Acute Right Heart Failure in ICU

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Introduction

- Acute right ventricle(RV) dysfunction causes and aggravates many common critical diseases (acute respiratory distress syndrome, pulmonary embolism, acute myocardial infarction, and postoperative cardiac surgery.

- Our role as ICU team is related to mechanical ventilation and fluid management, can make RV dysfunction worse, potentially exacerbating shock

- Touch upon the role of mechanical ventilation within the cascade of RVD, “RV protective ventilation” in ARDS.

Outline

- Definitions
- Epidemiology
- Pathophysiology
- Diagnosis
- Recommendations to guide management of acute RV dysfunction.

Definition of Acute Right Heart Failure in ICU

- Acute RV dysfunction (RVD)
- RV failure(RVF)
- Right heart failure(RHF)

Right heart failure

- RHF can be defined by a clinical syndrome due to an alteration of structure and/or function of the right heart circulatory system(comprised from the systemic veins up to the pulmonary capillaries) that reduces the ability to propel blood to the pulmonary circuit and/or high systemic venous pressures at rest or with effort
- Failure of the RV is a frequent component of RHF but not a mandatory feature of the RHF syndrome.
- Most common cause of RHF is Left Heart failure

Acute RVD

- Defined as at least one of the following:
  - (i) On Echo, Acute occurrence of RV systolic dysfunction by measuring the longitudinal systolic displacement and dilation
  - (ii) Unexplained increase of natriuretic peptides in the absence of LV or renal disease
  - (iii) Electrocardiographic(ECG) RV strain patterns which are strong markers of moderate-to-severe RV strain. While specific, they are limited by a lack of sensitivity
  - Acute cor pulmonale (ACP) is a form of RVD due to an acute increase in RV afterload

Chronic Right Heart Failure

Table 1: Acute right ventricular dysfunction definition*.

Echo parameters		ECG signs	Biomarkers
RV systolic function	RV dilation		
TAPSE < 16 mm	ED RVD/LVD ratio > 0.9	Complete RBBB	BNP > 100 pg/mL
S < 10 cm/sec	ED RVA/LVA ratio > 0.6	Incomplete RBBB	NT-proBNP > 900 pg/mL
RV fractional area change < 35%	ED RVD > 42 mm (at the base)	Anteroseptal ST elevation	
RV ejection fraction < 45%	ED RVD > 33 mm (at the middle third of RV)	Anteroseptal ST depression	
	Septal dyskinesia in the RV focused view	Anteroseptal T-wave inversion	

BNP: B-type natriuretic peptide; ED RVD/LVD ratio: end-diastolic RV diameter/LV diameter ratio; ED RVA/LVA ratio: end-diastolic RV area/LV area ratio; ED RVD: end-diastole RV diameter; NT-proBNP: N-terminal pro-BNP; S: pulsed Doppler S wave; TAPSE: tricuspid annular plane systolic excursion. * At least one of the items must be present (echo parameters, ECG signs, and biomarkers) [30].

Acute Right Ventricular Failure (RVF)

- Acute RVF is defined as acute RVD plus low cardiac output (CO) and hypoperfusion with the consequent multiorgan dysfunction/failure.

- RVF occurs when the RV fails to provide enough blood flow to the pulmonary circulation to accomplish adequate LV filling

Epidemiology

Acute RVD is both common and possibly lethal in critically ill patients

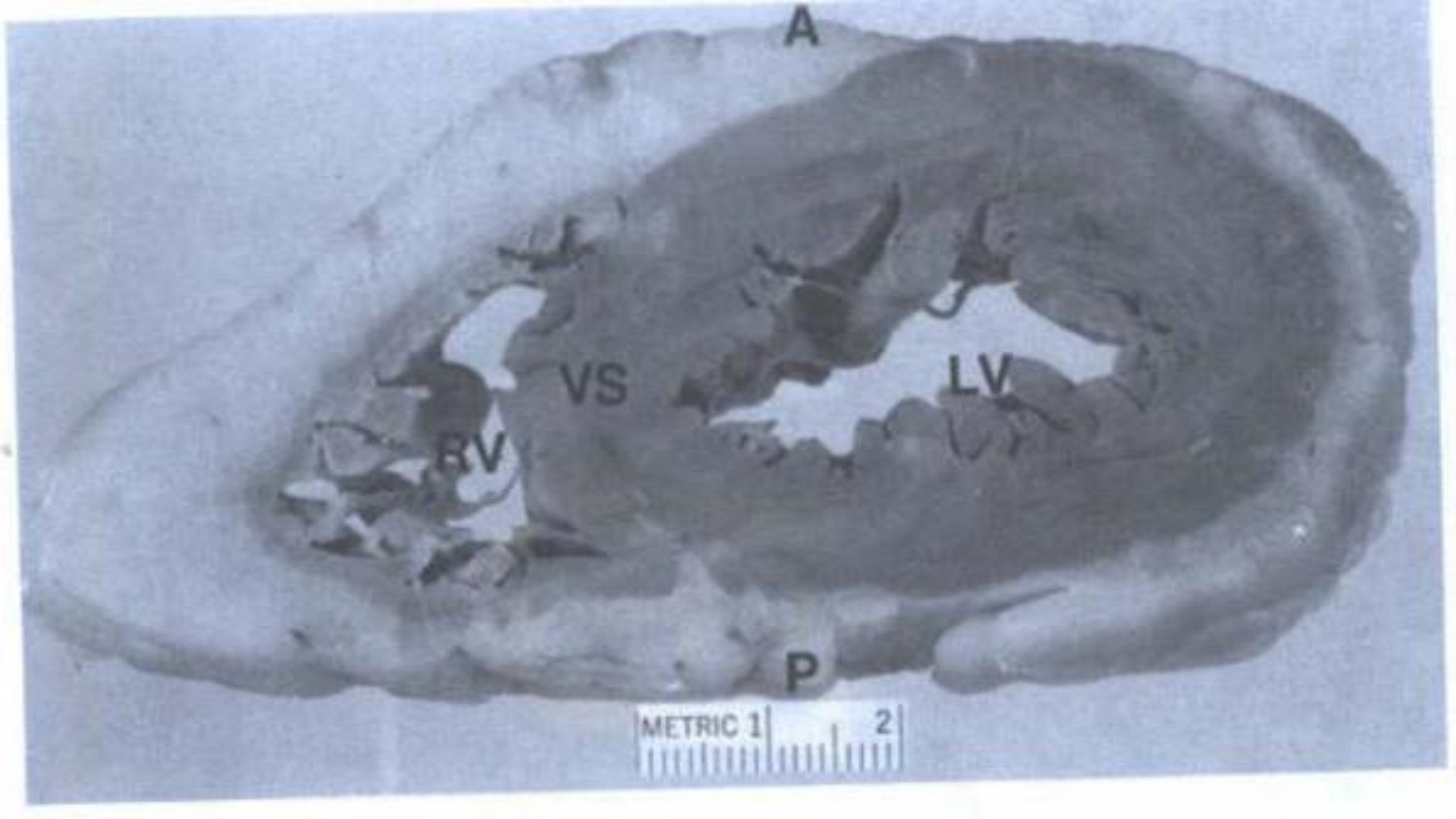
The clinically most important etiologies of acute RVD/RVF:

Acute PE

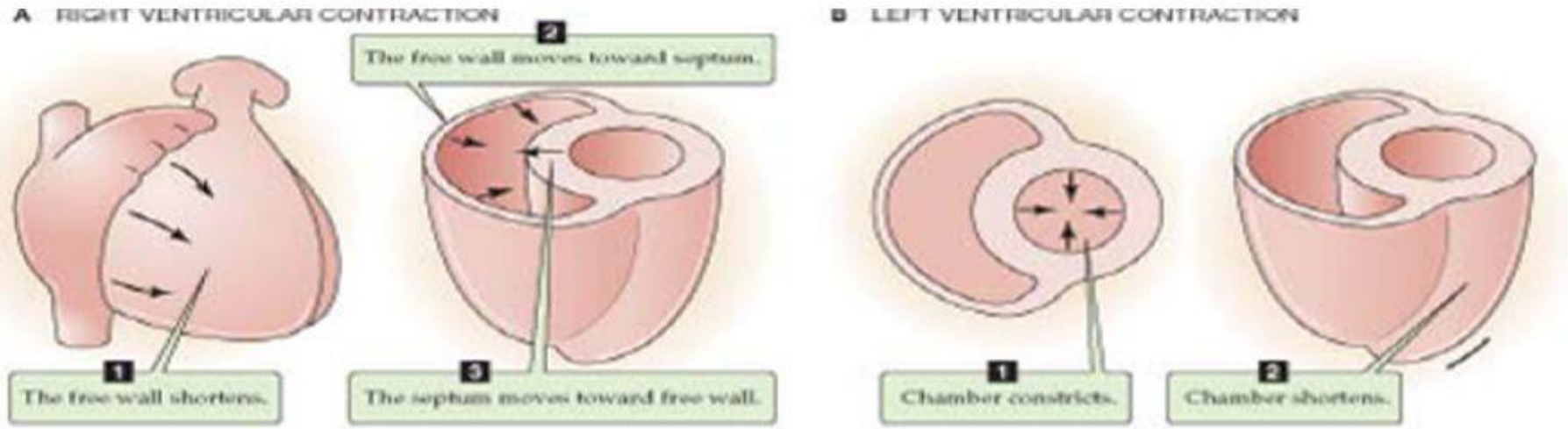
ARDS

RV myocardial infarction(RVMI)

After cardiothoracic surgery


A
VS
LV
RV
P
METRIC 1
2

Ventricular contraction: RV v/s LV

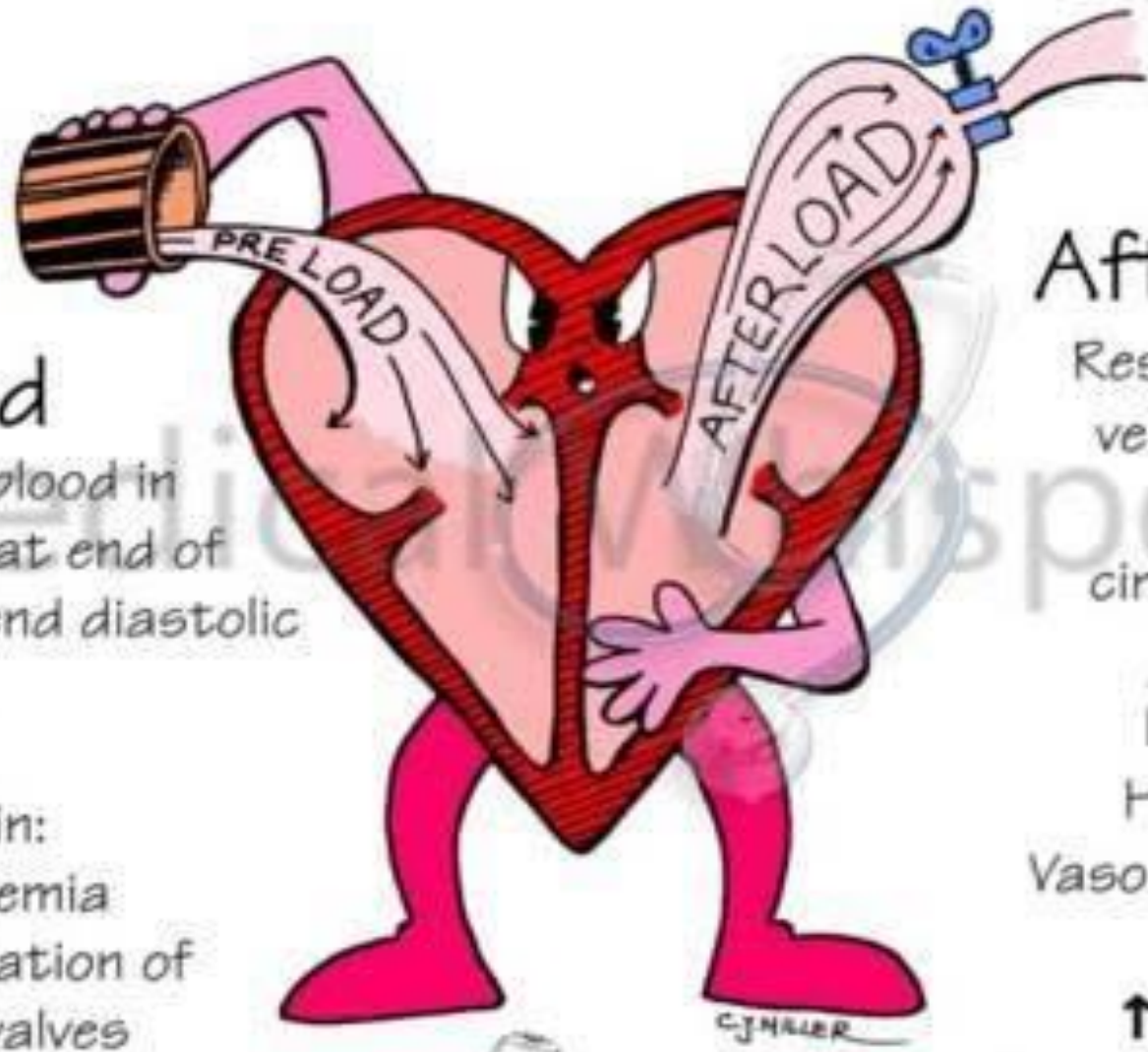


Pathophysiology of Acute RVD and RVF

- RV mechanics and function can be altered in the setting of either pressure/volume overload and primary reduction of contractility owing to myocardial ischemia.

- The compliant and thin walled RV can accommodate significant increases in preload but tolerates acute increases in afterload poorly.

PRELOAD AND AFTERLOAD



Preload

Volume of blood in ventricles at end of diastole (end diastolic pressure)

Increased in:

- Hypervolemia
- Regurgitation of cardiac valves
- Heart Failure

Afterload

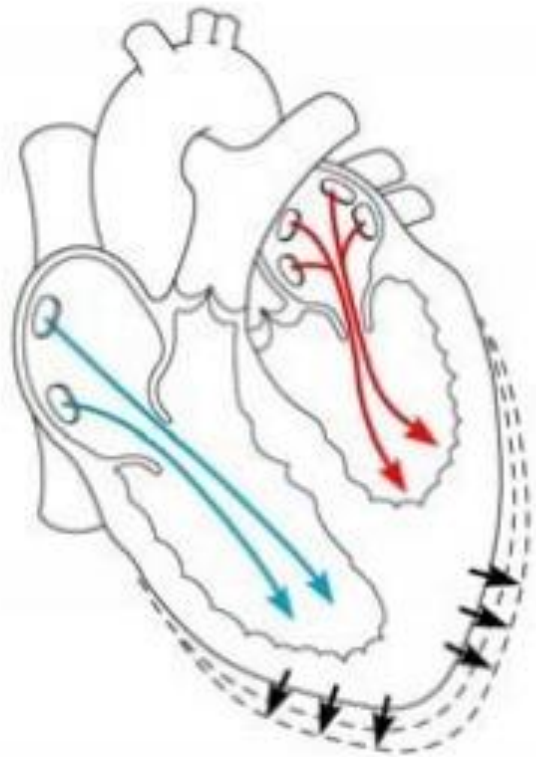
Resistance left ventricle must overcome to circulate blood

Increased in:

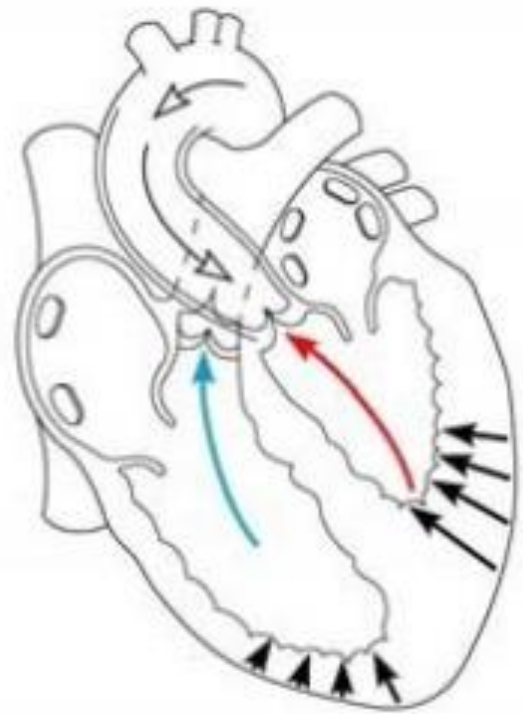
- Hypertension
- Vasoconstriction

↑ Afterload = ↑ Cardiac workload

PRELOAD AND AFTERLOAD DEPICTED



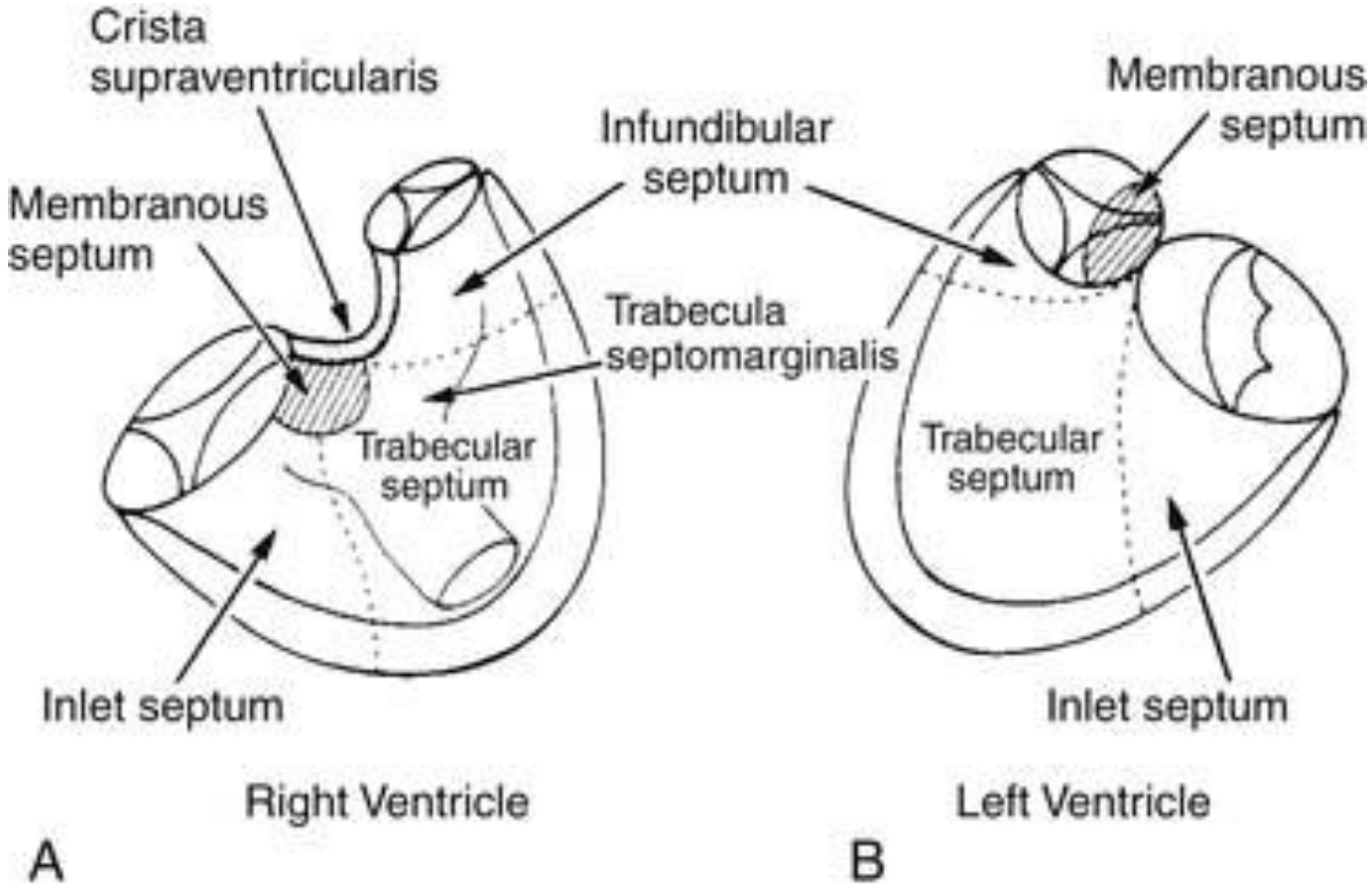
(a) Preload

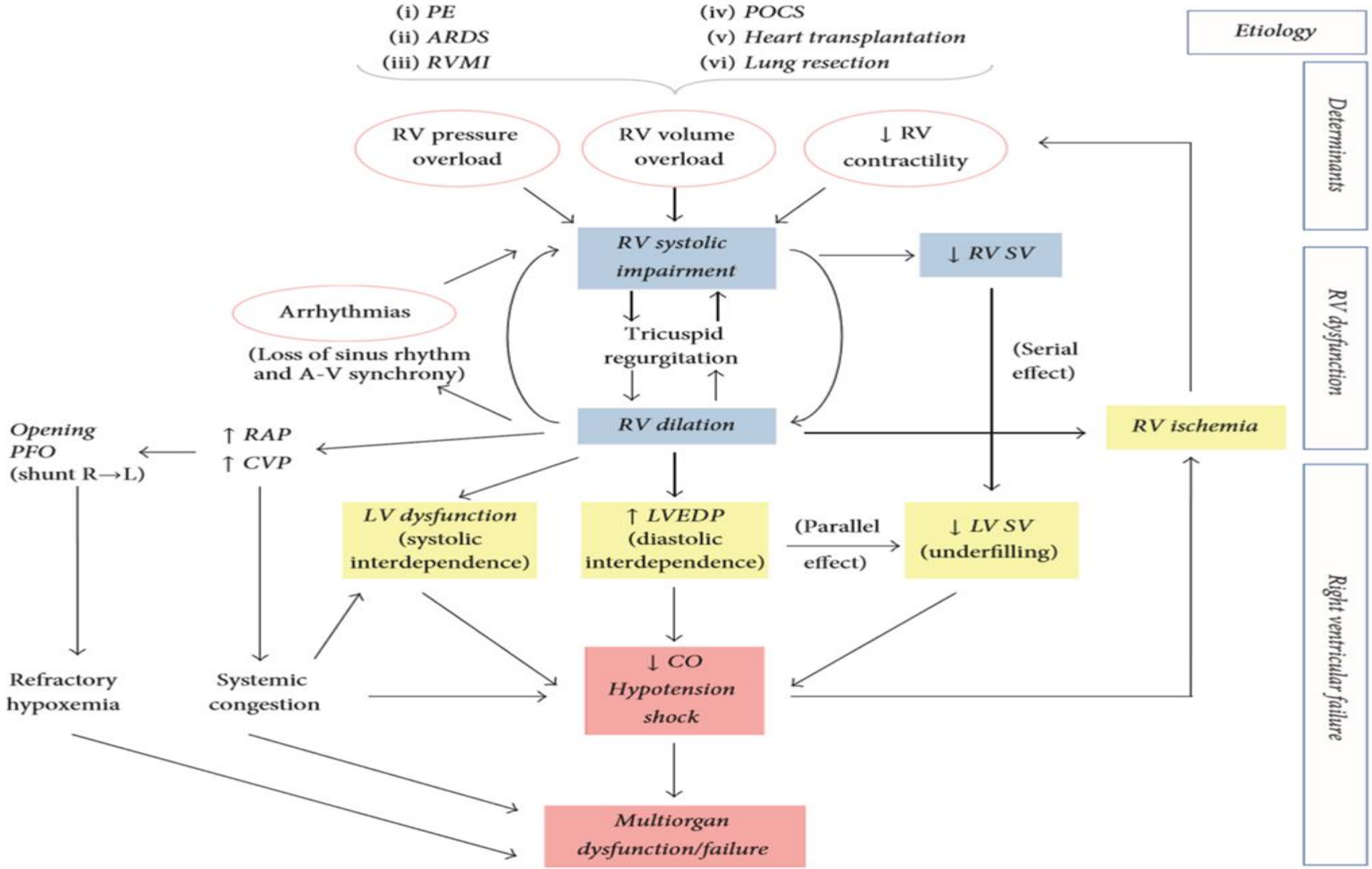


(b) Afterload

Anatomical considerations

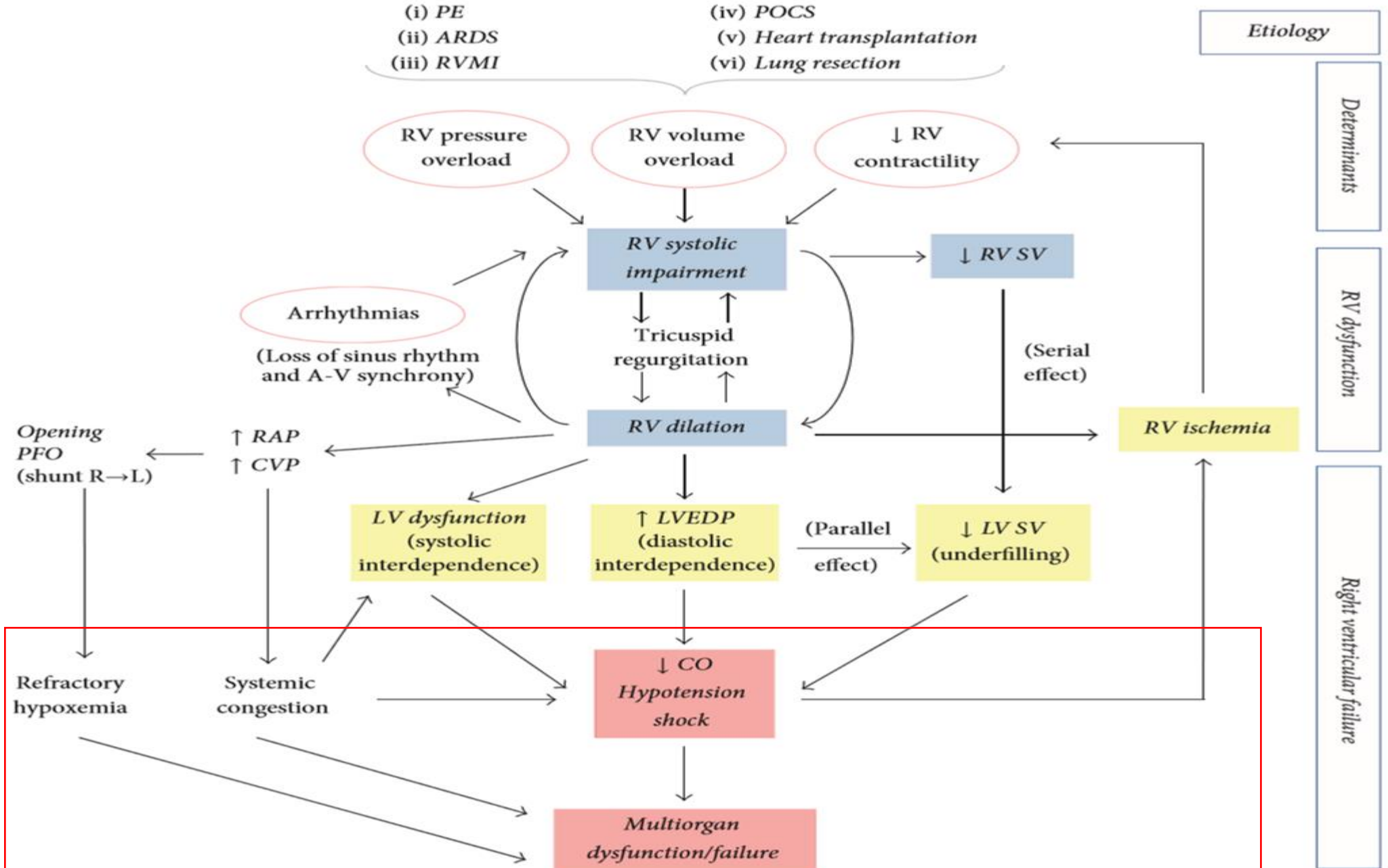
- Ellipsoidal shape of the LV; RV appears triangular and crescent-shaped
- RV can be described in three components:
  (1) The inlet, which consists of the tricuspid valve, chordal tendineae, and papillary muscles;
  (2) The trabeculated apical myocardium;
  (3) The infundibulum, outlet




(i) PE
(ii) ARDS
(iii) RVMI
(iv) POCS
(v) Heart transplantation
(vi) Lung resection
Etiology
Determinants
RV pressure overload
RV volume overload
↓ RV contractility
RV systolic impairment
↓ RV SV
RV dysfunction
Arrhythmias
(Loss of sinus rhythm and A-V synchrony)
Tricuspid regurgitation
(Serial effect)
RV dilation
RV ischemia
Opening PFO (shunt R→L)
↑ RAP
↑ CVP
LV dysfunction (systolic interdependence)
↑ LVEDP (diastolic interdependence)
(Parallel effect)
↓ LV SV (underfilling)
Right ventricular failure
↓ CO Hypotension shock
Refractory hypoxemia
Systemic congestion
Multiorgan dysfunction/failure

Clinical Presentation and Diagnosis of RVD/RVF in ICU

- The clinical presentation of acute RVF varies depending on the underlying cause, the presence of comorbidities, and the cardiovascular reserve of the right ventricle-arterial unit
- It can occur suddenly or catastrophic in a previously “healthy heart” or in a hidden way, worsening of compensated RVD in the setting of a chronic heart and lung disease.
- The diagnosis of acute RVF in ICU patients can become very difficult due to the presence of comorbid conditions that may cause organ hypoperfusion even in the absence of RVD (e.g., sepsis, LV dysfunction, and hypovolemia).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Clinical Presentation and Diagnosis of RVD/RVF in ICU

- Diagnosis typically relies on echocardiography.

- Echocardiography has become important not only for early detecting acute RVD in ICU patients but also for monitoring and guiding a rational therapy preventing RVF from occurring.

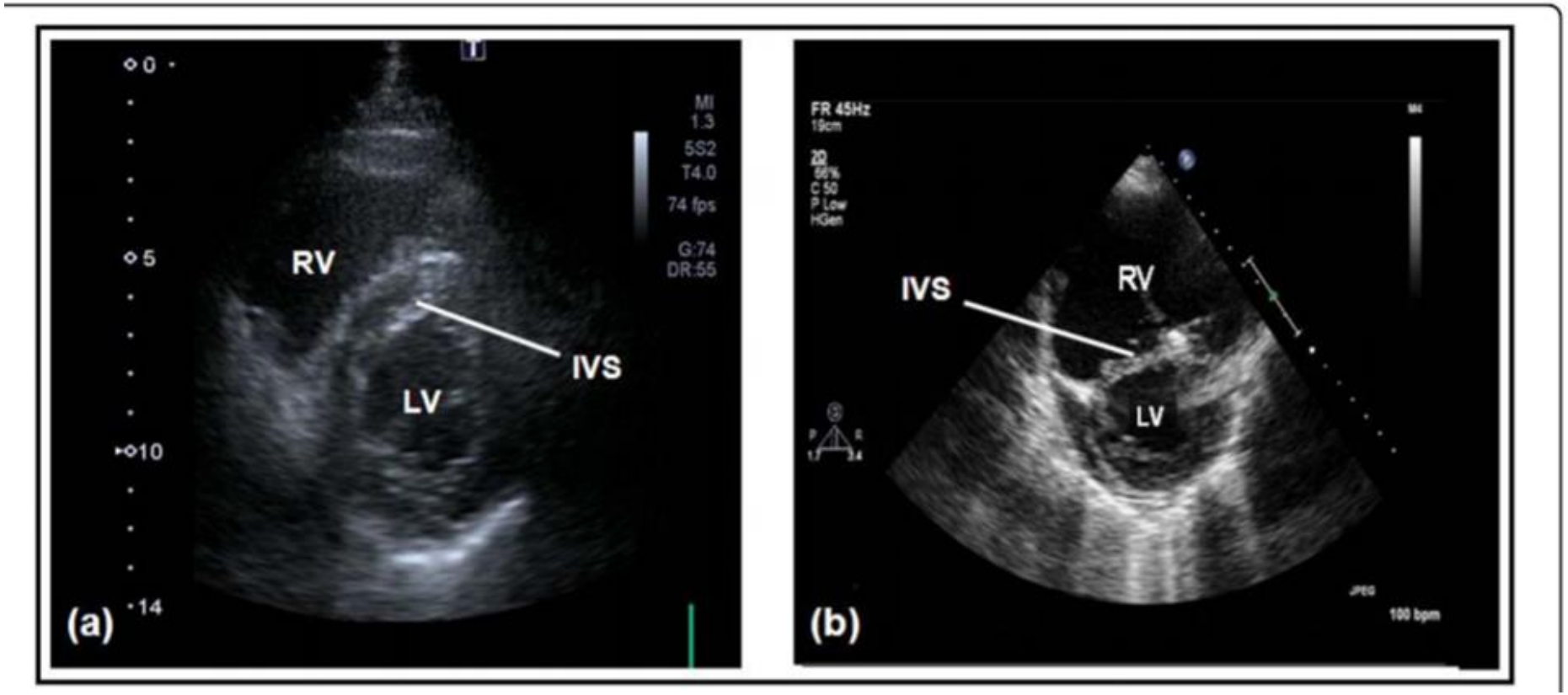
Echocardiographic Parameters of RVF

Table 2: Cut-off values of RV structural and functional parameters and RV afterload assessment.

RV structural parameters	RV functional parameters	RV afterload assessment
Basal RV diameter[§] > 42 mm	RV fractional area change ≥ 35%	AccT < 100 msec
RV mid-diameter[§] > 33 mm	MPI[§] > 0.43 (pulsed Doppler); >0.54 (tissue Doppler)	Shape of doppler RV outflow tract envelope[#]:
RV EDD/LV EDD[§] > 0.9	TAPSE[‡] < 16 mm	(i) No notch
RV/LV EDA[§] > 0.6	S wave[°] < 10 cm/s	(ii) Late notch
LV eccentricity index[†] > 1	Peak RV free wall 2D strain[*] > −20%	(iii) Midsystolic notch
McConnell's sign[§]		
RV wall thickness > 5 mm		

AccT: acceleration time of RV outflow tract flow; EDD: end-diastolic diameter; EDA: end-diastolic area; LV: left ventricle; RV: right ventricle; MPI: myocardial performance index (the ratio of the sum of isovolumic contraction plus relaxation time and ejection time intervals); S wave: peak velocity of systolic excursion at the lateral tricuspid annulus; TAPSE: tricuspid annular plane systolic excursion. [#]The presence and position of the systolic notching are related to the pulmonary dynamic afterload severity and RV dysfunction in patients referred for PH [31]. The presence of midsystolic notch is associated with the worst hemodynamic profile. [§]TTE: apical four-chamber; TEE: mid esophageal four-chamber; [†]TTE: parasternal midpapillary short axis; TEE: transgastric midpapillary short axis; [°]TTE: apical four-chamber; TEE: deep transgastric RV; [*]RV-focused four-chamber view. [‡]M-mode imaging at the lateral tricuspid valve plane.

Mcconel Sign



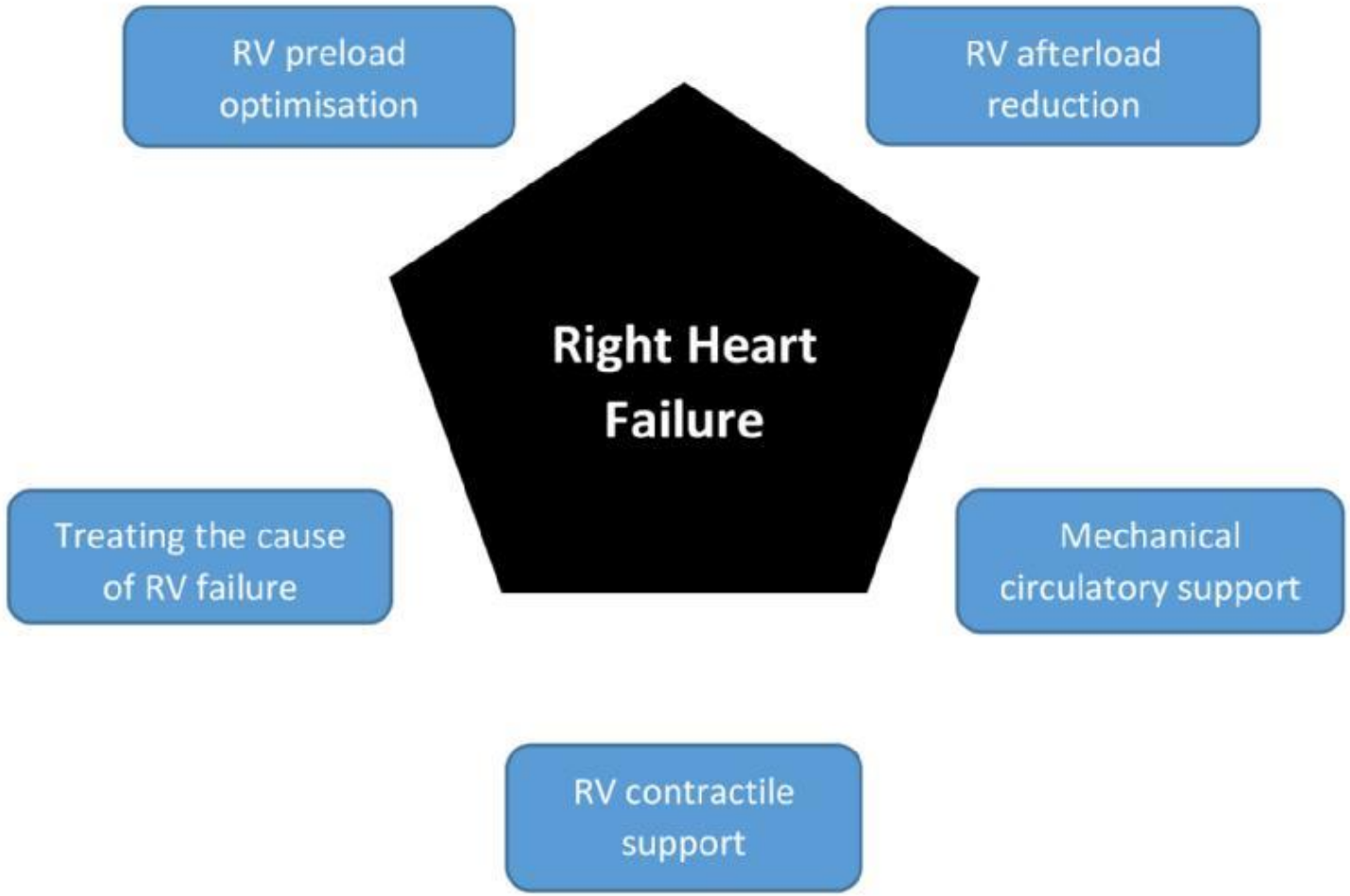
Pulmonary Artery Catheter (PAC)

- The availability of bedside echocardiography, the use of PAC is much less common nowadays. Post Operative Cardiac Surgery pts.
- PAC findings suggestive of acute RVD:
  - an elevated CVP (> 20mmHg),
  - An inverse pressure gradient(CVP>PAWP),
  - Low cardiac index(<2L/min/ m2),
  - Low stroke volume index (<30mL/m2),
  - Mixed-venous oxygen saturation(SvO2 <55%)

Diagnosis of RVD/RVF in ICU

- In summary, combining the use of real-time echocardiographic evaluation bedside with the knowledge of RV physiology is the desirable way to diagnose acute RVD/RVF in ICU patients.

- PAC might contribute to the monitoring and adjustment of the treatment.


RV preload optimisation
RV afterload reduction
Right Heart Failure
Treating the cause of RV failure
Mechanical circulatory support
RV contractile support

Treatment RVF outline

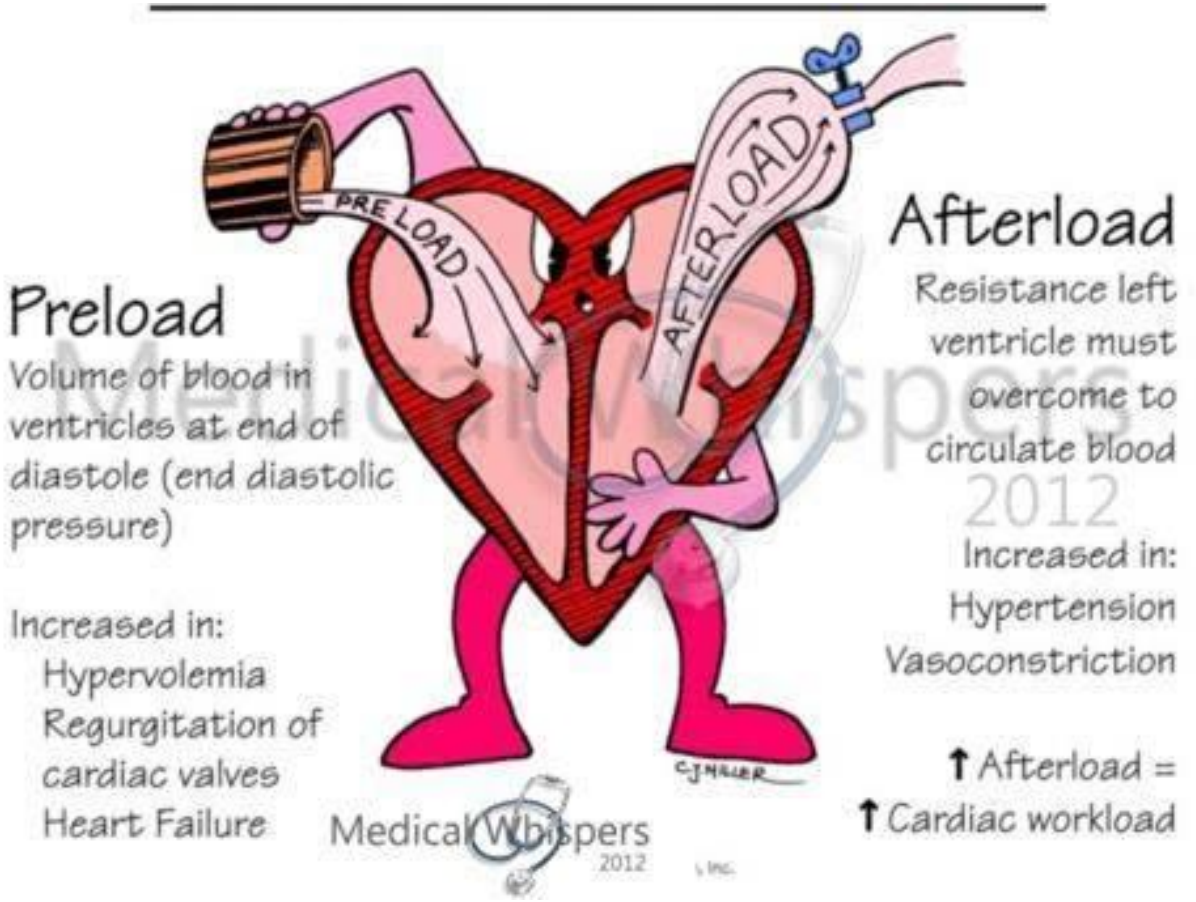
- Avoiding increasing RV afterload, decreasing RV contractility and optimization of RV preload
- Applying an “RV-protective” ventilation strategy.
- Maintaining sinus rhythm and atrioventricular synchrony;
- Pharmacological treatment with a guided inotropic and vasoactive supports;
- Mechanical circulatory support devices.
- Real-time monitor with bedside echocardiography assessment and the invasive hemodynamic monitoring remain the most valuable methods to guide a rational therapy of acute RVD/RVF in critically ill patients

Prevention of RVF in ICU

- The prevention of acute RVF in ICU begins with the identification of high-risk patients
- ARDS, Inferior wall MI and Cardiac surgery with long cardiopulmonary bypass times
- Once the severe RVD or RVF is recognized, we have to identify and treat any underlying reversible conditions that are either primarily responsible for (triggering factors)or contributing to the progressive impairment of RV function.

Fluid Management in RVD

- Proper management of volume status is essential for the failing RV, as both hypovolemia and hypervolemia may result in reduced CO
- When volume overload is present, the use of diuretics or renal replacement therapy is required



Fluid Management in RVD due to Increased Afterload

- RV preload requirements differ substantially based on whether afterload is normal or increased.
- Acute RVD in the setting of increased RV afterload, we should be restrictive with volume management.
- Increasing blood volume to an already overloaded RV(e.g.,PE,ARDS) will not only improve perfusion but also impair CO.
- Aggravating RV dilatation increasing tricuspid regurgitation and right-sided venous congestion underfilling of the LV hypoperfusion and multiorgan dysfunction Shock State.

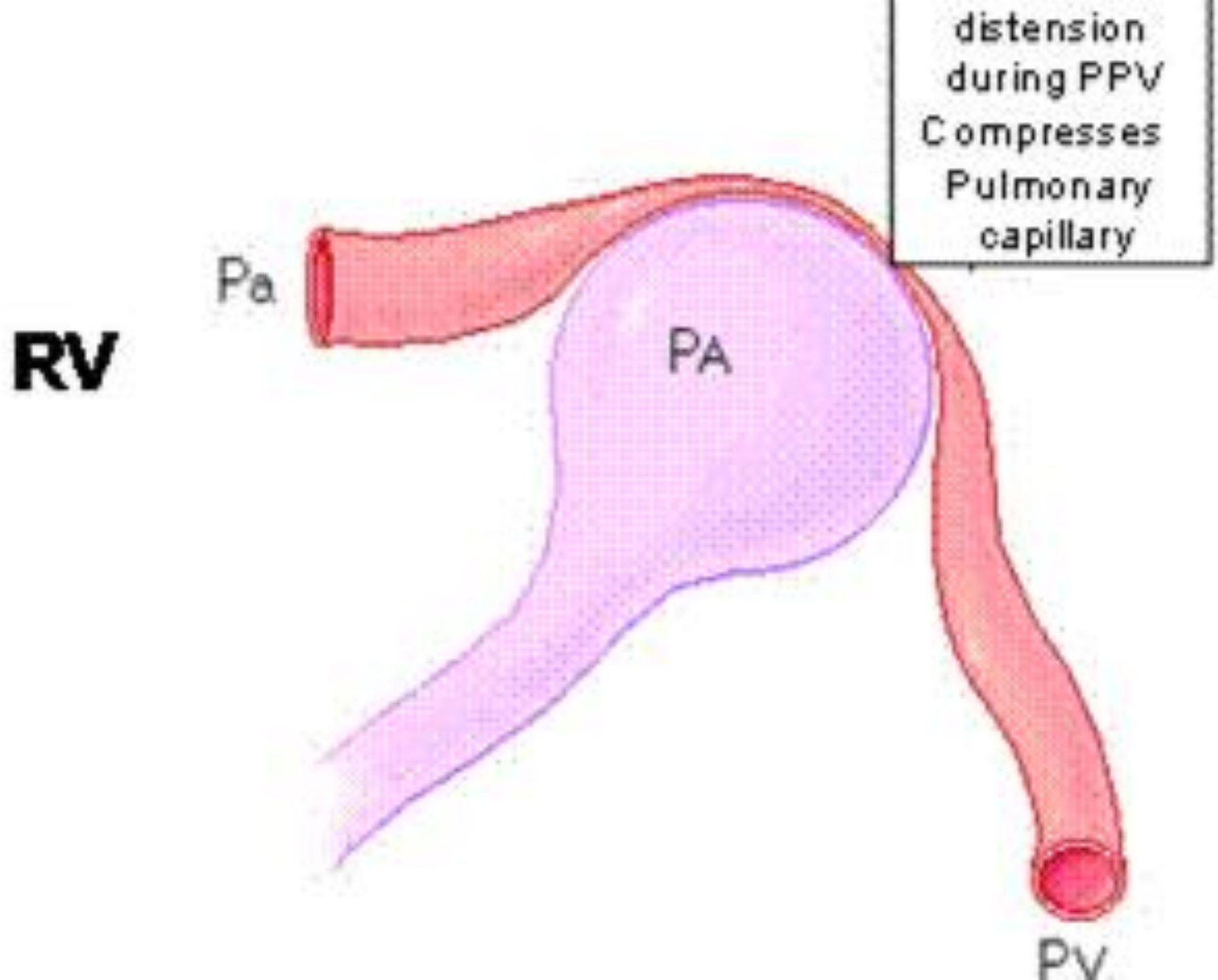
Fluid Management in RVD due to decreased Afterload

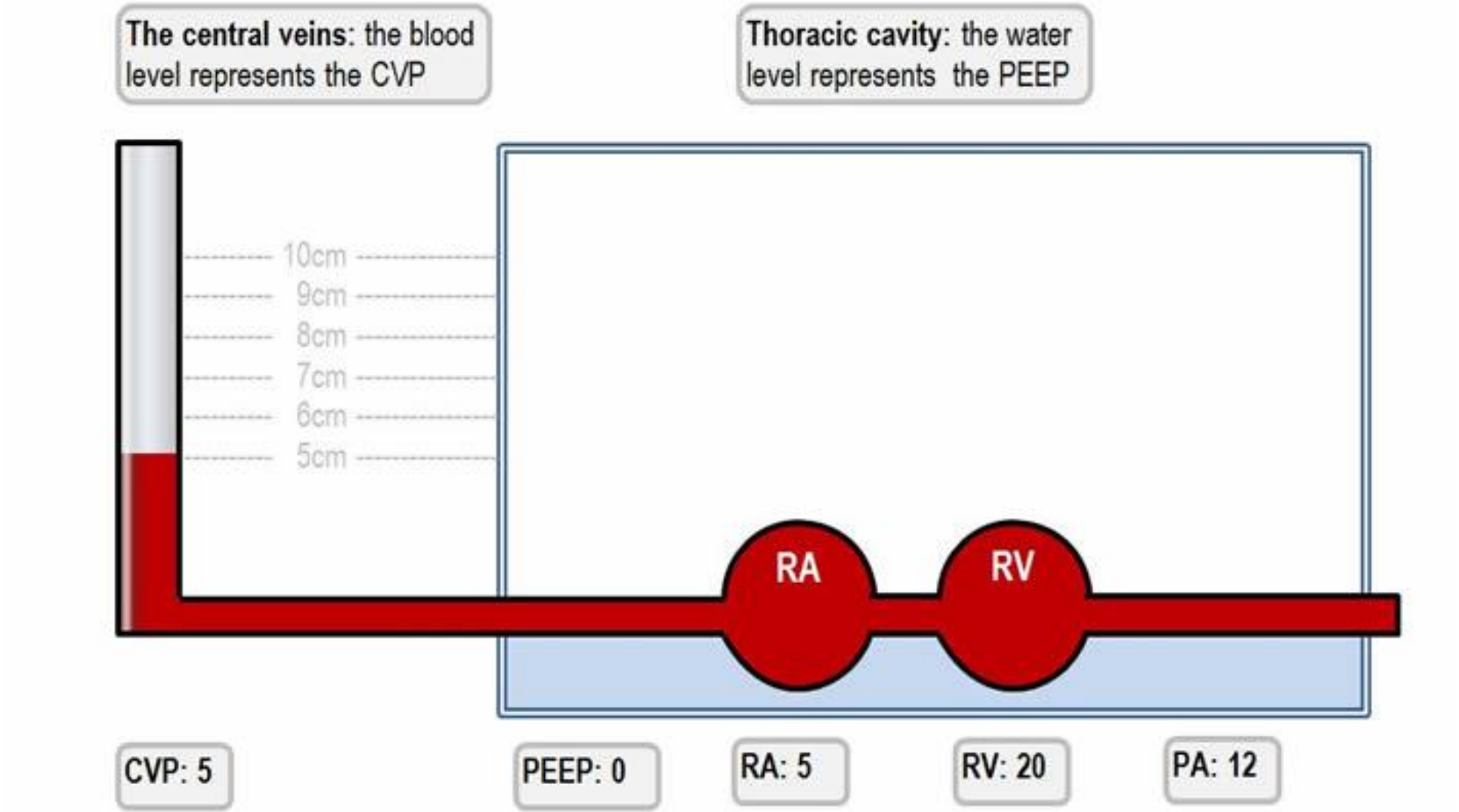
- On the contrary when acute RVD occurs in the setting of normal pulmonary vascular resistance (e.g., RV myocardial infarction),we can be more liberal with fluid reposition to maintain CO.

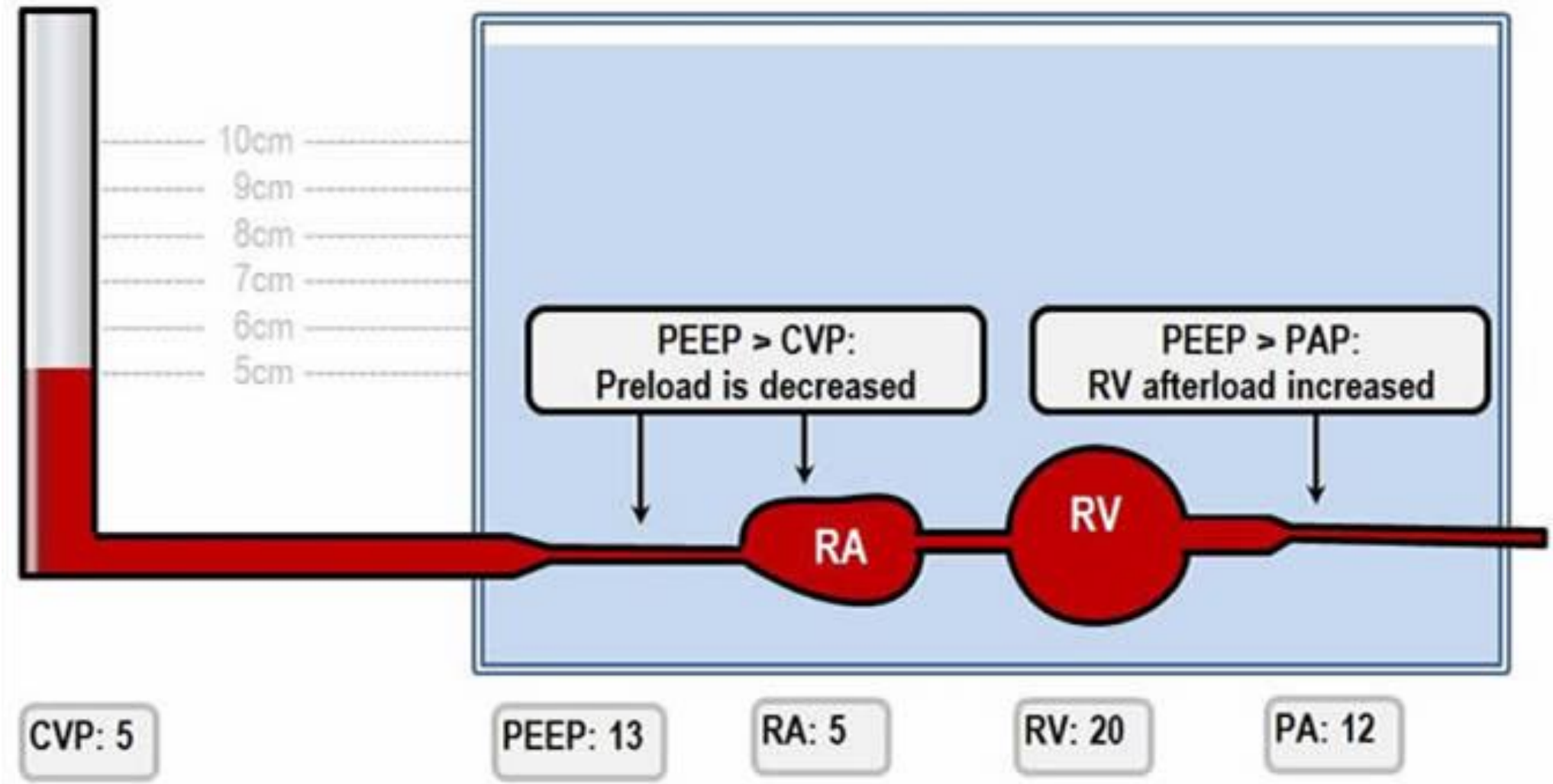
Mechanical Ventilation and RV

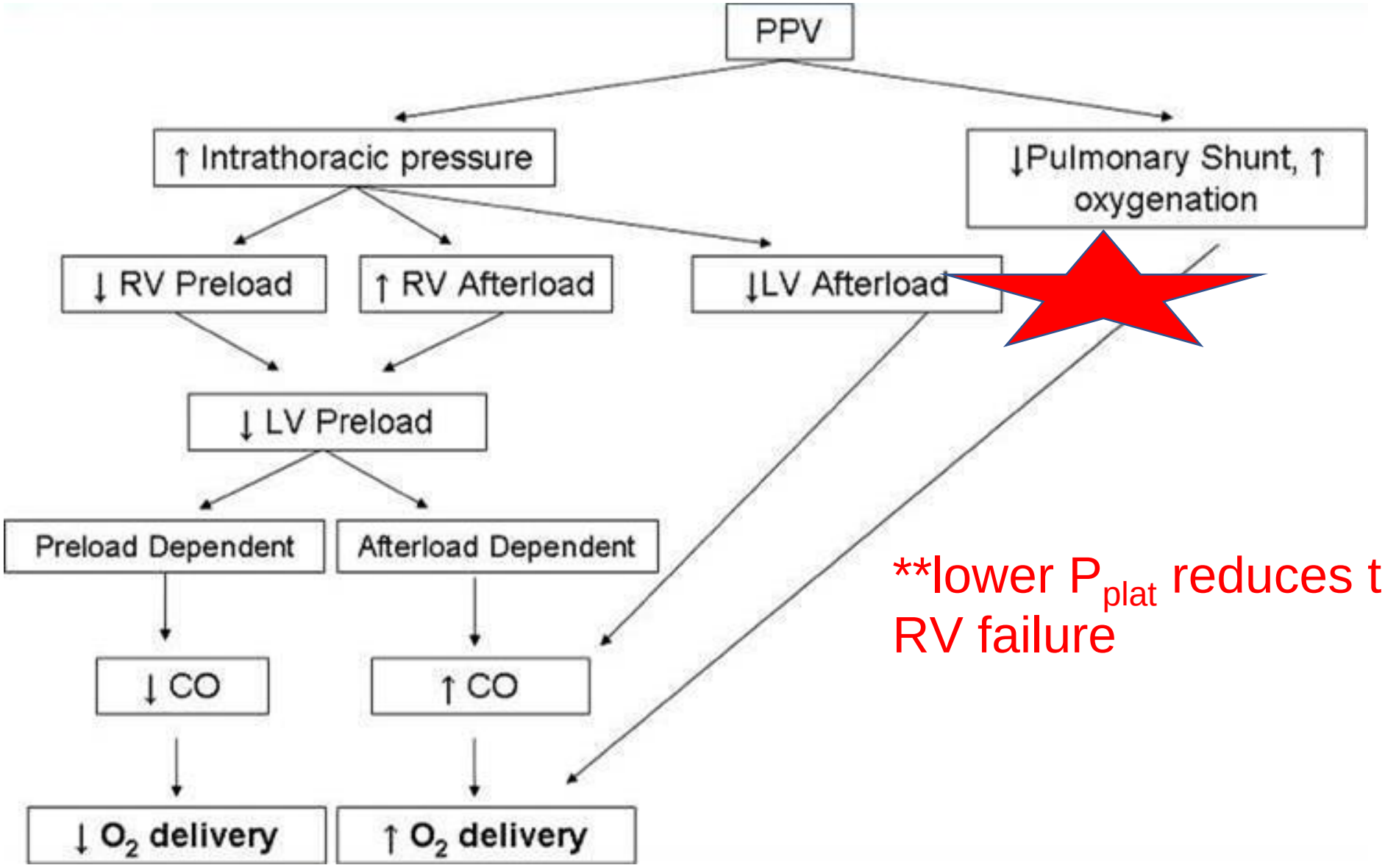
- The dominant RV effects of mechanical ventilation are to reduce the preload and raise the afterload, which in the setting of acute RVD maybe a critical issue.
- The ventilatory strategy is the main nonpharmacological treatment of the RV afterload through the control of hypoxemia, hypercapnia, acidemia, and inspiratory airway pressure.
- Before the era of protective ventilatory strategies in ARDS, the incidence of acute RV failure was 60% and has since decreased to 10% to 25%. (A discussion on ARDS ventilation strategies to follow with Dr Shaleileh)

By increasing airway pressure, PEEP may overdistend alveoli and compress alveolar capillaries, resulting in an increase in pulmonary vascular resistance and hence RV afterload.


distension
during PPV
Compresses
Pulmonary
capillary
Pa
PA
RV
Pv


The central veins: the blood level represents the CVP
Thoracic cavity: the water level represents the PEEP
10cm
9cm
8cm
7cm
6cm
5cm
RA
RV
CVP: 5
PEEP: 0
RA: 5
RV: 20
PA: 12


10cm
9cm
8cm
7cm
6cm
5cm
PEEP > CVP:
Preload is decreased
PEEP > PAP:
RV afterload increased
RA
RV
CVP: 5
PEEP: 13
RA: 5
RV: 20
PA: 12



**lower P_{plat} reduces the incidence of RV failure

Other treatment modalities

- Vasopressors
- Pulmonary vasodilators drugs
- Mechanical Circulatory Support (ECMO)
- Etiology specific management
- RV protective mechanical ventilation

Venoarterial ECMO	Venovenous ECMO
Higher PaO_2 is achieved	Lower PaO_2 is achieved
Lower perfusion rates are needed	Higher perfusion rates are needed
Bypasses pulmonary circulation	Maintains pulmonary blood flow
Decreases pulmonary artery pressures	Elevates mixed venous PO_2
Provides cardiac support to assist systemic circulation	Does not provide cardiac support to assist systemic circulation
Requires arterial cannulation	Requires only venous cannulation

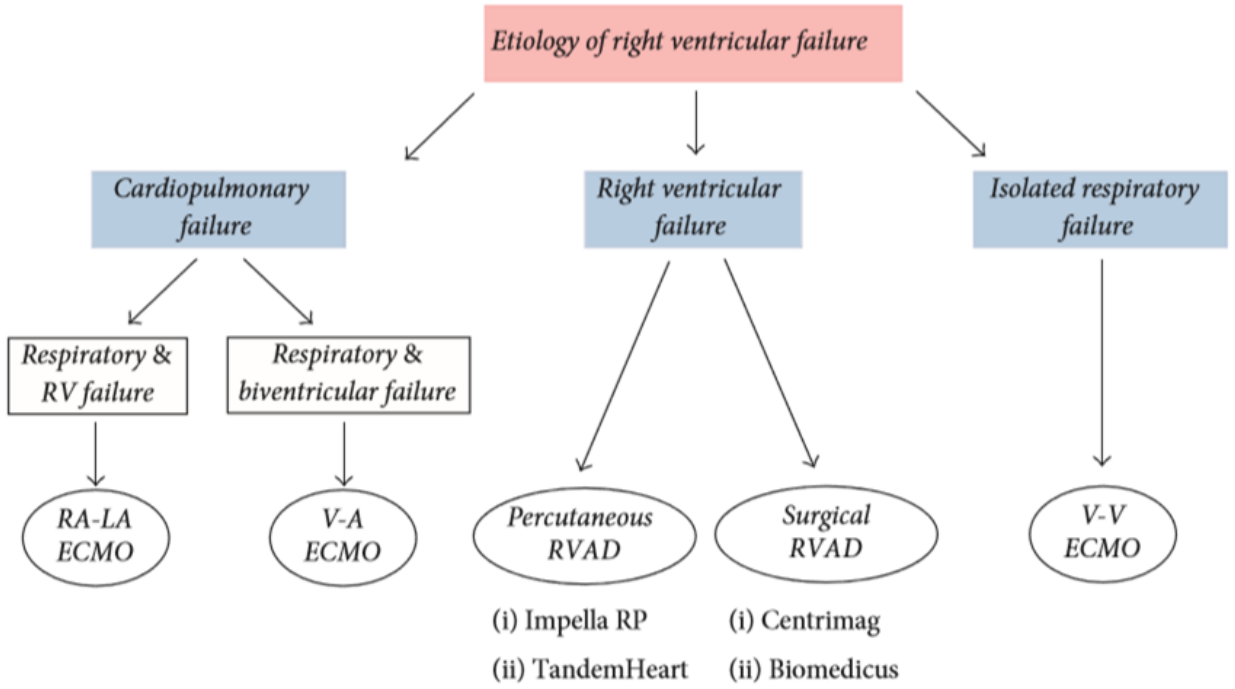


FIGURE 3: Schematic algorithm for selecting the appropriate extracorporeal life support in patients with refractory right ventricular failure. RA-LA: right atrial-left atrial; RVAD: right ventricular assist device; V-A: venoarterial; V-V: venovenous; ECMO: extracorporeal membrane oxygenation.

Table 5: Mechanisms and targeted management in specific clinical scenarios of acute RV failure.

Clinical scenario	Mechanism	Treatment
Right ventricular infarct	Decreased RV contractility	Early myocardial reperfusion (percutaneous coronary intervention, systemic thrombolysis)
Pulmonary embolism	Increase RV afterload (mechanical obstruction & vasoconstriction)	Systemic anticoagulation, systemic or catheter-directed thrombolysis, embolectomy
Decompensated PAH	Increase RV afterload	Parenteral prostanoids (with or without inhaled pulmonary vasodilators
ARDS	Increasing RV afterload/decreasing RV contractility	Limiting VT and PEEP, avoiding hypoxia, hypercapnia, and acidosis
Noncardiac surgery	Acute PH, decreasing RV contractility (RV infarct)	Pulmonary vasodilators, myocardial reperfusion, inotropic drugs
Cardiac surgery	Volume overload, myocardial ischaemia, preexisting RVD, arrhythmias	Diuretics, inotropic drugs, cardioversion, antiarrhythmic drugs

ARDS: acute respiratory distress syndrome; PAH: pulmonary arterial hypertension; RVD: right ventricular dysfunction.

Conclusion

- Acute RVD/RVF is seen with increasing frequency in the intensive care unit and causes or aggravates many common critical diseases.
- Bedside echocardiography assessment and invasive hemodynamic monitoring remain the most valuable methods to diagnose and to guide a rationale therapy of acute RVD/ RVF in critically ill patients.
- General precautionary measures, early diagnosis of RVD, and etiology-specific therapy may reduce the appearance of RVF.
- Supportive therapies focused on improving RV function via optimization of preload, enhancing contractility, and reducing afterload are the key principles in the management of acute RVF.