>
<tr>
<td><strong>Grade 3</strong> (Severe)</td>
<td>70-110</td>
<td>36-45</td>
<td>&gt;50</td>
</tr>
<tr>
<td><strong>Grade 4</strong> (Systemic or supra systemic)</td>
<td>&gt;110</td>
<td>46-55</td>
<td>&gt;60</td>
</tr>
</tbody>
</table>

*Data from 100 patients of PAH and rheumatic heart disease, Quintile 1 & 2 (Grade 1) quintile 3 & 4 (Grade 2) quintile 5 (Grade 3) top 3% (Grade 4)*
Eisenmenger Syndrome

Definition:

Pulmonary hypertension at or near systemic level with reversed or bidirectional shunt between the pulmonary and systemic circulation and pulmonary vascular resistance above 800dyn/cm$^5$ (10 Wood Units)

Eisenmenger Syndrome - A Progressive Disease

Left-to-right shunt

- Increased pulmonary blood flow (shear stress/circumferential stretch)
  - Endothelial dysfunction and vascular remodeling
    - Smooth muscle cell proliferation, increase in extracellular matrix, intravascular thrombosis
    - Increase in PVR
    - Inverted shunt: right-to-left
  - Cyanosis (Eisenmenger syndrome)
Maternal Cardiac Physiology
Cardiovascular Alterations

- Decreased systemic vascular resistance
- Decreased blood pressure
- Decreased pulmonary vascular resistance
- Increased heart rate
- Increased cardiac output

\[ \text{CO} = \text{SV} \times \text{HR} \]
Maternal Cardiac Physiology
Cardiovascular Alterations

The graph shows the percent changes in blood volume from nonpregnant levels as a function of weeks of gestation. The changes are measured over the course of pregnancy and within 6 weeks postpartum. The lines represent different components:

- Blue line: Blood volume
- Orange line: Plasma volume
- Red line with triangle markers: Red cell volume

The graph indicates a significant increase in blood volume and plasma volume during pregnancy, peaking around the 30th week of gestation, with a sharp decline after delivery.
Maternal Cardiac Physiology
Cardiovascular Alterations

![Chart showing cardiac output liters per minute across different stages of pregnancy and labor.](chart.png)

- Cardiac output liters per minute:
  - Nonpregnant: 5
  - 20–24 weeks: 6.8
  - 28–32 weeks: 7.1
  - 38–40 weeks: 5.8
  - Early labor: 6.2
  - Late labor: 7.2
  - 2nd stage: 8.9
  - Immediately postpartum: 9.3

Weeks’ gestation

Labor
Supportive Therapy and General Measures in PAH

- Anticoagulants (IPAH/HPAH)
  - Favorable data primarily from retrospective trials

- Diuretics
  - Standard of care for right-heart failure
  - Clinician preference on choice of agents

- Oxygen
  - Low-flow supplemental O2 improved outcome in clinical case series; maintain SaO2 >92%
    - Not evaluated in randomized controlled trial

- Digoxin
  - Modest increase in cardiac output
  - No data available on long-term management

- Supervised exercise program rehabilitation

ACCP Recommendations for Vasodilator Testing During RHC

1. Administer appropriate pulmonary vasodilator
   a) Measure hemodynamics every 10 – 15 min

2. Record changes in hemodynamics
   a) Focus on changes in mPAP, PCWP, and CO
   b) Monitor changes to BP, HR, and O₂ saturation to ensure patient safety

3. Interpret change in hemodynamics
   a) Acute response: ≥10 mm Hg ↓ in mPAP to mPAP ≤40 mm Hg with normal or increased CO

ACCP=American College of Chest Physicians; RHC=right heart catheterization; mPAP=mean pulmonary arterial pressure; PCWP=pulmonary capillary wedge pressure; CO=cardiac output; BP=blood pressure; HR=heart rate.
Mechanisms of Pathology for PAH

Endothelin pathway

Preproendothelin → Proendothelin

Endothelin-1

Endothelin-receptor A

Endothelin-receptor B

Endothelin-receptor antagonists

Vasoconstriction and proliferation

Nitric oxide pathway

L-arginine

Nitric oxide

cGMP

Endothelin

Endothelin-receptor A

Endothelin-receptor B

Exogenous nitric oxide

Vasodilatation and antiproliferation

Phosphodiesterase type 5

Phosphodiesterase type 5 inhibitor

Prostacyclin pathway

Arachidonic acid → Prostaglandin I₂

Prostaglandin I₂

cAMP

Vasodilatation and antiproliferation

Prostacyclin derivates

## Updated Guidelines: PAH-Specific Therapies Available in the US

<table>
<thead>
<tr>
<th>Strength of Recommendation</th>
<th>WHO Class II</th>
<th>WHO Class III</th>
<th>WHO Class IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Ambrisentan, bosentan, sildenafil</td>
<td>Ambrisentan, bosentan, epoprostenol IV, iloprost inh, sildenafil</td>
<td>Epoprostenol IV</td>
</tr>
<tr>
<td>B</td>
<td>Tadalafil</td>
<td>Tadalafil, treprostinil SC</td>
<td>Iloprost inh</td>
</tr>
<tr>
<td>C</td>
<td></td>
<td>Treprostinil IV</td>
<td>Treprostinil SC</td>
</tr>
<tr>
<td>E/B</td>
<td></td>
<td>Treprostinil IV</td>
<td>Treprostinil IV, initial combo tx</td>
</tr>
<tr>
<td>E/C</td>
<td></td>
<td>Treprostinil inh</td>
<td>Ambrisentan, bosentan, sildenafil, tadalafil</td>
</tr>
<tr>
<td>Recently approved</td>
<td></td>
<td>Treprostinil inh</td>
<td>Treprostinil inh</td>
</tr>
</tbody>
</table>

Vasodilator Agents

- Nitric Oxide:
  - Inhaled gas, short half life, prospective studies, 20-80ppm for 3-5 minutes

- Flolan:
  - 2-10 ng/kg/min
  - Increase by 2ng/kg/min until goal
  - SE: headache, flushing, nausea

- Response: mean PAP <40mmHg, 10 mmHg average drop, maintain CO
Pregnancy outcomes in pulmonary arterial hypertension in the modern management era

Xavier Jais**,‡,†,#,++; Karen M. Olsson†,†,‡; Joan A. Barbera§; Isabel Blanco§; Adam Torbicki†; Andrew Peacock**, C. Dario Vizza**, Peter Macdonald†,‡; Marc Humbert*,†,‡,§,++; and Marius M. Hoeper**

ABSTRACT: Previous studies have reported mortality rates of up to 56% associated with pregnancy in pulmonary arterial hypertension (PAH) but the management of this disease has changed considerably in recent years.

We compiled a multinational, prospective registry to examine the contemporary outcome of pregnancies in patients with PAH.

During a 3-yr period, the 13 participating centres reported 26 pregnancies. Three (12%) females died and one (4%) developed right heart failure requiring urgent heart–lung transplantation. There were eight abortions; two spontaneous and six induced. 16 (62%) pregnancies were successful, i.e. the females delivered healthy babies without complications. These females had well controlled PAH (pulmonary vascular resistance (PVR) 500 ± 352 dyn·s·cm⁻⁵); eight of them were long-term responders to calcium channel blockers. In contrast, the females who died or required transplantation had poorly controlled PAH (PVR 1,667 ± 209 dyn·s·cm⁻⁵).

Pregnancy remains associated with a substantial mortality rate in PAH. However, our results indicate that the outcome of pregnancy in PAH has improved, at least when PAH is well controlled, and particularly in long-term responders to calcium channel blockers. These data must be confirmed by larger series before the general recommendation to avoid pregnancy in all patients with PAH is reconsidered.

KEYWORDS: Hypertension, pregnancy, pulmonary, right heart failure
Choice of Anesthetic Technique

- HEMODYNAMIC GOALS
  - Maintain Preload
  - Maintain Afterload
  - Maintain Contractility
  - Maintain Sinus Rhythm
  - Avoid Increase in PVR
Factors Which Increase Pulmonary Vascular Resistance (and Decrease PBF)

- Hypoxia
- Hypercarbia
- Hypotension
- Hypovolemia
- Acidosis
- Mechanical Ventilation
- Sympathetic stimulation
PERI-OPERATIVE HEMODYNAMIC GOALS

Systolic blood pressure ≥ 110 mmHg and/or 40 mmHg above sPAP
MAP ≥ 75 and/or 20 mmHg above mPAP
mPAP < 35 mmHg or 25 mm HG lower than MAP
PVR/SVR ratio < 0.5 or aim for pre-operative PVR/SVR ratio RAP the lowest possible that maintains
MAP > 75 mmHG
Cardiac index ≥ 2.2 l.min⁻¹ .m²

sPAP, systolic pulmonary artery pressure; MAP, mean arterial pressure, mPAP, mean pulmonary artery pressure; PVR/SVR ratio, pulmonary vascular resistance/systemic vascular resistance ratio; RAP, right atrial pressure.
Elective Cesarean Section at 36 weeks

**General Considerations**
- Continue with Epoprostenol Infusion
- Sildenafil and Digoxin PO
- Stop Lovenox X 24 hours
- Air Filters in all IVs and CVP
- Labs: CBC, BMP, ABG, BNP, PT, PTT, ACT, TEG
- Blood Bank: PRBCs and Platelets
- Excellent IVs
- Left Uterine Displacement
- Avoid: Hypoxemia, Hypercapnia, Acidosis, Pain, Stress and drugs increase PA pressure

**Monitoring:**
- ASA Standard Monitors
- Arterial Line with EV 1000
- Oxymetric CVP
- Continuous TTE
Evolution of Continuous Cardiac Monitoring

Swan Ganz Catheter introduced in 1970

FloTrac introduced in 2005

PreSep
The *FloTrac* sensor... why should I use it?

- CO = Cardiac Output
- CI = Cardiac Index
- SV = Stroke Volume
- SVI = Stroke Volume Index
- SVR = Systemic Vascular Resistance
- SVRI = Systemic Vascular Resistance Index
- SVV = Stroke Volume Variation
System Configuration

*PreSep* Catheter
((ScvO₂)

Venous Oximetry

**EV1000**
Monitor

*FloTrac* Sensor
(peripheral artery)
CASE PRESENTATION

- Multiple infusions:
  - Vasopressors
    - Phenylephrine
    - Norepinephrine
    - Vasopressin
  - Inotropics
    - Dobutamine
    - Milrinone
  - Oxitocin
CASE PRESENTATION

- SVD/Elective Cesarean Section and BTL
  - Loss of resistance with Saline
  - CSE Early in Labor with Fentanyl/Duramorph
  - Slow Epidural
    - Bupivacaine 0.125% + Fentanyl 2 mcg/ml
    - Lidocaine 2% or Ropivacaine 0.5%
    - Post Operative Pain Management with Neuraxial Opioids (Morphine, Butorphanol)
- Obstetrical ICU for 72 Hours
CASE PRESENTATION

▪ Urgent Cesarean Section and BTL
  ▪ Modified RSI: Remifentanil, Etomidate, Rocuronium
  ▪ Avoid Nitrous Oxide
  ▪ Oxytocin Slow Infusion
  ▪ Low Tidal Volumes and PEEP
  ▪ TAP Block for Post Operative pain Management
  ▪ Obstetrical ICU for 72 Hours
### ANESTHETIC TECHNIQUE UTILISED FOR CESAREAN DELIVERY ACCORDING TO URGENT CATEGORY

<table>
<thead>
<tr>
<th></th>
<th>Category 1 (n=2)</th>
<th>Category 2 (n=2)</th>
<th>Category 3 (n=9)</th>
<th>Category 4 (n=24)</th>
<th>All (n=37)</th>
</tr>
</thead>
<tbody>
<tr>
<td>General anesthesia</td>
<td>2 (100%)</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2 (5%)</td>
</tr>
<tr>
<td>Single-shot spinal</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>6 (25%)</td>
<td>6 (16%)</td>
</tr>
<tr>
<td>Epidural de novo</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>12 (50%)</td>
<td>13 (35%)</td>
</tr>
<tr>
<td>Epidural in situ</td>
<td>0</td>
<td>0</td>
<td>8 (89%)</td>
<td>0</td>
<td>7 (19%)</td>
</tr>
<tr>
<td>Low-dose CSE</td>
<td>0</td>
<td>1 (50%)</td>
<td>0</td>
<td>3 (13%)</td>
<td>4 (11%)</td>
</tr>
<tr>
<td>Standard CSE</td>
<td>0</td>
<td>1 (50%)</td>
<td>1 (11%)</td>
<td>3 (13%)</td>
<td>5 (14%)</td>
</tr>
</tbody>
</table>

CSE, combined spinal-epidural.
# EFFECT OF ANAESTHETIC AGENTS ON RIGHT VENTRICULAR (RV) CONTRACTILITY AND PULMONAR VASCULAR RESISTANCE

<table>
<thead>
<tr>
<th>Anaesthetic Agent</th>
<th>Isoflurane</th>
<th>Desflurane</th>
<th>Sevoflurane</th>
<th>Nitrous oxide</th>
<th>Thiopental</th>
<th>Etomidate</th>
<th>Ketamine</th>
<th>Propofol</th>
<th>Opiods</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV Contractility</td>
<td>↓↓↓</td>
<td>↓↓↓</td>
<td>↓↓↓</td>
<td>↓↓↓</td>
<td>↓↓↓</td>
<td>↓↓</td>
<td>↓↓</td>
<td>↓↓</td>
<td>↔</td>
</tr>
<tr>
<td>Pulmonary vascular resistance</td>
<td>↑↑</td>
<td>↔</td>
<td>↑↑↑</td>
<td>↔↑↑↑</td>
<td>↔↑↑↑</td>
<td>↓↑</td>
<td>↑↑ Adult</td>
<td>↓↑↑↑</td>
<td>↔</td>
</tr>
</tbody>
</table>

- ↓↓↓-marked decrease; ↑↑↑-marked increase; ↑↑-increase; ↓↓-decrease; ↔-no change; - not known.
Consequences of PAH

↑ PVR

↑ RV afterload

↓ RV ejection (CO) and PBF

RV hypertropy and dilation

RV failure

Death

PVR=pulmonary vascular resistance; RV=right ventricular; CO=cardiac output; PBF=pulmonary blood flow.
Treatment Acute R.Ventricular Failure

- REVERSE HYPOTENSION
  - Right Ventricular Ischemia
    - Right Coronary Flow in Systole and Diastole
    - Increased Oxygen Consumption
    - Cycle of Ischemia and Failure

- Role Interventricular Septum
### Etiologies of Hypotension

<table>
<thead>
<tr>
<th></th>
<th>CVP</th>
<th>PAP</th>
<th>PAOP</th>
<th>CO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased preload</td>
<td>↓↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Decreased contractility</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Decreased SVR</td>
<td>→</td>
<td>→</td>
<td>→ or ↓</td>
<td>↑ or →</td>
</tr>
<tr>
<td>Increased PVR</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td></td>
</tr>
</tbody>
</table>
MANAGEMENT OF HYPOTENSION

Is CVP decreased?  Yes  Volume
   ↓  No

Is PAP decreased?  Yes  Inotropes
   ↓  No

Are there reversible causes of increased PVR?  No  ↓  Yes

Is cardiac output decreased?  Treatment
   ↓  No

Systemic  ↓  Yes
Vasoconstrictors  Inotropes and/or
Pulmonary vasodilators
MANAGEMENT OF HYPOTENSION

Is CVP decreased?  Yes  Volume
  ↓ No

Is PAP decreased?  Yes  Inotropes
  ↓ No

Are there reversible causes of increased PVR?  ↓ No
  ↓ Yes

Is cardiac output decreased?  Treatment
  ↓ No

Systemic  ↓ Yes
Inotropes and/or
Vasoconstrictors  Pulmonary vasodilators
Inhaled
CONCLUSIONS

- Recognize potential for PH based on signs and symptoms
- Identify etiology of PH and treat reversible causes
- Use right heart cath to confirm dx, and help risk stratify
- Once start therapy have close clinical f/u to follow response
- Decide on treatment goals
- Consider referral to PAH center or clinic
- Future may be combination therapy