ACUTE RIGHT VENTRICULAR FAILURE

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Definition

« 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 »
Why Focus on Acute Heart Failure?

- There are currently no national guidelines for acute heart failure management
- Hospital readmissions
  - 20% at 30 days
  - 50% at 6 months
- Mortality
  - 11.6% at 30 days
  - 33.1% at 12 months
- Clinical trials in heart failure:
  - Focus on...
  - Omit...
  - Stable outpatients
  - Criteria for admission to hospital
  - Systolic dysfunction
  - Treatments for acute heart failure
  - Enroll relatively younger patients
  - Exclude many pts
  - Diastolic dysfunction
  - Pts with co-morbidities

Anatomy

- The RV is triangular in shape
- Divided into three regions:
  - Inlet
  - Apex
  - Infundibulum or Conus
- Both Ventricles are composed of a 3D network of muscle fibers

Circulation 2008;117:1436-1448
Effect of Ventricular Contraction

- **Right ventricular Contraction** is sequential, starting with the inlet and ending at the infundibulum.
  - Inward movement of the free wall (bellows effect)
  - Contraction of the longitudinal fibers, which shortens the long axis.
  - Traction of the free wall caused by contraction of the LV. (wringing effect)
- Due to the compliance of the infundibulum the peak pressure is reduced and prolonged.
- This leads to near complete emptying of the RV>>reduced RVEDP>>Optimal Venous return.

Ventricular interdependence

- During systole, LV protrudes in RV
- Surrounding pericardium with limited distensibility
- Compliance of one ventricle can modify the other = Diastolic ventricular interaction
Oxygen Delivery and Blood Supply to the RV

• Right Ventricular Perfusion
  – In a right dominant system;
    • Lateral Wall- Marginal branches of RCA
    • Posterior and Inferoseptal- PDA
    • Anterior and Anteroseptal- Branches of LAD
• RV is relatively resistant to ischemia compared to the LV.
  – Lower oxygen consumption
  – More extensive collateral system
  – The ability to increase its O2 extraction during increased demand.
• Normally proximal RCA flow occurs in both diastole and systole. Unlike LCA flow to the LV. This is secondary to the transmural pressure generated during systole.

Physiology of the normal pulmonary circulation

• Low pressure system: Prv (syst) = 25 mmHg / Plv (syst) = 120 mmHg
• The pressure in the pulmonary system depends on cardiac output, resistance and compliance
• The pulmonary vascular resistance has a particular dependency on alveolar oxygen tension, whereby alveolar hypoxia leads to pulmonary arterial vasoconstriction
• High compliance of the pulmonary vessels with large diameter and thin wall
Etiology, Diagnosis and Pathophysiology

- Cardiac versus pulmonary causes
- RV previously healthy (Acute)
- Chronically impaired RV function (Acute on Chronic): RV hypertrophy / dilatation

RIGHT HEART FAILURE:
Acute right heart failure less common than left heart failure.
Most commonly seen as acute on chronic heart failure, 2º to any insult to cardiac function.
Specific causes of right heart failure:
- Right ventricular involvement of inferior MI
- Acute pulmonary embolus
Main circumstances in ICU

- Severe pulmonary embolism
- ARDS
- Sepsis induced RV dysfunction
- Exacerbation of medical conditions leading to chronic pulmonary hypertension
- Right ventricle infarction
- Pericardial diseases
- RV failure after cardiac surgery
- After cardiac transplant

Diagnostic Tools

- Biomarkers and ANP.
- ECG and X-ray.
- Echocardiography.
- CT and MRI.
- CVP and arterial line.
- PAC.
- Cardiac catheterisation.
CVP and Arterial Line

- Calculation of Fick CO:
  \[ CO(L/min) = \frac{VO_2}{13.4 \times Hgb \times (SaO_2 - SvO_2)} \]
- Correlates well with thermodilution at low CO <5L/min, larger variation at higher CO's
- In setting acute RHF the response to a small initial fluid bolus of 250cc can be informative.
  - A rise in CVP without a concamitant rise in CO may indicate when further volume would be detrimental.

Differentiating features between RHF with or without cor-pulmonale/pulmonary arterial hypertension

<table>
<thead>
<tr>
<th>RHF without pulmonary hypertension</th>
<th>Cor pulmonale/pulmonary hypertension present</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Chest x-ray:</strong> Enlargement of pulmonary arteries (uncommon), oligemic peripheral lung fields (rare)</td>
<td><strong>Chest x-ray:</strong> Right-sided cardiac enlargement, enlargement of pulmonary arteries, oligemic peripheral lung fields</td>
</tr>
<tr>
<td><strong>Echocardiography:</strong> No evidence of increased pulmonary pressure. Septal flattening during diastole but not systole</td>
<td><strong>Echocardiography:</strong> Evidence of increased pulmonary pressure. Septal flattening during systole</td>
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<td><strong>Physical examination:</strong> Evidence of underlying pulmonary pathology if cor pulmonale present (but not in primary PAH)</td>
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Auto-Aggravation: The Vicious Cycle

Diagnosis: Nonspecific Findings

Table 1—Symptoms of RV Failure

<table>
<thead>
<tr>
<th>Acute</th>
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<tbody>
<tr>
<td>Dizziness</td>
</tr>
<tr>
<td>Lightheadedness</td>
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<tr>
<td>Syncope</td>
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<tr>
<td>Chest discomfort</td>
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<table>
<thead>
<tr>
<th>Chronic</th>
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</thead>
<tbody>
<tr>
<td>Acute symptoms</td>
</tr>
<tr>
<td>Right-upper-quadrant abdominal pain</td>
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<tr>
<td>Lower-extremity swelling</td>
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Table 2—Signs of RV Failure

<table>
<thead>
<tr>
<th>Acute</th>
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<tbody>
<tr>
<td>Hypotension</td>
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<tr>
<td>Tachycardia</td>
</tr>
<tr>
<td>Tachypnea</td>
</tr>
<tr>
<td>Cyanosis</td>
</tr>
<tr>
<td>Elevated jugular venous pressure</td>
</tr>
<tr>
<td>Parasternal heave</td>
</tr>
<tr>
<td>RV third-heart sound</td>
</tr>
<tr>
<td>Tricuspid regurgitation</td>
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</tbody>
</table>

<table>
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<tr>
<th>Chronic</th>
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</thead>
<tbody>
<tr>
<td>Acute signs</td>
</tr>
<tr>
<td>Hepatic enlargement and ascites</td>
</tr>
<tr>
<td>Lower-extremity edema</td>
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</tbody>
</table>

Chest. 2005;128:1836-1852
Management

- Treat the underlying cause if possible
- Fluid Management
  - Volume Loading or Diuresis?
  - Maximize Myocardial O$_2$ Supply/Demand
    - Supplemental Oxygen
    - Maintaining Systemic Pressures
      - Norepinephrine
      - Phenylephrine
    - Avoiding Tachycardia
    - Blood transfusion?
- Increasing Contractility
  - Dobutamine
  - Milrinone
  - Calcium Sensitizers
- RV Afterload Reduction
  - Milrinone
  - Nitro Oxide
  - IV Prostacyclin
  - Avoid Mechanical Ventilation if possible
Treating the cause of the RV failure, if possible

- Treatment of Pulmonary Arterial Hypertension
- Pericardiotomy/drainage
- Thrombolysis/embolectomy
- Thrombolysis/angioplasty
- Thromboendarteriectomy
- Atrial septostomy
- Transplantation
- Arrhythmias
- Infections

General Measures

- Adequate oxygenation PO2>92% to avoid PHV.
- Careful volume management:
  - Optimize the preload → low fluid challenge → high diuretics.
  
  Restoration of sinus rhythm and treatment of other arrhythmias.
Strategies that improve RVEF, increase the RV perfusion pressure and decrease the afterload

- Dobutamine
- Nor-epinephrine
- Levosimendan
- Milrinone

Dobutamine

- β1 adrenergic stimulation
- ↑ CI ↓ PVR at 5 μg/kg/mn
- At higher dose ↑ HR without subsequent ↓ in PVR
- Experimental models Dobutamine > Norepinephrine to improve right-ventricular – pulmonary artery coupling
- Improves CI, PVR and PaO2/FiO2 in combination with Inhaled nitric oxide
Prospective, controlled, randomized, animal study

- 22 dogs underwent transient PA constriction (90mn)
- Dobutamine 5 and 10 µg/kg/mn, norepinephrine 0.1 to 0.5 µg/kg/mn
- A transient increase in PA pressure persistently worsens PA hemodynamics, RV contractility, RV-PA coupling, and cardiac output.
- Dobutamine restores RV-PA coupling and cardiac output better than norepinephrine because of its more pronounced inotropic effect.

Norepinephrine

- $\alpha_1$ and $\beta_1$ adrenergic stimulation
- Increases mPAP and PVR
- But marked improvement in CO
- Useful in combination with Dobutamine for hypotensive patients
- Causes less tachycardia than other inotropes
- Second choice after Dobutamine in normotensive patients
Levosimendan

- Calcium sensitizer: increases the sensitivity of troponin C for Ca2+ within cardiac myocyte
- Dilatation of pulmonary vasculature by activation of adenosin tri-phosphate potassium channel
- Animal studies and pilot studies support its efficacy in right ventricle failure associated with pulmonary hypertension

Milrinone

- PDE-3 inhibitor.
- Vasodilator and inotropic.
- Increase RVEF and decrease PVR.
- Hypotensive.
- Can be combined with iNO.
- Inhaled form is better to avoid hypotension
Strategies that decrease RV afterload (pulmonary vasodilators)

- Inhaled NO.
- Prostacyclins.
- Endothelin receptor antagonist.
- PDE-5 inhibitors (seldinafil).

Inhaled nitric oxyde

- Dilate pulmonary vessels in ventilated units of the lung
- Reverses hypoxic pulmonary vasoconstriction
- In acutely decompensated RV improves PVR, increase CO improve PaO2/FiO2 (Benker KA et Al. Am J Crit Care. 1997 Mar;6(2):127-31)
- Beware of methemoglobinemia (high concentration, prolonged use)
Prostanoids

- Intravenous Epoprostenol
- Effect on survival in stable patients with PAH
- Reduces mPAP and improves CO
- Systemic side effects
- Worsening PaO2/FiO2
- Systemic effects (hypotension)

Sidenafil

- Phosphodiesterase-5 inhibitor
- Approved for treatment of PAH (stable patients)
- May be useful for weaning from inhaled nitric oxide
- Effect start 15mn after administration, peak effects within 30-60mn
- Systemic hypotension
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)

Surgical and Interventional Therapies

- Atrial septostomy.
- Correction of congenital or valvular lesions
- LVADs.
- BVADs.
- RVADs.
- Pulmonary thrombo-endarterectomy.
- Heart-lung transplantation.
- Lung transplantation.