5th iHEF CARD

The 5th Indonesian Symposium on Heart Failure and Cardiometabolic Disease

Getting Right the RV Failure

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June, 12-14 2025

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Outline

- Introduction
- Pathophysiology of RV Failure
- Diagnostic approach
- Management of RV Failure
- Conclusion

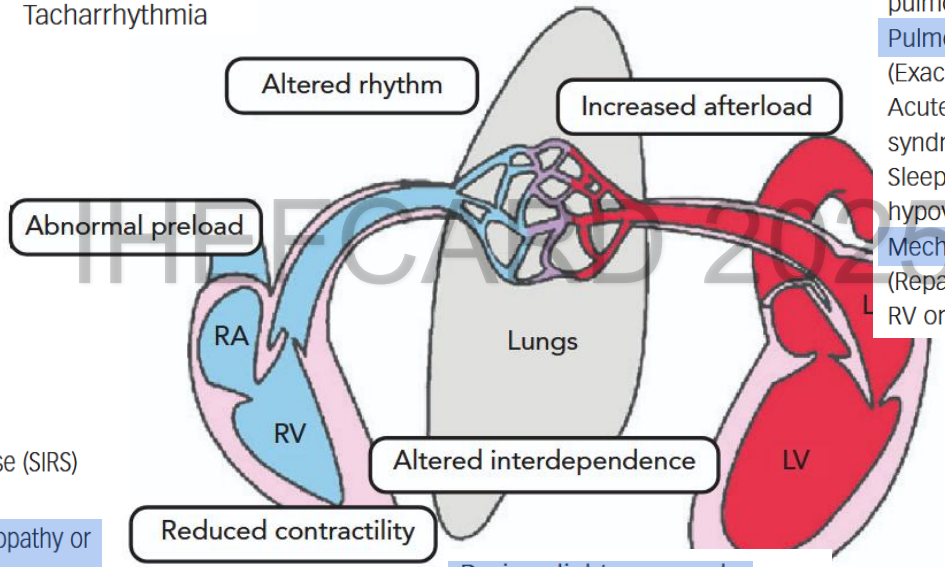
Introduction

- **RV Dysfunction (RVD):** evidence of **abnormal RV structure or function**, associated with poor clinical outcomes independently of the underlying mechanism of disease
- **RV failure (RVF)**: as a **clinical syndrome** with signs and symptoms of HF resulting from RVD
- Diagnosis is **subtle** than overt LV dysfunction, often delayed, which worsens the prognosis
- Acute RVF is observed in 3%–9% of acute heart failure admissions, and the inhospital mortality ranges from **5% to 17%** . In the special case of RVF after LVAD implantation, the prevalence ranges from 9% to 40%

Mechanism of RVD

Bradyarrhythmia
Tacharrhythmia

Hypo- or hypervolaemia
LV forward failure
Pericardial tamponade
Mechanical ventilation
Chronic left-to-right shunt



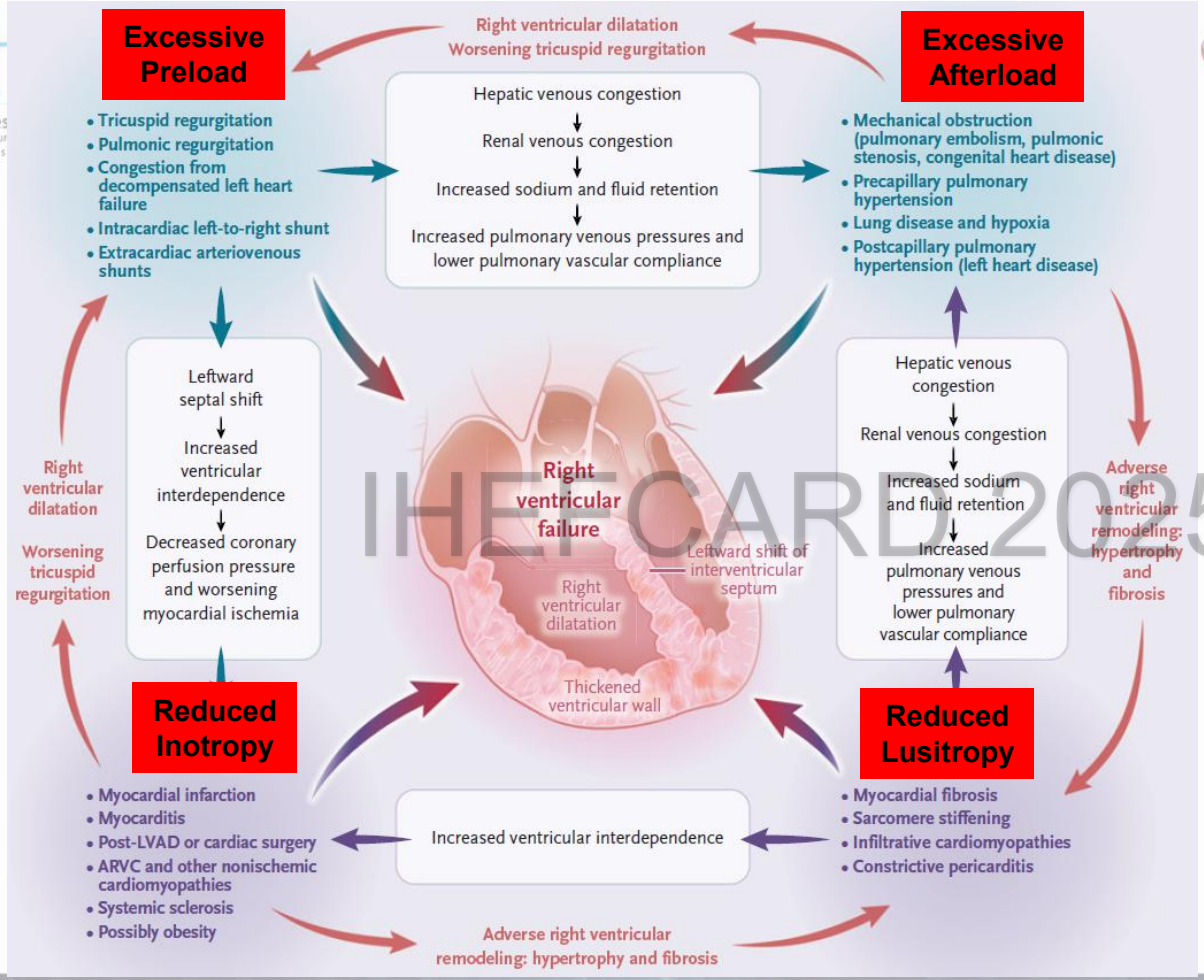
LV backward failure (pulmonary hypertension associated with left-sided heart disease)
Pulmonary embolism, chronic thromboembolic pulmonary hypertension
Pulmonary artery hypertension
(Exacerbated) chronic pulmonary disease
Acute lung injury/acute respiratory distress syndrome
Sleep-related breathing disorders, obesity-hypoventilation syndrome
Mechanical ventilation
(Repaired) congenital heart disease with systemic RV or RV outflow obstruction

RV ischaemia/RV infarction
RV injury, systemic inflammatory response (SIRS)
Myocarditis
Cardiomyopathies (e.g. dilated cardiomyopathy or hypertrophic cardiomyopathy)
Arrhythmogenic RV cardiomyopathy, Uhl's anomaly

Pericardial tamponade
Pericardial disease
Septal shift

Arrigo, M., *et al.*, Cardiac Failure Review 2019;5(3):140–6

Mechanical Mechanism of RVF



Houston, B.A., *et al.*, N Engl J Med 2023;388:1111-25.

Causes of Right Ventricular Failure

Acute RVF
Volume overload
Acute left-sided heart failure
LVAD implantation
Pressure overload
Acute pulmonary embolism
Hematological disorders (e.g., acute chest syndrome in sickle cell disease)
Decreased contractility
Acute myocardial ischemia
Fulminant myocarditis
Pericardial disease (tamponade)
Sepsis (can cause increased venous return and volume overload)
Post-cardiotomy shock
Reduced pericardial compliance

Chronic RHF
Exacerbation of chronic lung disease and/or hypoxia
Chronic pulmonary hypertension (groups 1–5)
Pericardial disease (constrictive pericarditis)
Arrhythmias (supraventricular or ventricular tachycardia)
Congenital heart disease (e.g., atrial or ventricular septal defect, Ebstein's anomaly)
Valvulopathies (e.g., tricuspid valve regurgitation, pulmonary valve stenosis)
Cardiomyopathies (e.g., arrhythmogenic right ventricular dysplasia, familial, idiopathic)
Myocarditis or other inflammatory diseases

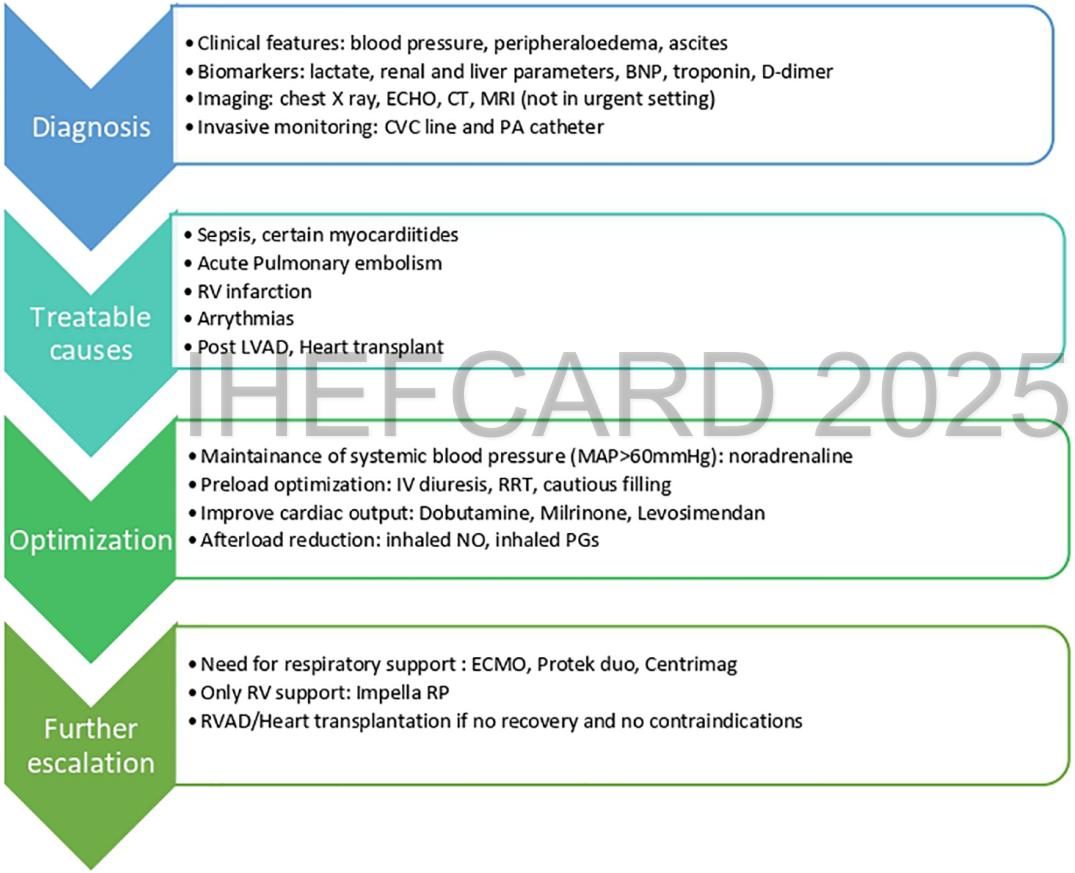
Correlation between pathophysiological mechanism and diagnostic tools

Ecocardiography	MRI	Haemodynamic markers
• RV dilatation • Hypertrophy • Systolic/diastolic dysfunction	• Fibrosis • Hypertrophy • Interventricular septum flatenning • Wall thinning • Angiogenesis	• Pressure overload • Volume overload • RV-PA coupling

Diagnostic of RVF

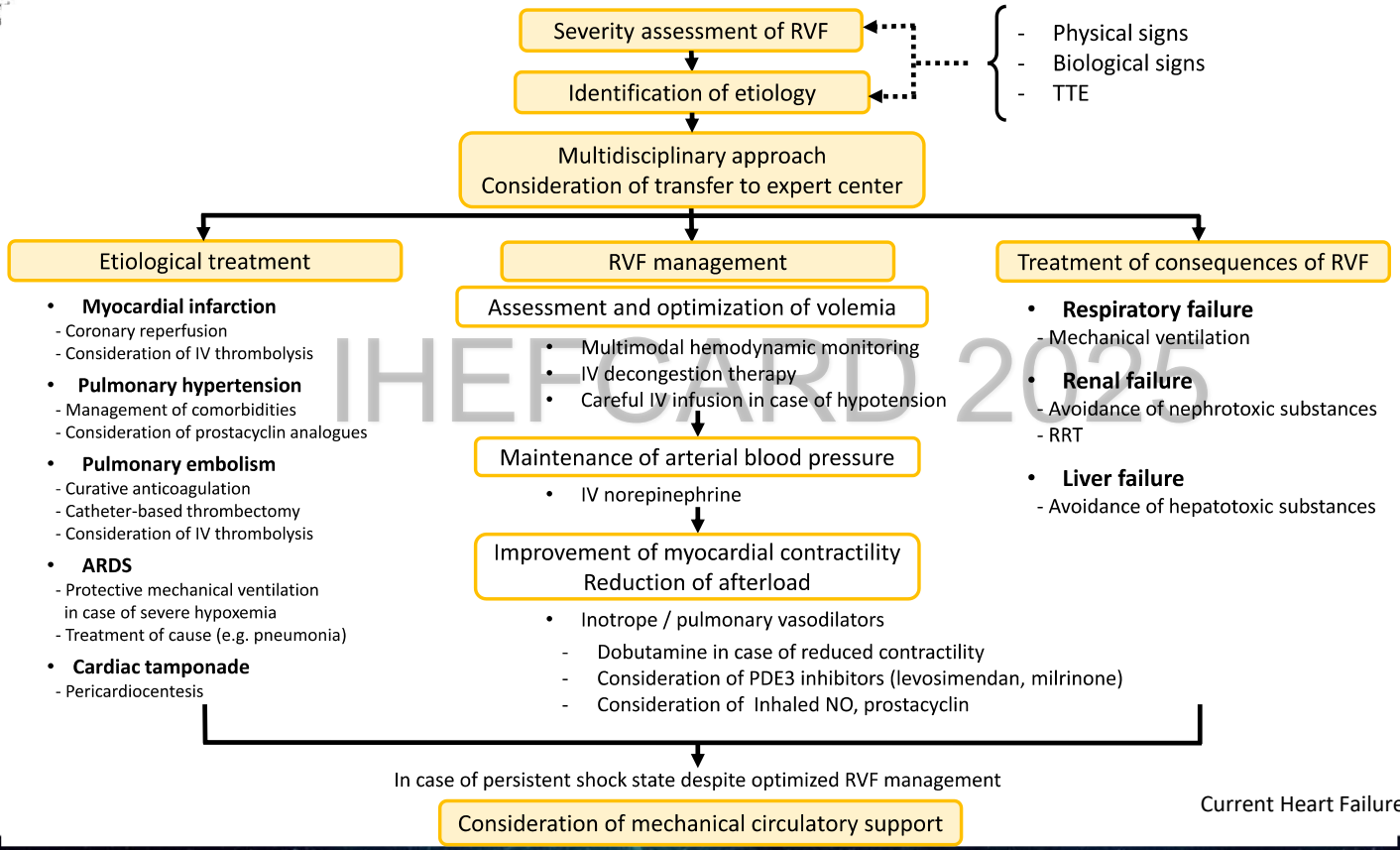
Clinical	Biomarkers	Echocardiography	Hemodynamic Parameter	MRI
• Raised JVP • Ascites • Peripheral edema • Liver congestion • Low cardiac output state • Hypoxemia	• BNP • **Cardiac troponin** • Lactate • **D-Dimer** • Liver biochemistry • Renal Function	• TAPSE <17 mm • RV dilatation • RVEDD / VEDD >1 • IVS flattening, shift: LV D shaped • TR Vel > 2.8 m/s • IVC diameter > 21 mm, collapsibility < 50% • FAC <35% • 3D RVEF < 45% • Pericardial fluid > 5 mm • RV Wall thickness > 5mm	• RAP or CVP > 15 mmHg • PAPI:< 1 in acute MI; < 1.85 in post LVAD • RAP/PCWP: > 0.86 in acute MI; > 0.63 in post LVAD • RV stroke work index < 0.25 – 0.30 mmHg.L/m^2 • PA compliance < 2.5 mL/mmHg • PVR > 3.6 WU in post LVAD	• RV Volume • RV mass • RV EF • RV fibrosis • RV volume changes and intermittent interventricular septal flattening

The management of RVF



Monteagudo-Vela et al.
Cardiovasc. Med. 10:998382.
doi: 10.3389/fcvm.2023.998382

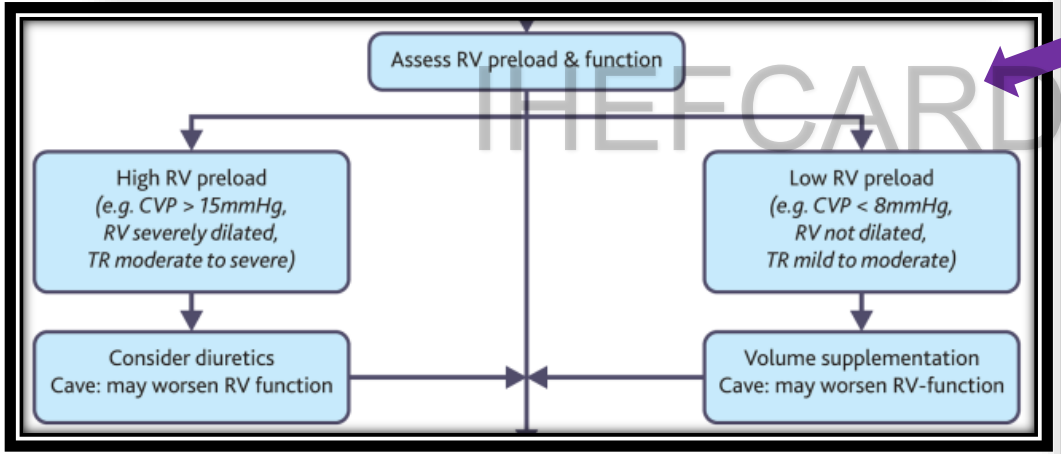
Management of Acute RVF

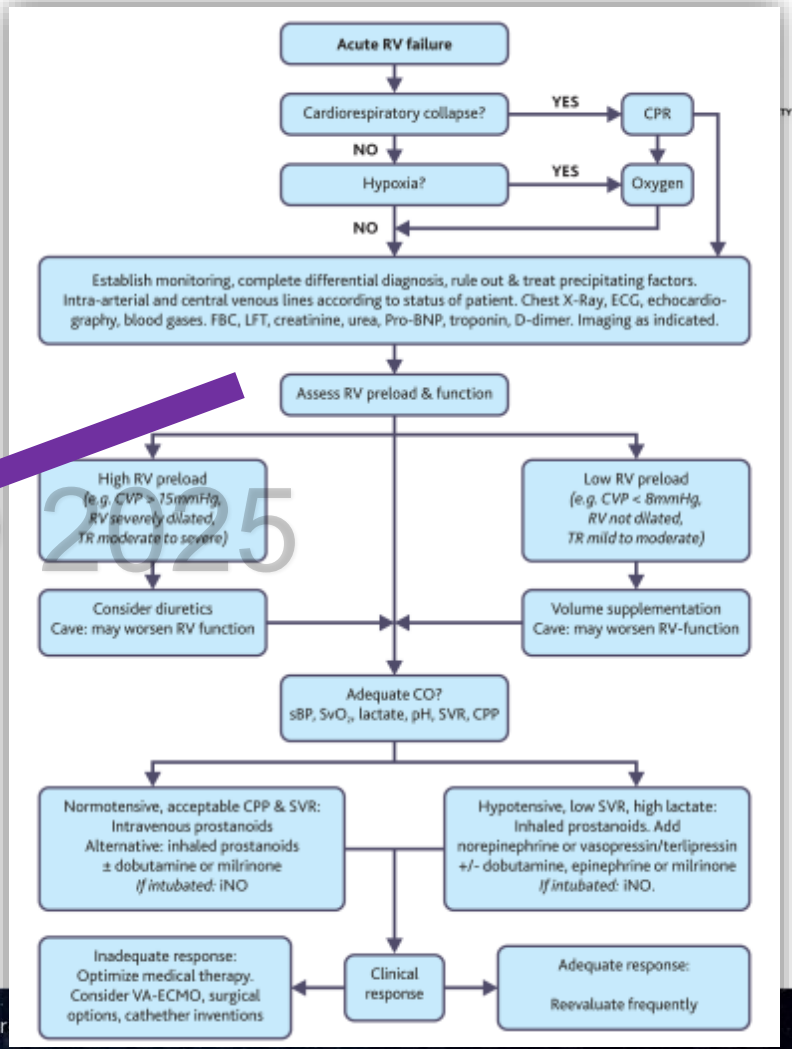


Current Heart Failure Reports (2023) 20:218–229

Volume optimization

Optimize fluid status:
- IV diuretics if volume overload
- RRT if situation insufficiently managed with diuretics
- Cautious fluid filling if low CVP; avoid overfilling





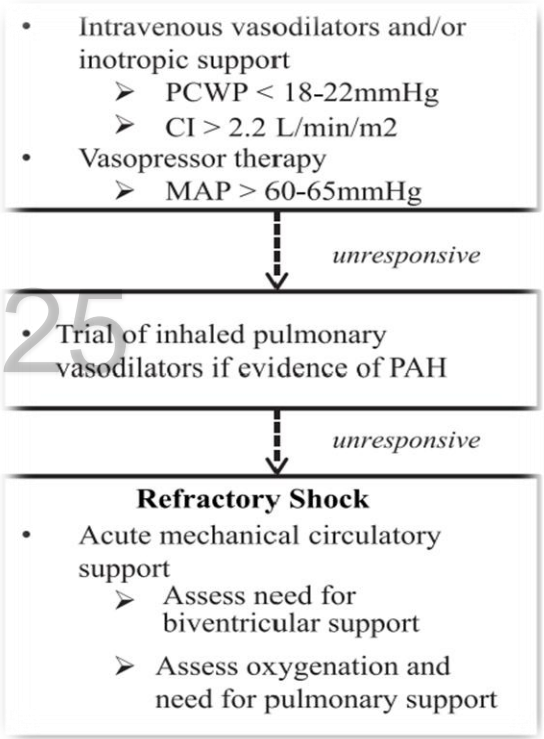
[1] Wilcox SR, Kabrhel C, Channick RN, 2015. Pulmonary Hypertension and Right Ventricular Failure in Emergency Medicine, *American College of Emergency Physicians*

[2] Kaestner M, et al. Heart 2016;102:ii57–ii66. doi:10.1136/heartjnl-2015-307774

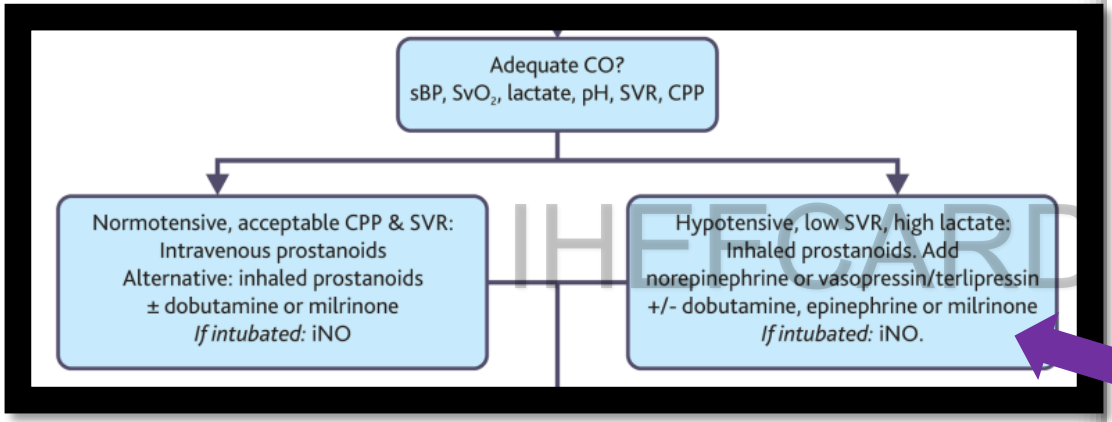
Maintenance perfusion (MAP & CI)

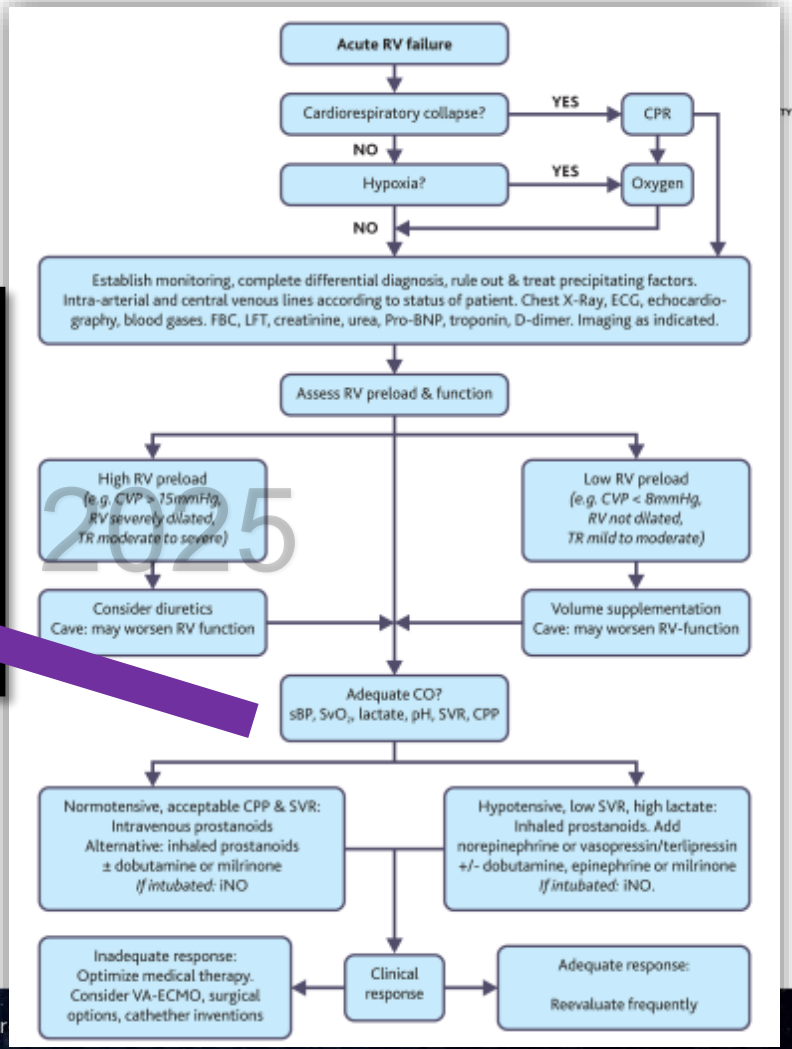
GOAL: **reducing RV afterload, enhancing forward flow, and augmenting RV perfusion**

- **Afterload reduction** → correct hypoxemia and acidosis, NTG or sodium nitroprusside, inhaled and parenteral epoprostenol and nitric oxide
- **Augment contractility** → inotropes (milrinone or dobutamine)
- **Maintain perfusion** → dopamine, norepinephrine, and epinephrine



Maintenance perfusion





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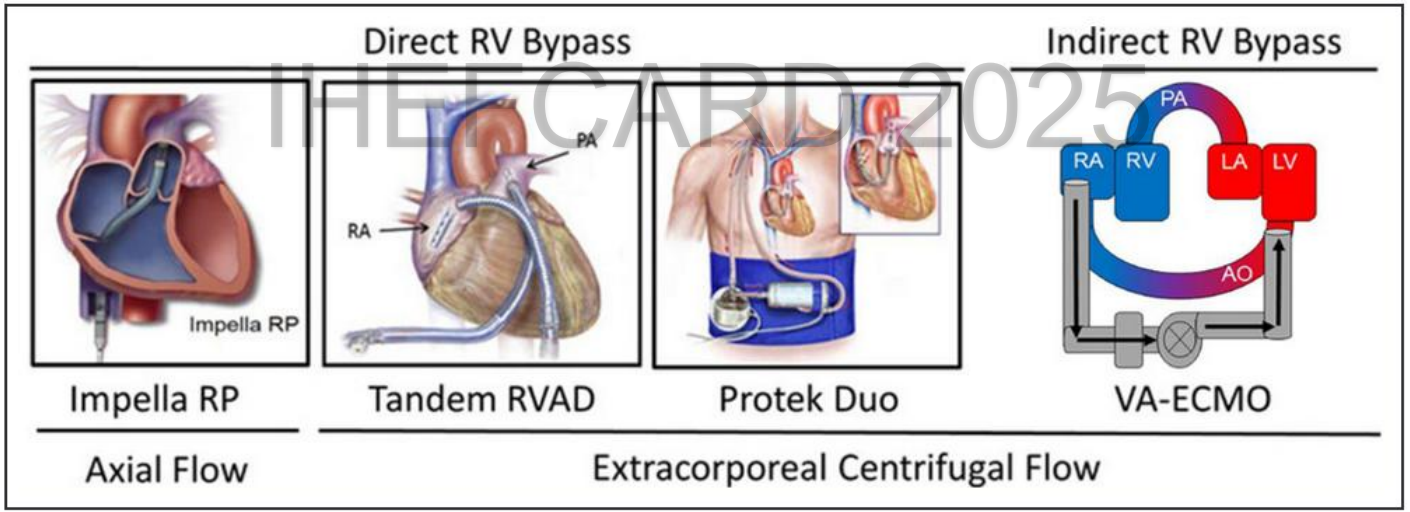
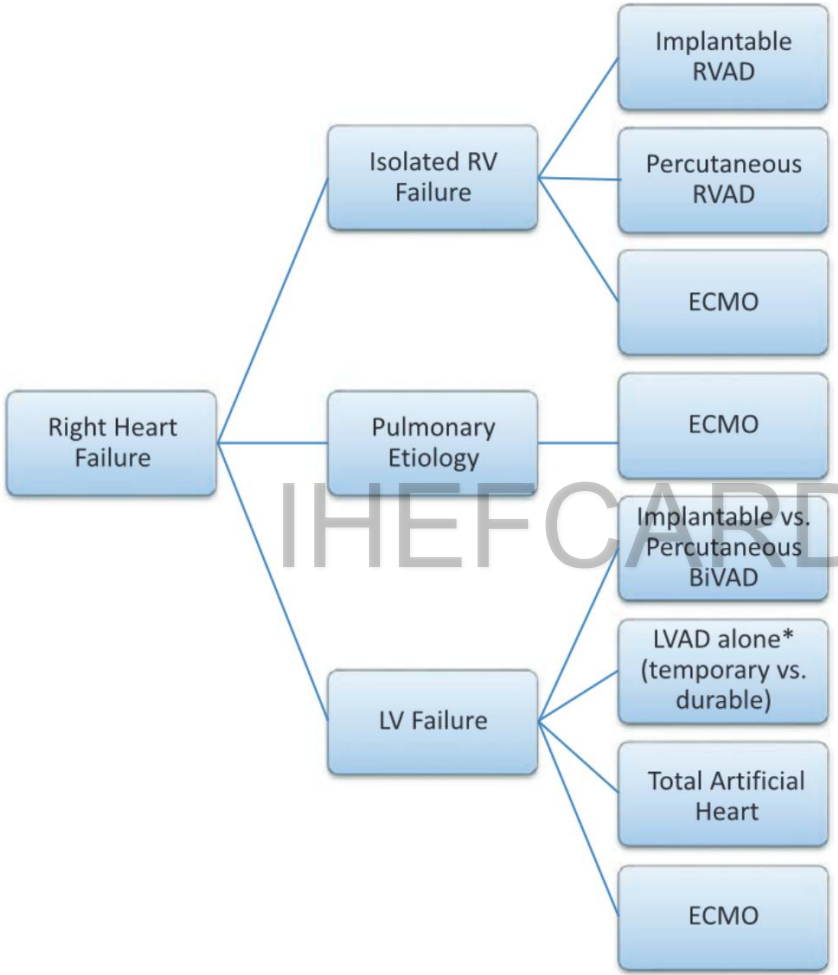
Mechanical Circulatory Support

- Mechanical Circulatory support (MCS) indicated for patient that refractory to optimal medical management
- Used for bridge to recovery; bridge to heart and/or lung transplantation; or as permanent therapy



Konstam, M.A., *et al*., Circulation 2018; 137(20):e578-e622

Mechanical Circulatory Support based on RVF Pathogenesis



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Management of Chronic RV Failure

- **Diuretics**
  - Maintain sufficient preload
  - Reduce RV volume overload, ventricular interdependence, and congestion.
  - Often require high dose diuretics
  - Combination therapy loop diuretics with thiazides to augment natriuresis
- Sodium and fluid Restriction
  - Sodium < 3 g/day
  - Fluid 1.5 – 2 L/day, when patients had refractory congestion and hyponatremia

Management of Chronic RV Failure

- RAAS-inhibitor, β-Blocker, Hydralazine:
  - **Not recommended** in patients with PH regardless of RHD/RVF, unless associated with hypertension, coronary artery disease, or LHF
  - Although **angiotensin-converting enzyme inhibitors** (ACEIs) have demonstrated positive impact on filling pressures and end-diastolic volume of RV, no improvement in hemodynamics or exercise capacity is demonstrated

- Digoxin
  - Meta-analysis did not find digoxin to be associated with improvement in RVEF, exercise capacity, or New York Heart Association class

- The function of beta-blockers in RVF improvement **is still controversial** as also the use of nesiritide, a BNP, **demands more research**

Management of Chronic RV Failure

- **Pulmonary Vasodilators:**
  - ↓ RV afterload → ↑ outcomes among group 1 PH
  - Riociguat: ↑ exercise capacity and PVR in patients with persistent CTEPH
  - Prostacyclin analogs:
    - Benefit in PH group I
    - Contraindicated in PH group 2
  - PDE5i:
    - Contraindicated in combination with nitrates
    - Beneficial in PH group 1
    - Uncertain outcome PH group 2
  - ERA:
    - Improvements in HF symptoms, exercise capacity, hemodynamics, and time to clinical worsening in PH group 1
    - Monitor liver function

Pulmonary Hypertension Medications

Table 4. Pulmonary hypertension medications

Selective pulmonary vasodilators	Clinical application	Observations
Calcium channel blockers • Nifedipine • Amlodipine • Diltiazem	• IPAH, HPAH, DPAH with positive vasoreactivity testing • High doses needed (ie, amlodipine 15-30 mg/d)	• Adverse effects: systemic hypotension and peripheral edema
Endothelin receptor antagonists • Bosentan (dual) • Ambrisentan (A receptor) • Macitentan (dual)	• PAH • Improve symptoms, exercise capacity, hemodynamics, and time to clinical worsening[137-139]	• Teratogenic effects • Peripheral edema (ambrisentan) • Transaminitis (bosentan) • Important drug interactions • Anemia (macitentan)
Phosphodiesterase 5 inhibitors • Sildenafil • Tadalafil	• PAH • Improve symptoms, exercise capacity, hemodynamics, and time to clinical worsening (tadalafil)[140,141]	• Hypotension when used with nitrates • Side effects: headache, epistaxis
Guanylate cyclase stimulators • Riociguat	• PAH and CTEPH • Improves exercise capacity, hemodynamics, functional class, and time to clinical worsening[142]	• Side effects: headache, epistaxis
Prostacyclin analogues • Epoprostenol (I.V., inhaled) • Iloprost (inhaled) • Teprostinil (inhaled, SC, I.V., enteral) • Beraprost (enteral)	• IPAH and systemic sclerosis PAH • Requires continuous infusions • Improved symptoms, exercise capacity, hemodynamics, and mortality (epoprostenol)[104] • Long-term efficacy in PAH	• Inhibit platelet aggregation • Side effects: jaw pain, diarrhea, infusion site pain • Pump and/or insertion site complications
Prostacyclin receptor agonist • Selexipag	• PAH • Reduced the risk of morbidity and/or mortality by 40%[143]	• Side effects: headache, jaw pain, diarrhea, nausea

CTEPH, chronic thromboembolic pulmonary hypertension; DPAH, drug-associated pulmonary arterial hypertension; IPAH, idiopathic pulmonary arterila hypertension; I.V., intravenous; HPAH, heritable pulmonary arterial hypertension; PAH, pulmonary arterial hypertension; SC, subcutaneous.

Available therapy for RVF

Treatments	Therapeutic effect
Diuretics [8]	Preload optimization
Nitric oxide [63]	Afterload optimization
Resynchronization therapy [63]	Ejection fraction
Dobutamine or (dobutamine + nitric oxide) [63]	Improved contractility
Anticoagulants [63]	Pulmonary embolism
ACEI, ARB [63]	Neurohormonal responses
Oxygen therapy, transplantation, RV assist device, ventilation [63]	Improvement of cardiac function

Chronic RV Failure in Congenital Heart Diseases

- No adequately powered clinical trials have been completed for medical therapies in RVF with CHD
- RVF result form pressure and volume afterload can be seen after repair TOF, pulmonary atresia, Ebstein anomaly, and pulmonary valvotomy for congenital PS
- CRT in CHD: preliminary studies demonstrated clinical improvement and improvement in RV function
- Current data do not support the routine administration of standard HF drug therapies to patients with CHD with either a single RV or systemic RV or in patients with a pulmonary RV at risk for RV failure
- Might require heart or heart-lung transplant

Surgical Management of RVF with VHD

Pulmonal Regurgitation

- symptoms or signs of RVD have occurred and PR is severe, the setting of severe RV dilation or dysfunction (cardiac MRI–derived RV end-diastolic volume index >150 mL/m2, RV end-systolic volume index >80 mL/m2, RVEF <47%) or symptomatic atrial and ventricular arrhythmias.
- Current guidelines support surgery for severe PR along with (1) moderate to severe RVD (**Class IIa**; Level of Evidence B), (2) moderate to severe RV enlargement (Class IIa; Level of Evidence B), (3) symptomatic or sustained atrial and ventricular arrhythmias (Class IIa; Level of Evidence C), or (4) moderate to severe TR (Class IIa; Level of Evidence B)
- Transcatheter PV replacement is also now possible

Pulmonal Stenosis

- PS may be treated with either percutaneous balloon PV commissurotomy or valve replacement
- Surgical ther- apy, as opposed to percutaneous therapies, is recom- mended for patients with severe PS and associated hy- poplastic pulmonary annulus, severe PR, subvalvular PS, or supravalvular PS.

Tricuspid Valve

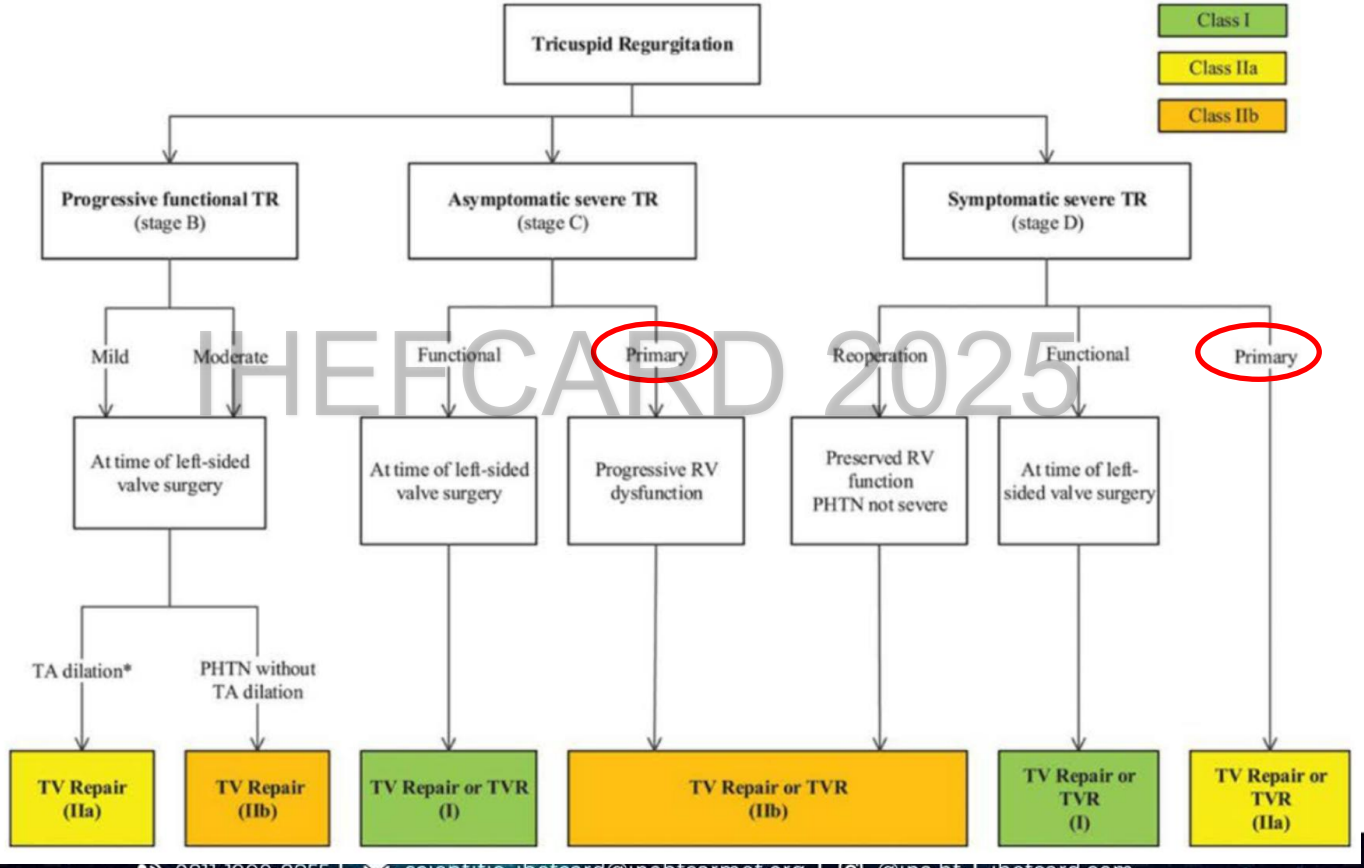
Tricuspid Stenosis

- Severe TS is generally performed in conjunction with surgery for left-sided valve disease, most commonly MS - RHD

Tricuspid Regurgitation

- The severity of TR affects prognosis even when controlling for LV dysfunction or PH. In a study of 5223 patients at 3 Veterans Affairs medical centers, 1-year survival rates were 92%, 90%, 79%, and 64% in patient groups with no, mild, moderate, or severe TR, respectively.
- Moderate or greater TR was associated with increased mortality regardless of PASP or LVEF.
- Severe TR, older age, lower LVEF, inferior vena cava dilation, and moderate or greater RV enlargement were associated with worse survival.

Surgical Management of RVF with VHD



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Take Home Message

- ✓ Diagnosis RVF often missed and delayed which will worsen its prognosis
- ✓ Diagnostic approach of RVF included clinical sign and symptoms, biomarkers, echocardiography, hemodynamics parameter, and MRI
- ✓ Cornerstone management of acute RVF are:
  - ✓ Establishing diagnosis and treat the specific underlying causes
  - ✓ Optimalization of volume status
  - ✓ Maintain perfusion and myocardial contractility
- ✓ RVF in VHD can be treated percutaneously or surgically depending of cause, severity, and concomitant lesion