

# ***Acute Right Ventricular Failure***

***Ahmed El Hawary MD  
Suez Canal University***

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## **Definition**

**RVF results from any structural or functional process decreasing the ability of the RV to pump blood into the pulmonary circulation**

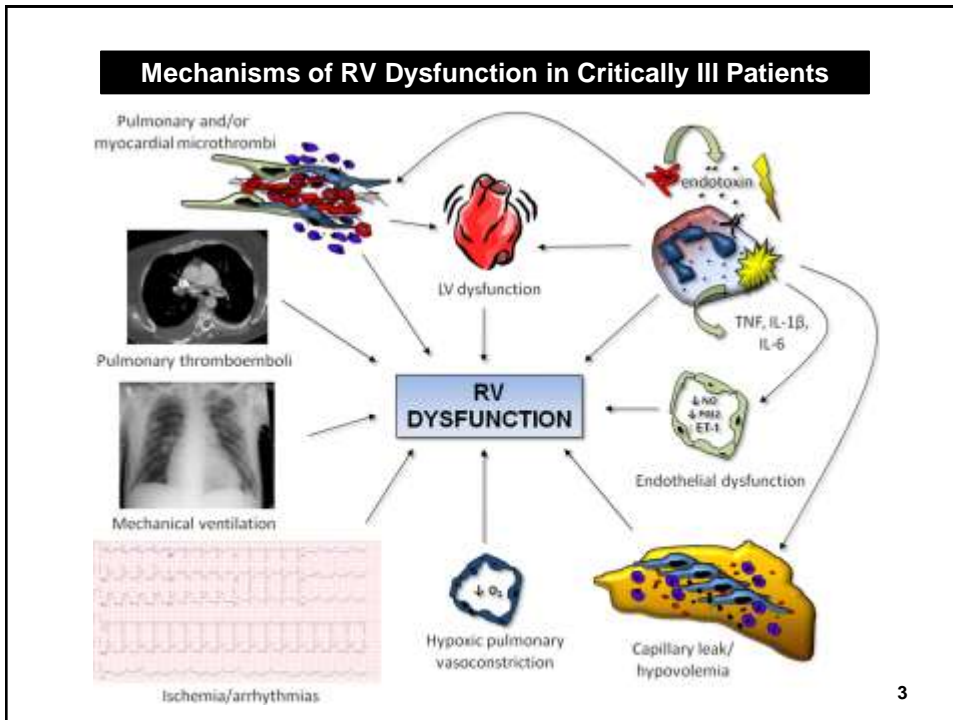
### **Causes**

1. Alterations in preload and diastolic filling.
2. Decreases in inotropy.
3. Increases in afterload.

### **The most common etiologies of RVF in the ICU are**

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| <ol style="list-style-type: none"> <li>1. LV failure.</li> <li>2. RV ischemia.</li> <li>3. Acute pulmonary embolism.</li> <li>4. Pulmonary hypertension.</li> <li>5. Sepsis.</li> </ol> | <ol style="list-style-type: none"> <li>6. Acute lung injury.</li> <li>7. Cardiac tamponade.</li> <li>8. post-cardiothoracic surgery.</li> <li>9. Arrhythmias.</li> <li>10. Congenital, and/or valvular heart disease.</li> </ol> |
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### Diagnosis of RVF in the ICU

- No specific biomarker for RVF, but aid in prognosis.
- ECG, although specific, lacks sensitivity.
- Chest X-ray or CT showed signs of advanced RV dysfunction.

### Pulmonary artery catheters (PACs)

**PACs directly measure**

1. PAP.
2. Capillary wedge pressure.
3. Right atrial pressure (RAP).
4. Cardiac output (CO).
5. Oxygen saturation.
6. Pulmonary vascular resistance (PVR).
7. RV stroke work index.
8. Evaluate response to pharmacologic therapies and drug titration.

## TTE

1. Estimates of RV size and function.
2. Elucidate precipitating factors for RV failure.

RV systolic function can be assessed by tricuspid annular plane systolic excursion (longitudinal systolic displacement of the RV base toward the RV apex).

This is simple, highly reproducible, and correlate well with RV EF (normal value 2.4–2.7 cm & value below 1.8 cm has poor prognosis)

## Cardiac magnetic resonance imaging

is the most sensitive method to assess RV function; however, due to logistical issues, it is rarely used for critically ill patients.

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## Treatment of Acute RVF

Major components include:

1. General supportive ICU care.
2. Volume optimization.
3. RV inotropes (vasopressor and/or inotropic therapy).
4. RV afterload reduction (selective pulmonary vasodilators).
5. Surgical and/or mechanical interventions.
6. If possible, measures against underlying etiology

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## General supportive ICU care

1. Infection prevention measures.
2. Thromboembolism and peptic ulcer prophylaxis.
3. Nutritional support.
4. Glucose control.
5. In stable mechanically ventilated patients, daily interruptions of sedation with spontaneous breathing trials.
6. Optimize hemoglobin level.
7. Sodium restriction (in volume overload states).
8. Daily monitoring of body weight and volume status.

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## Treatments that attenuate HPV, optimize volume status, and target arrhythmias.

Adequate oxygenation to avoid afterload increases due to HPV (aim for oxygen saturations of 92%).

**If pre-load is too low**

- RVEF will not be adequate.
- Careful administration of fluid.

**For volume overload**

1. Diuretics.
2. Venovenous ultrafiltration for decompensated LV.

**Arrhythmias**

Restoration of sinus rhythm and/or atrioventricular synchrony.

- Digoxin marginally improves CO in severe PH in the short term.
- BBs and ACEIs improve RV hemodynamics in biventricular failure.

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### Strategies that avoid negative effects of mechanical ventilation on RV pre-load and afterload.

#### **V<sub>T</sub> & PEEP**

1. The lowest V<sub>T</sub>, and PEEP should be used.
2. High V<sub>T</sub> and PEEP increase PAP and RAP, worsen TR, and increase RV afterload.
3. Lower V<sub>T</sub> decrease endothelial dysfunction
4. Excessive hypercapnia should be avoided.

#### **Hyperventilation**

1. Attenuates acidosis-induced vasoconstriction and decreases PAP.
2. Can be used to lower PAP acutely, but should not be performed at the expense of a high V<sub>T</sub>.

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### Strategies that improve RV EF

Inotropes improve cardiac contractility and CO.  
Vasopressors increase RV perfusion pressure.

#### **Dobutamine**

- Stimulate  $\beta$ 1 increases myocardial contractility.
- Stimulate  $\beta$ 2 induces vasodilation and decreases afterload.
- In acute PH, low-dose (2 to 5  $\mu$ g/kg/min) increases CO and decreases PVR.
- Combination of dobutamine and inhaled nitric oxide (iNO) improved CO, decreased PVR.

#### **Norepinephrine**

- Increases inotropy through  $\beta$ 1 agonism.
- Stimulate  $\alpha$ 1 increases RV perfusion pressure and CO.
- May be beneficial in hypotensive and tachycardic patients not tolerating dobutamine

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### Strategies that improve RV EF

#### Milrinone

- A selective PDE-3 inhibitor with inotropic and vasodilatory properties.
- Decrease PVR and increase RVEF.
- Can be combined with iNO to augment pulmonary vasodilation.
- Inhaled milrinone minimizes hypotension.

#### Levosimendan

- Has inotropic, vasodilator and anti-ischemic properties.
- Increases CO, decreases PVR, and improves perfusion.
- Protect against endothelial dysfunction.
- Use is limited by hypotension and arrhythmias.

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### Strategies that decrease RV afterload

#### iNO

- Pulmonary vasodilator.
- Decrease HPV, PAP and PVR, and improve oxygenation.
- Decreases inflammatory cytokine production.
- Use is limited by potential methemoglobinemia.
- Of benefit when combined with dobutamine or milrinone.

#### Prostacyclins epoprostenol

- Pulmonary and systemic vasodilator and platelet inhibitor.
- The short half-life (3 to 6 min) and potent effects make epoprostenol the preferred prostacyclin in the ICU.
- It decreases PAP and PVR and increases CO.
- Side effects; hypotension, GIT symptoms, headaches.
- Nebulized or inhaled prostacyclins avoid systemic side effects.

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## Strategies that decrease RV afterload

### Endothelin antagonists

- Increasing CO and decreasing PAP in PH.
- Use in is limited by long half-lives (5 h for bosentan)
- Hepatotoxic

### PDE-5 inhibitors

- Block degradation of cGMP.
- Decrease PAP and increase CO in acute and chronic PH.
- Beneficial for HPV.
- Effects of oral sildenafil occur after 15 to 30 min, with peak effects after 30 to 60 min.
- An IV form of sildenafil with shorter half-life is also available.

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## Surgical and interventional therapies

Indicated for patients with potentially reversible RVF unresponsive to or intolerant of medical therapy or for those with disease progression despite maximal medical therapy.

1. Surgery for valvular or congenital heart disease.
2. Thrombendarterectomy for chronic thromboembolic PH.
3. Surgical embolectomy for acute massive pulmonary embolism.
4. BAS (right-to-left-shunt) to unload the RV.
5. Mechanical circulatory support LVAD, Bi ventricular AD, RVAD.
6. Extracorporeal membrane oxygenation.
7. Heart, lung, or combined heart-lung transplantation.

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## Future Directions

Therapies specifically targeting the diseased RV.

1. Metabolic modulators aimed at reversing mitochondrial dysfunction.
2. Stem cell therapy in ischemic and PAH-related RVF.
3. Tyrosine kinase inhibitors in severe PAH with RVF.

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*Thanks For Attention*

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