Right Ventricular Failure and Pulmonary Hypertension 2011

George G. Sokos, DO FACC
Assistant Professor of Medicine, Temple University
Director, Advanced Heart Failure and Cardiac Transplant Fellowship
Allegheny General Hospital
Pittsburgh, PA
BIGEMINAL PRODUCTIONS
PRESENTS
Living Arrhythmias
Overview of Right Ventricular Failure and Pulmonary Hypertension

- Normal Right Ventricle
- Definition of RV Failure
- Pathophysiology of RV Failure
- Etiology
- Diagnosis
- Treatment
Historical Perspective of RV function

- Physiology has been poorly understood
- Harvey (1616) first described RV and its function
- Right Ventricle thought to be a passive conduit for many years (Starr et al 1943)
- Cohn renewed interest in 1974 with recognition of RV infarct in Inferior wall MI
- NIH special emphasis area concentrating on RV Function
Normal RV characteristics

- RV is not simply weak Left Ventricle
- Normal RV wall thickness: 2-3 mm
- Normal LV wall thickness: 8-11 mm
- RV contraction: peristaltic-like beginning at apex and moves in a wave toward the outflow tract

- Coronary perfusion:
  - Vascular supply: 2/3 RCA, 1/3 left branches
  - LV confined to diastole
  - RV with continuous perfusion (low pressure)

- Less O2 requirements than the LV: less myocardic mass, less pre load and afterload
Right Ventricular Physiology

Figure 1. Illustration of how the geometry of the right ventricle (RV) changes with contraction and is affected by pressure overload. Top, the crescentic RV flattening in systole, leading to a large-volume change with minimal change in RV free wall area. Bottom, how a shift of the interventricular septum during acute pressure overload permits RV end-diastolic volume to increase with no change in end-diastolic RV free wall area, a decrease in interventricular septal surface area, and a corresponding decrease in left ventricle (LV) end-diastolic volume. Because the RV free wall does not stretch, there is no recruitment of RV function via the Frank-Starling mechanism, while at the same time there is a loss of function via the Frank-Starling mechanism in the LV.
Normal RV and LV Hemodynamics

| Table 1. Typical values for systemic and pulmonary pressure and resistance |
|--------------------------------------------------|------------------|
|                                                   | Pulmonary/RV/RA | Systemic/LV/LA |
| Pressure, mm Hg, average ± range                  |                  |                |
| Atrial mean                                       | 2–7              | 2–12           |
| Ventricular systolic                              | 15–28            | 90–140         |
| Ventricular diastolic                             | 0–8              | 4–12           |
| Vascular mean                                     | 10–16            | 65–105         |
| Resistance, dyne-sec-cm⁻⁵·M², average ± sd        |                  |                |
| Vascular                                          | 123 ± 54         | 2130 ± 450     |

RV, right ventricle; RA, right atrium; LV, left ventricle; LA, left atrium.
Data from Grossman and Baim (23), Davidson and Bonow (24), and others.
Hemodynamic Values at Rest: Normal Ranges\textsuperscript{1-3}

\[ TPG = mPAP - \text{mean PCWP} \]

**Normal TPG** = <12 mm Hg

\[ PVR = \frac{TPG}{CO} \]

\[ \times 80 = \text{dyne\textperiodcentered sec\textperiodcentered cm}^{-5} \]

**Normal PVR** = <3 Wood units

(<240 dyne\textperiodcentered sec\textperiodcentered cm\textsuperscript{-5})

\[ PCWP = \text{LAP} \]

CO, cardiac output; LAP, left atrial pressure; mPAP, mean pulmonary arterial pressure; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; TPG, transpulmonary gradient.

Characterization of the Pulmonary Circulation

- Low pressure system
  - One fifth the pressures of systemic circulation, despite same CO as systemic circulation

- Low resistance
  - ~One seventh the resistance of systemic circulation

- High capacitance
  - Accommodates 5- to 6-fold ↑ in blood flow with only 2-fold ↑ in PAP

- Dynamic vascular bed
  - V:Q matching; vasodilatation and recruitment

Pulmonary vascular system

CO, cardiac output; PAP, pulmonary arterial pressure; V:Q, ventilation-perfusion.
Right Ventricular Failure

- **Multiple Definitions**
  - Results from structural or functional process decreasing the ability of the RV to pump blood into the pulmonary circulation
  - The clinical syndrome resulting from the inability of the right ventricle to provide adequate blood flow to the pulmonary circulation at a normal central venous filling pressure
Pathophysiologic changes seen in RV failure resulting from increased afterload. Adapted with permission from Lualdi and Goldhaber. ¹⁰
Vicious cycle of auto-aggravation

RV pressure overload

Reduced cardiac output

Systemic hypotension

Reduced RV tissue perfusion

RV free wall ischemia

Reduced RV free wall contractility

Pathophysiology of Failing RV

Ventricular Interdependence

- During systole, LV protrudes in RV
- Surrounding pericardium with limited distensibility
- Compliance of one ventricle can modify the other = Diastolic ventricular interaction
# Etiologies of RV Failure

<table>
<thead>
<tr>
<th>Table 1 Causes of Acute RV Failure in the Intensive Care Unit</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left ventricular dysfunction</strong></td>
</tr>
<tr>
<td>RV co-involvement in structural or ischemic heart disease or indirect RV dysfunction due to ventricular interdependence, pulmonary venous congestion, and/or arrhythmias</td>
</tr>
<tr>
<td><strong>RV ischemia (via negative effects on inotropy and/or relaxation or via arrhythmias)</strong></td>
</tr>
<tr>
<td>Relative RV Ischemia secondary to RV pressure or volume overload</td>
</tr>
<tr>
<td><strong>Afterload increase (endothelial dysfunction, vasoconstriction, and/or mechanical obstruction)</strong></td>
</tr>
<tr>
<td>Hypoxic pulmonary vasoconstriction</td>
</tr>
<tr>
<td>Post-cardiothoracic surgery (CABG, corrective surgery for CHD, heart/lung transplantation, pneumonectomy)</td>
</tr>
<tr>
<td>Pulmonary embolus</td>
</tr>
<tr>
<td>Pulmonary microthrombi (sepsis and acute lung injury)</td>
</tr>
<tr>
<td>Pulmonary stenosis/RV outflow tract obstruction</td>
</tr>
<tr>
<td>Acute chest syndrome in sickle cell disease</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
</tr>
<tr>
<td><strong>Pre-load decrease (via effects on RV fiber length and contractility)</strong></td>
</tr>
<tr>
<td>Superior vena cava syndrome</td>
</tr>
<tr>
<td>Tricuspid stenosis</td>
</tr>
<tr>
<td>Cardiac tamponade (inhibition of diastolic filling)</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
</tr>
<tr>
<td><strong>Intrinsic myocardial disease</strong></td>
</tr>
<tr>
<td>Arrhythmogenic RV dysplasia</td>
</tr>
<tr>
<td>Sepsis (cytokine-induced myocardial depression)</td>
</tr>
<tr>
<td><strong>Congenital and valvular heart disease</strong></td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
</tr>
<tr>
<td>Transposition of the great arteries</td>
</tr>
<tr>
<td>Atrial septum defect</td>
</tr>
<tr>
<td>Anomalous pulmonary venous return</td>
</tr>
<tr>
<td>Tricuspid regurgitation</td>
</tr>
<tr>
<td>Pulmonary regurgitation</td>
</tr>
<tr>
<td>Mitral valve disease</td>
</tr>
<tr>
<td><strong>Pericardial disease (via negative effects on diastolic filling)</strong></td>
</tr>
<tr>
<td><strong>Arrhythmias</strong></td>
</tr>
</tbody>
</table>

CABG = coronary artery bypass grafting; CHD = congenital heart disease; PH = pulmonary hypertension; RV = right ventricular.
Factors affecting RV Function

Figure 1  Mechanisms of RV Dysfunction in Critically Ill Patients

Right ventricular (RV) dysfunction occurs directly due to cardiodepressant effects of proinflammatory cytokines, cardiac microthrombi, and ischemia and/or arrhythmias or indirectly due to left ventricular (LV) dysfunction, afterload increases from endothelial dysfunction, hypoxic pulmonary vasoconstriction, pulmonary omboli, and/or pulmonary microthrombi, as well as pre-load decreases (induced or aggravated by capillary leak syndrome). Mechanical ventilation contributes to RV dysfunction by negatively affecting pre-load and/or afterload. Endotoxin and proinflammatory cytokines negatively affect RV function on several levels. ET = endothelin; IL = interleukin; NO = nitric oxide; O2 = oxygen; PG I2 = prostacyclin; TNF = tumor necrosis factor.
Effects of Mechanical Ventilation

- Increased RV afterload due to positive pressure ventilation
- Hemodynamic failure frequently refractory in PAH patient put on MV
- In ARDS increase in mPAP while increasing tidal volume and PEEP
- Permissive hypercapnia is deleterious (increase in mPAP)
- Decreased venous return
Effects of PEEP on RV performance
Effect of high PEEP on RV
Pulmonary Hypertension:
PAH Differentiation From PH (Dana Point Definitions)

<table>
<thead>
<tr>
<th>PAH</th>
<th>PH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean PAP $\geq 25$ mm Hg</td>
<td>Mean PAP $\geq 25$ mm Hg</td>
</tr>
<tr>
<td>+</td>
<td></td>
</tr>
<tr>
<td>PCWP/LVEDP $\leq 15$ mm Hg</td>
<td></td>
</tr>
</tbody>
</table>

LVEDP, left ventricular end-diastolic pressure; PAP, pulmonary arterial pressure; PCWP, pulmonary capillary wedge pressure; PH, pulmonary hypertension.

PAH Definition

- Mean PAP $>25$ mm Hg
- PCWP $\leq 15$ mm Hg
- Increased pressure load on RV

mPAP, mean PAP; PAP, pulmonary arterial pressure; PCWP, pulmonary capillary wedge pressure; RV, right ventricle; RVSP, right ventricular systolic pressure.

PAH Background

- Rare disease (orphan designation) of the pulmonary microvasculature affecting 15 to 50 people per million inhabitants in the Western world\(^1\)
  - Affects all races
  - Affects all ages; however, most prevalent in 4th and 5th decades of life
  - Higher prevalence in females
- Global burden of PAH may be underestimated because of:\(^1,2\)
  - Underdiagnosis (eg, nondescript symptoms)
  - Misdiagnosis (eg, asthma, left-heart disease)
  - Increasing risk factors (eg, HIV infection, schistosomiasis)

HIV, human immunodeficiency virus.

### WHO Clinical Classification of PH: Dana Point 2008

<table>
<thead>
<tr>
<th>Group 1—PAH</th>
<th>Group 2—PH owing to left heart disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic PAH</td>
<td>Systolic dysfunction</td>
</tr>
<tr>
<td>Heritable</td>
<td>Diastolic dysfunction</td>
</tr>
<tr>
<td>BMPR2</td>
<td>Valvular disease</td>
</tr>
<tr>
<td>ALK-1, endoglin (with or without HHT)</td>
<td></td>
</tr>
<tr>
<td>Unknown</td>
<td></td>
</tr>
<tr>
<td>Drug- and toxin-induced</td>
<td></td>
</tr>
<tr>
<td>PAH associated with:</td>
<td></td>
</tr>
<tr>
<td>Connective tissue diseases</td>
<td></td>
</tr>
<tr>
<td>HIV infection</td>
<td></td>
</tr>
<tr>
<td>Portal hypertension</td>
<td></td>
</tr>
<tr>
<td>Congenital systemic to pulmonary shunts</td>
<td></td>
</tr>
<tr>
<td>Schistosomiasian</td>
<td></td>
</tr>
<tr>
<td>Chronic hemolytic anemia</td>
<td></td>
</tr>
<tr>
<td>Persistent pulmonary hypertension of newborn</td>
<td></td>
</tr>
<tr>
<td>Pulmonary veno-occlusive disease or pulmonary</td>
<td></td>
</tr>
<tr>
<td>capillary hemangiomatosis</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Group 3—PH owing to lung diseases or hypoxia</th>
<th>Group 4—Chronic thromboembolic PH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td></td>
</tr>
<tr>
<td>Interstitial lung disease</td>
<td></td>
</tr>
<tr>
<td>Other pulmonary diseases with mixed restrictive</td>
<td></td>
</tr>
<tr>
<td>and obstructive pattern</td>
<td></td>
</tr>
<tr>
<td>Sleep-disordered breathing</td>
<td></td>
</tr>
<tr>
<td>Alveolar hypoventilation disorder</td>
<td></td>
</tr>
<tr>
<td>Chronic exposure to high altitude</td>
<td></td>
</tr>
<tr>
<td>Developmental abnormalities</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Group 5—PH with unclear multifactorial mechanisms</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Hematologic disorders</td>
<td></td>
</tr>
<tr>
<td>Systemic disorders</td>
<td></td>
</tr>
<tr>
<td>Metabolic disorders</td>
<td></td>
</tr>
<tr>
<td>Others</td>
<td></td>
</tr>
</tbody>
</table>

ALK-1, activin receptor-like kinase 1; BMPR2, bone morphogenetic receptor type 2; HHT, hereditary hemorrhagic telangiectasia; HIV, human immunodeficiency virus; PH, pulmonary hypertension; WHO, World Health Organization. a Myeloproliferative disorders, splenectomy. b Sarcoidosis, pulmonary Langerhans cell histiocytosis. c Glycogen storage disease, Gaucher disease, thyroid disorders. d Tumoral obstruction, fibrosing mediastinitis, chronic renal failure. Simonneau et al. *J Am Coll Cardiol.* 2009;54(1 suppl S):S43-S54.
Diagnostic Studies

- Echo
- MRI
- Right Heart Cath
Key Role of Echocardiography

- Screening tool for PAH
  - In conjunction with symptoms, chest x-ray, ECG, heart sounds, etc
  - Exclude secondary causes of PH
  - Detect preclinical disease
- Predicts prognosis
- Monitors efficacy of therapy

ABG, arterial blood gas; ANA, antinuclear antibody; cath, catheterization; CHD, congenital heart disease; CPET, cardiopulmonary exercise testing; CT, computed tomographic; CTD, connective tissue disease; CXR, chest x-ray; ECG, electrocardiogram; Echo, echocardiogram; RAE, right atrial enlargement; RV, right ventricle; RVE, right ventricle enlargement; RVSP, right ventricular systolic pressure; TEE, transesophageal echocardiography; VHD, valvular heart disease.

LA, left atrium; LV, left ventricle; PA, pulmonary artery; RA, right atrium; RV, right ventricle; RVSP, right ventricular systolic pressure; TR, tricuspid regurgitation.

RV Undervalued in Echocardiography

- Relative to left heart disease, most echocardiography texts dedicate little space to evaluation of
  - Pulmonary hypertension
  - Right-sided cardiac function
  - Right-sided valvular function

- Typical echo report provides **no** objective measurements of RV or RA size or RV function

Physician may want to alert the lab if pulmonary hypertension is suspected to ensure appropriate focus on the RV and other important structures and parameters.
Representative Echocardiographs: Normal Versus PAH in Apical View

Images and video courtesy of Paul Forfia, MD, Hospital of the University of Pennsylvania Heart and Vascular Center.
Echocardiographic Characterization of PAH Disease Progression

Early stage PAH
Moderate severity PAH
Severe PAH

Images and video courtesy of Paul Forfia, MD, Hospital of the University of Pennsylvania Heart and Vascular Center.
Echocardiographic Features of (Idiopathic) PAH

- 98% of patients demonstrated RV enlargement
- 92% had RA enlargement
- 90% exhibit systolic (interventricular) septal flattening
- 76% had (qualitative) RV systolic dysfunction
- 70% of patients had “grade I” diastolic dysfunction (E<A)
- All patients demonstrated normal LV function
- <2% of patients had >mild mitral regurgitation

LV, left ventricular; PVD, pulmonary vascular disease; RA, right atrial; RV, right ventricular.

Echocardiography: TR Jet Velocity

- TR jet velocity = RV pressure – RAP
- RVSP = 4(TR jet velocity)^2 + RAP
- RAP estimated from
  - RA size
  - IVC size
  - IVC inspiratory collapse

Doppler TR Jet Velocity

Multicolored high flow jet with severe regurgitation

IVC, inferior vena cava; RA, right atrium; RAP, right atrial pressure; RV, right ventricle; RVSP, right ventricular systolic pressure; TR, tricuspid regurgitation.

Image courtesy of Mardi Gomberg-Maitland, MD, MSc, University of Chicago Medical Center.
Advantages of Echocardiography in PAH

- Readily available
- Noninvasive
  - Uses ultrasound only
  - No ionizing radiation or contrast dye
  - No contraindications due to pacemakers
- Early detection of abnormalities
- Easy to apply serially to monitor disease status

Limitations of Echocardiography in PAH

- Experienced technicians and interpreting physicians are essential
- Consistency of skilled technicians/readers
  - Applies to all imaging modalities
- Images can be limited in some patient populations
- The RV, the chamber of highest concern in PAH, is the least emphasized on “standard” echocardiography exam
- TR jet may be absent in some patients, thus precluding PASP assessment
- May overestimate or underestimate actual pulmonary arterial pressure
- Can estimate LVEDP (PCWP) or CO/CI, but may prove impractical

CI, cardiac index; CO, cardiac output; LVEDP, left ventricular end diastolic pressure; PASP, pulmonary arterial systolic pressure; PCWP, pulmonary capillary wedge pressure; RV, right ventricle; TR, tricuspid regurgitation.
Cardiac MRI
Key Roles of Right Heart Catheterization

- Confirm diagnosis
  - *Gold standard*
- Evaluate severity of PAH
- Assess congenital heart defects
- Exclude left-sided heart disease
- Assess response to vasodilator challenge
- Assess key hemodynamic parameters

Importance of Right Heart Catheterization

- Vast majority of PH cases are not in WHO group I
- **PAH** characterized by
  - $\uparrow PVR$
  - $\uparrow TPG$
  - *Normal left-sided filling pressures*
- **PVH** characterized by
  - $\uparrow PCWP$, *usually normal TPG and PVR*

LAP, left atrial pressure; LVEDP, left ventricular end diastolic pressure; PCWP, pulmonary capillary wedge pressure; PVH, pulmonary venous hypertension; PVR, pulmonary vascular resistance; TPG, transpulmonary pressure gradient; WHO, World Health Organization.
WHO Group I (PAH) Versus Non–WHO Group I: Anatomical Considerations

Group I—PAH
- Idiopathic PAH
- Familial PAH
- PAH associated with:
  - Collagen vascular disease
  - Congenital systemic to pulmonary shunts
  - Portal hypertension
  - HIV infection
  - Drugs and toxins
  - Other
- PAH associated with significant venous or capillary involvement
- Pulmonary veno-occlusive disease
- Pulmonary capillary hemangiomatosis
- Persistent pulmonary hypertension of newborn

Group II—Pulmonary venous hypertension
- Left-sided atrial or ventricular heart disease
- Left-sided valvular disease

Group III—PH associated with lung diseases and/or hypoxemia
- Chronic obstructive pulmonary disease
- Interstitial lung disease
- Sleep-disordered breathing
- Alveolar hypoventilation disorder
- Chronic exposure to high altitude

Group IV—PH due to chronic thromboembolic and/or embolic disease
- TE obstruction of proximal pulmonary arteries
- TE obstruction of distal pulmonary arteries
- Pulmonary embolism (tumor, parasites, foreign material)

VC RA RV PA PC PV LA LV Ao

Ao, aorta; HIV, human immunodeficiency virus; LA, left atrium; LV, left ventricle; PA, pulmonary artery; PC, pulmonary capillary bed; PV, pulmonary vein; RA, right atrium; RV, right ventricle; TE, thromboembolic; VC, vena cava; WHO, World Health Organization.

a Glycogen storage disease, Gaucher disease, hereditary hemorrhagic telangiectasia, hemoglobinopathies, myeloproliferative disorders, splenectomy.

Key PAH Assessments During RHC

- PAP
- RAP
- RVSP
- PCWP
- Calculations: TPG, PVR, CO, CI


CI, cardiac index; CO, cardiac output; mPAP, mean PAP; PAP, pulmonary arterial pressure; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; RAP, right atrial pressure; RHC, right heart catheterization; RVSP, right ventricular systolic pressure; TPG, transpulmonary gradient.

*RHC to measure oxygen saturations, generate Fick CO, and confirm wedge position.
**RHC: Important Findings**

- Elevation in PCWP
- Elevation in RAP
- Decrease in CO/Ci
- Elevation of PCWP with vasodilator
  - *Pulmonary veno-occlusive disease (PVOD)*
  - *LV diastolic dysfunction*

**PVH: limits treatment for PAH**

**Aggressive early management suggested**

**CI, cardiac index; CO, cardiac output; LV, left ventricular; PCWP, pulmonary capillary wedge pressure; PVH, pulmonary venous hypertension; RAP, right atrial pressure.**
Treatment of RV Failure

- General Supportive ICU Care
  - *Infection control*
  - *DVT Prophylaxis*
  - *Daily interruptions of sedation*
  - *Na and fluid restriction*

- Optimize volume status
  - *Hemodynamic guidance helpful*
  - *Avoid boluses if able*

- Attenuate Hypoxic Pulmonary vasoconstriction

- Mechanical Ventilation Strategies

*J Am Coll Cardiol 2010;56:1435–46*
Strategies to Improve RV Function

1. Interventions that improve RV inotropy and/or lusitropy
2. Interventions that decrease LV systolic and/or diastolic dysfunction
3. Interventions that optimize volume status
4. Interventions that decrease effects of endotoxin/cytokines
5. Interventions that attenuate endothelial dysfunction
6. Interventions that decrease capillary leak
7. Interventions that decrease hypoxic pulmonary vasoconstriction
8. Interventions that target RV ischemia and arrhythmias
9. Interventions that minimize effects of mechanical ventilation
10. Interventions that target pulmonary/myocardial microthrombi
11. Interventions that reduce pulmonary thromboemboli
Acute right heart failure

**Treat underlying disease**

- **Pulmonary hypertension:** ± diuresis, inhaled NO, intravenous/inhaled prostacyclins (avoid subcutaneous route in severe RHF), PDE5 inhibitors, ET-1 receptor antagonists (1, 3, 5, 7, 10)
  - **Pulmonary embolus:** anticoagulation, thrombolysis, thrombectomy (surgical or catheter-directed) (5, 7, 11)
  - **CTEPh:** thrombendarterectomy (5, 11)
  - **RV infarction:** PCI, thrombolysis (8)
  - **LV dysfunction:** afterload reduction, diuresis, inotropes, nesiritide, IABP, LVAD (2)

- **CHD/VHD:** surgical or percutaneous correction (2, 3, 7, 8)

- **Sepsis/acute lung injury:** volume resuscitation, broad spectrum antibiotics, activated protein C, lung protective ventilation strategy (1, 2, 3, 4, 5, 6, 9, 10)

- **Post cardiothoracic surgery:** inhaled NO, inhaled/intravenous prostacyclins, milrinone, PDE5 inhibitors (1, 2, 3, 4, 7, 8)

**Volume optimization**

- **Volume overload**
  - Salt restriction, daily weights (3)
  - Diuretics (3)
    - Goal: net loss of 500 – 1000 ml/day
    - Consider continuous infusion of loop diuretics or combination of diuretics if nonresponsive to moderate doses of intermittently given diuretics
  - Continuous or intermittent RRT (3)

- **Hypovolemia**

**Hemodynamic support**

- **RV infarct**
- **Acute PE**
- **Hypotension/Shock**
  - Volume challenge (1, 3)
    - 500 – 1000 ml; no further volume challenge if no effect
  - Vasopressors/inotropes/inodilators:
    - Dobutamine, milrinone, levosimendan, norepinephrine, low-dose vasopressin? (1, 2, 8)
    - Avoid: dopamine, phenylephrine
  - Consider combination therapy with inhaled NO or inhaled/intravenous prostacyclins

**Rhythm stabilization:** cardioversion, antiarrhythmics, pacemaker, resynchronization (1, 2, 8)

**Rescue therapies:**
- Atrial septostomy, RVAD, ECMO, transplantation
Summary

- Right Ventricular Failure significant in ICU Setting
- Diagnosis of Pulmonary Hypertension is very common in obese patient
  - OSA is predominant underlying condition
  - Compliance
  - Echo is good screening tool
  - RHC is gold standard
- PAH diagnosis merits referral to specialized treatment center